**Thesis Ref. No_____**

# COMPARATIVE PATHOLOGICAL STUDY ON LUNG, HEART, KIDNEY AND LIVER OF CATTLE AND DROMEDARY CAMEL SLAUGHTERED AT AKAKI ABATTOIR AND ASSESSMENT OF ASSOCIATED FINANCIAL LOSSES

**MVSc THESIS**


**BY**

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**COLLEGE OF VETERINARY MEDICINE AND AGRICULTURE**

**DEPARTMENT OF PARASITOLOGY AND PATHOLOGY**


**MVSC PROGRAM IN VETERINARY PATHOLOGY**

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**BISHOFTU, ETHIOPIA.**

# COMPARATIVE PATHOLOGICAL STUDY ON LUNG, HEART, KIDNEY AND LIVER OF CATTLE AND DROMEDARY CAMEL SLAUGHTERED AT AKAKI ABATTOIR AND ASSESSMENT OF ASSOCIATED FINANCIAL LOSSES

**MVSc THESIS**

**A Thesis Submitted to Addis Ababa University College of Veterinary Medicine and Agriculture in Partial Fulfillment of the Requirements for the Degree of Master of Veterinary Science in Veterinary Pathology**

**By**

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**APPROVAL**

**ADDIS ABABA UNIVERSITY**
**COLLEGE OF VETERINARY MEDICINE AND AGRICULTURE**
**DEPARTMENT OF PARASITOLOGY AND PATHOLOGY**

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As members of the Examining Board of the final MVSc open defense, we certify that we have read and evaluated the Thesis prepared by: Emebet Etisa Demisse entitled **"COMPARATIVE PATHOLOGICAL STUDY ON LUNG, HEART, KIDNEY AND LIVER OF CATTLE AND DROMEDARY CAMEL SLAUGHTERED AT AKAKI ABATTOIR AND ASSESSMENT OF ASSOCIATED FINANCIAL LOSSES"** and recommend that it be accepted as fulfilling the thesis requirement for the degree of: Masters of Veterinary Science in Veterinary Pathology

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**STATEMENT OF THE AUTHOR**

I, the undersigned, the author of this thesis, hereby declare that I am the sole author of this thesis. To the best of my knowledge this thesis contains no material previously published by any other person except where due acknowledgement has been made. This thesis contains no material which has been accepted as part of the requirements of any other academic degree or non-degree program, in English or in any other language. This thesis has been submitted in partial fulfillment of the requirements for master of veterinary science degree in veterinary pathology at Addis Ababa University, College of Veterinary Medicine and Agriculture and is deposited at the University/College library to be made available to borrowers under rules of the library.

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| | |
|---|---|
| CBPP | Contagious Bovine Pleuropneumonia |
| Btb | Bovine Tuberculosis |
| CC | Cholangiocellular Carcinoma |
| Ccl4 | Carbon Tetrachloride |
| ER | Endoplasmic Reticulum |
| FAO | Food and Agriculture Organization |
| FNH | Focal Nodular Hyperplasia |
| H and E | Hematoxylin and Eosin |
| MOLS-TR | Market Ordinated Livestock Thematic research |
| PAS | Periodic Acid–Schiff |
| SC | Sclerosing Cholangitis |
| WHO | World Health Organization |

**ABSTRACT**

Lung, heart, kidney and liver are vital organ of the body and susceptible to various disease conditions and parasites that affect the total health status of cattle and camel. There is an increase in demand of carcass and organ consumption but the production capacity become decreasing due to the presence of endemic disease and varieties of pathological conditions. This study was carried out to investigate the pathological changes present in lungs, hearts, kidneys and livers of cattle and dromedarius camels slaughtered at Akaki abattoir. Such studies are very important in epidemiological surveys of animal diseases. Cross-sectional study design with purposive sampling method was used to study pathological findings on lung, heart, kidney and liver of cattle and camels slaughtered at Akaki abattoir. 3520 cattle and 560 camels were involved in the study. The organs from these animals were grossly inspected and the representative tissue samples in 10% neutral buffered formalin were collected for histopathological lesions characterization. Pulmonary hydatidosis was the highest findings in the lung of cattle 39% while emphysema was the highest lesion in camel 30.4%. Grossly the hydatidosis was characterized by having variable size yellowish to grey colored cysts embedded at different depths of lung and microscopically by the presence cystic wall and infiltration of inflammatory cells. The emphysema was characterized grossly by enlargement of the lung due to gas accumulation and microscopically by distention of the alveoli and interlobular septa. The least pathological changes in the lung of cattle were granulomatous pneumonia and CBPP both occurred at same rate 0.12%. Granulomatous pneumonia was characterized grossly by dark red area of streak, dilation of interlobular septa with mucoid like exudates and yellowish to gray caseous and calcified materials in the mediastinal lymphnode. Microscopically, there were granulomatous like lesions around the bronchioles and infiltrated with inflammatory cells and mediastinal lymphnode were necrotized and mineralized. In case of CBPP grossly there was fibrinous pleuritis and marbling appearance and microscopically, interlobular septa were markedly expanded with abundant edema and inflammatory cells. Serous cyst and calcification were the highest finding in heart of cattle and occurred at same rate 33.3% while pericarditis with cysticercosis was the only finding in camel's heart. The cysticercosis was characterized grossly by hard small size cyst like structure on myocardium

of the heart and microscopically by the presence of cyst in the myocardium, with egg in the reproductive tract of female nematode. In cattle kidneys, cysts were the highest pathological condition to be occurred 34% and hypertrophy were the least in occurrence 9.6%. Grossly, there were several small cysts on the cortex of the kidney and in other case single cyst on the medulla of the kidney. Microscopically the cyst was lined by cuboidal type of cells and other lined by single cell layer of epithelium However, abscess and pyelonephritis were highest in camel and occurred at same rate 50%. In camel pyelonephritis was characterized grossly by enlarged kidney due to accumulation of fluid and internal swelling and microscopically by distended tubules contained hyaline cast and heavily infiltrated interstitium. In cattle livers, fasciolosis were the highest pathological findings 52.1% and grossly it was characterized by deposition of rusted colored materials and immature and mature fasciola in the bile duct and microscopically by fibrosis and haemorrhagic portal tracks, metastasis bile duct in to mucus producing like cells and infiltration of inflammatory cells. In camel cystic liver with fatty change was the highest 60% and grossly it was characterized by variable size cysts and slightly elevated nodular like structure on the parietal surface of liver and microscopically by presence of several cystic area lined by single layer of epithelium accompanied by fatty change and Von Meyenburg complex. The monitory losses as the result of organ condemnation were 877,587.08 ETB in cattle and 169,309.03 ETB in camel. In conclusion, the pathological changes observed in one species were not seen in other species and in other cases same pathological changes were seen in both species and 1,046,896.11 ETB lost due to the organs condemnation from both species as the result of these pathological changes. We recommend further study to be conducted on the root ethological causes of the pathological changes observed in this study.

**Key Words:** *Abattoir, Cattle, Dromedarius Camels, Pathology, Vital Organs*

## 1. INTRODUCTION

Throughout long history, Ethiopians have constantly relied on livestock in order to survive. Livestock in Ethiopia are extremely important as they serve a wide variety of functions in society from social to subsistence purposes (Kimball, 2011).

Cattle are kept for all purpose. However, the purposes of keeping cattle vary with production systems. Traction ranked highest, followed by milk and reproduction/breeding in both crop-livestock and agro pastoral systems. In contrast, reproduction and breeding requirements received higher ranks in pastoralist systems and, for female, requirements for breeding outranked the importance of milk production (Tonamo, 2015).

The Dromedary one-humped camel (*Camelus dromedaries*) is most widely distributed in the hot arid areas. Camels are multipurpose animals with females used primarily as milk producers, the males for transport or draught and both sexes providing meat as tertiary product. The dromedary camel (nicknamed as ship of desert) is a good source of meat especially in areas where the climate adversely affects the performance of other meat animals (Kadim *et al.,* 2005).

However, camels are generally raised in less developed countries and research for improving their reproductive and productive characteristics (Skidmore, 2005) particularly organ diseases has been limited. Diseases of various etiological agents are the major problems faced by camel producing communities. Most of these diseases either directly or indirectly affect the organs of camels (Belina *et al.,* 2015).

Renal function is essential for life. When the kidneys fail the ionic balance will be affected and also excretory wastes will not be disposed. But a problem in one usually leads to problems in the other. Several factors as well as etiological agents are blamed in initiating kidney affections, such factors are nutritional status and toxic agents either of exogenous or endogenous origin together with specific infectious agent (Salem and Hassan 2011).

Liver is considered the most important organ for animal health production and reproduction as many of the metabolic activities of the body occurred in the liver (Radostits, 2000). Hepatocellular injury is one of the pathologic condition affecting domestic animals including camels. Any disease of liver can prove fatal for the animal owing to its imperative role in the body. The root cause of liver condemnation through post-mortem examination are diseases caused by parasites mainly *Fasciola spp.,* bacteria and viruses. The liver disease are reflected by gross and histopathological changes involving size, shape, color, texture and continuity due to inflammation, degeneration which may be congenital, nutritional, vascular or neoplastic (Dar *et al.,* 2018).

The respiratory system is a major system in the animal body which communicates directly with the external environment. The responses of the respiratory tract to injury, and the resulting patterns of diseases, are determined largely by the structural and functional complexity of the system. Pulmonary lesions have been reported to cause decreased productivity and huge economic loss to farmers, because it can interfere with pulmonary functions especially oxygenation of blood and supply of oxygen. Pneumonia (of different types), pulmonary abscess, pleurisy (adhesion), emphysema, hydrothorax, empyema, pulmonary tuberculosis and parasitic infestations are the main pulmonary pathology (Zubair *et al.,* 2004).

Congenital and acquired disorders of the heart have been observed in large animals. Some of the abnormalities cause no clinical signs and they may be picked up incidentally for some other reasons (Shekarforoush *et al.,* 2006). Cardiac diseases in cattle remain medically challenging for the clinician both to detect and to treat. Bacterial endocarditis, traumatic reticulo pericarditis, lymphoma involving the myocardium, dilated cardiomyopathy, ventricular septal defect and other congenital heart diseases are most commonly reported (Buczinski *et al*., 2010).

According to (Berhanu *e al.,* 2015) hydatid cyst, cirrhosis, hepatitis, pneumonia emphysema are the most and major causes for organs condemnation in camel at Akaki abattoir. Fasciolosis, hydatidosis, pneumonia, emphysema, cirrhosis, calcification, nephritis, splenitis,

edema tuberculosis, hemorrhage and abscess are major causes of total condemnation of organs (Hersi 2017).

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In Ethiopia most of the slaughtering taking place at the back yard (AACCSA, 2015). Abattoirs are good sources of valuable information on the incidence of animal diseases and conditions. Prevalence of less acute, chronic and mild diseases with no clinical signs can be detected at slaughterhouses. An abattoir is a focal point to detect diseases of public health importance (Mummed and Webb, 2015). Any observation and information obtained at slaughterhouse can contribute to the understanding of slaughtered animals' diseases. The pathological examination represents a useful tool to make a diagnosis within the slaughter line (Alsari *et al.,* 2017).

Several researches have been done at various abattoirs in Ethiopia on prevalence of different lesions and diseases encountered in different organs only by observing the gross changes. But there are few researches on macroscopic changes of these lesions and diseases in parallel to their microscopic changes. On the top of this, there is no research on comparative pathological study on vital organs of camel and cattle. Therefore, such study is very important to design future approach for control of diseases in animals and humans.

Therefore the objectives of this study are,

- ✓ To describe the gross and microscopic characterization of lesions in lung, heart, kidney and liver of camel and cattle
- ✓ To identify if there is any similarity or difference on lesions or diseases occurrence between camel and cattle
- ✓ To assess monetary losses result from condemnation of organs

## 2. LITERATURE REVIEW

### 2.1. Pathology of Lung

The respiratory system in general constitutes the most extensive surface and gets exposed directly to the environment. Any sudden change in the environment precipitates the infection by interfering with the local defense and rendering the system more susceptible to infections. Various infectious and noninfectious agents can damage lungs and produce significant lesions (Mekuriaw *et al.,* 2016). This implies that the respiratory tract is more prone to injurious agents although major airways and lung parenchyma neutralize or remove the infectious agents that are deposited (Gabinaitiene *et al.*, 2011).

Different infectious agents such as bacteria, viruses and parasites, play a synergistic and interactive role in the etiology of respiratory system diseases observed in cattle. Out of them, bacterial diseases have drawn attention due to variable clinical manifestations, severity of diseases, and reemergence of strains resistant to a number of chemotherapeutic agents (Chakraborty *et al.,* 2014). Even though the primary factors are infectious agents, environmental and management factors are also important in clinical development and rise in economic losses (Pinar and kadir, 2012). The trend of these animals to huddle and group rearing practices further predispose ruminants to infectious and contagious diseases (Kumar *et al.,* 2016).

Although camels are well adapted to their environment and seem to be spared from devastating epidemic infections which threaten other livestock species in the same region, there are however a number of economically important diseases that affect camels. Pulmonary diseases are among the emerging problems of camels that are causing considerable loss in production and death (*Abubakar et al.,* 2010).

*2.1.1. Tuberculosis*

Bovine tuberculosis (bTB) is a major disease of cattle that can also affect humans, and many other livestock and wild animal species. Human infection has not been a major public health problem in developed countries since the introduction of milk pasteurization. Advanced cases in cattle experience loss of condition, and this directly affects the economic value of the animal, but in most developed countries detection of infection leads to movement restrictions being placed on the herd, mandatory slaughter and considerable indirect losses for the farmer (Godfray *et al.,* 2013).

Mycobacteria of the Mycobacterium tuberculosis complex cause tuberculosis in various mammalian hosts but exhibit specific host tropisms. The two major pathogenic species in this complex are *M. tuberculosis* and *M. bovis*, the causative agents of tuberculosis in humans and cattle, respectively. However, it is well known that *M. bovis* is zoonotic, while infection with *M. tuberculosis* has been sporadically reported in domestic and wild animal species, most frequently in animals living in prolonged, close contact with humans. Among domestic animals, infection with *M. tuberculosis* has been most frequently identified in cattle (Ameni *et al.,* 2011). According to Mamo *et al.,* (2011) in Ethiopia they isolate *M. bovis* strain (SB0133) from camel which is similar to cattle strain in pastoral area of East Africa, implies the existence of potential inter-species transmission of the strain among livestock of pastoral area.

Mycobacterium bovis can be shed from virtually any body orifice. The respiratory route is consistently described as the major route of infection although oral infection is also common. This inference is largely based on location of lesions. Once bacteria entered through aerosolized droplets or ingestion it is established in a herd of cattle. The incubation period can range from months to years with the severity depending on the immune system of each individual animal. The bacteria usually enter the respiratory system of a cow and settle in the lungs. Macrophages in lungs are then responsible for phagocytizing the organism. The organism replicates intracellularly after it has been taken up by the macrophages. A granuloma or tubercle forms as the body tries to wall off the infected macrophages with fibrous tissue. The infection can spread hematogenously to lymph nodes and other areas of the body and

cause smaller, two to three mm in diameter, tubercles. The formation of these smaller tubercles is known as “miliary tuberculosis” (Jemal, 2016).

The disease characterized by a gross pathology named tubercles mostly found in the lungs and lymph nodes. However, it occasionally affects other tissues such as the pleura, lactating glands, gastrointestinal and urogenital tracts (Ahmad *et al.,* 2019). The most common form is pulmonary tuberculosis, characterized pathologically by necrotizing granulomas, associated pneumonia (Gupta *et al.,* 2016), collection of epithelioid and giant cells surrounded by a layer of fibroblasts and lymphocytes, having the center of the granuloma necrotic and calcified (Headley, 2002).

The disease in camel was reported in Ethiopia by Richard, and a spontaneous case of camel tuberculosis in Somaliland has been described by Pellegrini. It was characterized by progressive debility, coughing and death within six months. Caseous nodules were found in the lungs, liver, spleen and lymph nodes. Granulomatous masses with caseation were present in mediastinal lymph nodes, and grape-like lesions in the pleural cavity (Mustafa, 1987).

*2.1.2. Contagious bovine pleuropneumonia*

Contagious bovine pleuropneumonia (CBPP), also called cattle lung disease, is a highly contagious disease of cattle and water buffaloes caused by a bacterium (Waithanji *et al.,* 2015). CBPP is caused by *Mycoplasma mycoides subsp. mycoides*, a member of the *Mycoplasma mycoides* cluster, comprising four additional closely related mycoplasmas, i.e., *M. mycoides subsp. capri, M. capricolum subsp. capricolum, M. capricolum subsp. capripneumoniae,* and *M. leachii*, all causing diseases in ruminants (Heller *et al.,* 2016). The bacteria are primarily transmitted through the exchange and inhalation of infectious aerosols when animals are in close contact with each other. The bacteria can be found in nasal discharges, saliva, urine, fetal membranes, and uterine excretions. Transplacental transmission can also occur. Infected CBPP cattle without clinical signs may shed the bacteria when stressed (FADPREP, 2017).

Many authors were reported that CBPP is typical example of multi-factorial diseases, where factors such as inter-current infections, crowding, inclement climatic conditions, age, genetic constitution and stress from transportation, handling and experimentation are important determinants of the final outcome of infection. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, probably prior to the development of pneumonic lesions. Natural infection is by inhalation and results in Bronchitis, alveolitis, bronchiolitis with predominantly neutrophils and mononuclear cellular response constitute the very early inflammation in Mycoplasma pneumonia It is lobar variety of pneumonia in which the inter lobular septa are dilated and prominent due to a great out pouring of plasma and fibrin in to them and it this dilated septa that give the "marbling" effect to the lung in these area (Abera *et al.,* 2016).

The pathological changes, generally characterized by the involvement of only one lung and its gross macroscopic aspects, are well known and may be used for disease surveillance at abattoirs. However, the pathogenetic mechanism of the disease is not yet fully understood. It has been suggested that auto immune and hypersensitive reactions are essential in the development of lesions. CBPP induced lesions vary during the course of the disease (Provvido *et al.,* 2017).

Gross pathological lesions in the acute stage are characterized by fibrinous deposits on the parietal surfaces of lungs and distension of the interlobular spaces with straw colored serofibrinous exudate. Lesions are usually unilateral, localized in the diaphragmatic lobe and present a characteristic marbling appearance. Lesions are detectable on palpation, and upon incision, red and grey areas of hepatization are revealed. In subacute cases, lesions are characterized by necrosis organized within lobules and interlobular septa and early sequestrum formation. Lesions in the chronic stage are characterized by well-defined sequestra surrounded by fibrous capsules. Adhesions, connecting thickened viscera and parietal pleura, are common (Malamsha, 2009).

The lesions develop first in the lymphatic system. Thrombi that develop in the lymphatics cause coagulation of lymph, distension of interlobular septa and focal perivascular round cell

infiltration. The formation of cuffs of round cells around the arterioles is the only histological pathognomonic characteristic of CBPP. The secondary lesion is characterized by alveolar involvement due to the accumulation of exudate from the foregoing changes. Necrotic foci surrounded by a band of polymorphonuclear granulocytes often develop. These foci may develop into sequestra in chronic cases (Laak, 1992).

*2.1.3. Emphysema*

Pulmonary emphysema is anatomically defined as the ‘‘abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls” (Fehrenbach, 2006). Pulmonary emphysema is distension of the lung caused by over distension of alveoli with rupture of alveolar walls with or without escape of air into the interstitial spaces. Over inflation describes the situation in which there is enlargement of airspaces without tissue destruction. Pulmonary emphysema is always secondary to some primary lesion which effectively traps an excessive amount of air in the alveoli. It is a common clinic pathological finding in many diseases of the lungs of all species and is characterized clinically by dyspnea, hyperpnea, poor exercise tolerance and forced expiration (Radostits, *et al.,* 2006).

Pulmonary emphysema is an important lesion only in cattle, although occasional cases occur in pigs. The bovine lung is highly susceptible to the development of emphysema from many different causes, not all of them respiratory in origin. In those of respiratory origin it is common to find pulmonary emphysema when the primary lesion in the lung causes trapping of air in alveoli or terminal bronchioles. Acute interstitial pneumonia, parasitic pneumonia with pulmonary, edema in acute anaphylaxis, Perforation of the lung by foreign body as in traumatic reticuloperitonitis are recorded as causing pulmonary emphysema in cattle. An imbalance between protease and antiprotease activity in the lung is proposed as the major mechanism resulting in emphysema. The imbalance is mostly due to an increase in the numbers of alveolar macrophages and neutrophils. Emphysema can also develop from increased alveolar wall cell death and/or failure in alveolar wall maintenance. Chronic

inflammation and increased oxidative stress contribute to increased destruction and/ or impaired lung maintenance and repair (Sharafkhaneh, *et al.*, 2007).

Chronic bronchitis or bronchiolitis can causes obstruction of airways on expiration due to exudates plugging airway passages. This causes airway imbalance where the volume of air entering exceeds the volume of air leaving the lung. "Check valve" lesion in which air is still able to enter alveoli on inspiration but is unable to leave freely". Pulmonary emphysema can be divided into two broad categories, alveolar and interstitial. In alveolar emphysema the alveoli are distended by excessive amounts of air pressure and often times rupture. In interstitial emphysema the air accumulates in the sub-pleural, interstitial, and intralobular regions of the lungs (Schroeder, 2005). The lungs do not collapse when the thorax is opened and often carry the impression of the ribs. Air pockets are often present and these may be partially filled with coagulated blood. The emphysema may extend to the mediastinal space and reach the musculature and subcutaneous tissue of the dorsal cervical and thoracic regions. Edema is present in the lungs and the air passages contain frothy fluid. The pericardial sac often contains gelatinous transudate. Histopathologically, there is edema and interstitial and alveolar emphysema of the lungs (O'Donoghue, 1960).

*2.1.4. Edema*

Pulmonary edema is the accumulation of excess fluid in the extravascular space of the lungs (McGavin and Zachary, 2012). Edema of the lung was, in many respects, similar to edema of other tissues, and is governed by the permeability of the vascular wall and by Starling forces- the balance of hydrostatic and osmotic pressures between the intravascular and interstitial compartments. Pulmonary edema is often complication of many diseases and is therefore one of the most commonly encountered pulmonary abnormalities. If severe, pulmonary edema has a catastrophic effect on lung function by reducing pulmonary compliance, blocking ventilation of the alveoli, obstructing gas exchange across the alveolar septa, and reducing the surface area of the air-liquid interface in the alveoli. In addition, proteins present in the edema fluid interfere with surfactant function, further reducing compliance and contributing to pulmonary dysfunction (Jubb *et al.*, 2005). Diffuse pulmonary edema was the predominant change in

cattle. Edema is prominent in the pleura and the pulmonary interstitium, and may form shallow pools in the hilus of the lung or the mediastinum (Muhammad *et al.,* 2010).

Edematous lungs macroscopically characterized by large, firm, dark red, heavier than normal and incision results in flow of fluid from cut surface (Jubb *et al.,* 2005). Section of the lung with edema histologically characterized by faintly pink stained edematous fluid is acidophilic granular material with in alveoli except for occasional discrete holes that represent trapped air bubbles and fibrosis in chronic cases. Chronic edema is accompanied by a diffuse increase in the number of alveolar macrophages, and in heart failure these may contain phagocytosed erythrocytes or hemosiderin (Rashid *et al.,* 2013).

*2.1.5. Hemorrhage*

Haemorrhage is escape of the blood from a vessel. It classified as haemorrhage by rhexis when there is rupture of a blood vessel; and haemorrhage by diapedesis when blood leaves through intact blood vessels. Pulmonary hemorrhages are associated with conditions affecting the blood vessels and conditions affecting the blood like severe septicemia or traumatic lesion to the lung (McGavin and Zachary, 2012).

In addition they can cause by lacerations, vasculitis, infarction, ruptured aneurysms, trauma, haemophilia, tumors that have undergone necrosis, or drug reactions. Aspiration of blood is frequent at slaughter, and has a characteristic pattern of multiple, small, bright-red foci with feathery or indistinct borders. Pulmonary hemorrhages vary from petechiation to massive filling of large regions by blood. Affected animals may be found dead with blood flowing from the nares. Agonal hemorrhages resulted from seizures and struggling during slaughter also results in pinpoint hemorrhage over lung surface particularly in the anterior lobes. Histological alterations are resulted from intra alveolar hemorrhage rather than the cause, and can be produced by intrapulmonary injection of auto logos blood and the affected regions characterized by hemosiderin-laden macrophages in airspaces and in the interstitium, alveolar septal fibrosis, and mild bronchiolitis and bronchiolar fibrosis (*Jubb et al.,* 2005). Grossly a

haemorrhagic lung appears as variable size red, brown or grey discoloration of the lung or as patchy, blue-brown subpleural foci (McGavin and Zachary, 2012).

### *2.1.6. Hydatidosis*

Hydatidosis (cystic echnococcusis) is one of the most important parasitic diseases of ruminants responsible for huge economic losses due to reduction in carcass weight gain and condemnation of organs. Hydatidosis is a zoonotic parasitic disease caused by larval stages (hydatid cysts) of cestodes belonging to the genus *Echinococcus* and the family *Taeniidae*. Hydatid cyst, which is the larval stage of Echinococcus, is a bladder like cyst formed in various organs and tissues following the growth of the oncospheres of an Echninoccus tape worm in that specific organ or tissue Martinma (Abegaz and Mohammode, 2018).

Certain deep-rooted traditional activities have been described as factors associated with the spread and high prevalence of the disease in some areas of the country. These can include the widespread backyard slaughter of animals, the corresponding absence of rigorous meat inspection procedures, the long standing habit of feeding domesticated dogs with condemned offal and the subsequent contamination of pasture and grazing fields. This can facilitate the maintenances of the life cycle of *E. granulosus* which is the causative agent of cystic hydatidosis and consequently the high rate of infection of susceptible hosts (Getachew *et al.*, 2012).

The life cycle of hydatidosis involves two mammalian hosts. The adult cestode inhibits the small intestine of carnivores (definitive host) and produces eggs containing infective oncosphers. Cestodes segments, proglotids containing eggs (free eggs) released from the intestinal tract of final host in to the environment. After ingestion of eggs by food animals (intermediate host) such as cattle, sheep, goats, swine and camel, the larval stage (metacestod), develops in the visceral organs typically the matured metacestod produces numerous protoscolices, each having the potential to develop into an adult cestode after being ingested by the carnivore definitive host. Accidentally, ingestion of the eggs infects humans and other

aberrant hosts (Abegaz and Mohammode, 2018). Condemnation of edible offal unfit for human consumption is the major economic loss incurred by Hydatidosis (Erbet *et al.,* 2010).

Ibrahem *et al.,* (2016) reported hydatid cyst wall with intact or disrupted laminated membrane moderate to broad fibrous capsule. Protoscolices, congestion, haemorrhage, fibrosis, and heavy cellular infiltration composed of mononuclear cells mainly lymphocytes. Hyperplasia of the bronchial and bronchiolar epithelium, atelectasis and emphysema were seen in distant lung tissues.

*2.1.7. Cryptococcosis*

Cryptococcosis is a chronic, subacute to acute pulmonary, systemic or meningitic disease, initiated by the inhalation of basidiospores and/or desiccated yeast cells of *Cryptococcus neoformans*. Primary pulmonary infections have no diagnostic symptoms and are usually subclinical. On dissemination, the fungus usually shows a predilection for the central nervous system, however skin, bones and other visceral organs may also become involved. *C. neoformans* and *C. gattii* are regarded as the two principle pathogenic species. Outside the host, *C. neoformans* is believed to exist as a poorly or moderately encapsulated spherical to oval structure with a diameter ranging from 2 to 10 μm. The pathogenesis *of C. neoformans* infection is mediated by four main virulence factors that allow it to survive within the host environment; these include: The ability to grow at $37^0$C, synthesis of an extracellular capsule, production of melanin and secretion of extracellular proteases (Refai *et al.,* 2014).

Histologically the cryptococcosis classified in to two categories there reactive pattern and minimally reactive pattern. Reactive pattern is characterized by a granulomatous inflammatory response composed of histiocytes, multinucleated giant cells and lymphocytic infiltration. Regions of necrosis are occasionally associated with neutrophilic infiltrates. Fibrotic nodules cryptococcomas, considered to be a variant of the reactive pattern-are also found. The minimally reactive pattern is characterized by minimal or no inflammatory response. Numerous spherical microorganisms or oval microorganisms (or a combination of the two) of 2-20 μm in diameter, surrounded by a light halo and arranged extracellularly, are seen. In

some cases, complete destruction of tissue architecture is observed. According to some authors, the minimally reactive inflammatory pattern can be suggestive of poor prognosis (Severo *et al.,* 2009).

### *2.1.8. Atelectasis*

Development of atelectasis is associated with decreased lung compliance, impairment of oxygenation, increased pulmonary vascular resistance, and development of lung injury (Duggan and Kavanagh, 2005).

Lung atelectasis is the failure of alveoli to open or the alveoli are collapsed and thus do not have air. Atelectasis is the collapse of certain portion of pulmonary tissue in absence of air content in alveoli. These lesions are classically localized in the apical and cardiac lobes, and more rarely in the diaphragmatic one. Seem to come with infections with Mycoplasma and Pasteurella species. Macroscopically atelectatic lung characterized by depressed relative to aerated lung, homogeneously dark-red and the texture is fleshy or more firm and non-spongy than normal lung. Section of atelectic lung appears as slightly congested alveolar walls lying in close apposition with cleft-like residual lumina having sharp angular ends. Atelectatic alveoli often contain scant edema fluid and excess alveolar macrophages. The edema may result from hypoxic damage, hypoxic vasoconstriction, or reduced surfactant activity (Jubb *et al.,* 2005).

### *2.1.9. Pneumonia*

Pneumonia is an inflammation of the tissues of the lungs that results from the response of the animal to an infectious agent, either a virus or bacteria, or in most cases both. Common viruses that can initiate pneumonia in cattle include: infectious bovine rhinotracheitis virus, bovine respiratory syncytial virus, parainfluenza 3 viruses, bovine virus diarrhea virus, certain rhino viruses, and a host of uncommon viruses that can affect cattle. Often the virus infection will cause damage to the lung tissue and then bacteria will invade the compromised tissues. The bacteria most often involved include Mannheimia hemolytica (formerly Pasteurella

hemolyticum), Pasteurella multocida, and Histophilus somni (formerly Hemophilus somnus). These bacteria are never far from cattle and are particularly adept at invading lung tissue damaged by viruses. Other bacteria commonly involved in pneumonia include Mycoplasma bovis and Arcanobacterium pyogenes (formerly Actinomyces pyogenes). These are more Latin names than anyone really wants to consider; however, the principal organism involved can influence (Maas, 2008).

Pneumonias in domestic animals can be classified based on texture, distribution, appearance and exudation into four morphologically distinct types: bronchopneumonia, interstitial pneumonia, embolic pneumonia, and granulomatous pneumonia. By using this classification, it is likely to predict with some degree of certainty the likely causes (virus, bacteria, fungi, parasites) and routes of entry (aerogenous versus hematogenous). On the other hand, overlapping of these four types of pneumonias is possible, and sometimes two morphologic types may be present in the same lung (McGavin and Zachary, 2010).

Pneumoconiosis was a common environmental health hazard for camels. This condition was probably associated with the increasingly dusty environment where camels are reared. It can also predispose camels to secondary infections, as there had been associated bronchopneumonia, by interfering with the defense mechanisms of the lungs (El-mahdy *et al.*, 2013).

*2.1.9.1. Aspiration pneumonia*

Pulmonary aspiration in bovines is the inhalation of secretions, forestomach contents or foreign material into the larynx and the lower respiratory tract. The presence of abnormal substances in the airways and alveoli as a result of inhalation is usually referred to as aspiration pneumonia. Injury to the lung will depend, however, on the amount and nature of the aspirate, the frequency of aspiration, the distribution within the respiratory tract and the host's response to the aspirated material (Marik, 2001).

In mature bovines, because of the size of the rumen and the magnitude of contents that can be regurgitated, overwhelming aspiration of regurgitated contents will cause instant death as a result of mechanical asphyxiation. The pH of these contents, even with a severe ruminal acidosis, is more alkaline than that seen in monogastrics, and the chemical burn as seen in the latter is unlikely. However, contamination of the pulmonary tree with pathogenic bacteria is a distinct possibility and even small amounts aspirated can cause aspiration pneumonia after a day or two (Shakespeare, 2012).

Microscopic finding during aspiration pneumonia could be acute inflammatory cells and eosinophilic amorphous exudate in the bronchioles and alveolar space, foamy cells (lipid-laden macrophages) or foreign body giant cell and thickening alveolar septum due to edema and inflammation (Robbins and Cotran, 2005).

*2.1.9.2. Interstitial pneumonias*

Interstitial pneumonias are inflammatory conditions in which the predominant exudative and proliferative responses involve alveolar walls. Grossly, the lesions are distributed widely throughout the lungs. Interstitial pneumonias comprise a significant proportion of cattle respiratory diseases known by different names, such as acute bovine pulmonary emphysema and edema, fog fever, atypical interstitial pneumonia and cow asthma, the condition seems to occur predominantly in late summer or fall (Kerr, 1969).

It is often caused by a blood-borne insult, but can also be aerogenous. Instead, the whole lung seems just bigger and firmer than normal, sometimes even rubbery. The lesions are really easy to see histologically though. The focus of damage is on and within the alveolar walls. There is a wide variety of causes of interstitial pneumonias. Inhalation of high concentrations of toxic gases or fumes will cause interstitial pneumonia. Many of the viruses that arrive at the lung, either from the blood stream, or from the air, settle at the bronchoalveolar junction, and from there quickly move to the alveoli, creating an interstitial pattern (Sorden *et al.,* 200).

*2.1.9.3. Granulomatous pneumonia*

This is a particular type of pneumonia where a pathogen, either inhaled or arriving at the lung via the bloodstream, settles out in the parenchyma to incite a typical chronic granulomatous inflammation. Fungal diseases are most commonly the reason, but also some of the higher bacteria, such as mycobacteria or Rhodococcus equi, will do this as well. Distribution is multifocal, without regard for cranioventral or caudodorsal they can settle out anywhere (Elsiddig and Elsiddig, 2003).

Grossly it is identified by the presence of granulomas in the lung and sometimes in other organs too. Be aware that granulomatous pneumonia can resemble lung cancer and may require histopathological confirmation. Histopathology: Variable size nodules with a necrotic center infiltrated by macrophages and giant cells and surrounded by connective tissue mixed with lymphocytes and plasma cells (López, 2012).

*2.1.9.4. Embolic pneumonia*

This term can be used to include pneumonias caused by any circulating particulates. Lungs are a biologic filter for circulating particulate matter. Causes of embolic pneumonia include those bacteria that tend to travel as septic aggregates. The offending agent could have been inhaled or could have arrived at the lung from the circulation (Millar *et al.*, 2017).

Embolic pneumonia is the result of a showering of septic thrombi into the pulmonary arterial system from major veins such as the caudal vena cava, mammary, uterine and jugular veins. Septic thrombi result from bacterial infections in tissues, resulting in erosion into veins and release of thrombi into the circulatory system. Animals surviving the thromboembolic showering event may later develop pulmonary arterial aneurysms, which may rupture into a bronchus causing rapid blood loss and death. At necropsy, there is severe widespread discoloration, abscessation, and haemorrhage affecting the lungs. Further dissection may find an abscess at the hilus of the liver that has eroded into the vena cava, or an infection of other organs and tissues that has entered other large veins (jubb *et al.*, 1970).

*2.1.9.5. Bronchopneumonia*

The hallmark feature of bronchopneumonia is that the inflammation originates in the bronchial tree. As would be expected, the origin of bronchopneumonia is aerogenous something nasty comes down the conducting tree. Bronchopneumonia frequently comes from something being brought down the trachea into the lungs. In this type of pneumonia injury and the inflammatory process take place mainly in the bronchial, bronchiolar and alveolar lumens (Fulton, 2009).

Within 2-3 days of bacteria becoming established at the bronchioloalveolar junction, there is red consolidation evident. Leukocytes migrating in in large numbers will change the color of the exudates to more of a gray appearance in 5-7 days. It may begin to resolve by 7-10 days, with slow turnover of type II alveolar cells back to the more efficient type I variety. The lung can return to normal by 3-4 weeks. Or, on the other hand, the whole lung can go to heck in a hand basket and the end result will be available for full viewing in the necropsy room within days, with all shades of red and gray, consolidation and fibrin exudation (Britton and Zabek, 2012).

Bronchopneumonia grossly characterized by is of irregular consolidation in cranioventral regions. The cranial and middle lobes are most often affected in those species having well defined lobation (Ertan, 2006). Consolidated lungs vary from dark red, through gray pink, to more gray, depending on the age and nature of the process. Consolidation of the tissue is the single most important gross criterion of pneumonia (Goodwin, 2005). The cut surface of infected lungs shows the variability of involvement seen on the pleural surface. In catarrhal or suppurative bronchopneumonia, the section of consolidated lobules is moist and mucopurulent or purulent material can be expressed from small airways. The cut surface of fibrinous inflammation has a dull and dry appearance (Sorden *et al.,* 2000; Ertan, 2006).

## 2.2. Pathology of heart

Cardiac diseases of livestock may involve valvular structures, myocardium, pericardium, or blood vessels (McGuirk, 1991). Heart disease in cattle remains medically challenging both to diagnose and to treat. This is in part due to its low incidence in the bovine species but also because the prognosis is typically guarded to poor (Buczinski *et al.,* 2010).

### *2.2.1. Pericarditis*

Pericarditis, an inflammation of the pericardium with accumulation of serous or fibrinous inflammatory products, is usually associated with progressive disturbances in the heart function and almost always results in sudden death. It is usually caused by sharp foreign bodies (wire, needles, and nails) which penetrate the reticulum, diaphragm and pericardial sac with a resultant traumatic pericarditis (Ibrahim and Gomaa, 2016). There are three general forms of pericarditis, effusive, fibrinous and constrictive although combination of three can also occur. Effusive pericarditis is characterized by accumulation of a protein rich fluid within the pericardial sac. Subsequent fibrin deposition will lead to fibrinous pericarditis and if fibrin within the pericardial sac matures to fibrinous tissue and fibrosis of the pericardium or epicardium then constrictive pericarditis will result (Perkins *et al.,* 2004).

It is the most common pericardial disorder in cattle. In cattle, it is often attributable to a reticular foreign body that has penetrated the reticular wall, diaphragm and pericardial sac. It is associated with progressive disturbances in heart function and almost always results in death (Braun, 2009). It is acute, subacute or chronic inflammation of the pericardium due to penetration of pericardium by sharp foreign body. Since distance from the reticulum to the pericardium is only a few cms, sharp contaminated foreign body can easily pierce the diaphragm and enter the pericardium. Rarely, traumatic pericarditis may be caused by the penetration of the wire through the skin, with subsequent migration into the sternebrae and pericardial sac (Athar *et al.,* 2012).

Pericarditis attributable to hematogenous spread of infectious diseases (such as colibacillosis, pasteurellosis, salmonellosis and anaerobic infections) is much less common and is usually masked by signs of septicemia (Ueli, 2009).

*2.2.2. Serous cysts*

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According to Marcatoc *et al.,* (1996) grossly the serous cysts were always sessile and roundish or oval and protruding above the atrial surface of the valvular leaflets. They were 1 mm to 3 cm in diameter and histologically they contained a homogenous acidophilic and weakly Periodic acid Schiff (PAS) positive material, generally devoid of cells except for occasional macrophages containing granules of a yellowish PAS-positive and iron-negative pigment. The wall of the serous cyst was always thinner than that of the blood cyst and lacked dense collagen. No blood vessels were found in the wall. However, a few small endothelium-lined channels were observed, connected with the lumen and directed toward the valve base. No direct connection was observed between cyst lumina and the ventricular cavities.

**2.3. Pathology of Liver**

The liver is a vital organ of the body and susceptible to various disease conditions and parasites that affect the total health status of the animal. It is a complex organ that performs many metabolic functions which are dependent upon the integrity and interaction of four anatomic subunits: the hepatic parenchymal cells, the biliary system, the hepatic vascular system, and the Kupffer or reticulo endothelial system. The liver is one of the main metabolic organs and, as such, is positioned between the digestive tract and the systemic circulation. The majority of compounds absorbed from the gastrointestinal tract reach the liver directly through the portal circulation. The liver is the regulatory site of carbohydrate, protein and lipid metabolism. In addition, the liver is an organ responsible for excretion (bile for the digestion of fats), a storage organ (glycogen, vitamins, trace elements), an organ where synthesis occurs (albumin, fibrinogen, prothrombin), and it participates in immune regulation (Kupffer's cells) (Runnels *et al.,* 1965).

Liver is considered to be the most important organ for mammalian metabolism thus any disturbance in this organ will reflect on the general health causing great economic losses in animal production. Liver lesions are common and they indicate the presence of disease in other organs and systems as the liver acts as a catchment for the vast absorptive area of the gut, with all its resident microorganisms ( Borai *et al.,* 2013). Liver is highly susceptible for parenchymal, vascular and biliary system lesions. Bacterial, chemical, viral, toxic or immune mediated insults may cause focal or diffuse hepatic abnormalities or lesions. Liver function is only impaired once more than 80% of the liver has been damaged. However, the liver does possess a unique capacity for maintaining its specific functions and simultaneously repairing and regenerating its own tissue ( Mellau *et al.,* 1987).

*2.3.1. Abscess*

Liver abscesses can occur at all ages and in all types of cattle, including dairy cows, but they have the greatest economic importance for grain-fed cattle. Liver abscesses in feedlot cattle result from aggressive grain feeding programs and are influenced by a number of dietary and management factors. Generally, the incidence and severity of abscesses increase as roughage level in the diet decreases. High roughage levels promote more stable ruminal fermentation and decrease the variation in feed intake, thereby lowering the incidence of acidosis and rumenitis. The ruminal wall that is damaged from acidity or penetration of foreign objects becomes susceptible to invasion and colonization by *F. necrophorum* and subsequently shed bacterial emboli to the portal circulation, leading to infection and abscess formation. Moreover, abscess could be developed as a sequelae of traumatic reticuloperitonitis in cattle (Abdelaal *et al.,* 2014).

Undoubtedly, the virulence factors of *F. necrophorum* play a critical role in the penetration and colonization of the ruminal epithelium and entry and establishment of infection in the liver. The protease activity, dermonecrotic activity, and cytotoxic effect of leukotoxin on ruminal cells may aid in penetration and colonization of the ruminal wall. The *F. necrophorum*, being an anaerobe, has to overcome both high oxygen concentrations and

phagocytic mechanisms in order to survive, proliferate, and initiate abscess formation (Nagaraja and Chengappa, 1998).

The etiopathogenesis of liver abscesses in cattle fed with diets high in readily fermentable carbohydrates and low in roughage is known as the acidosis-rumenitis-liver abscess complex. Bacteria reach the liver through the portal vein and cause a focal infection. Other ports of entry to the liver include the hepatic artery, the umbilical vein, and the biliary tree. Cattle suffering from liver abscesses exhibit no or vague clinical signs, and the final diagnosis is often made at postmortem examination (Dore *et al.,* 2007).

Histologically, liver abscesses have a necrotic center containing leukocytes, hepatocytes and cellular debris. The area surrounding the necrotic center contains macrophages and multinucleated giant cells. The next layer contains plasma cells, degenerating hepatocytes, immature fibroblasts, neutrophils, macrophages, and immature collagen strands. The capsule consists of fibrous connective tissue. At necropsy, the liver may contain a single abscess or numerous abscesses that range in size from pinpoint to over 15 cm in diameter. Peritonitis may be present if an abscess has ruptured into the abdominal cavity. The liver may be adhered to the diaphragm with fibrous connective tissue (Fairchild and White, 2019).

### *2.3.2. Fasciolosis*

Fasciolosis is a liver parasitic infection affecting mainly both domestic and wild ruminants, but monogastrics and even humans can be infected (Arjmand *et al.*, 2015). According to a World Health Organization (WHO) report in 2007, the infection was limited in the past to specific and typical geographical areas (endemiotopes) but is now widespread throughout the world, with human cases being increasingly reported from Europe, the Americas, and Oceania (where only *F. hepatica* is transmitted) and from Africa and Asia (where the two species overlap). Fasciolosis is endemic in 61 countries and has become a food-borne infection of public health importance in parts of the world (Magaji *et al.,* 2014).

The adult flukes in the bile ducts produce eggs which are passed in the faeces. The eggs hatch when separated from fecal material. The miracidia released invade the lymnaeid snails. The tadpole-like cercariae leave the snails and encyst on vegetation, forming metacercariae, which are the infective stage of the fluke. The ingested metacercariae cyst in the small intestine and the released immature flukes penetrate the intestinal wall into the abdominal cavity. The young flukes penetrate the liver capsule and migrate through the liver tissue, entering the bile ducts to become adult flukes (Hutchinson and Stephen, 2007).

Fasciolosis is characterized by increase in the size of the organ due to inflammatory changes in the parenchyma and fibrosis of the bile ducts containing the adult fluke. The histopathological changes in chronic Fasciolosis were characterized by infiltration of fibroblasts admixed with lymphocytes and few mononuclear cells in the area previously migrated by young flukes. Huge proliferations of fibrous connective tissue associated with infiltration of lymphocyte and plasma cells in the portal area have also been reported (Okaiyeto *et al.,* 2012).

In acute fasciolosis, there may be an outbreak of the disease following a massive but relatively short term intake of metacercariae. The high intake is the result of certain seasonal and climatic conditions combined with a lack of fluke control measures; typically, stock forced to graze in heavily contaminated wet areas as a result of overstocking and/or drought. Animals suffering from acute fasciolosis may not show any obvious symptoms. Some animals may show abdominal pain and may become jaundiced. Death is usually due to blood loss resulting from haemorrhage in the liver. The liver haemorrhage is the result of the immature fluke burrowing through the liver. Subacute fasciolosis characterized by jaundice, some ill thrift and anaemia. The burrowing fluke causes extensive tissue damage, leading to hemorrhaging and liver damage. The outcome is severe anaemia, liver failure and death in 8–10 weeks (Hutchinson and Stephen, 2007).

*2.3.3. Hydatidosis*

Hydatidosis (cystic echnococcusis) is one of the most important parasitic diseases of ruminants. Hydatidosis is a zoonotic parasitic disease caused by larval stages (hydatid cysts) of cestodes belonging to the genus Echinococcus and the family *Taeniidae Thompson* and *McManus* (Getachew *et al.,* 2012). The life cycle of hydatidosis involves two mammalian hosts. definitive host and intermediate host (Abegaz and Mohammode, 2018).

Singh *et al.,* (2016), reported that histologically there was slight hemorrhage, leucocyte infiltration and mild hepatocellular degeneration in the liver. Due to pressure effects from developing cysts, adjacent hepatic parenchyma showed atrophy, variable degeneration and lympho mononuclear infiltration. The parenchyma adjacent to cysts was markedly congested and showed multiple small haemorrhagic areas. The cyst may present as a liver abscess and large cyst can produce localized or diffuse hepatomegaly. Local pathological effects depend on the site of the hydatid cyst; ruptured liver cyst through the diaphragm can produce a pleural effusion or broncho biliary fistula .The parasite destroys the liver parenchyma, bile ducts and blood vessels resulting in symptoms of biliary obstruction, portal hypertension and necrosis of the central portion of the cyst with abscess formation (Al Se´adawy and AlKaled, 2012).

*2.3.4. Focal nodular hyperplasia*

Focal nodular hyperplasia (FNH) is the second most frequent benign tumor of the liver (Teixeira *et al.,* 2007). The clinical course of FNH is usually asymptomatic in animals and humans; and its diagnosis is often an incidental finding during surgery, necropsy/autopsy, or imaging procedures for unrelated symptoms. Despite the lack of clinical significance or malignant transformation potential, FNH is an important differential diagnosis of hepatocellular adenoma, well differentiated hepatocellular carcinoma, nodular regenerative hyperplasia and metastatic disease (Souza *et al.,* 2018).

The pathogenesis is not fully understood, but it is highly accepted that an arterial abnormality, often a malformation, causing hypo or hyperper fusion, which triggers reactive hyperplasia of

otherwise normal hepatocytes. This hypothesis is strengthened by the association of FNH with hereditary hemorrhagic telangiectasia and hepatic hemangiomas. However a vascular malformation is not identified in all FNH. Indeed, some lack a dominant feeding artery and are hypovascular or have a peripheral rather than a central blood supply. The liver cells of FNH have been shown to be polyclonal in more than 50% of cases. No somatic mutations in genes have, thus far, been identified in FNH (Geller and de Camposc, 2014).

The gross appearance was that of lobulated, well circumscribed masses, lacking a fibrous pseudocapsule and showing a central scar with fibrous septa running to the periphery and partially demarcating nodular structures. Microscopically, FNH showing a nodular hyperplastic parenchyma with a typical central fibrous scar, containing a proliferation of small bile ducts, irregular tortuous arteries with thickened walls, veins and capillaries. A discrete inflammatory infiltrate filled the fibrous septa surrounding the hepatocytic nodules (Farruggia *et al.*, 2010).

*2.3.5. Cholangiocarcinoma*

Cholangiocellular carcinoma (CC) is a term used for malignant liver tumors originating from intrahepatic and extrahepatic bile duct epithelium (Aslan *et al.*, 2014). Cholangiocarcinoma is relatively uncommon in domestic animals, and the previous reports are documented more in dogs and cats, and less in other species (Azizi *et al.*, 2016).

Macroscopically, CC is usually a firm to hard, white to tan-white mass without extensive necrosis. CC can form a single mass with or without satellite nodules, in most cases not accompanied by cirrhosis. Less frequently, it consists of multiple nodules. Microscopically, classic CCs are adenocarcinomas consisting of tubules, acini, solid nests, or trabeculae, usually embedded in a desmoplastic stroma (Esposito and Schirmacher, 2008).

### *2.3.6. Calcification*

Calcification happens when calcium builds up in body tissue, blood vessels, or organs. This buildup can harden and disrupt your body's normal processes. Calcium I transported through the bloodstream. It's also found in every cell. As a result, calcification can occur in almost any part of the body. 99 percent of body's calcium is in teeth and bones. The other 1 percent is in the blood, muscles, fluid outside the cells, and other body tissues. The Causes of Hepatic calcifications could be infectious lesion, vascular lesion, benign tumor, primary malignant tumor, metastatic tumor and biliary lesion (Stoupis *et al.,* 1998)

### *2.3.7. Fatty liver*

The presence of excessive lipid within the liver is termed as fatty liver. Fatty liver occurs when the rate of triglyceride accumulation within hepatocytes exceeds either their rate of metabolic degradation or their release as lipoproteins. Fatty liver is not a specific disease entity but it occurs as a sequel to many perturbations of normal lipid metabolism which can be due to excessive entry of fatty acid into liver, abnormal hepatocyte function, excessive dietary intake of carbohydrate, increased esterification of fatty acids to triglycerides, decreased apoprotein synthesis and subsequent decreased production and release of lipoprotein and impaired secretion of lipoprotein from the liver (Smith, 2002).

The other mechanism for this is activation of free radicals. For instance, Carbon tetrachloride (CCl4) is converted to the toxic free radical trichloromethyl (CCl3), principally in the liver; causing autocatalytic membrane phospholipid peroxidation, with rapid breakdown of the Endoplasmic reticulum (ER). Hence decline in hepatic protein synthesis of enzymes and plasma proteins; swelling of the smooth ER and dissociation of ribosomes from the smooth ER have occurred. Thus, there is reduced lipid export from the hepatocytes, as a result of their inability to synthesize apoprotein to form complexes with triglycerides and thereby facilitate lipoprotein secretion; the result is the "fatty liver" of CCl4 poisoning (Kitila *et al.,* 2016).

Histological findings in animals with fatty liver include fatty cysts in liver parenchyma; increased volume of individual hepatocytes; mitochondrial damage; compression and decreased volume of nuclei, rough endoplasmatic reticulum, sinusoids, and other organelles; and decreased number of organelles (Kirovski and Sladojevic, 2017).

### *2.3.8. Eosinophilic hepatitis*

Eosinophils may be present as part of a mixed inflammatory infiltrate in portal and perivenous areas and less frequently within the sinusoids. They can be regarded as a nonspecific reactive hepatitis particularly associated with allergic conditions and hypereosinophilic syndromes (Cullen and Winkle, 2007).

### *2.3.9. Cirrhosis*

Cirrhosis is the end-stage of chronic hepatitis and is defined as a diffuse distribution characterized by fibrosis of the liver and the conversion of normal liver architecture into structurally abnormal nodules, micro- or macro-nodules (Elhiblu *et al.,* 2015). Liver cirrhosis results from different mechanisms of liver injury that lead to necroinflammation and fibrosis. Histologically, liver cirrhosis is characterized by diffuse nodular regeneration surrounded by dense fibrotic septa with subsequent collapse of liver structures and thus causes pronounced distortion of vascular architecture in the liver (Nishikawa and Osaki 2015)

### *2.3.10. Sclerosing cholangitis*

Sclerosing cholangitis (SC) is a chronic cholestatic liver and biliary tract disease that has a highly variable natural history. The pathogenesis of the disorder remains elusive, although the complications of the disease are a direct result of fibrosis and strictures involving intra and extrahepatic bile ducts. SC may be asymptomatic for long periods but may also have an aggressive course, leading to recurrent biliary tract obstruction, recurrent episodes of cholangitis, and may progress to end-stage liver disease (Lindor *et al.,* 2015). The classic

description of concentric ductal fibrosis (“onion skinning”) involving bile ducts within portal tract areas (Eaton *et al.,* 2013)

### 2.4. Pathology of Kidney

Generally, kidneys excrete the end-products of tissue metabolism and maintain fluid, electrolyte and acid-base balance via varying the volume of water and concentration of solutes in urine. Kidneys of camel are bean shaped with a very strong, thick and completely adhesive capsule and possesses anatomical requisites for production of hypertonic urine. The renal cortex in camel occupies about 50% of the kidney’s volume, and the ratio of thickness of the medulla to cortex has been evaluated about 4:1. The relative thickness of the medulla is about 7.89 cm. This parameter is an indicator of the length of Henle and vasa recta loops, and according to the countercurrent theory, is consequently an indicator of the kidney ability for urine concentrating. According to previous histopathological study on camel kidney, renal corpuscles and glomeruli are larger than those of other domestic animals. Reportedly, dehydrated camels had 73.00% decreases in tubular reabsorption of sodium leading to an increase of urinary sodium excretion by 42.00%. The kidney of camel is known to play a vital role in water conservation through the production of highly concentrated urine that may predispose animal to varieties of renal dysfunction (Kojouri *et al.*, 2014).

Diseases of the kidney are as complex as its structure but dividing them into those that affect the three basic morphologic components; glomeruli, tubules, interstitum; facilitates their study. This traditional approach is useful because the early manifestations of disease that affect each of these components tend to be distinctive. Further, some components appear to be more vulnerable to specific forms of renal in jury: for example, tubular and interstitial disorders are more likely to be caused by toxic or infectious agents. Nevertheless, some disorders affect more than one structure. In addition, the anatomic interdependence of structures in the kidney implies that damage to one almost always secondarily affects the others. Chronic renal disease can able to destroy all three components of kidney, culminating in chronic renal failure, and what has been called end-stage contracted kidneys (Tavassoly, 2003).

Urinary tract infection exists when bacteria adhere, multiply, and persist in a portion of the urinary tract. Cystitis and urethritis are more common in the female camel because of a shorter urethra and the possibility of retrograde invasion by bacteria. Like other species, urinary tract infection results in food animals most commonly from ascending infection of pathogenic bacteria normally inhabiting the genitourinary epithelium and gastrointestinal tract, or residing in the environment (Tharwat *et al.,* 2018).

*2.4.1. Cyst*

Cystic diseases of the kidney include various conditions characterized by one or more grossly visible cystic cavities in the renal parenchyma. No satisfactory classification of renal cysts exists, but location of cysts, mode of inheritance (or lack thereof), the presence of lesions in other organs, and the clinical course in affected animals are important aspects to consider (Jubb *et al.,* 2016).

Renal cysts can be acquired or congenital, solitary or multiple and involve either one or both kidneys. Furthermore these cysts can be classified as either simple or complicated, in which case they contain cells, bacteria or fungi. An autosomal dominant mode of inheritance for polycystic kidney disease has been demonstrated in Persian cats, and Bull terriers. Ultrasonographically simple renal cysts can mimic renal neoplasia and renal cortical and perinephric abscesses in humans. Simple renal cysts usually have a distinct sonographic appearance, which includes a round to oval contour, anechoic contents, smooth walls (with enhancement of the far wall) and marked distal acoustic enhancement. Cysts can have internal echoes that can be related to haemorrhage or cellular debris, or might be artefactual due to a slice thickness artefact mimicking sediment in the cyst (Kitshoff *et al.,* 2011).

Three mechanisms, which are not mutually exclusive, can lead to the formation of renal cysts: Renal cysts may be caused by obstructive lesions; examples are the acquired retention cysts of chronic renal disease, some dysplastic cysts, and possibly those of glomerulocystic Disease, a fundamental change, of unknown origin, may occur in the tubular basement membrane and result in formation of saccular or fusiform dilations of the tubules. Some dilated segments may

detach from the tubule and form spherical cystic structures. Likewise, detachment of the proximal tubule from the urinary pole will result in dilated Bowman's capsules and so-called "atubular glomeruli." And a disordered growth of tubular epithelial cells may lead to focal hyperplastic lesions and cyst formation (Jubb et al., 2016).

By far the most common cystic disorder of the kidney is the simple cyst. These cysts vary in size from a few centimeters in diameter to a mass holding a liter or more of liquid. From two-thirds to three-fourths occur at the lower pole of the kidney; the majority of the remainder occurs at the upper pole; and an occasional cyst arises from the midportion of the kidney. Characteristically the cyst tends to project from the surface of the kidney and "grow away" from the renal substance rather than encroach upon the parenchyma. For this reason actual destruction of kidney tissue is usually minimal. Grossly, the wall of the cyst is thin; histologically, it is seen to be composed of a single layer of low cuboidal or endothelial type of cell. The remainder of the wall is composed of fibrous tissue. The wall is intimately adherents to the underlying kidney parenchyma, and no well-defined plane of cleavage exists. The contained fluid usually is serous, but in perhaps 15% of the cases the fluid is bloody (Spence *et al.*, 1957).

### *2.4.2. Urolithiais*

Urolithiais was coined from two Greek words "Ouron" means urine and "Lithos" means stone, hence literary means formation of stone anywhere in urinary tract. Urolithiais generally includes nephrolithiasis (Renal calculi or kidney stones), Ureterolithiasis (Ureter calculi) and cystolithiasis (Bladder calculi) (Padma *et al.*, 2016).

Renal stone formation is a biological process that involves physicochemical changes and supersaturation of urine. As a result of supersaturation, solutes precipitate in urine leads to nucleation and then crystal concretions are formed. That is, crystallization occurs when the concentration of two ions exceeds their saturation point in the solution (Parmar, 2004). The transformation of a liquid to a solid phase is influenced by pH and specific concentrations of excess substances. The level of urinary saturation with respect to the stone-forming

constituents like calcium, phosphorus, uric acid, oxalate, cystine, and low urine volume are risk factors for crystallization (Malhotra, 2008). Thus, crystallization process depends on the thermodynamics (that leads to nucleation) and kinetics (which comprises the rates of nucleation or crystal growth) of a supersaturated solution (Kok, 1990).

### *2.4.3. Abscess*

Renal abscess, like any other abscess, is a collection of infective fluid in the kidney. It is usually a sequela of acute pyelonephritis, where severe vasospasm and inflammation may occasionally result in liquefactive necrosis and abscess formation. Abscesses infection of farm animals is detrimental to the livestock due to the tremendous economic losses of animals, meat, skin, and wool production associated with this affection. Camel infections with pyogenic bacteria such as *Corynebacterium pseudotuberculosis*, *C. pyogenes*, *group B Streptococci* and *Staphylococci* have been reported and common in many areas (Zidan *et al.,* 2013). Renal abscess and pyelonephritis are common renal diseases in ruminants, especially in cattle but rarely reported in camels (Tharwat *et al.,* 2018).

An abscess is a defensive reaction of the tissue to prevent the spread of infectious materials to other parts of the body. An abscess results from pus gathering in a tissue of the body that has formed a cavity due to an infection. The pathophysiology of an abscess is a series of immune responses beginning with the migration of white blood cells to the infection and the separation of a fluid-filled cavity from the surrounding, healthy tissue. Some abscesses result from blocked ducts in glands, while others are caused by infected injuries, frequently by the bacterium *Staphylococcus aureus*. The pathophysiology of abscesses begins in damaged tissue when the immune system prevents foreign substances and potentially harmful microorganisms from spreading. During infection, large numbers of white blood cells, particularly neutrophils, migrate to compromised tissue. They do these following signals from cytokines that alert them to cell death and injury. Pus, the mixture of dead cells and the chemical mediators of immune response, fills the area around the site, which is separated from healthy tissue by the formation of an abscess wall (Kojouri *et al.,* 2014).

# 3. MATERIALS AND METHODS

## 3.1. Study Population

The study populations were cattle and camels brought from different parts of Ethiopia for slaughter purpose at Akaki abattoir. 3,520 cattle and 560 camels were involved in the study regardless of their age, sex, body condition and origin.

## 3.2. Study Design and Sampling Method

In this study cross-sectional study design was used. The samples were collected between October 2018 to February 2019 and purposive sampling was used to collect the samples. Livers, lungs, hearts and kidneys with only gross lesions were collected and thoroughly examined. Organs without visible gross lesions were excluded from being study.

## 3.3. Macroscopic Examination

Macroscopic examinations were done according to FAO (2007). The livers, lungs, hearts and kidney were examined externally and internally for the presence of any changes. The post slaughter examinations involved visualization, palpation, incision and olfaction of target organs of this study and associated parts of these organs like mediastinal lymphnodes and the whole carcass. All lesions observed on the organs were recorded and coded. The representative sample with thickness of 5 mm were taken from the affected organs and mediastinal lymphnodes by doing single cut, put them immediately in to 10 percent neutral buffered formalin in a ratio of 10:1, formalin:tissue and labeled them (Brown, 2012). The samples transported to Addis Ababa University College of veterinary medicine and agriculture for storage.

### 3.4. Microscopic Examination

After proper fixation, the tissue were trimmed 2-3 mm to reach the adequate size and orientation of the tissue, put them in tissue castes and labeled the castes to make ready for automatic tissue processing procedure. In automatic tissue processing machine, the tissue dehydrated in ascending grades of ethanol, cleaning with xylene, impregnated with molten paraffin wax then embedded with molten paraffin wax to make tissue block, thin tissue sectioned made at 5 microns in thickness from the tissue block to make tissue ribbon, the tissue ribbon on microscopic slides stained with hematoxylin and eosin stain and mounted with drop of DPX (a mixture of distyrene, plasticizer and xylene) for general microscopic examination details on annex I (Slaoui and Fiette, 2011). The tissue processing was done at University of Gondar College of Veterinary Medicine and Animal Sciences pathology laboratory.

### 3.5. Assessment of Financial Loss

The total financial loss due to organ condemnation was computed based on the average cost of each type of examined organs, average number of animals slaughtered in the abattoir per year from retrospective data of the abattoir and condemnation rate of each organ. Average local market prices of each organ were collected by asking different butcheries' houses in Addis Ababa and took the average price (Ogunrinade and Ogunrinade 1980).

$EL = \Sigma srx * Coy * Roz$

Where:

EL = financial loss estimated due to organ and carcass condemnation from market.
Σsrk = Annual cattle and camels slaughter rate of the abattoir
Coy = Average cost of liver, kidney, heart and lung for cattle and liver, kidney and heart for camel because the lungs of camel were disposed weather they are abnormal or normal

Roz = Condemnation rates of liver, kidney, heart and lung for cattle and liver, kidney and heart for camel

### 3.6. Data Analysis

Data obtained from abattoir and laboratory investigations were recorded, checked and coded using Microsoft excel. Qualitative methods were used to describe macroscopic as well as the microscopic change occurred in the lungs, hearts, kidneys and livers of cattle and camels. Descriptive statics were used to summarize data.

### 3.7. Ethical Clearance

Ethical clearance was not necessary as study was based on only post-mortem examination of organs from the animals slaughtered for food purpose

## 4. RESULTS

### 4.1. Pathological Changes Encountered in Lungs of Cattle

Out of 3520 examined cattle 23.2% (820/3520) had different pathological changes in their lungs. The frequency of tuberculosis like lesions were 0.24% (2/820), granulomatous pneumonia 0.12% (1/820), CBPP 0.12% (1/820), hydatidosis 39% (320/820), emphysema 23.4% (192/820), edema 7.8% (64/820), hemorrhage 24.1% (198/820) and bronchopneumonia 5.1% (42/820). Among these hydatidosis the most frequently occurred pathological change in the lung followed by hemorrhage, emphysema, edema, bronchopneumonia, tuberculosis and the least pathological changes were granulomatous pneumonia and CBPP.

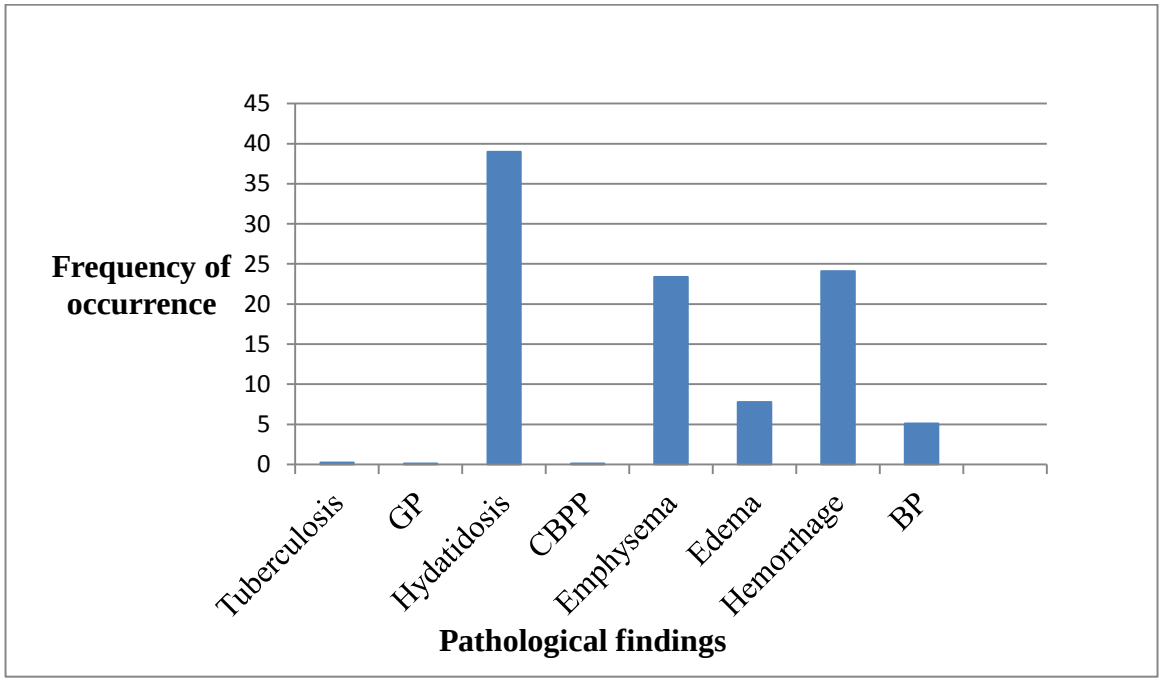



**Figure 1:** Frequency of pathological findings in lung of cattle

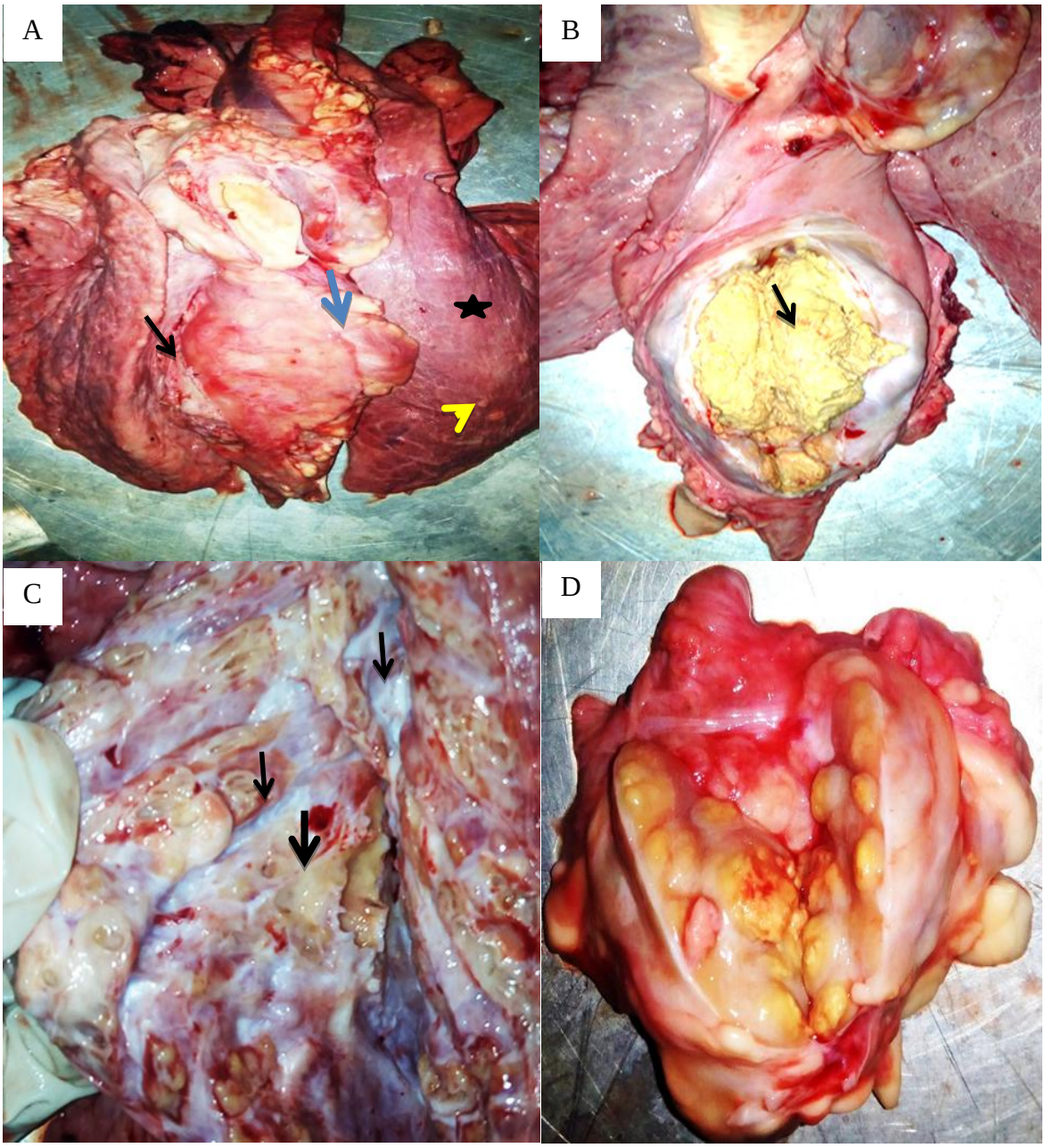
Key GP=granulomatous pneumonia

BP=bronchopneumonia

*4.1.1. Cavitary tuberculosis*

The rate of occurrence of cavitary tuberculosis was 0.12% (1/820). Grossly, the lung had meaty nature and rounded to sharp lobs (Figure 2 A) there were multiple dark to pale, irregular, raised nodule like structure on pleural surfaces of all lob of the lung (Figure 2 B). Between left and right lobs there was large sack like structure containing doughy like yellowish material inside it (Figure 2 C). Upon incision of lung there were multiples markedly dilated or cavities like structures filled with caseous materials (Figure 2 D). The mediastinal lymph node was enlarged and replaced by multifocal, yellowish materials (Figure 2 E).

Histologically, there were multiple granulomatous lesions with caseous central necrosis. There was fibrosis of lung parenchyma (Figure 2 E). The granulomatous areas were infiltrated with foamy macrophage, multi nucleated giant cell and cellular debris (dead neutrophils) (Figure 2 F and G), the bronchiole was dilated (bronchiectasis) and there was focal granulomatous lesion on the smooth muscle of bronchioles (Figure 2 H).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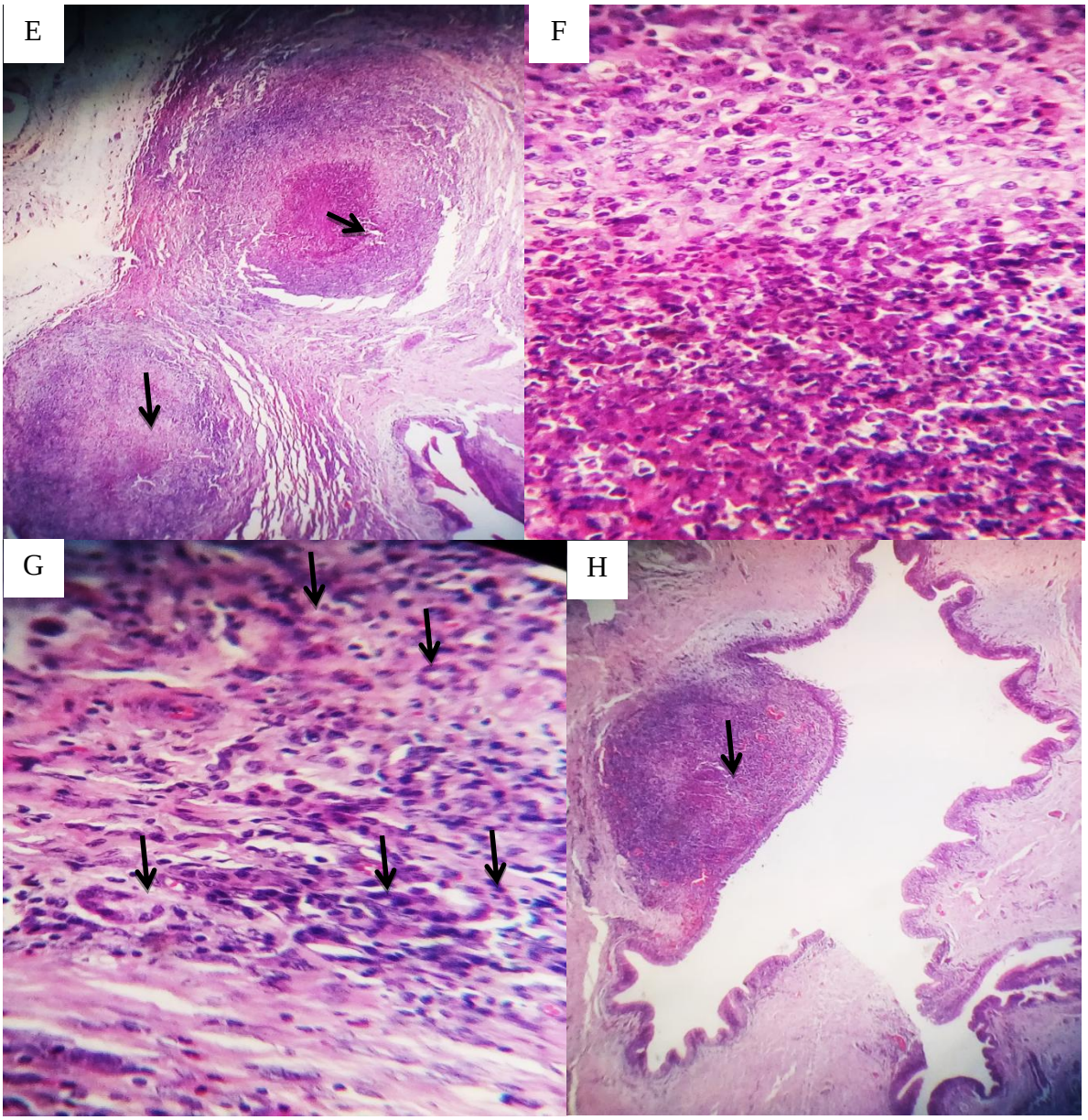


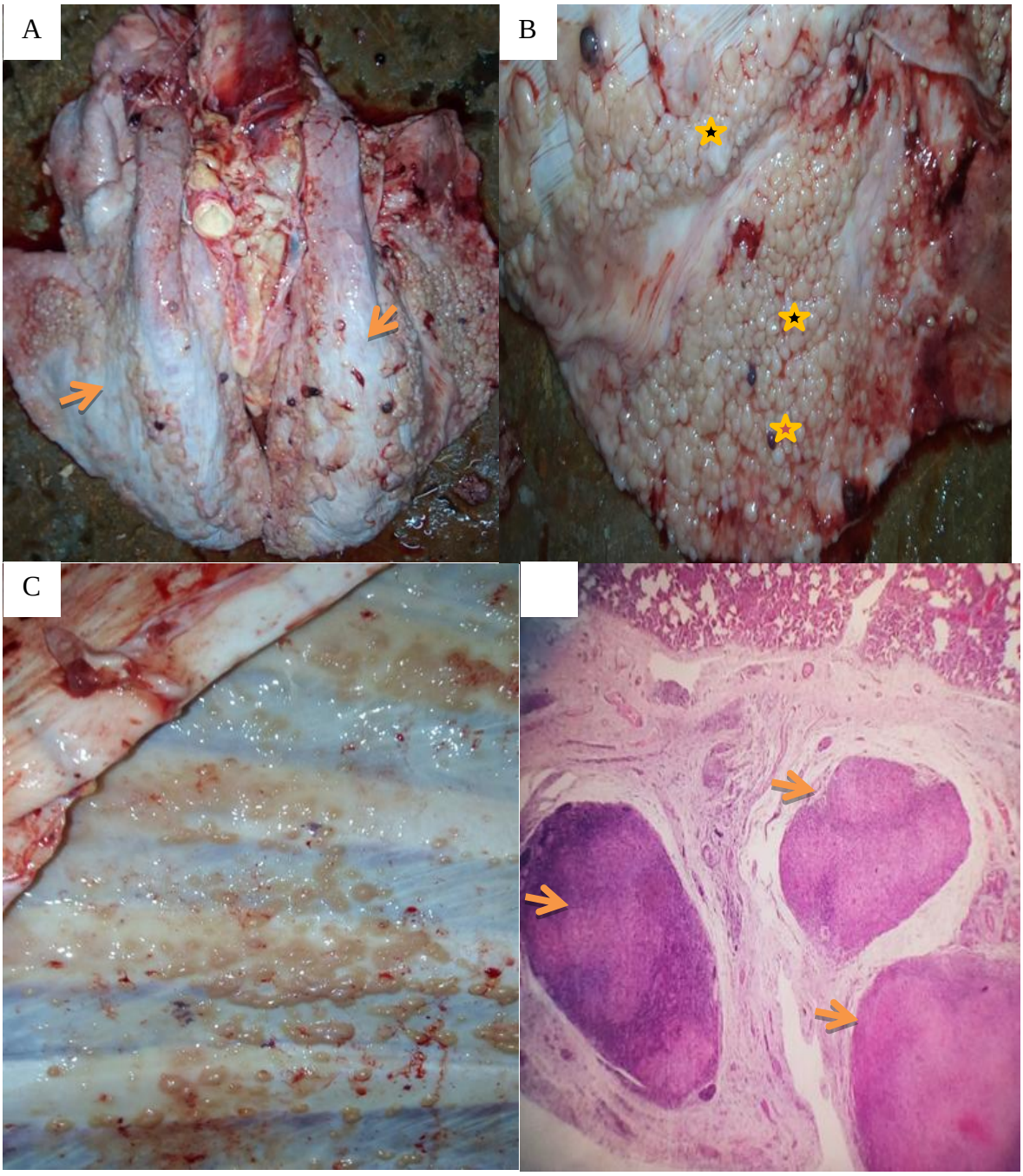

**Figure 2:** Cavitary tuberculosis cattle, macroscopic and microscopic change

(A) Lung showing meaty appearance with irregular raised nodules arrow (arrow head), rounded (star) and sharp lob (arrow) and swollen area between the lobs (blue arrow). (B) Doughy material in sack like structure. (C) Dilated bronchi filled with caseous exudate. (D) Mediastinal lymphnode with multifocal caseous nodules. (E) Granulomatous lesions with caseous central necrosis (arrows). Inflammatory cells (F). G) Multinucleated giant cells (arrows) and (H) Bronchiectasis with granulomatous like lesion (arrow).

*4.1.2. Miliary tuberculosis*

The rate of occurrence of miliary tuberculosis was 0.12% (1/820). Grossly, the whole pleura surface of the lung was covered by fibrinous structure and there were 1 mm to 2 mm whitish nodules of miliary tuberculous granulomas on left and right lobs of the lung (Figure 3 A). On left dorsal area of the lung the nodules were slightly larger and irregular and the nodules were extensively seen on the left lob. The nodules were firm in consistency. The visceral and parietal pleura were thickened, firmly attached to the lung and had ligament like appearance (Figure 3 B). The mediastinal lymph node was relatively normal except having very minute size of yellowish nodules in some area. Variable small sizes of nodules that had rounded structure and firm consistency were also seen in the thoracic cavity (Figure 3 C).

Histologically, there were multiple granulomatous lesions with central necrotic area (Figure 3 D). The central necrotic areas were infiltrated by dead neutrophils, macrophage and to the periphery lymphocytes (Figure 3 E) and multinucleated giant cells (Figure 3 F)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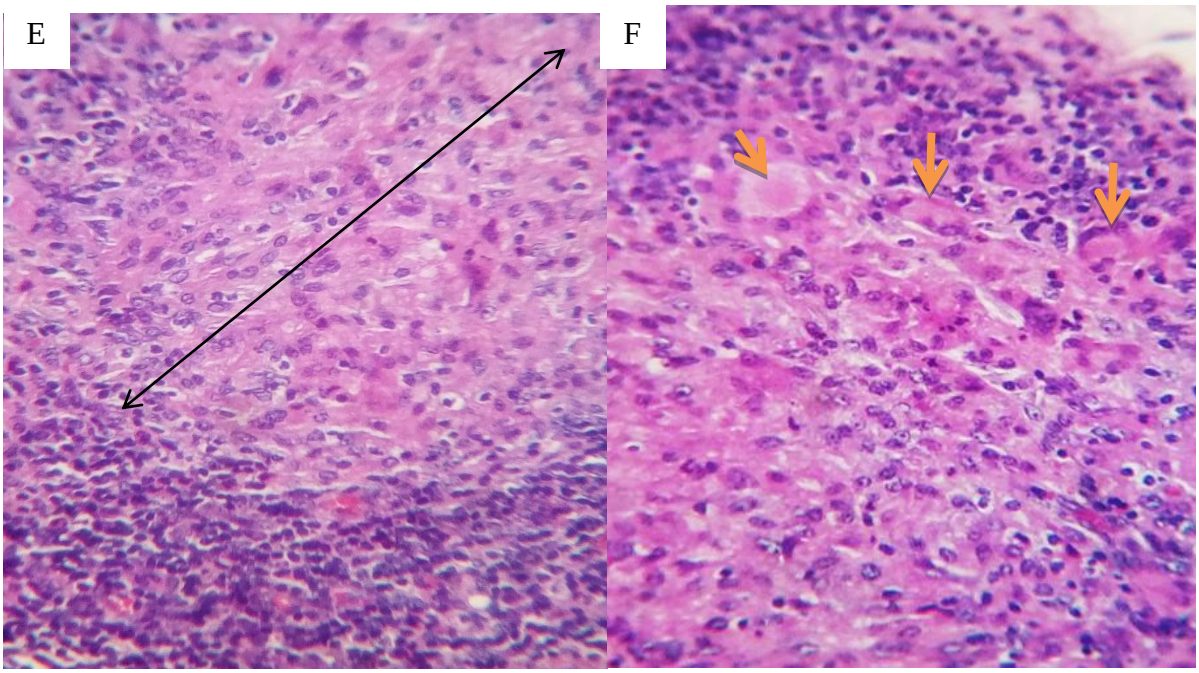


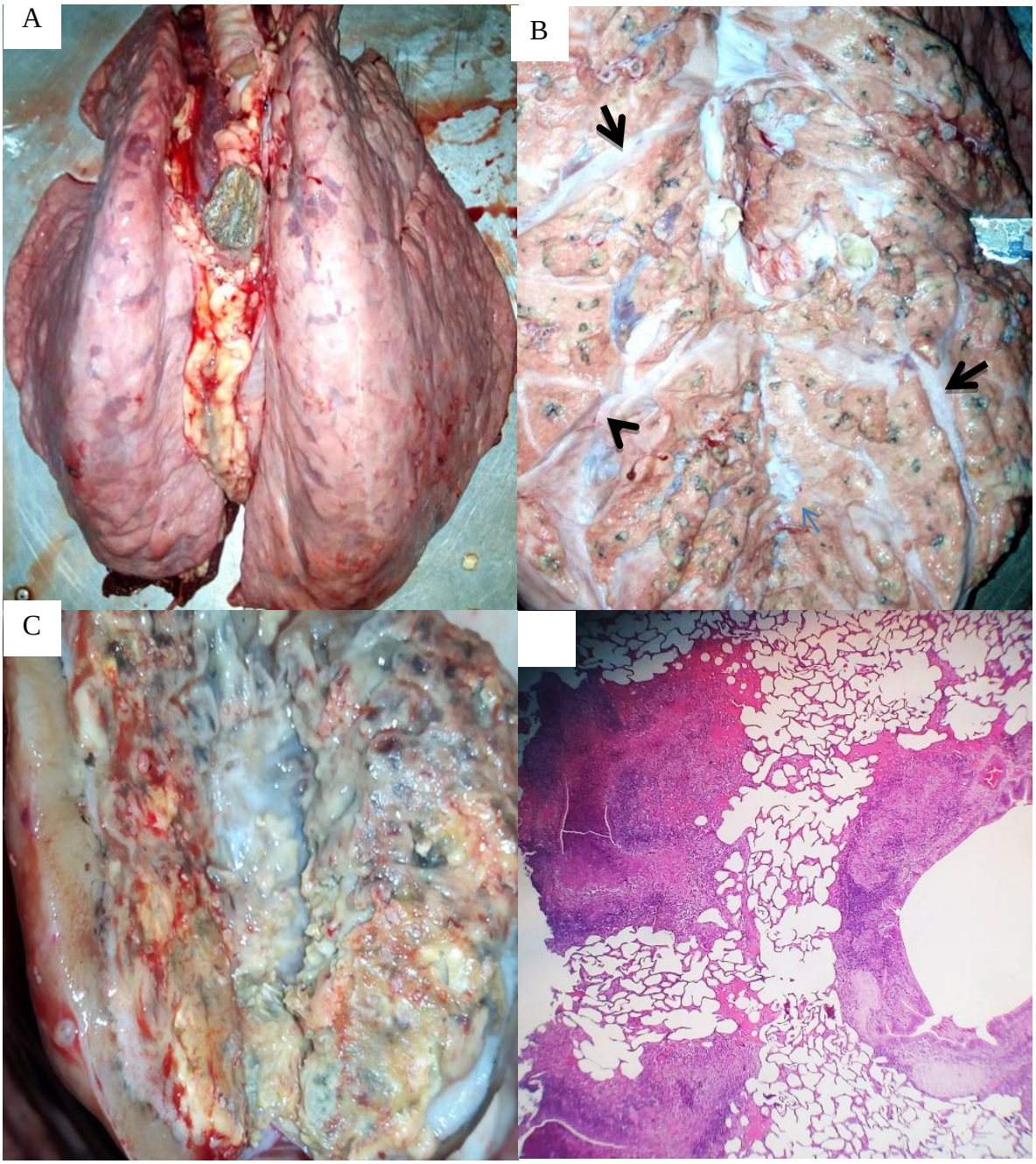

**Figure 3:** Miliary tuberculosis cattle, macroscopic and microscopic change

(A and B) thickened pleura surface of the lung (arrows) with variable size of nodules (star). (C) Nodules in the thoracic cavity. (D) Granulomatous lesions with central necrotic area (arrow). (E) Central necrotic area surrounded by inflammatory cells. (F) Multinucleated giant cells (arrows).

*4.1.3. Granulomatous pneumonia*

The rate of occurrence of granulomatous pneumonia was 0.12% (1/820). Grossly, the lung was enlarged, it was non-collapsed and had dark red area of streak or discoloration all over the lung (Figure 4 A). Upon incision, it was moist. Interlobular space was dilated with mucoid like exudates; the bronchioles were dilated because of accumulation of garish to yellowish caseous to materials inside it (Figure 4 B). The mediastinal lymph node was enlarged and on cut it had yellowish to gray caseous and highly calcified materials (Figure 4 C).

Histologically, there were variable size irregular granulomatous like lesions around the bronchioles, there were expansions of bronchioles and the parenchyma of the lung surrounded the bronchioles were necrotized, edematous and infiltrated with inflammatory cells like dead neutrophils, lymphocytes and macrophage (Figure 4 D and E) and giant cells (Figure 4 F). The alveolar septa were distended and filled with eosinophilic fluids (Figure G). The mediastinal lymphnodes were necrotized, its the normal structure were lost and had deposition of minerals (Figure 4 H).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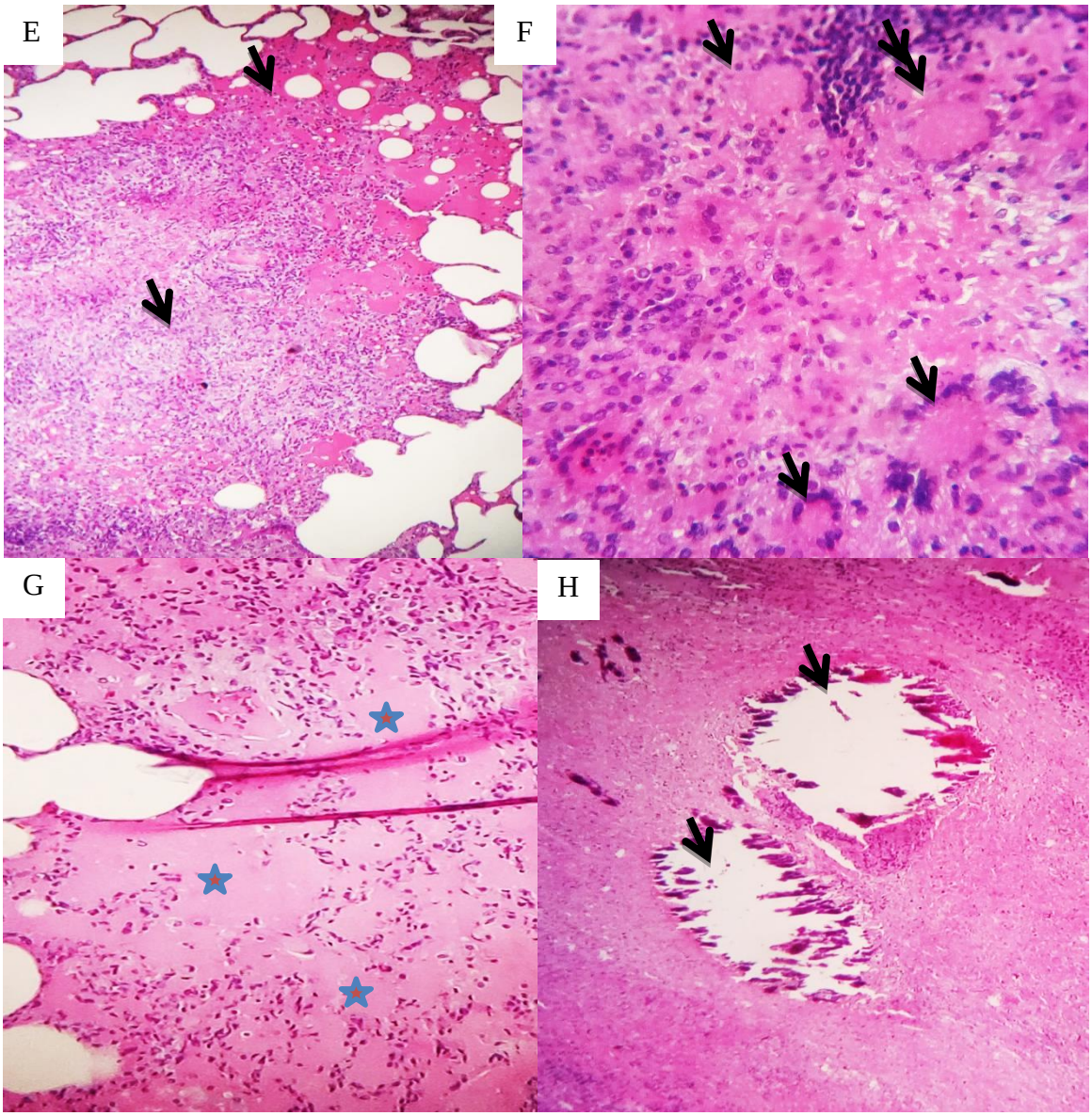


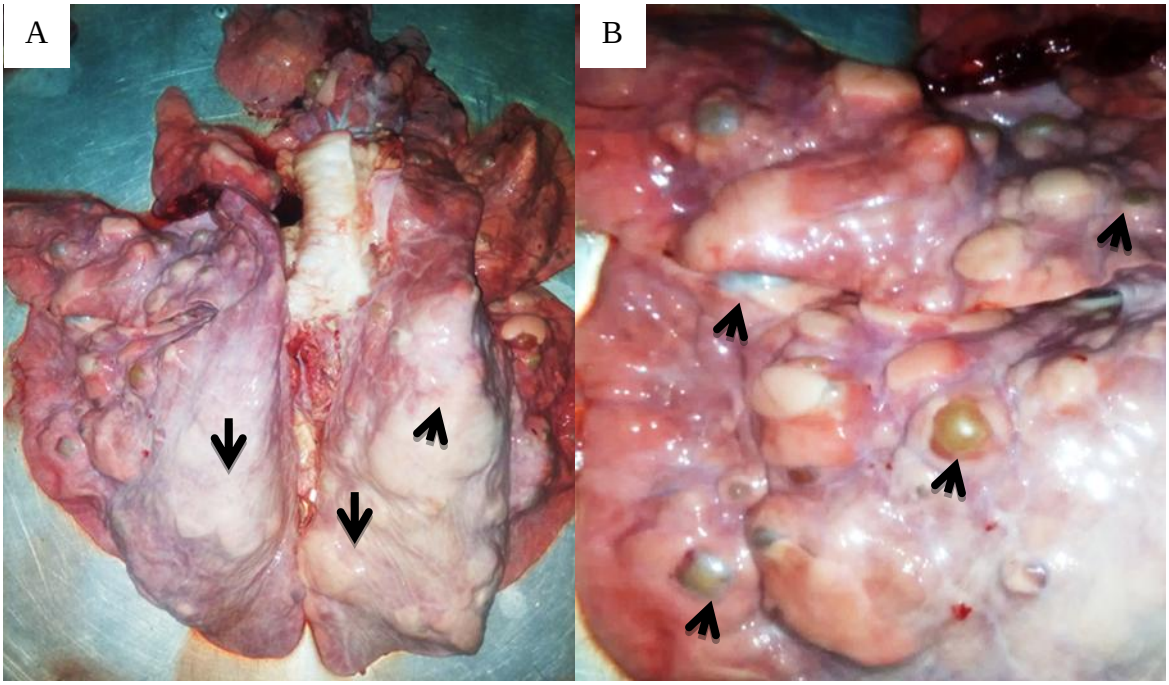

**Figure 4:** Granulomatous pneumonia cattle, macroscopic and microscopic change

(A) Dark red area of streak. (B) Dilation of interlobular septa with mucoid like exudates (arrow) and garish to yellowish caseous materials in the bronchioles (arrowhead). (C) Yellowish to gray caseous and calcified materials in the mediastinal lymphnode. (D) Granulomatous like lesions around the bronchioles (arrows). (E) The granulomatous lesion which is necrotized, edematous and infiltrated with inflammatory cells. (F) Giant cells. (G) Edema in alveoli. (H) Necrotized and mineralized mediastinal lymphnode.

*4.1.4. Pulmonary hydatidosis*

The prevalence of occurrence of hydatidosis was 39% (320/820). Grossly, the lung was pale and had variable sized cysts protruding on the surface. The cysts of varying sizes were also found deep in the lung parenchyma resulting in gross enlargement of the lung. The cysts were yellowish and grey in colored and filled with clear to turbid fluids (Figure 5 A and B).

Histologically, the cyst had three layers: outermost pericyst is fibrous, middle ectocyst layer is laminated, hyaline and acellular and the inner endocyst is the germinative layer which consists of daughter cysts and brood capsules (Figure 5 C). The parenchyma of the lung that surrounded the cyst was fibrosis, edematous, had accumulation of calcified material to the periphery and infiltrated with macrophage, lymphocytes and eosinophil (Figure 5 D). Higher magnification of macrophage, lymphocytes and eosinophil (Figure 5 E).



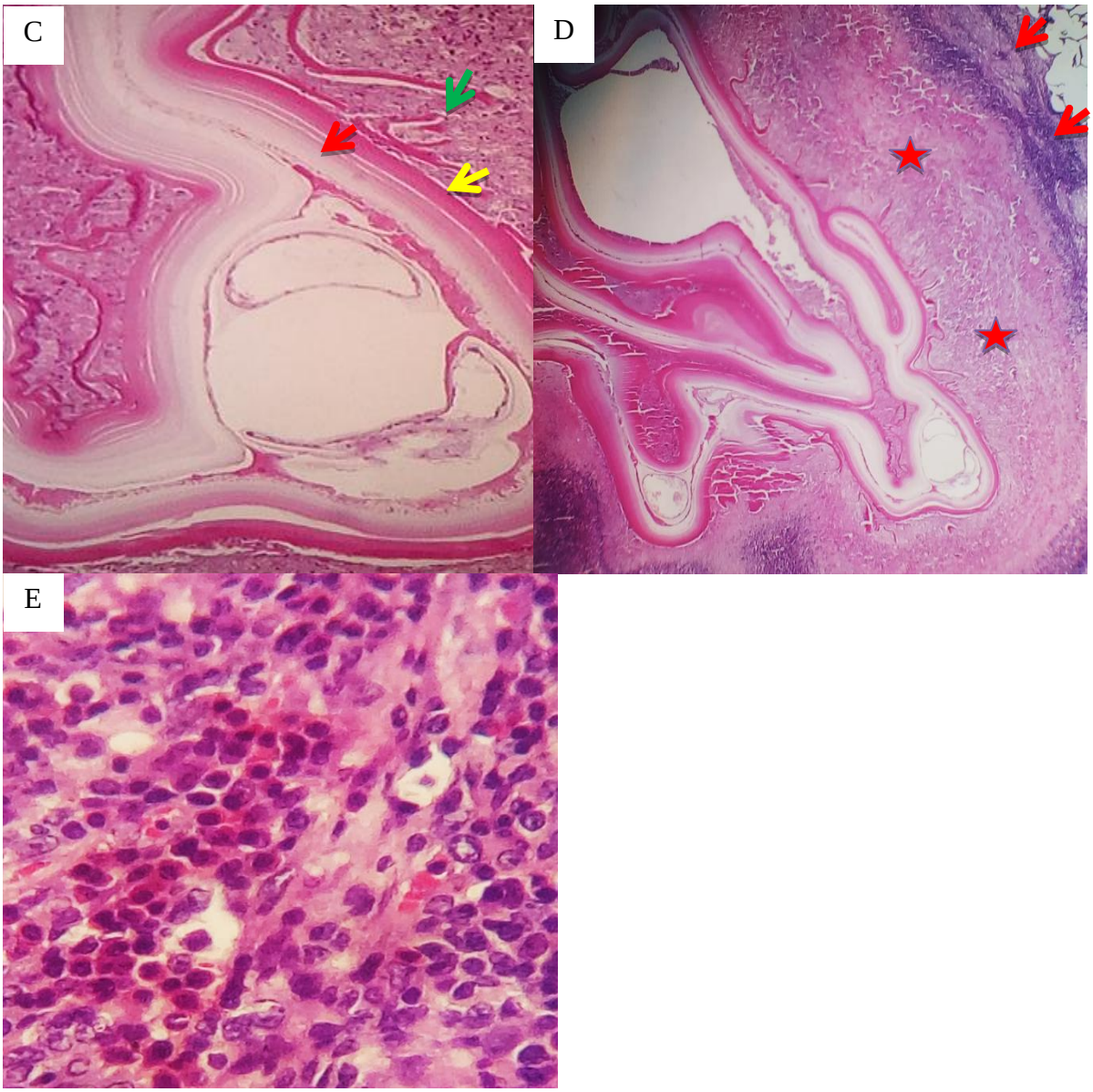


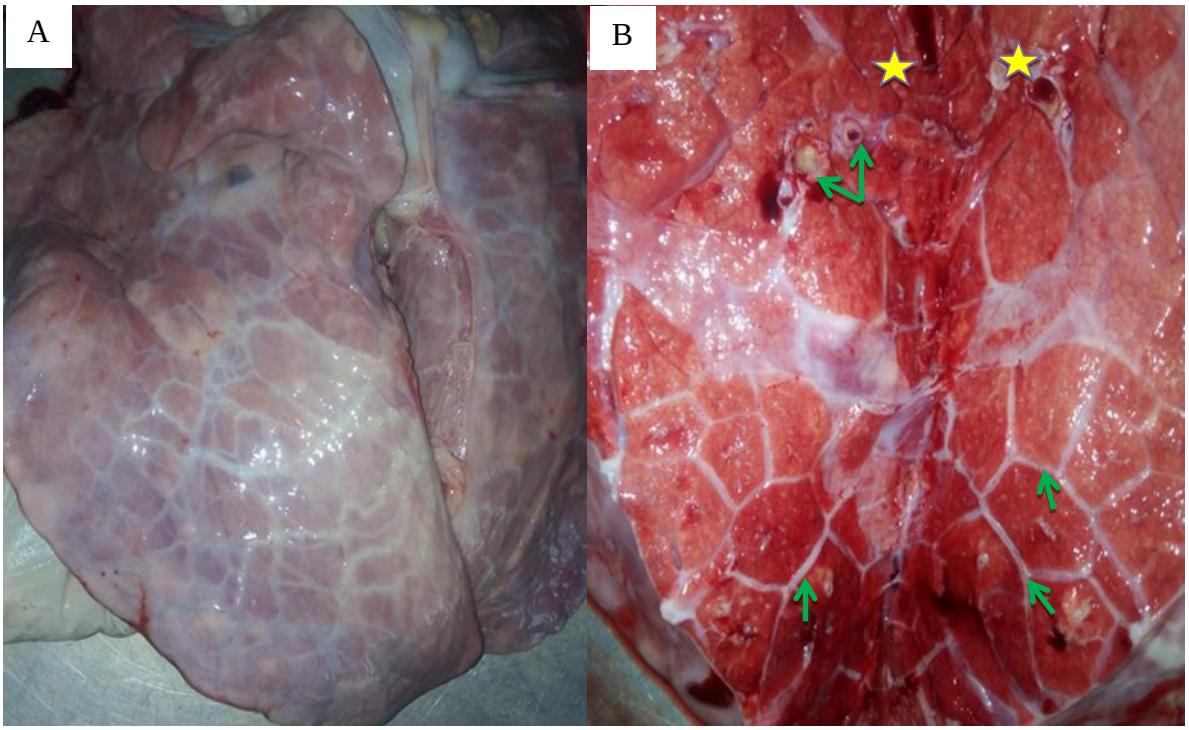

**Figure 5:** Pulmonary hydatidosis cattle, macroscopic and microscopic change

(A) Variable sized cysts. (B) cysts embedded at different depths of lung. (C) Cyst with outermost pericyst (green arrow), middle ectocyst layer (yellow arrow) and inner endocyst with daughter cysts and brood capsules (red arrows). (D) Fibrosis parenchyma of lung, edematous and infiltrated with plasma cell and macrophages (stars) and calcified material (arrows). (E) Eosinophils, macrophage and lymphocyte infiltration.

*4.1.5. Acute contagious bovine pleuropneumonia*

The rate of occurrence of CBPP was 0.12% (1/820). Grossly, the left and right lobs of the lung had prominent fibrinous pleuritis (Figure 6 A) and on cut section, a small portions of the healthy lung were seen, while the remaining surfaces of the lung showed thickening of interlobular septa with white networking appearance (so-called marbling appearance of the lung). Lobules were pale to red and wet (edematous) some of the bronchioles were filled with caseous materials (Figure 6 B).

Histologically, the interlobular septa were prominent and highly expanded than normal size. These expanded interlobular septa had abundant edema throughout the septa and infiltrated with, lymphocytes and macrophages (Figure 6 C and D). The lumen of pulmonary arteries was filed with blood (thrombosis) which is an essential part of the pathogenesis of the disease (Figure E). There were formation of cuffs of round cells around the arterioles which is the only histological pathognomonic characteristic of CBPP (Figure 6 F).



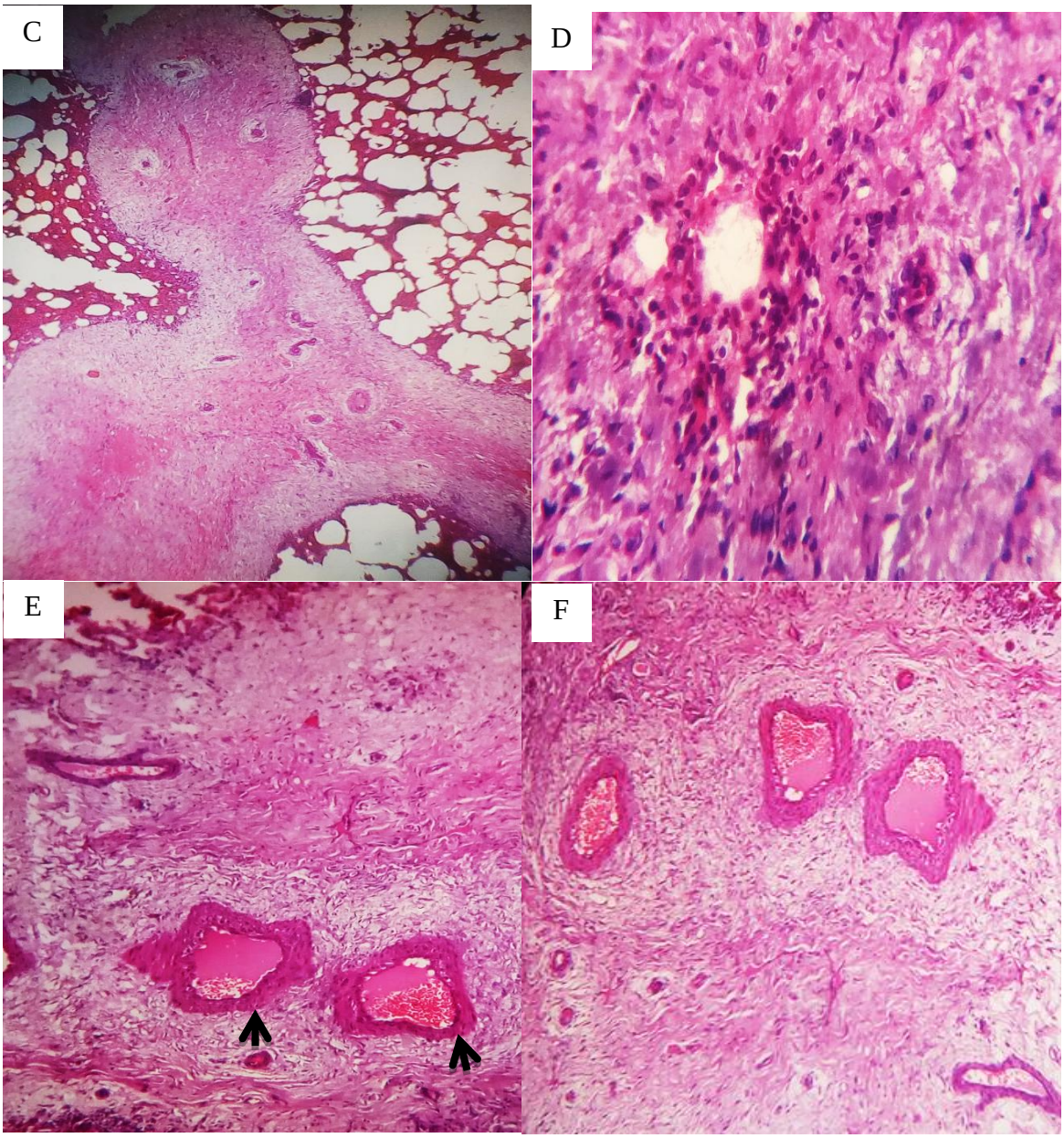


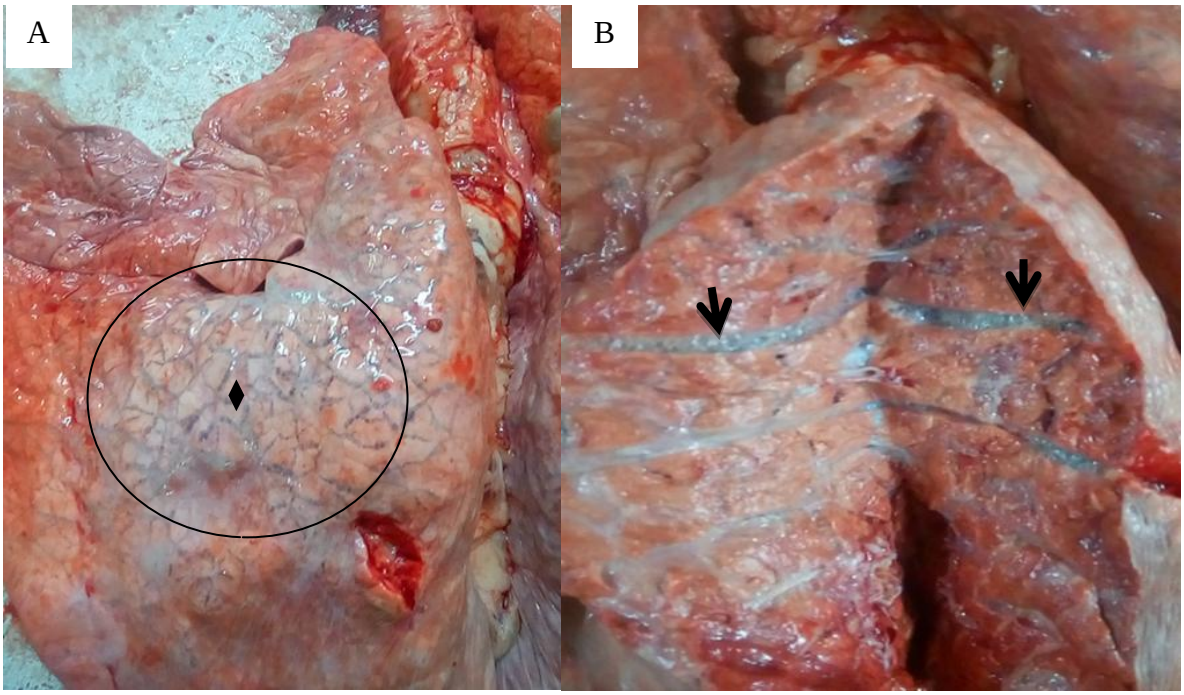

**Figure 6:** Contagious bovine pleuropneumonia, Macroscopic and Microscopic change

(A) Fibrinous pleuritis. (B) Slightly healthy lung (stars) and marbling appearance of the lung (arrows) and bronchioles filled with caseous exudates (arrow). (C) Markedly expanded interlobular septa with abundant edema. (D) Inflammatory cell on the septa (E) The lumen of pulmonary arteries filed with blood (arrows).

*4.1.6. Interlobular emphysema*

The rate of occurrence of different type of emphysemas was 23.4% (192/820), but histologically confirmed interlobular emphysema was 0.12% (1/820). Grossly, the right lob of the lung was distended and had pale area while the left lob seemed relatively normal. There were markedly visible numerous air bubbles from pleura surface of the lung (Figure 7 A) and on cut the interlobular space of the lung were distended with these air bubbles (Figure 7 B).

Histologically, the interlobular spaces were highly dilated (Figure 7 C), the adjacent bronchioles and the alveolus were distorted due to the pressure from the accumulation of the air between the interlobular spaces. The interstitial tissue were thickened, edematous, the capillary were distended with blood and infiltrated with inflammatory cells (Figure 7 D). The interlobular tissues were highly fibrosed (Figure 7 E). There was also interstitial pneumonia with thickening of alveolar septa and alveolar wall with infiltration of inflammatory cells and there were slightly hemorrhagic area on thickened alveolar wall (Figure 7 F).



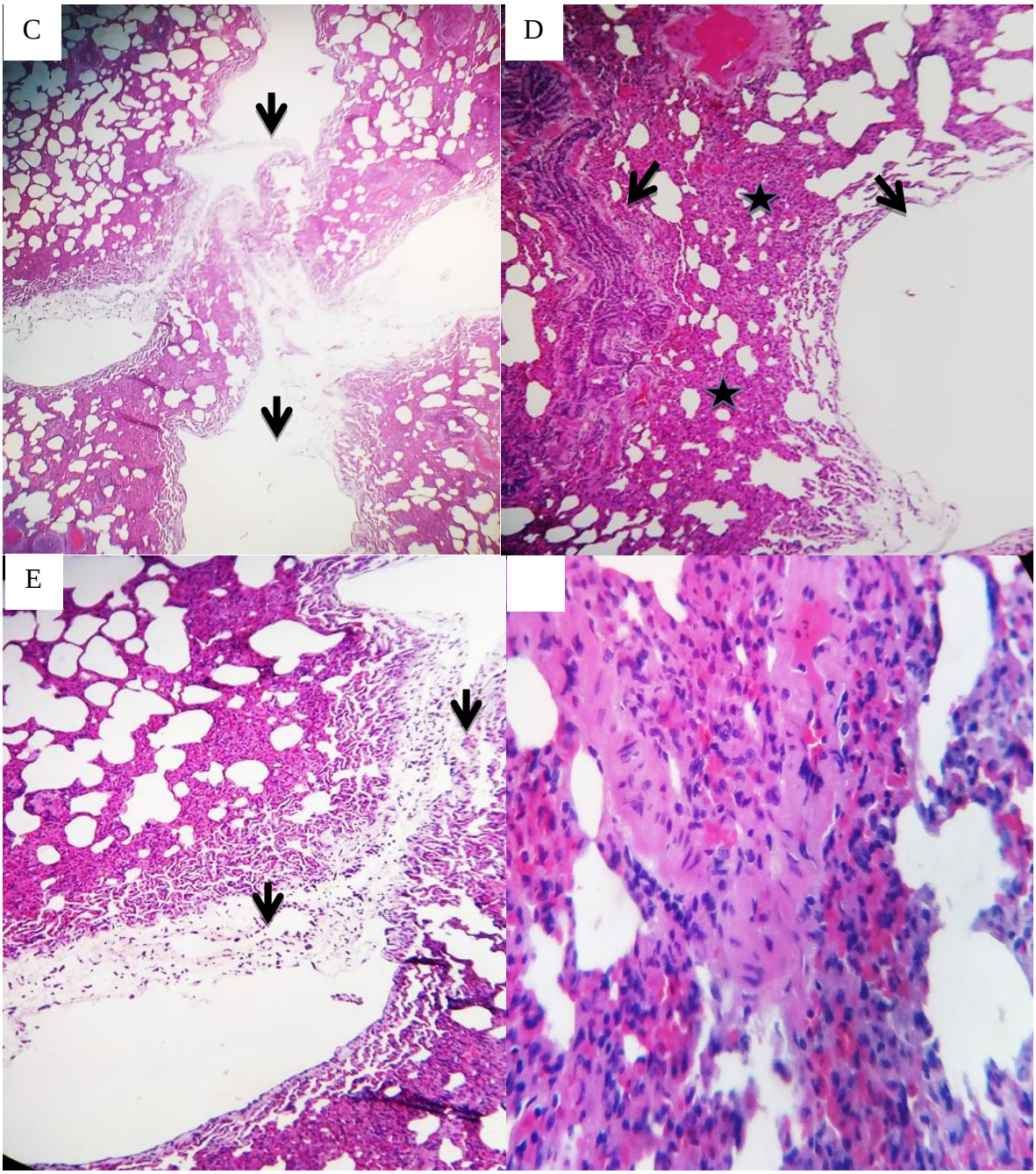



**Figure 7:** Interlobular emphysema in cattle, macroscopic and microscopic changes

(A) Pale and enlarged area of the lung (circle). (B) Distended interlobular space with air bubbles (arrow). (C) Dilated the interlobular spaces (arrows). (D) Distorted bronchioles and the alveolus (arrow) and thickened, edematous interstitial tissue (stars) distended capillary with blood. (E) Fibrosis interlobular tissue (arrow). (F) Interstitial pneumonia

*4.1.7. Interstitial edema*

The rate of occurrence of different type of edema was 7.8% (64/820), but histologically confirmed interstitial edema was 0.12% (1/820). Grossly, the overall surfaces of the lungs had meaty appearance, were moist, dull and shrunken (Figure 8 A), there was focal markedly prominent interlobular space on the border of the left lob due to accumulation of fluid. There tissues surrounding the interlobular space were loose (Figure 8 B) and upon incision clear fluids were oozed from it.

Histologically, the interlobular space of the lung was distended and had edematous parenchyma (Figure 8 C). There were thickening of the alveolar wall with infiltration of inflammatory cells. The interstitial tissue was covered with pinkish fluid (edema) and some of the bronchioles were thickened, fibrosis and collapsed (Figure 8 D).

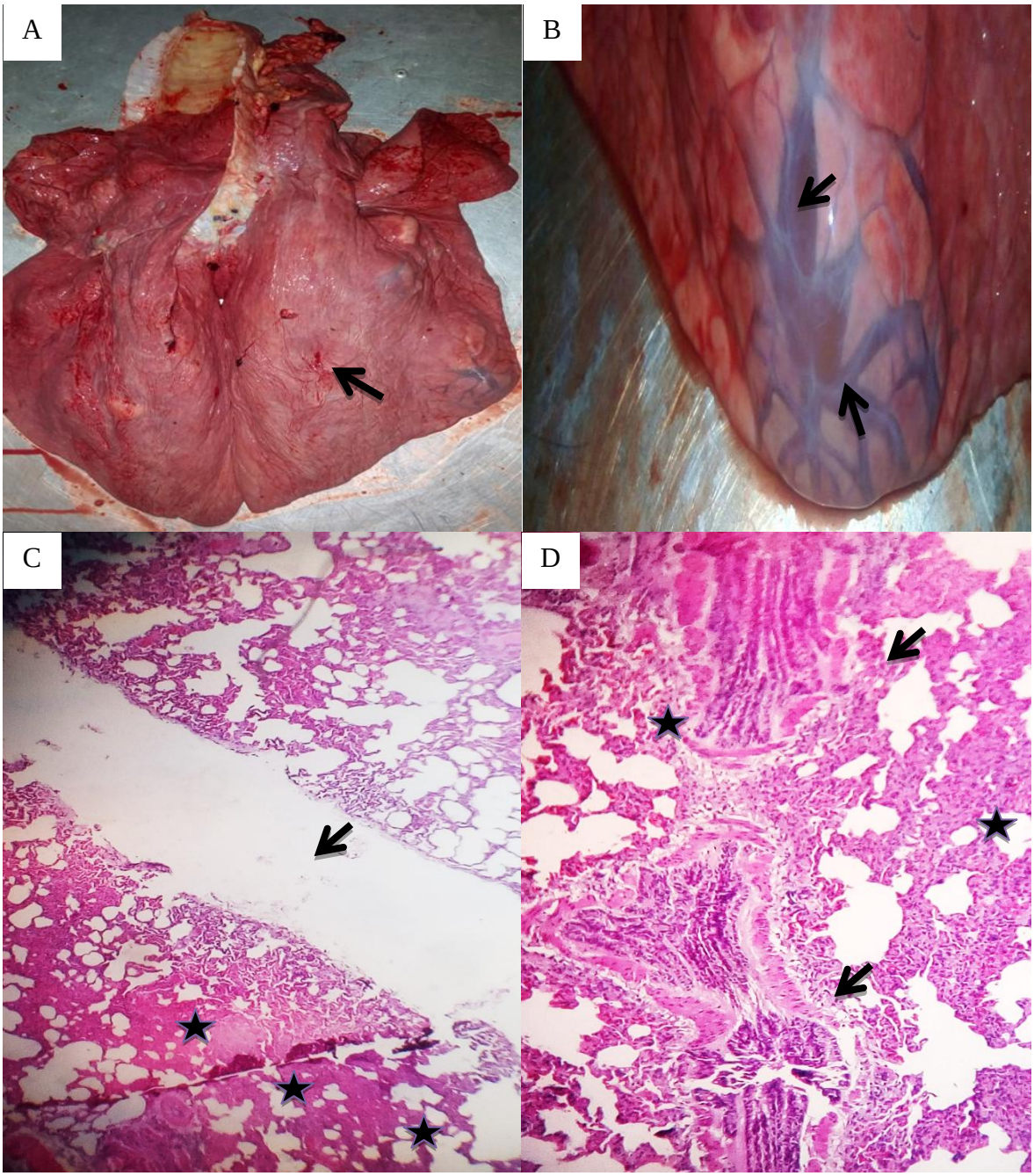


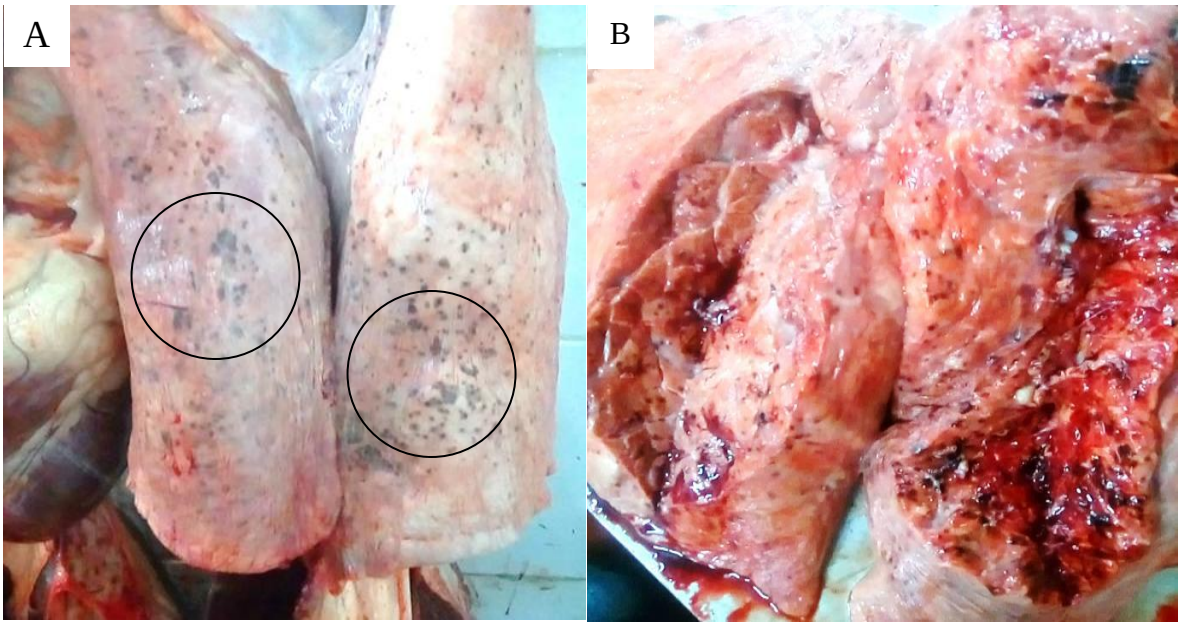

**Figure 8:** Interstitial edema lung cattle, macroscopic and microscopic change

(A) Meaty appearance and shrunken lung (arrow). (B) Prominent interlobular septa with slack tissue (arrow). (C) Distended interlobular space (arrow) with edematous issue (stars). (D) Edematous thickened alveolar wall (stars), thickened fibrosis and collapsed bronchioles (arrow).

*4.1.8. Diffuse alveolar hemorrhage*

The rate of occurrence of different type of hemorrhage was 24.1% (198/820), but histologically confirmed diffuse alveolar hemorrhage 0.12% (1/820). Grossly, there were small pin pointed diffuse dark spots on all lobs of the lung which were visible from pleura surface of the lung (Figure 9 A) and on cut, theses pint pointed dark spots later appeared as a petechial type of hemorrhage (Figure 9 B).

Histologically, some of the alveolar septa were markedly thickened, distended and the alveoli were filled with blood. The others were slightly thickened, rupture and filled with blood and adjacent alveoli were compressed due to the pressure result from accumulation of the blood in the alveoli (Figure 9 C and D). Some of pulmonary arteries filled with the blood (embolism) and infiltrated with inflammatory cells (lymphocytes) (Figure 9 E). Hyperplasia of the bronchioles was seen in some areas (Figure 9 F).



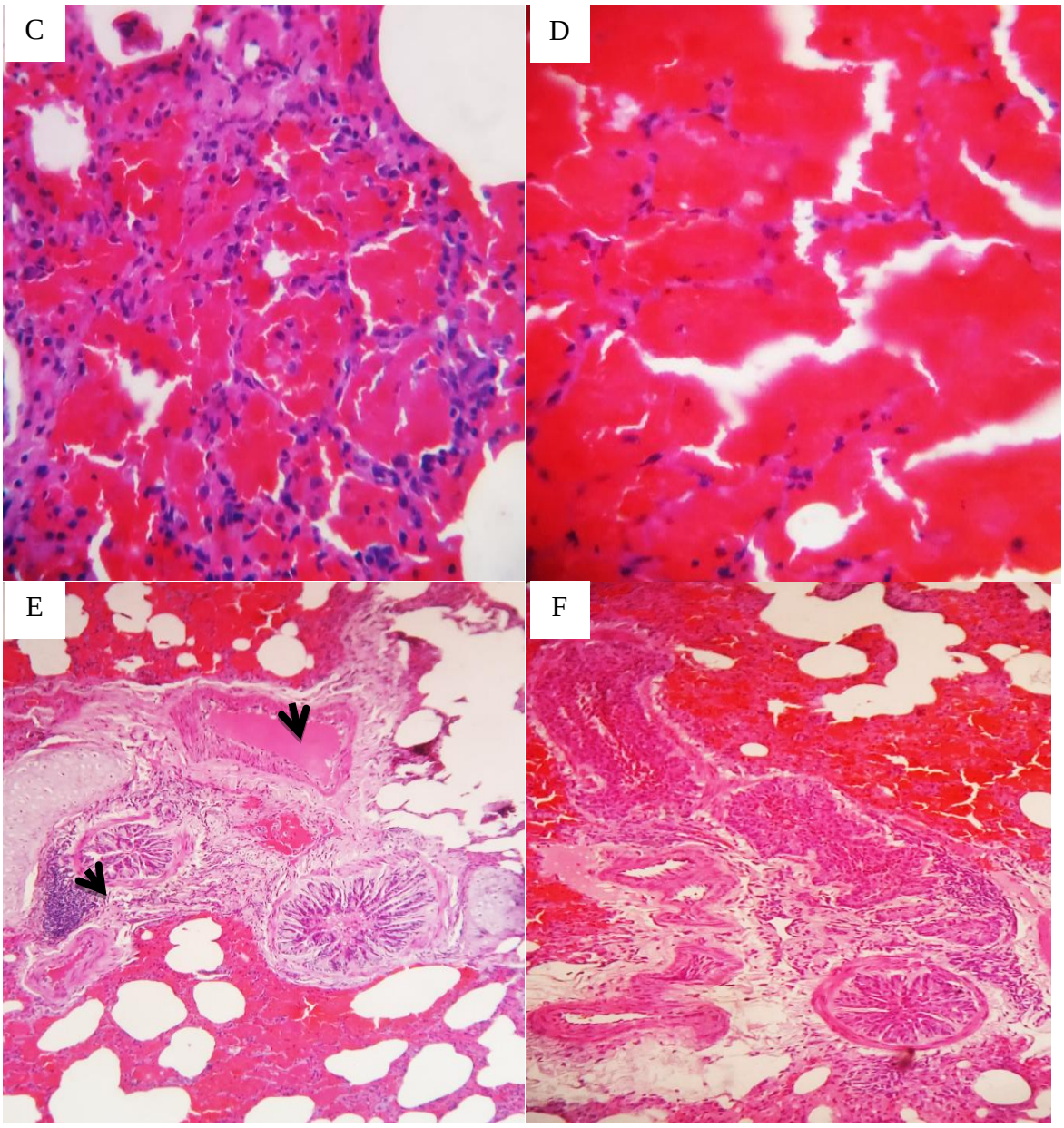



**Figure 9:** Diffuse alveolar hemorrhage cattle, macroscopic and microscopic change

(A) Pin pointed diffuse dark spots (circle). (B) Petechial type of hemorrhage (star). (C) Markedly thickened, distended alveolar septa and blood filled alveoli. (D) Slightly thickened, rupture alveolar septa and blood filled alveoli (E) pulmonary arteries filled with the blood (embolism) (arrow) and infiltrated with lymphocytes (arrow). (F) Hyperplasia of the bronchioles.

### 4.2. Pathological Changes Encountered in Lungs of Camel

Out of 560 camels examined, 57.5% (322/560) had different type of abnormalities in their lungs. Of theses 0.31% (1/322) had tuberculosis with suppurative bronchopneumonia, 0.31% (1/322) pulmonary cryptococcosis, 26.7% (86/322) pulmonary hydatidosis, 4.6% (15/322) aspiration pneumonia, 13.9% (45/322) atelectasis, 30.4% (98/322) emphysema of different type and 23.6% (76/322) hemorrhage. Among this pathological change emphysema was the highest followed by pulmonary hydatidosis, hemorrhage, atelectasis and aspiration pneumonia. The least pathological changes were tuberculosis with suppurative bronchopneumonia and pulmonary cryptococcosis.

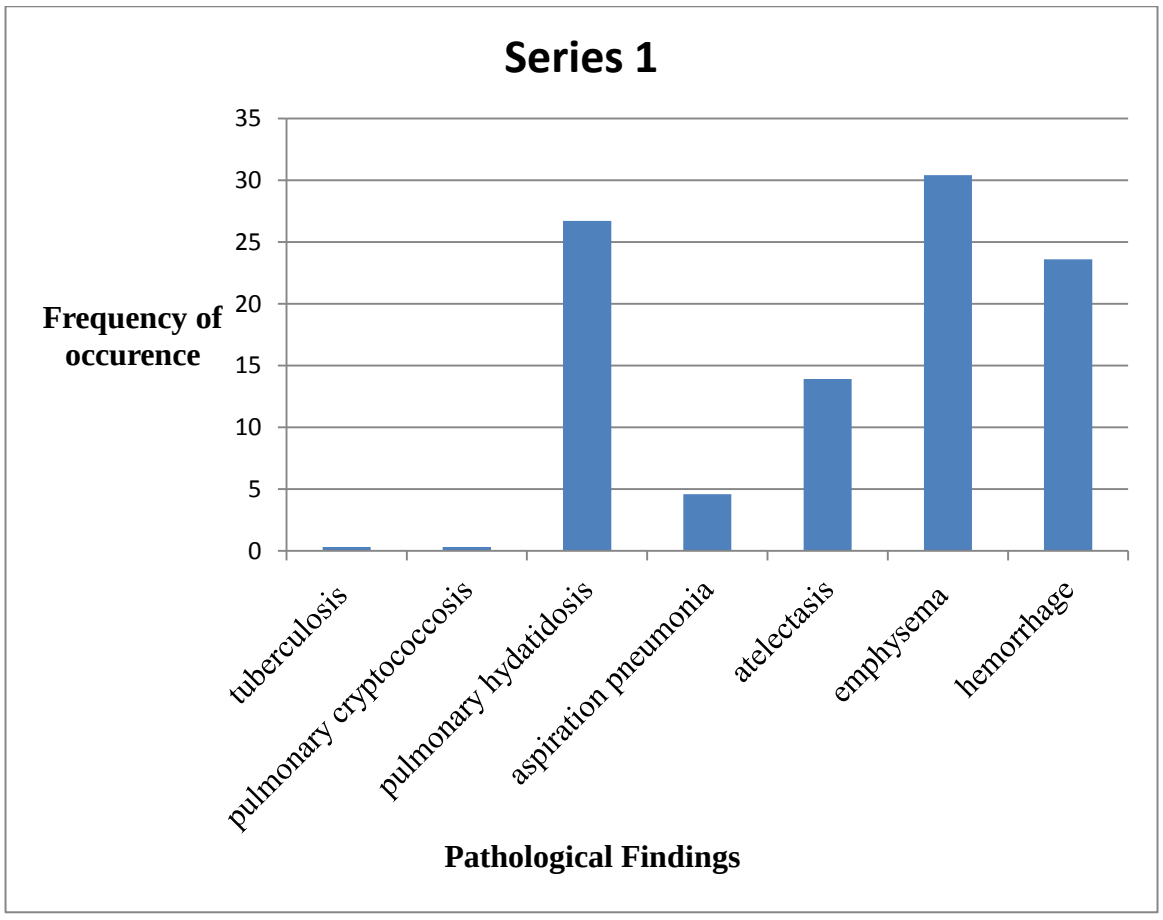
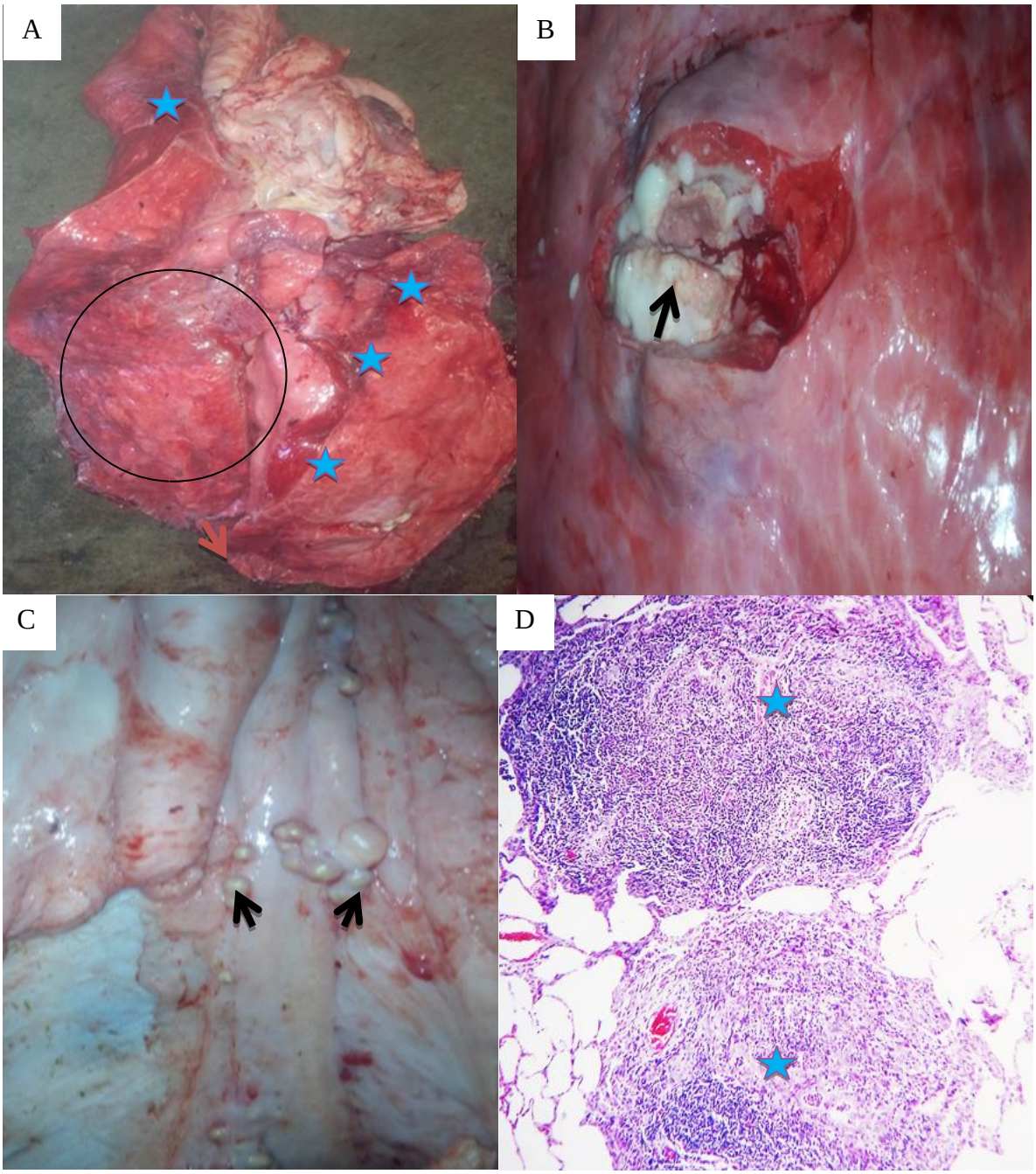



**Figure 10:** Frequency of pathological changes in the lung of camels

*4.2.1. Tuberculosis with suppurative bronchopneumonia*

The rate of occurrence of tuberculosis with suppurative bronchopneumonia was 0.31% (1/322). Grossly, the lung had hepatized appearance, slightly inflated, elevated nodular structure on pleura surface of the lung (Figure 11 A). On the caudal lob of the lung there was abscess which was whitish in color and inside this abscesssation there were calcified materials which were grey in color (Figure 11 B). The thoracic cavity had pea shaped hard moveable nodular structured (Figure 11 C).

Histologically, there were multi focal granulomatous lesions with central necrosis, infiltrated with macrophages, lymphocyte, dead neutrophils and giant cells (Figure 11 D, E and F). The lumens of bronchioles were infiltrated with inflammatory cells mainly neutrophils and the epitheliums were relatively normal (Figure 11 G and H).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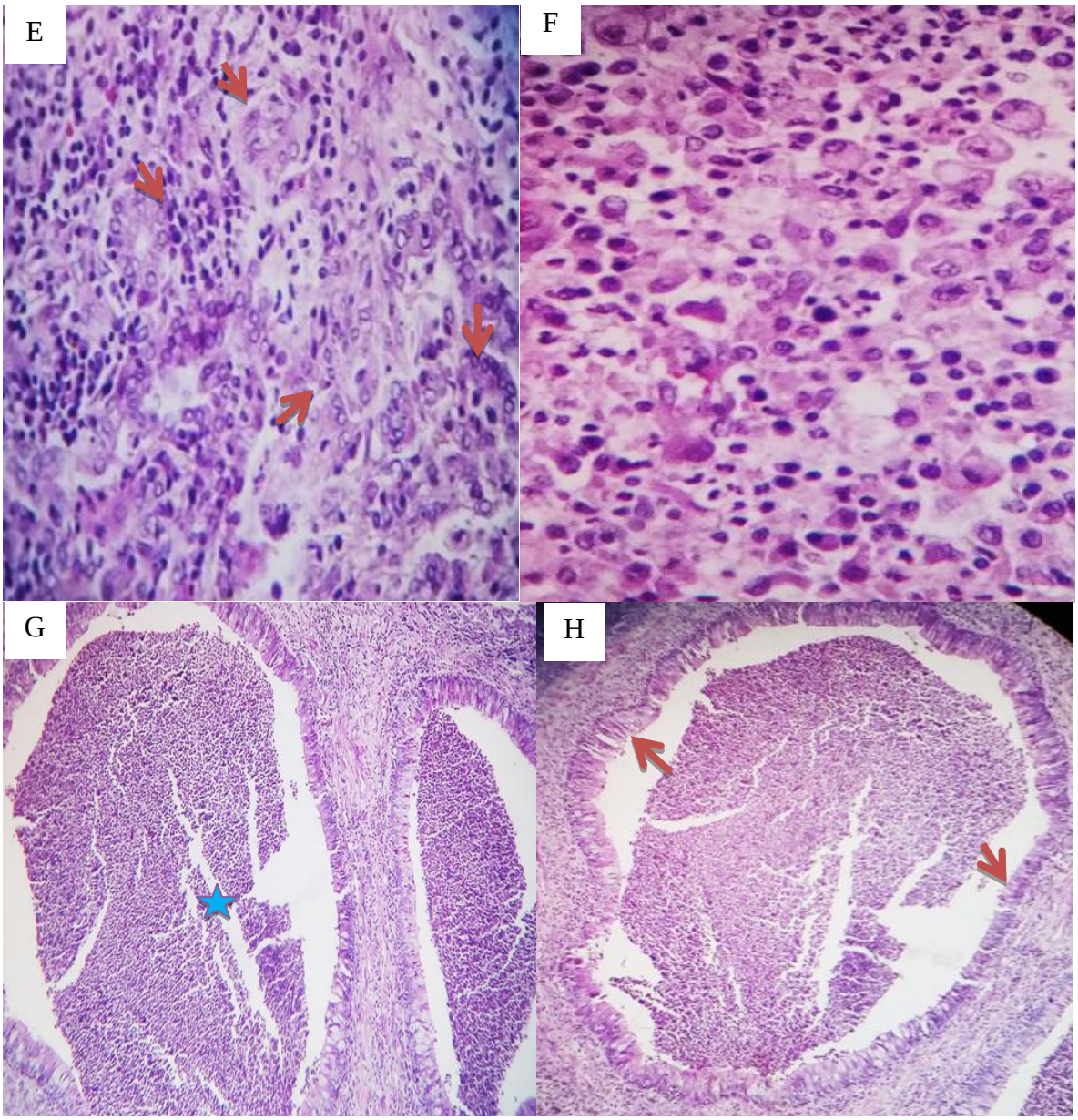


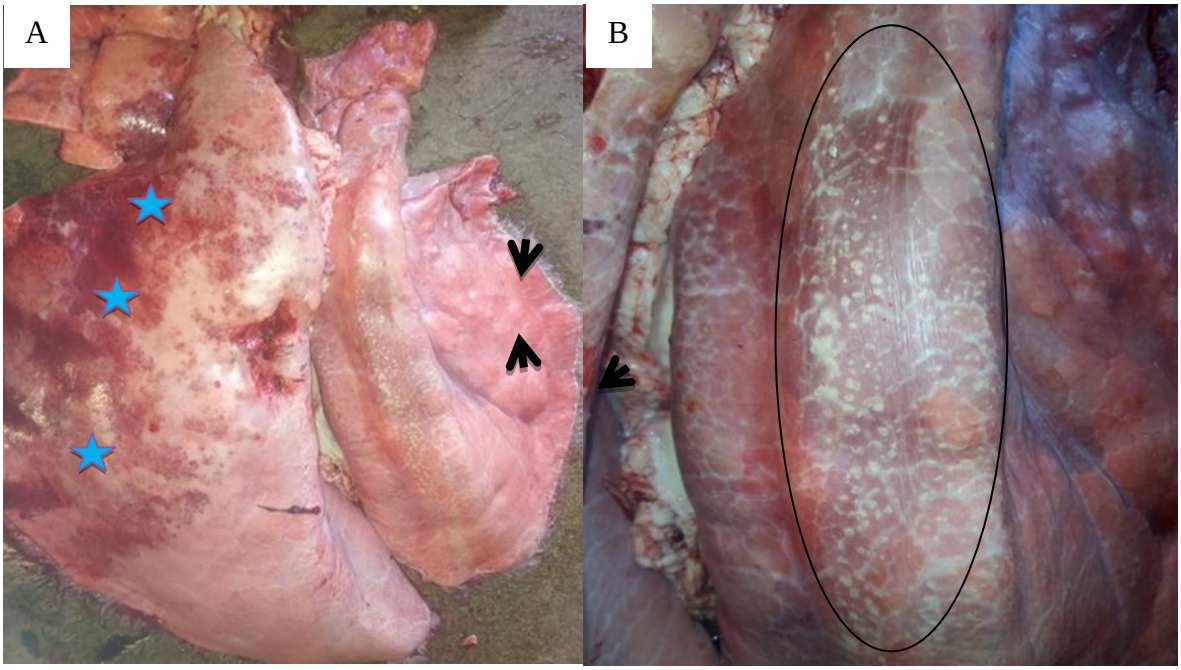

**Figure 11:** Tuberculosis with suppurative bronchopneumonia camel, macroscopic and microscopic changes

(A) Lung showing hepatized appearance (stars), elevated nodular structure (circle). (B) Abscess with grayish classified materials (arrow). (C) Variables size hard moveable nodular in thoracic cavity (arrows). (D) Granulomatous lesions with central necrosis (stars). (E) Giant cells (arrows). (F) Macrophages, lymphocytes and neutrophils. (G) The lumen of bronchioles infiltrated with inflammatory (star). (H) Infiltrated bronchiole with relatively normal epithelium (arrow)

*4.2.2. Pulmonary cryptococcosis*

The rate of occurrence of pulmonary cryptococcosis was 0.31% (1/322). Grossly, the left lob of the lung was smaller than the right lob. The posterior end of the left lob had very flat edge with few non uniformed bullous like emphysema. The right and anterior lobs were hemorrhagic, but it was severe on the right lob (Figure: 12 A). The left lob had pinpointed yellowish to whitish fungal like growth and on cut this fungal like growth slightly extend to the inside of the parenchyma of lung (Figure: 12 B).

Histologically, the alveolar septa were distended, rupture and contained the cryptococci (Figure 12 C) lower magnification. The cryptococci were multifocally distributed in the lung and involved many alveoli. They had soapy bubble appearance, large mucoid or mucinous unstained capsule around bluish round nucleus (Figure 12 D and E) higher magnification.



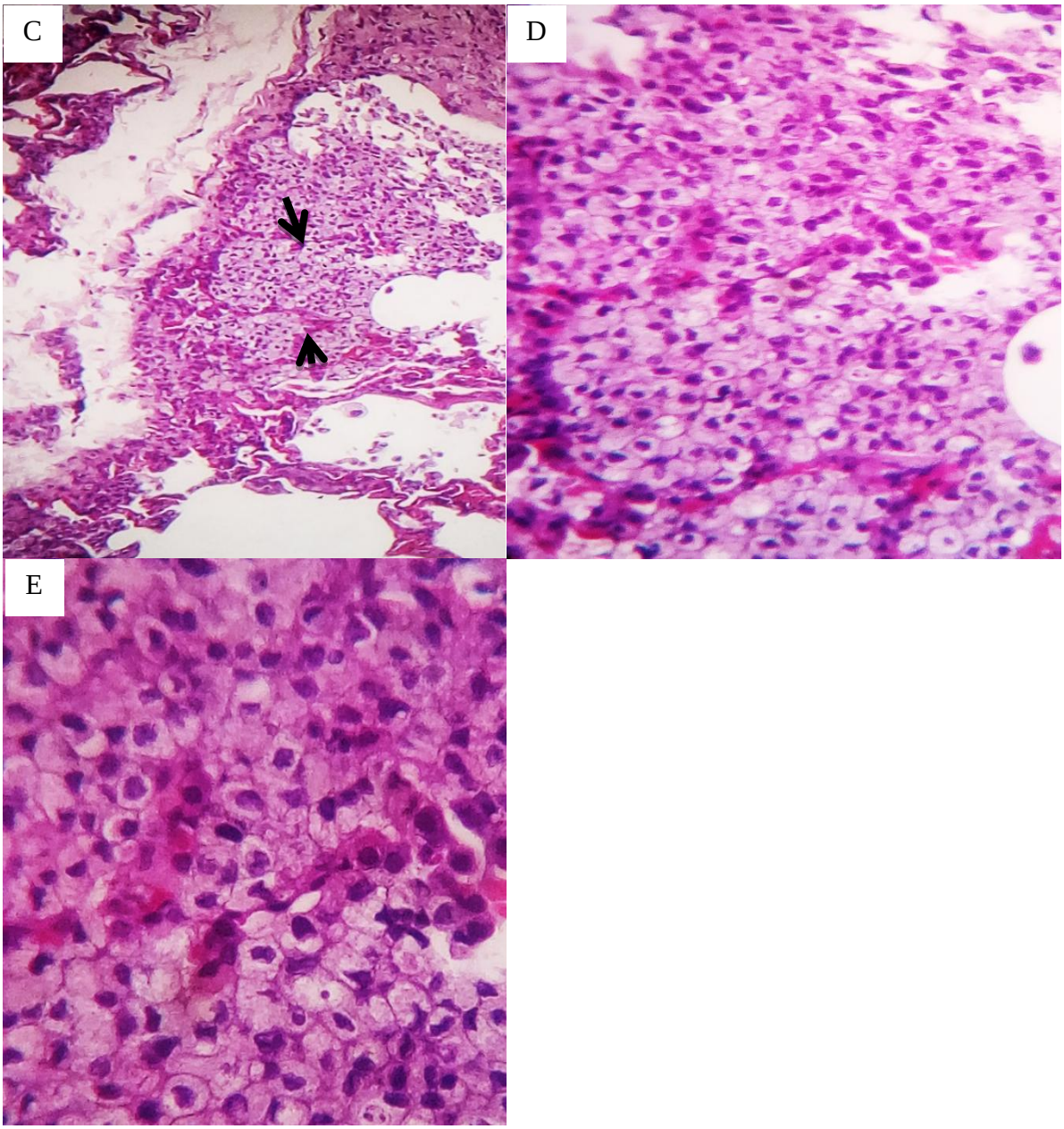


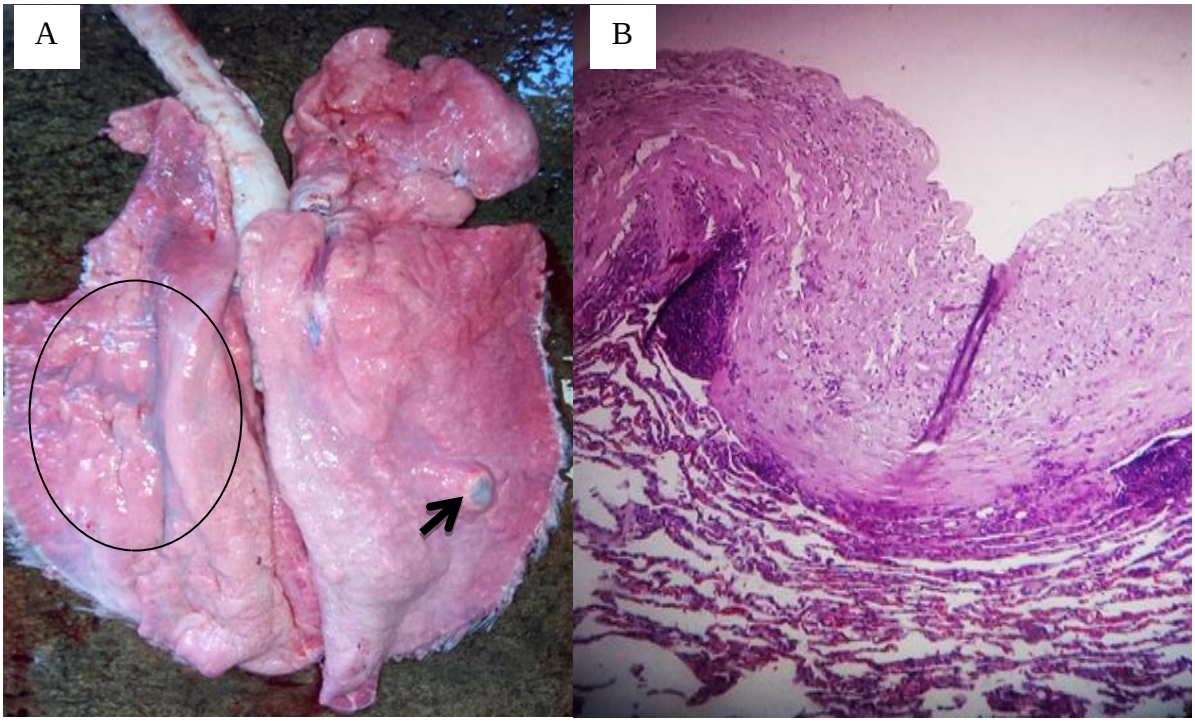

**Figure 12:** Pulmonary cryptococcosis camel, macroscopic and microscopic changes

(A) Flat edge lung with emphysema (arrow), Hyperemic lobs of lung (stars). (B) Pinpointed yellowish to whitish fungal like growth (elliptic). (C) Distended, rupture alveolar septa contained the cryptococci (arrow). (D and E) soapy bubble cryptococci with large mucoid unstained capsule around bluish round nucleus at different magnification

*4.2.3. Pulmonary hydatidosis*

The rate of occurrence of pulmonary hydatidosis was 26.7% (86/322). Grossly, the size of the left and the right lobs of the lung were different. The right lob was smaller than the left. Most parts of the lung were cloudy; the surface of the left lobs was covered with mucoid exudate and had darkened grooved areas. There were focal, cysts on the right lob of the lung. The cyst was protruding out of the lung parenchyma and had eye like structure (Figure: 13 A).

Histologically, the cyst walls were fibrosis and the alveoli were highly compressed, infiltrated with mononuclear cells (Figure 13 B). The three layer of the cyst outermost pericyst, middle ectocyst layer and the inner endocyst were seen (Figure 13 C). The migratory oncosphere was seen in the alveolar duct (Figure 13 D). The capillaries were distended and filled with the blood (Figure 13 E). Infiltration of mononuclear cells (Figure 13 F)



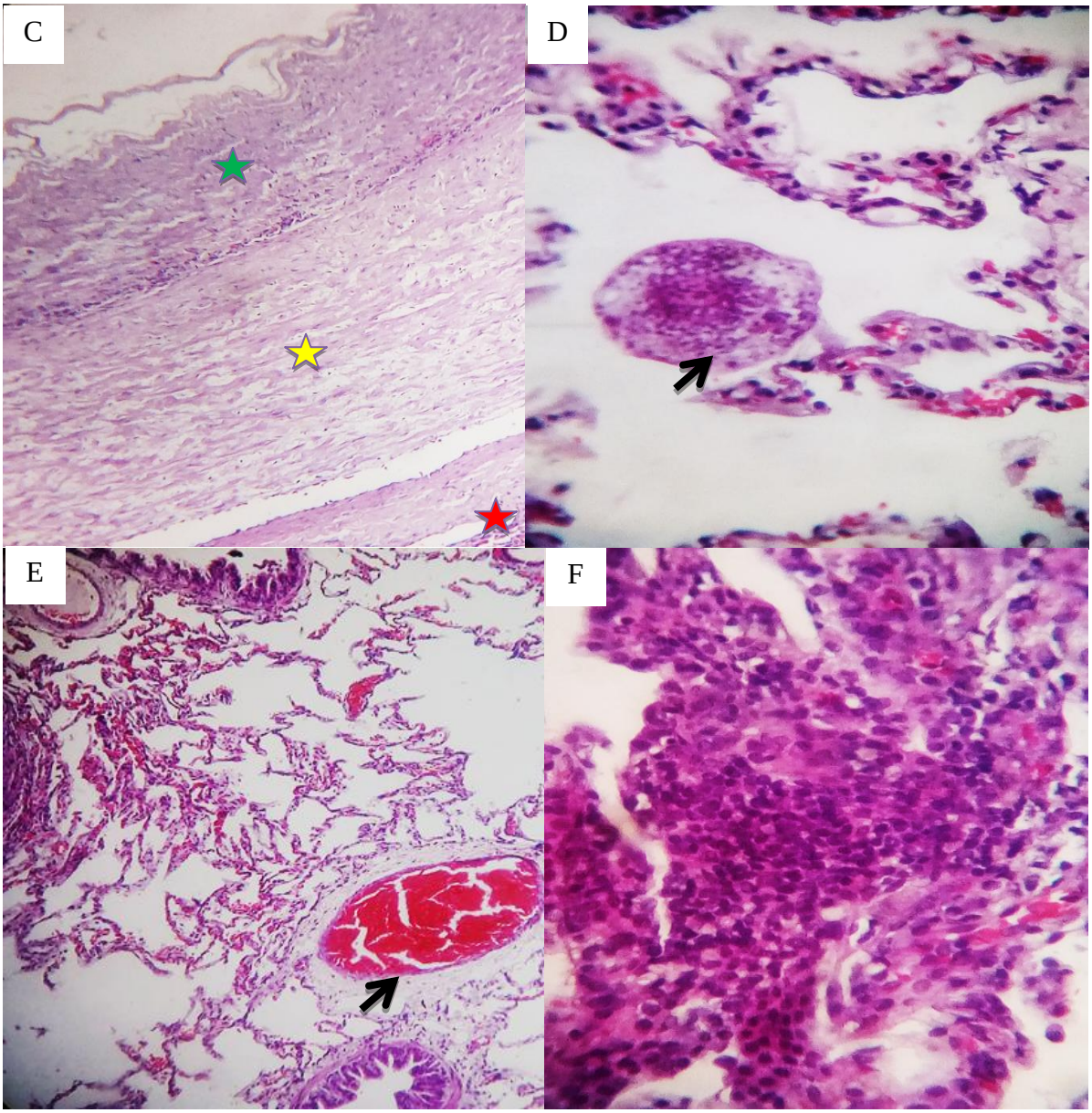


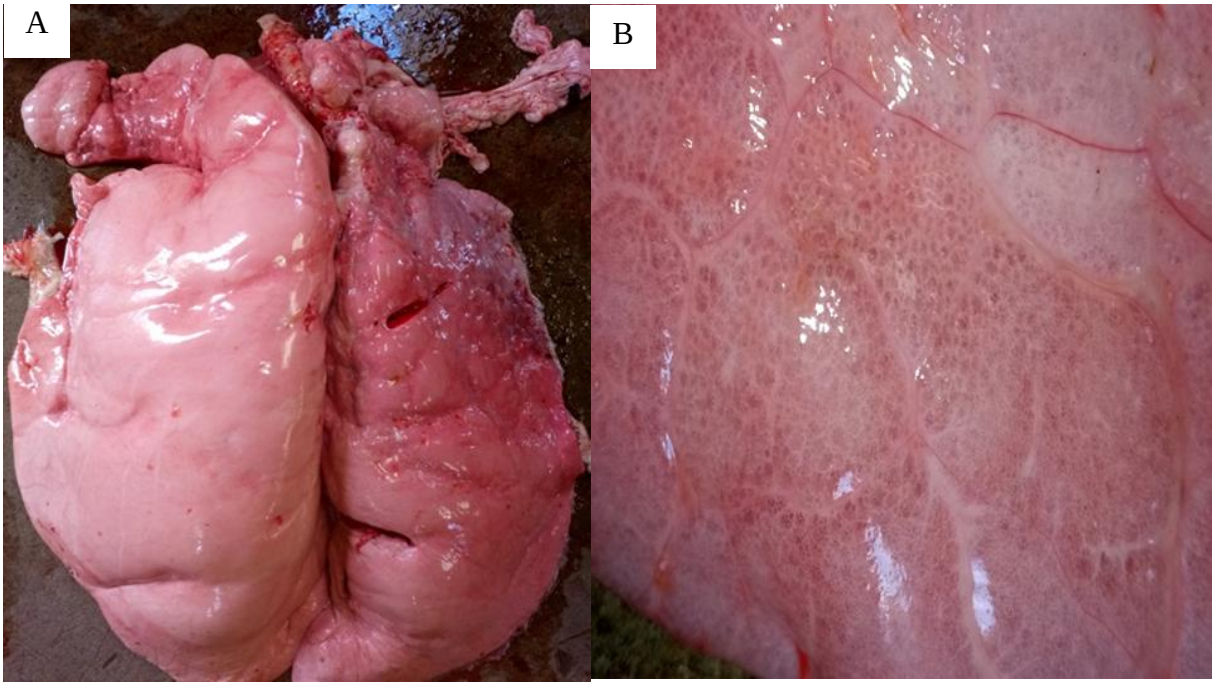

**Figure 13:** Pulmonary hydatidosis camel, macroscopic and microscopic changes

(A) Darken, flattened and grooved areas cover with mucoid like exudate (circle) and protrude cyst (arrow). (B) Fibrosis cyst walls which were infiltrated with mononuclear cells. (C) Cyst's wall with the three layers. (D) Migrated oncosphere (arrow). (E) Distended blood filled capillary (arrow). (F) Mononuclear cells infiltration.

*4.2.4. Aspiration pneumonia*

The rate of occurrence of aspiration pneumonia was 4.6% (15/322). Grossly, the lung was elevated on the right lobe while the left lob was slightly collapsed (Figure: 14 A). The right anterior lob was enlarged due to accumulation of air blebs on the pleural surface (Figure: 12 B). During incision ingesta were found along the bronchi of the right lob (Figure: 14 C).

Histologically, the walls of the bronchi were edematous, infiltrated with inflammatory cells. The forgings particles were seen in the lumen of bronchi (Figure 14 D) and in alveoli (Figure 14 E). The walls of the alveoli were expanded and some of the alveolar septa were rupture and some of them were thicken and fibrosis. Pulmonary capillaries were distained and filled with blood (Figure 14 F).



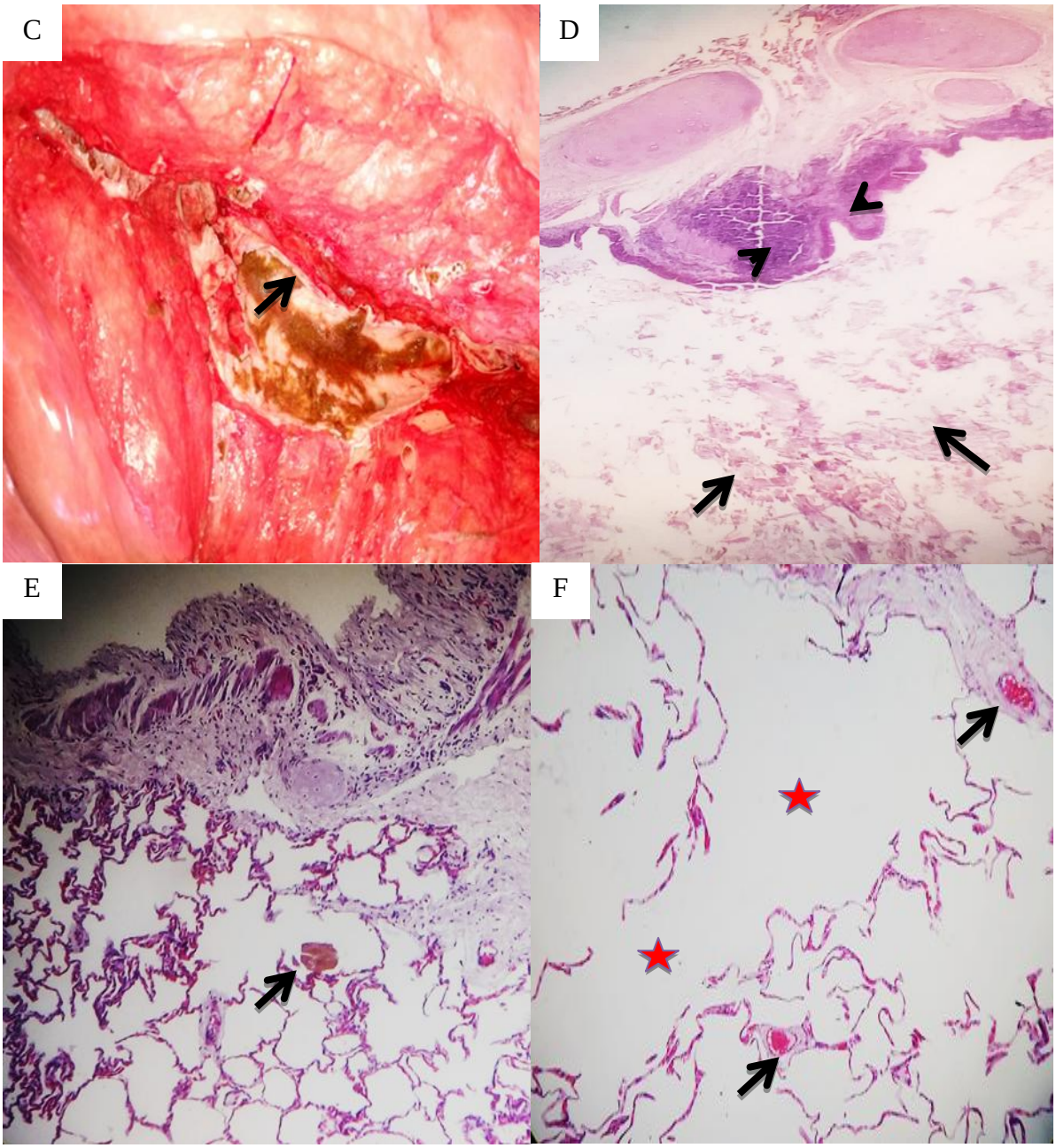


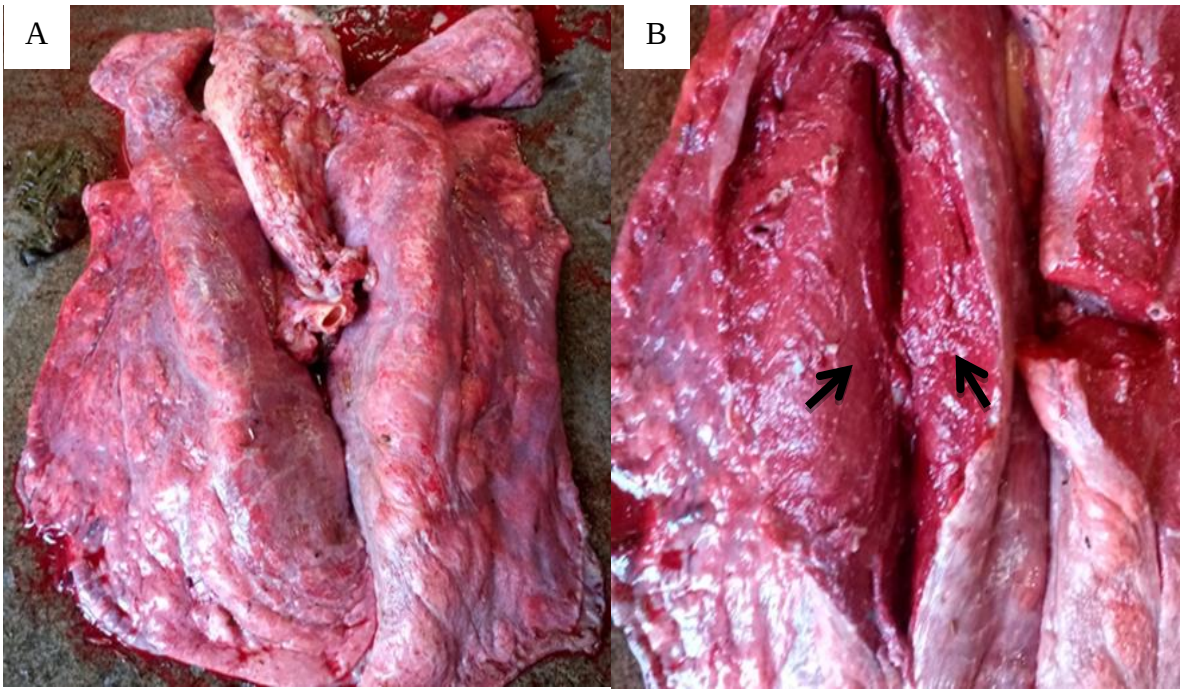

**Figure 14:** Aspiration pneumonia cattle, macroscopic and microscopic changes

(A) Slightly elevated lob of lung. (B) Air blebs on the pleural surface of right anterior lob. (C) Ingesta along the bronchi of the right lob (arrow) (D) Edematous bronchi infiltrated with inflammatory cells (arrowheads) and the lumen of bronchi contained food particles (arrow) and (E) forgings /food particles in alveoli (arrow). (F) Expanded alveoli with rupture septa (stars) and distended alveolar septa and capillary filled with blood (arrows).

*4.2.5. Atelectasis*

The rate of occurrence of atelectasis was 13.9% (45/322). Grossly, the whole parts of lung were dark red in color, smaller in size, depressed under the surface (Figure: 15 A). The lung had diffused soft raised structure on all lobs of the lung and cut section resembles liver (hepatized lung) and had elastic/rubbery nature (Figure: 15 B).

Histologically, the smooth muscle and the connective tissue of bronchi were highly fibrosised and infiltrated with inflammatory cells. . Its lumen were collapsed and filled with debris and inflammatory cells (Figure 15 C). Several bronchioles were collapsed and hypertrophied (Figure 15 D). The pulmonary arteries were hyperplastic, slightly collapsed, fibrosis of tunica intima and its lumen had few blood (Figure 15 E). The alveoli and bronchioles around them were collapsed and alveolar septa were thickened and haemorrhagic (Figure 15 F).



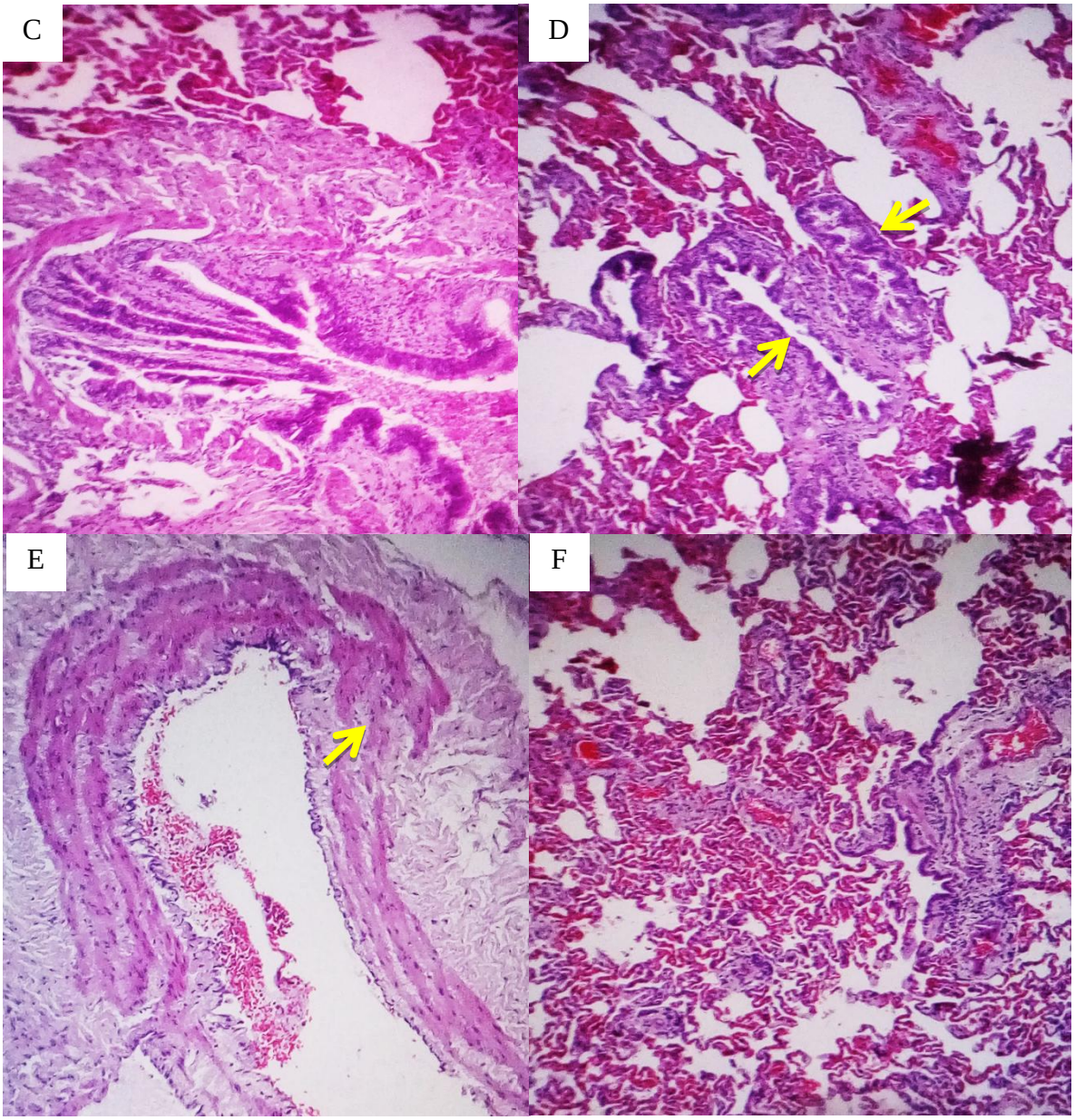


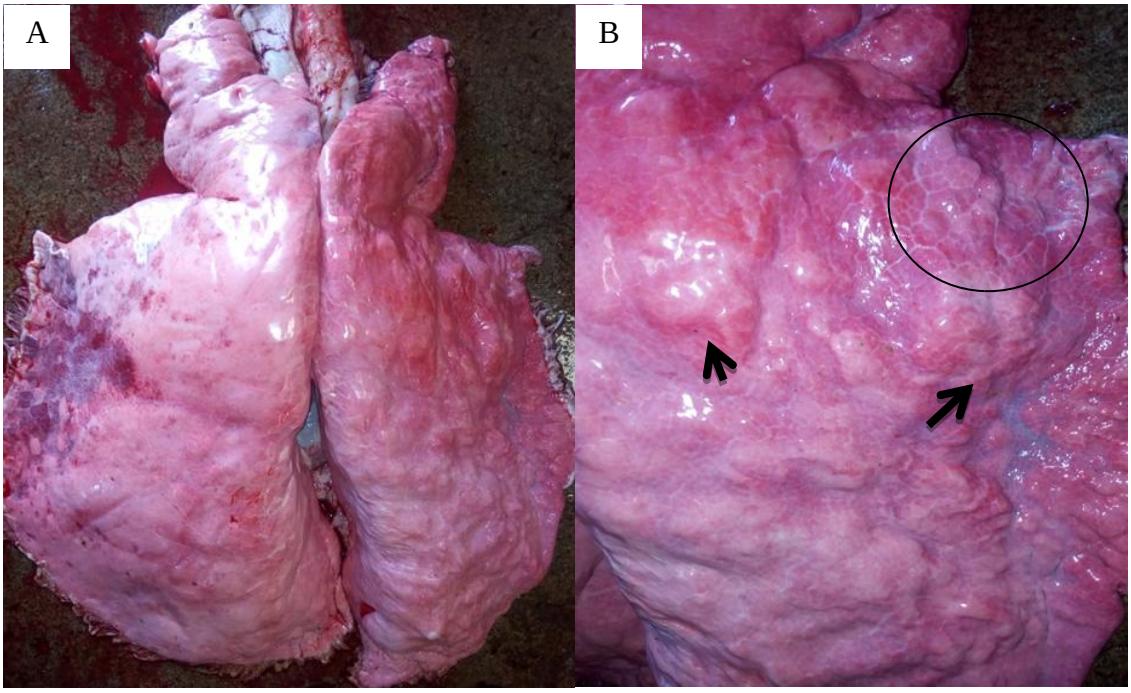

**Figure 15:** Atelectasis cattle, macroscopic and microscopic changes

(A) Dark red, smaller and depressed lung with diffused soft raised structure. (B) Hepatized lung (arrows). (C) Collapsed bronchi. (D) Collapsed bronchioles (E) Hyperplastic and slightly collapsed pulmonary arteries and fibrosis tunica intima (arrow). (F) collapsed alveoli and thickened and haemorrhagic alveolar septa.

*4.2.6. Bullous emphysema*

The rates of occurrence of different type of emphysema were 30.4% (98/322), but histologically confirmed bullous emphysema was 0.31% (1/322). Grossly, all lobs of the lung were enlarged; the right lob had hemorrhage on its edge. The left lob had diffused accumulated airs which were varying in size (Figure: 16 A). This accumulated airs had cone shape and raised above the surface of the lung parenchyma and there was slight thickening of the interlobular septa of the left lob (Figure: 16 B).

Histologically, several bronchioles were ruptured (Figure 16 C); some of the several alveoli were distended and arranged in the lobular form without having the alveolar duct (Figure 16 D). The adjacent alveoli and bronchioles around the emphysematous alveoli were compressed due to the pressure from distended alveoli (Figure 16 E and F).



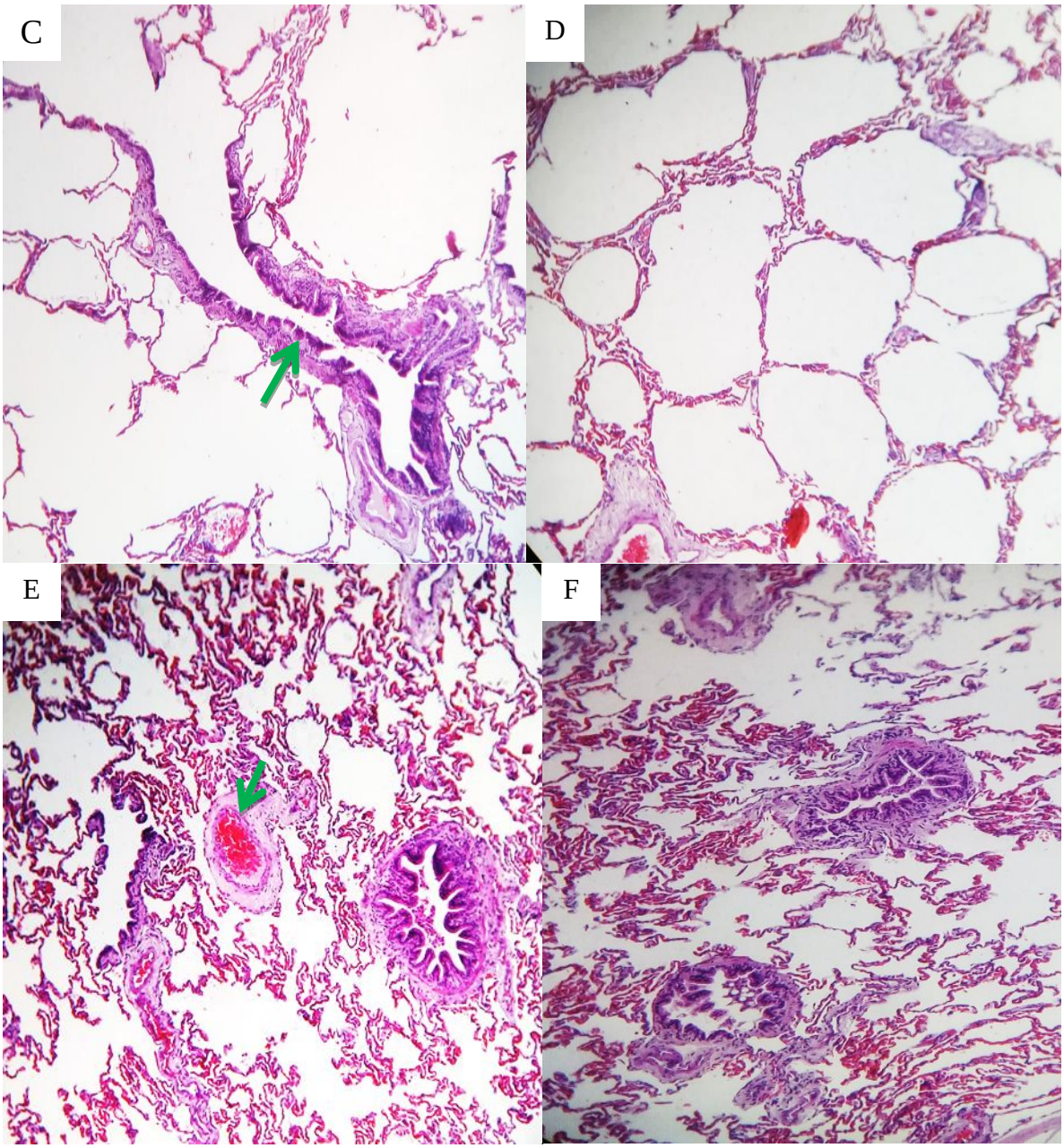


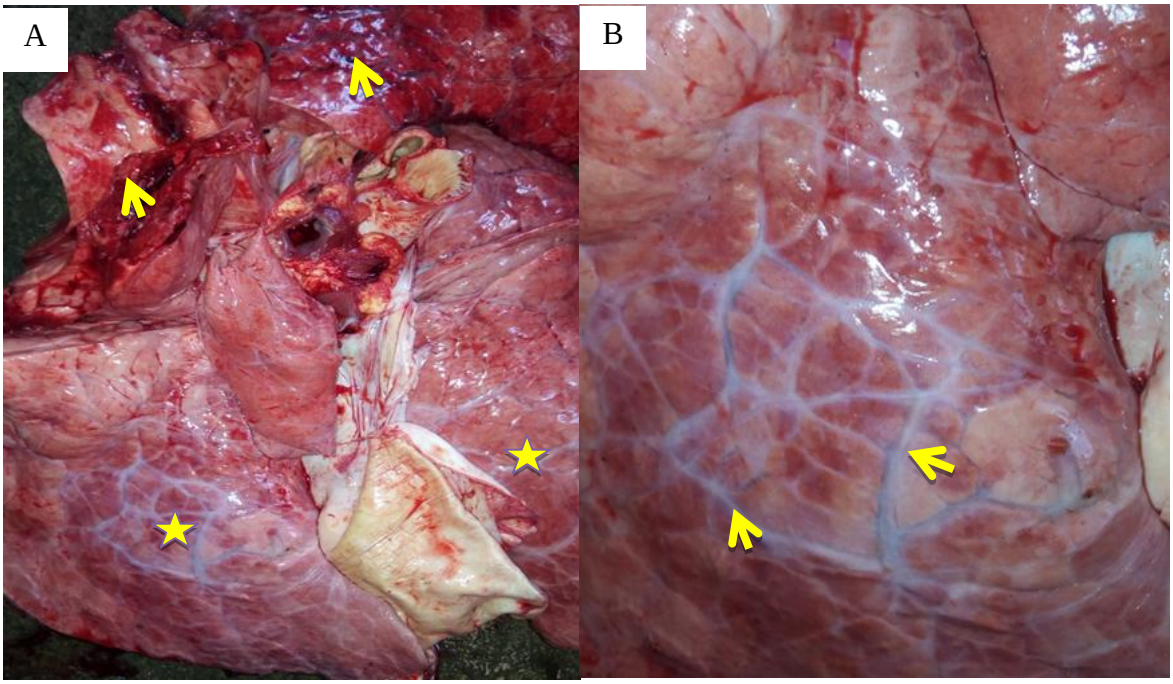

**Figure 16:** Bullous emphysema camel**,** macroscopic and macroscopic changes

(A) Enlarged and hemorrhagic lung (arrow). (B) Cone shaped raised air filled structure protruding above the surface of the lung parenchyma (arrow) and slight thickening of the interlobular septa (circle). (C) and (E) rupture bronchioles (arrows). (D) Distended and lobularly arranged alveoli. (E and F) compressed alveoli and bronchioles

*4.2.7. Interlobular emphysema*

The rate of occurrence of different type of emphysema was 30.4% (98/322), but histologically confirmed interlobular emphysema was 0.31% (1/322). Grossly, the dorso cranial areas of lung were cloudy on both left and right lobs of the lung. The anterior and some part of the right lobs were hemorrhagic (Figure 17 A). The pleura were slightly thickened. The lobules of the lung were separated by mucoid like exudates (Figure 17 B). At the cut surface, the interlobular spaces were distended by gelatinous or mucoid like materials (Figure 17 C).

Histologically, the interlobular spaces of the lung were distended and filled with exudates (Figure 17 D). There was inflammation and fibrosis of interstitial tissue (Figure 17 E).



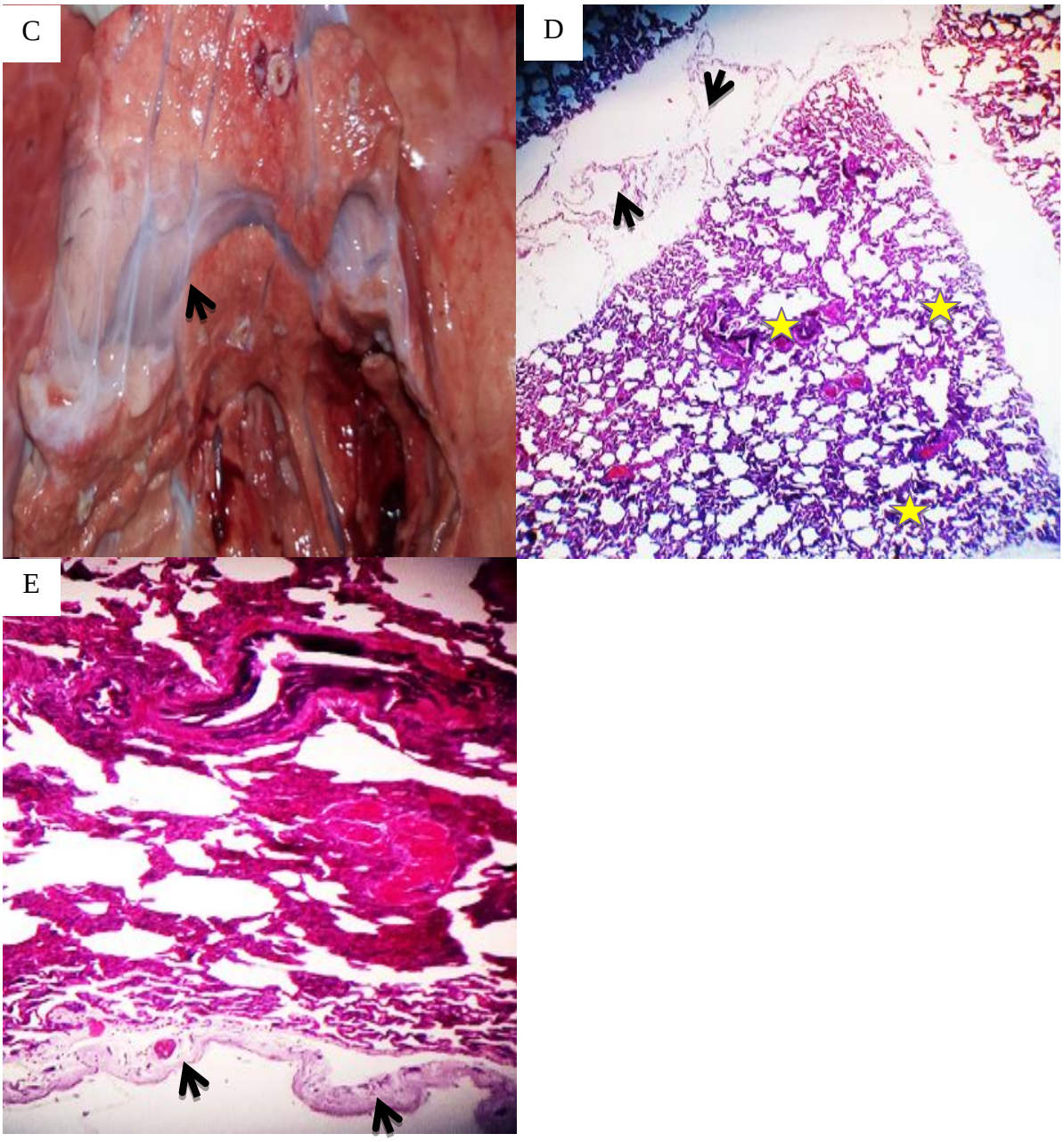



**Figure 17:** Interlobular emphysema camel, macroscopic and microscopic changes

(A) Cloudy appearance of right lobs (stars) and hemorrhagic lobs of lung (arrow). (B) Thickened pleura and the lobule separated by mucoid like exudates (arrow). (C) Distended interlobular spaces by gelatinous materials (arrow). (D) Distended interlobular spaces of the lung (stars) filled with exudates and compressed alveoli (arrows). (E) Fibrosis interlobular septa (arrows).

*4.2.8. Intra-alveolar hemorrhage*

The rate of occurrence of different type of hemorrhage was 23.6% (76/322), but histologically confirmed intra-alveolar hemorrhage was 0.31% (1/322). Grossly, the right lob of the lung was collapsed, pale in color and it had large and small sized cysts on it. The cysts capsules were hard (Figure: 18 A). The left lung was voluminous and had emphysema with petechial type of hemorrhage which was more predominated on the apical lobe of the lung (Figure 18 B).

Histologically, the alveolar septa were highly distended than normal size (Figure 18 C). Some of the Intra-alveolar space were distended and ruptured and filled with blood (Figure 18 D).

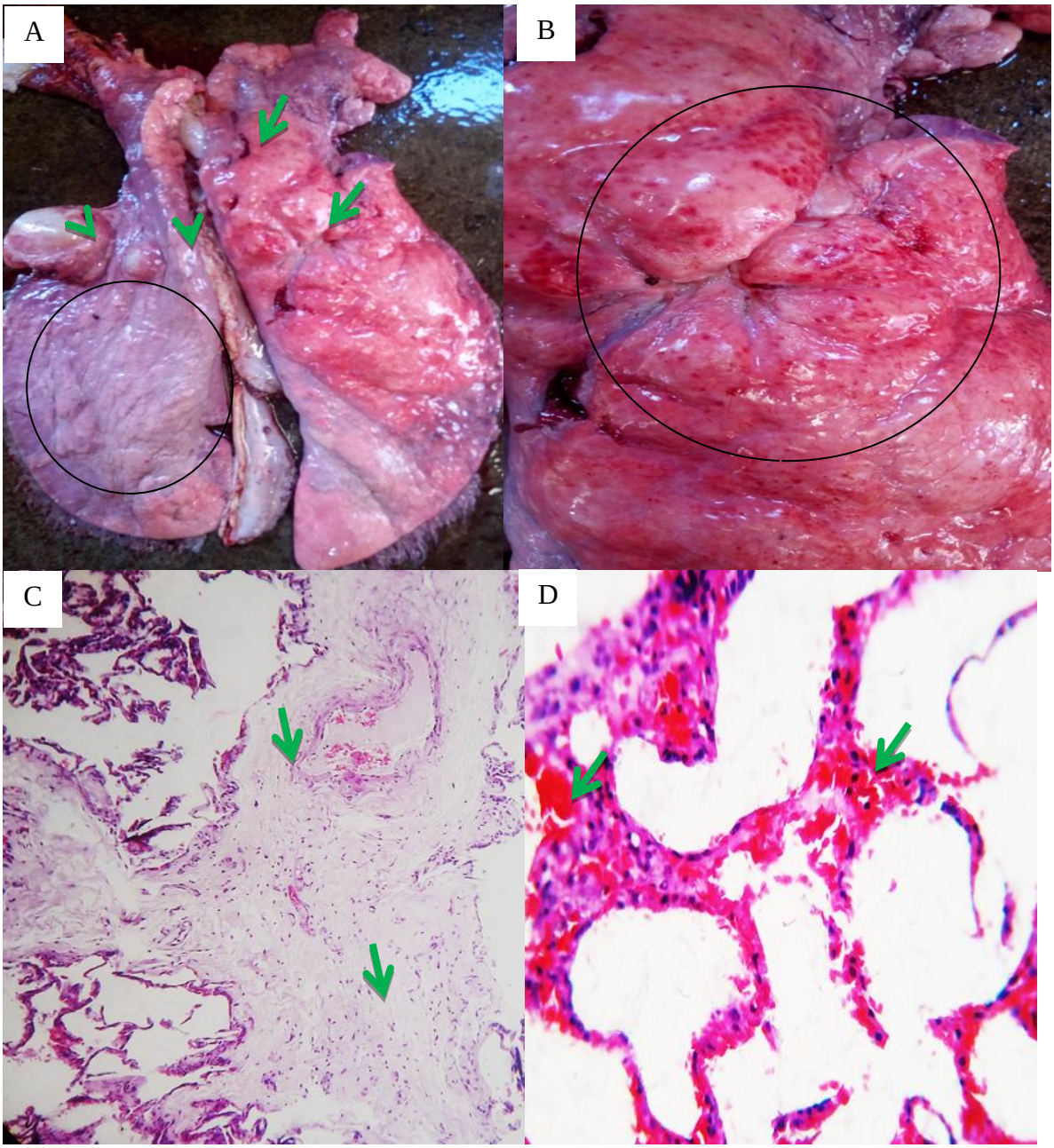



**Figure 18:** Intra-alveolar hemorrhage cattle, macroscopic and microscopic changes

(A) Elevated left lob of lung (arrow), collapsed and pale right lob of lung (circle) and different sized cysts on it (arrowhead). (B) Petechial type of hemorrhage on the apical lobe of the lung (circle). (C) Widened alveolar septa (arrows). (D) Thickened and distended intra-alveolar space filled with blood (arrows).

### 4. 3. Pathological Changes Encountered in Heart of Cattle

Out of 3520 cattle examined 0.39% (14/3520) cattle were found with different abnormalities of harts. 28.5% (4/14) had serous cysts, 28.5% (4/14) calcification, 21.4% (3/14) sarcocystis and 21.4% (3/14) pericarditis with myocardial infraction. Serous cysts and calcification were the highest in occurrence followed by pericarditis and sarcocystis.

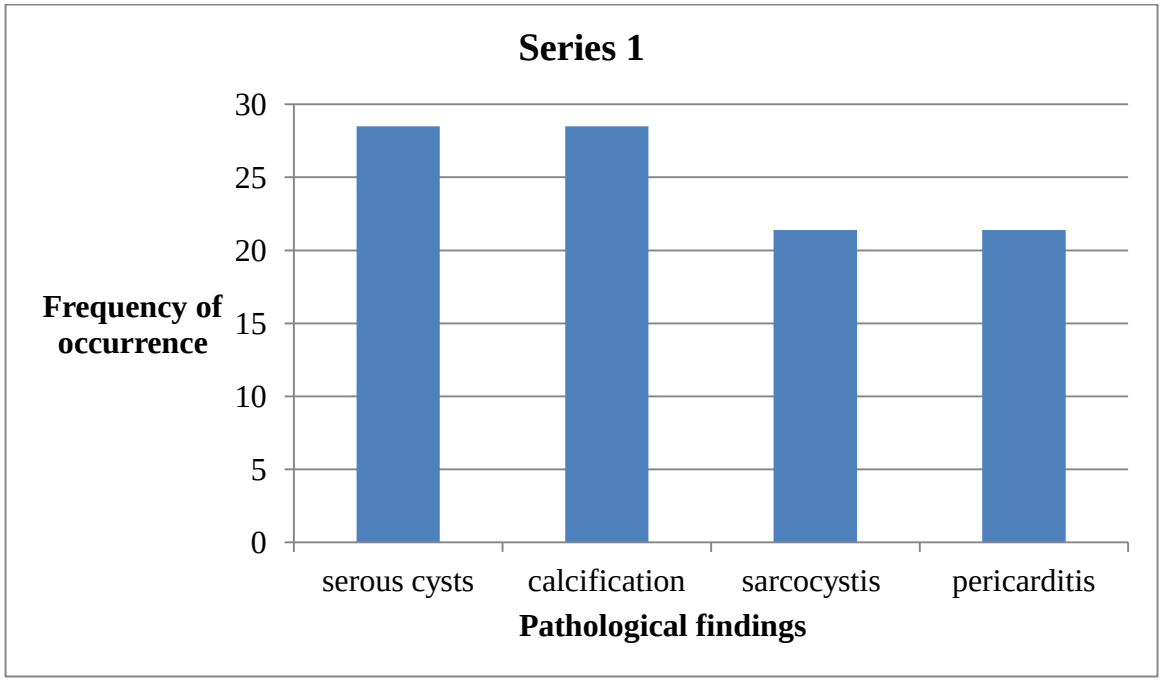



**Figure 19:** Frequency of pathological changes in heart of cattle

*4.3.1. Serous cyst*

The rate of occurrence of serous cysts was 28.5% (4/14). Grossly there was immoveable cyst about 1 cm in diameter on mitral value of the heart. The wall of the cyst was thin and contained yellowish serous fluids (Figure 20 A).

Histologically, the cysts wall was edematous and surrounded by plasma cells, lymphocytes and macrophages (Figure 20 B and C). There were pale areas on myocardial region which indicates necrosis (Figure 20 D).

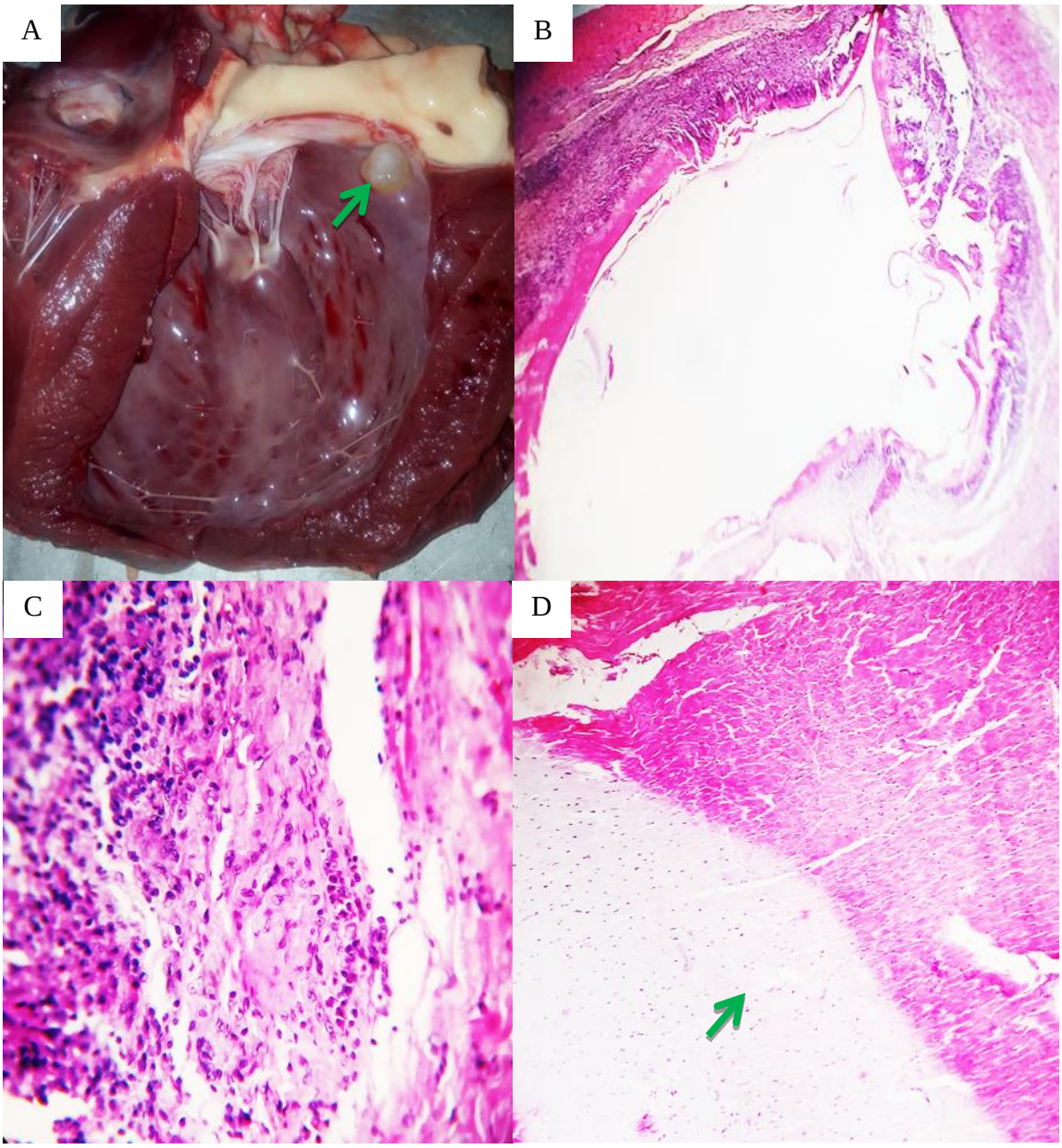



**Figure 20:** Serous cyst heart cattle, macroscopic and microscopic changes

(A) The cyst filled fluid (arrow). (B) Edematous cysts wall surrounded by inflammatory cells. (C) Area surrounded the cyst infiltrated with plasma cells, lymphocytes and macrophages X 40. (D) Pale areas on myocardial region (arrow)

### *4.3.2. Calcification*

The rate of occurrence of calcification was 28.5% (4/14). Grossly, there was yellowish raised calcified material on the left ventricle near to apex (Figure 21 A).

Histologically, the calcified materials were seen as purple and deposited on the endocardia area (Figure 21 B). The endocardium surrounding the calcified materials were fibrosis, necrotized and infiltrated with inflammatory cells mostly lymphocytes, but also there were few macrophages (Figure 21 C). The adjacent myocardium were necrotized and infiltrated with lymphocytes (Figure 21 D).

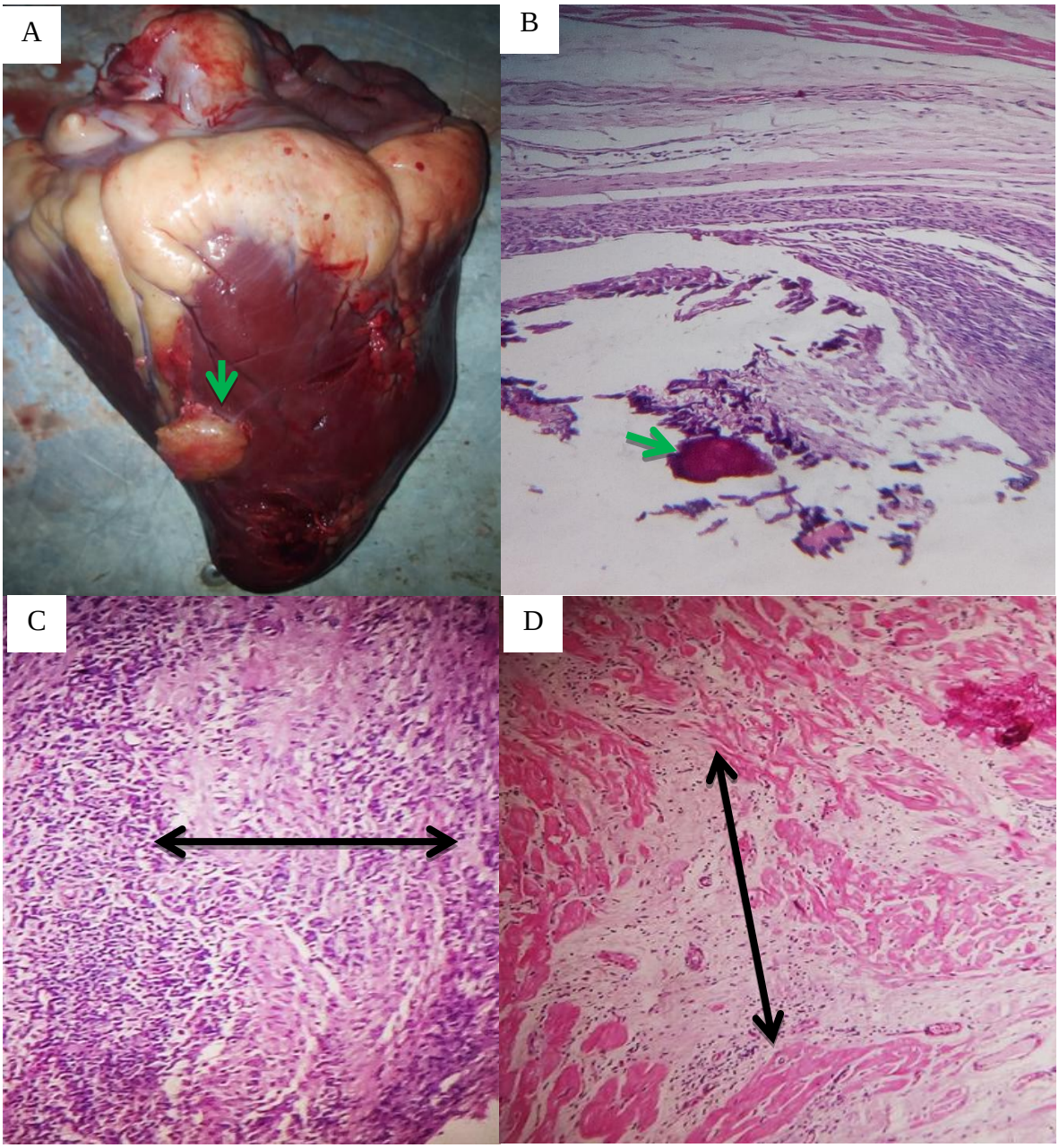



**Figure 21:** Calcification of heart cattle, macroscopic and microscopic changes

(A) Yellowish calcified material on the left ventricle (arrow). (B) Calcified materials deposited on the endocardium (arrow). (C) Necrotized endocardium and infiltrated with inflammatory cells. (D) Myocardium, necrotized and infiltrated with lymphocytes

*4.3.3. Sarcocystis*

The rate of occurrence of sarcocystis was 21.4% (3/14). Grossly the heart had focal glistening small sized yellowish semi soft consistency rounded structure on the surface of right ventricle of the heart (Figure 22 A).

Histologically, the longitudinal section of heart tissue showed oval shaped sarcocyst which appeared as basophilic bodies surrounded by thin outer wall. The encysted parasite in the myocardium showed internal bradyzoite separated by internal septations (Figure 22 B).

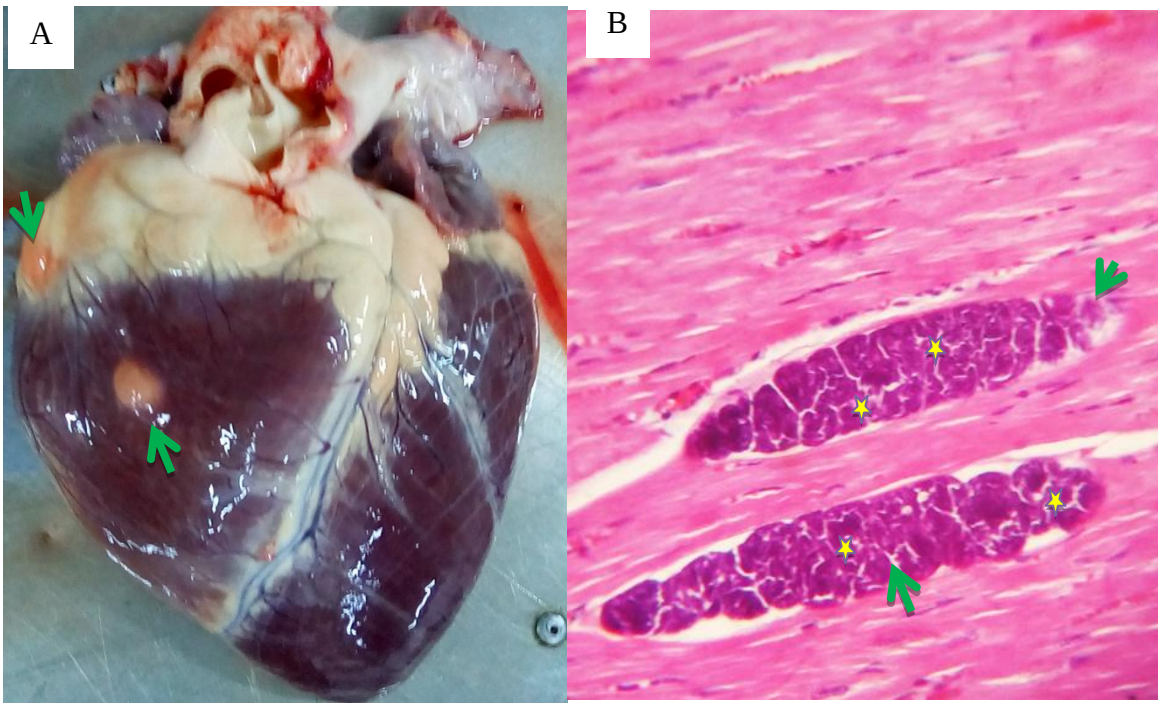



**Figure 22**: Sarcocystis in heart cattle, macroscopic and microscopic changes

(A) The cysts on right ventricle (arrows). (B) Oval shaped sarcocyst with basophilic bodies (arrows) contained bradyzoite separated by internal septations (stars).

*4.3.4. Fibrinous pericarditis with Myocardial infarction*

The rate of occurrence of pericarditis with myocardial infraction was 21.4% (3/14). Grossly, the pericardium of the heart was tightly attached to the heart. The pericardium had yellowish highly fatty nature (Figure 23 A).

Histologically, edematous pericardium with fibrinous exudate and infiltrated with lymphocytes (Figure 23 B). The myocardium of the heart was necrotized and infiltrated with few inflammatory cells (Figure 23 C).

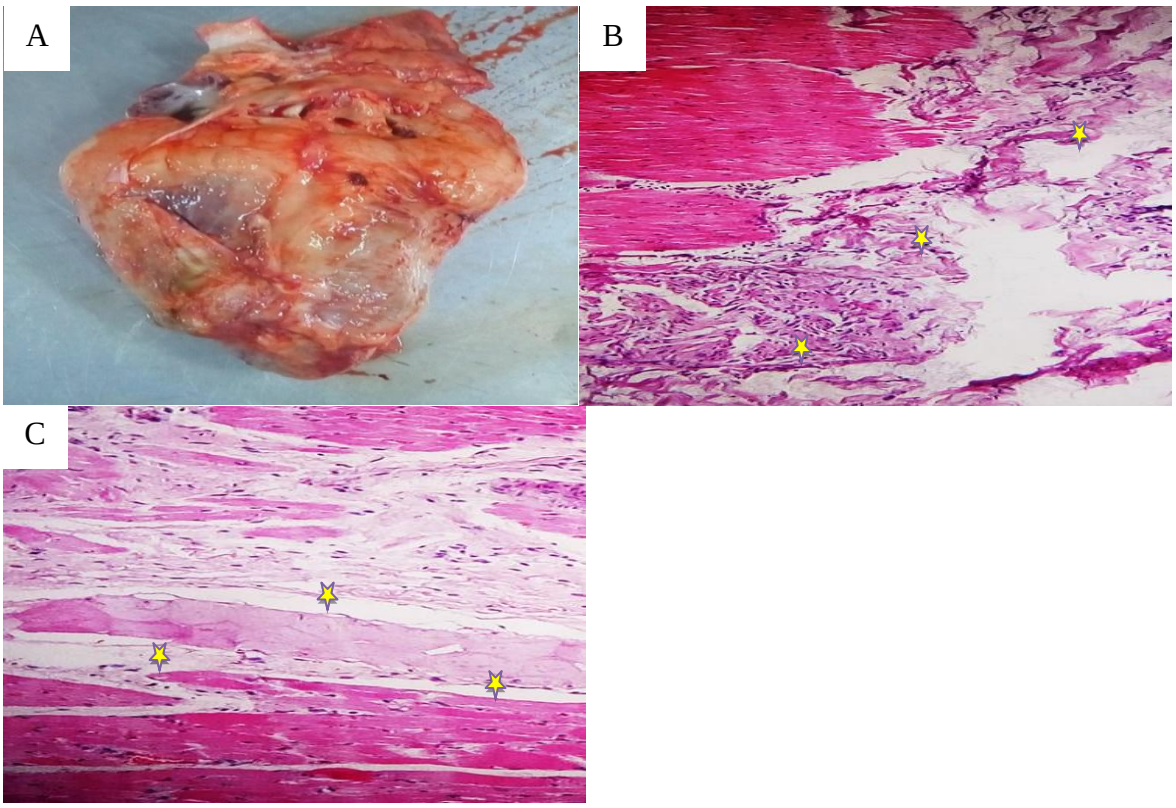



**Figure 23:** Fibrinous pericarditis with Myocardial infarction cattle, macroscopic and microscopic changes

(A) Pericardium attached to heart. (B) Fibrinous strand (stars) (C) Necrotized myocardium of the heart that was infiltrated with few inflammatory cells (stars).

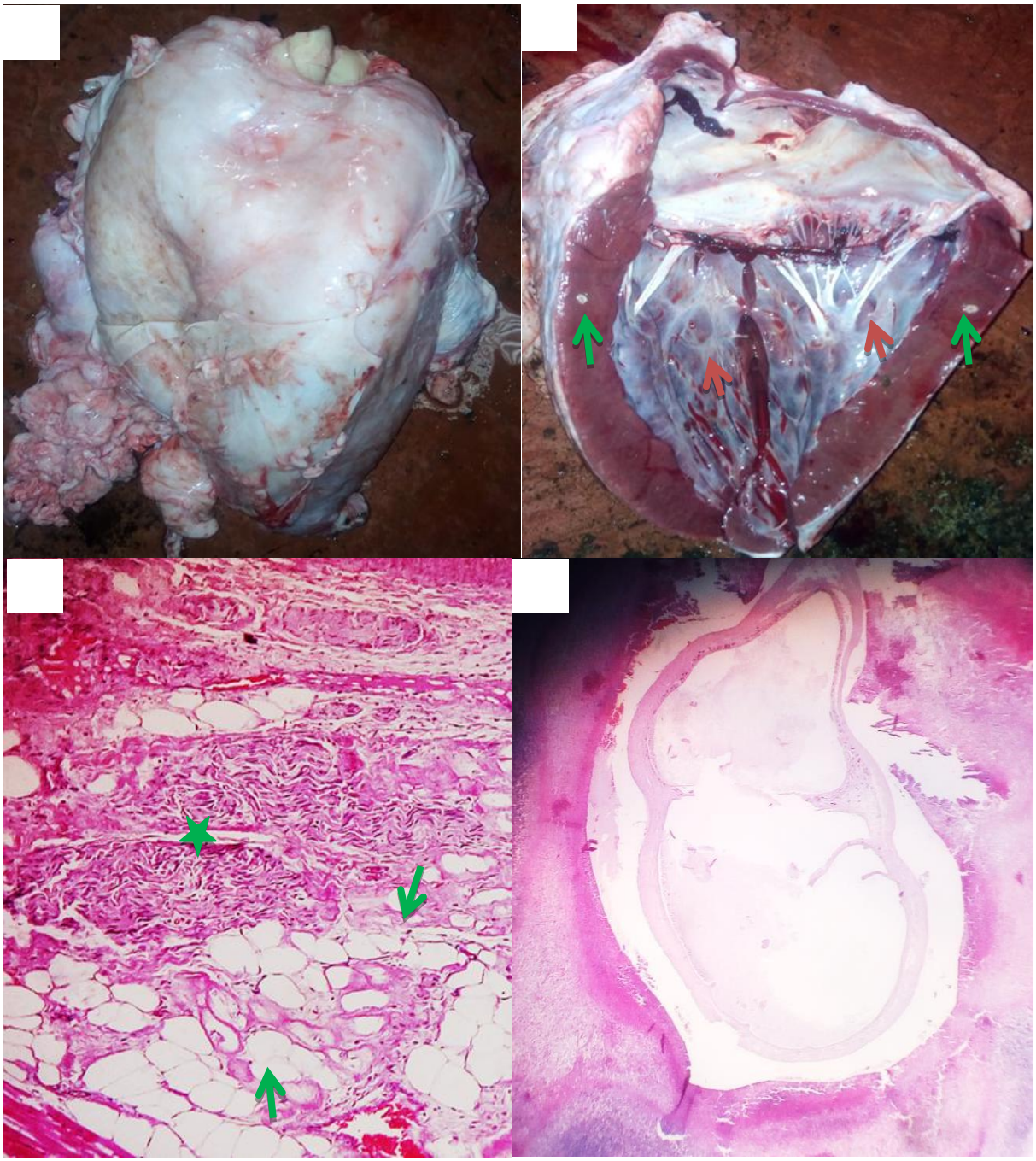
**4.4. Pathological Changes Encountered in Heart of Camel**

Out of 560 examined camels only 0.17% (1/560) was found to be affected by cysticercosis with pericarditis. The rest were grossly normal

*4.4.1. Cysticercosis with pericarditis*

Grossly, the pericardium of the heart was thick and adheres to the heart (Figure 24: A). On cut, there was thickened small size cyst like structure on myocardium. The whole ventricle was diffusely and uniformly whitish and opaque (Figure 24 B).

Histologically, there were fat deposits infiltrate between the bundles of heart wall. The muscle sounding the fat deposits were degenerate (Figure 24 C). The cysticercus cyst was seen in the myocardium of heart and surrounded by fibrosis and edematous tissue. There were large numbers of eggs in female nematode reproductive tract (Figure D and E). The myocardium surrounded the cyst were infiltrated with eosinophils, lymphocytes and macrophages (Figure 24 F and G) and the pericardium was totally lost and infiltrated with few inflammatory cell (Figure 24 H).



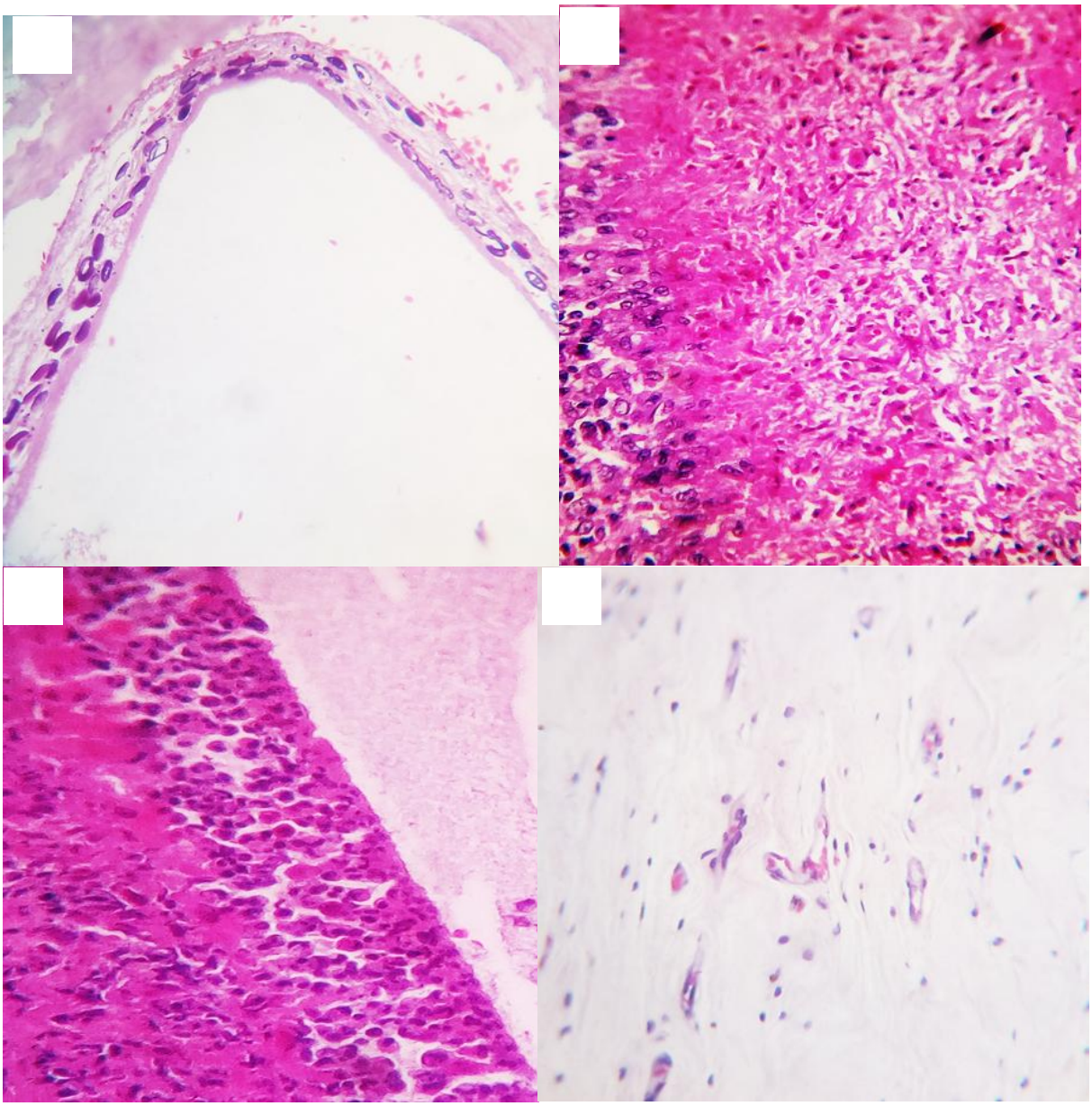


**Figure 24:** Cysticercosis with pericarditis cattle, macroscopic and microscopic changes

(A) Pericardium adheres to the heart (B) Thickened small size cyst like structure on myocardium (blue arrow) and whitish and opaque ventricles (brown arrow). (C) Degenerate bundles of heart muscle (star) with fat deposits (arrow). (D) The cyst in the myocardium. (E) The egg in the reproductive tract. (F) and (G) eosinophil Infiltration. (H) Necrotized pericardium with infiltration of few inflammatory cells.

### 4.5. Pathological Changes Encountered in Kidney of Cattle

Out of 3520 examined cattle 19.4% (685/3520) had different kind of abnormalities in their kidneys. Of these 34% (233/685) had cyst, 14.1% (97/685) nephritis, 14.8% (102/685) atrophy, 11.3% (78/685) urolithiais, 15.9% (109/685) hemorrhage and 9.6% (66/685) hypertrophy. The occurrence of cysts were highest followed by hemorrhage, atrophy, nephritis, urolithiais and hypertrophy.

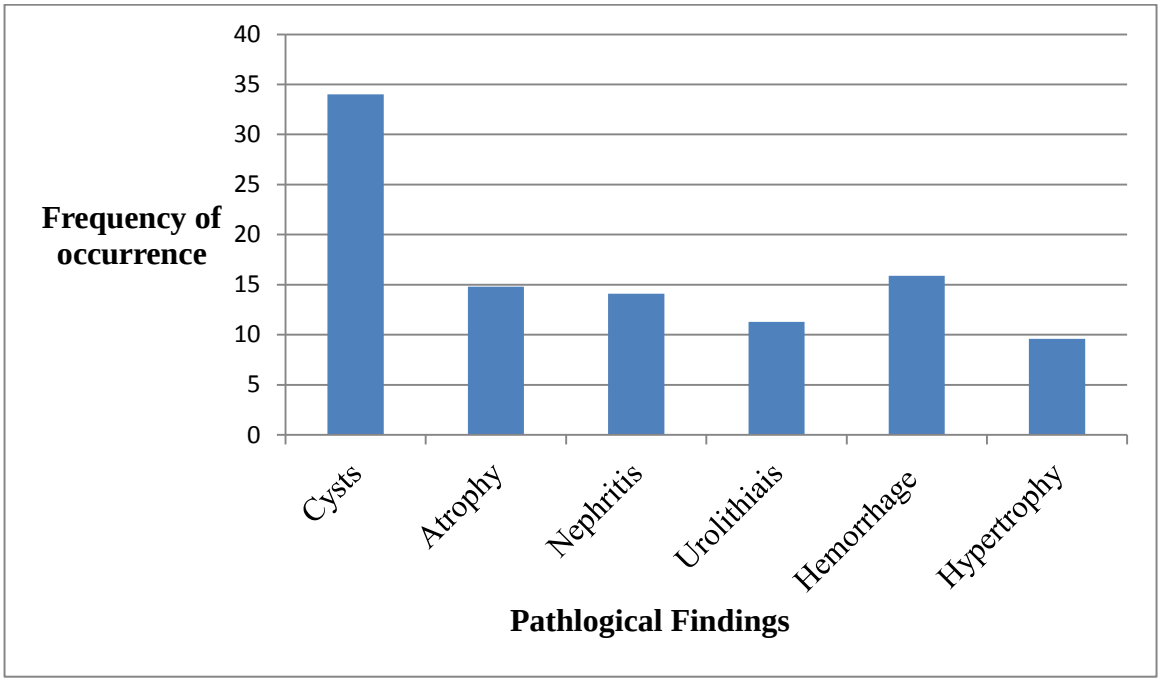



**Figure 25:** Frequency of pathological changes in the kidney of cattle

*4.5.1. Collecting duct cyst*

The rate of occurrence of cyst was 34% (233/685), but histologically confirmed collecting tubule cyst was 0.14% (1/685). Grossly, there was single, cyst on the pole of left kidney which is rounded, dark brownish and fitted between the surrounding lobules of the kidney (Figure: 26 A). Upon cut this cyst contained brownish or coffee color fluids. The cyst wall was thin and had honey comb structure on area in which the cyst wall attached to the kidney. They cyst's capsule was easily detached from the kidney. On overall view, the kidney had normal size and structure except having the cyst (Figure: 26 B).

Histologically, circular to tubular arranged cysts were seen in the medulla of the kidney (Figure 26 C). The cysts walls were lined by cuboidal type of cells and there was fibrosis and necrosis of interstitial tissues of kidney (Figure 26 D).

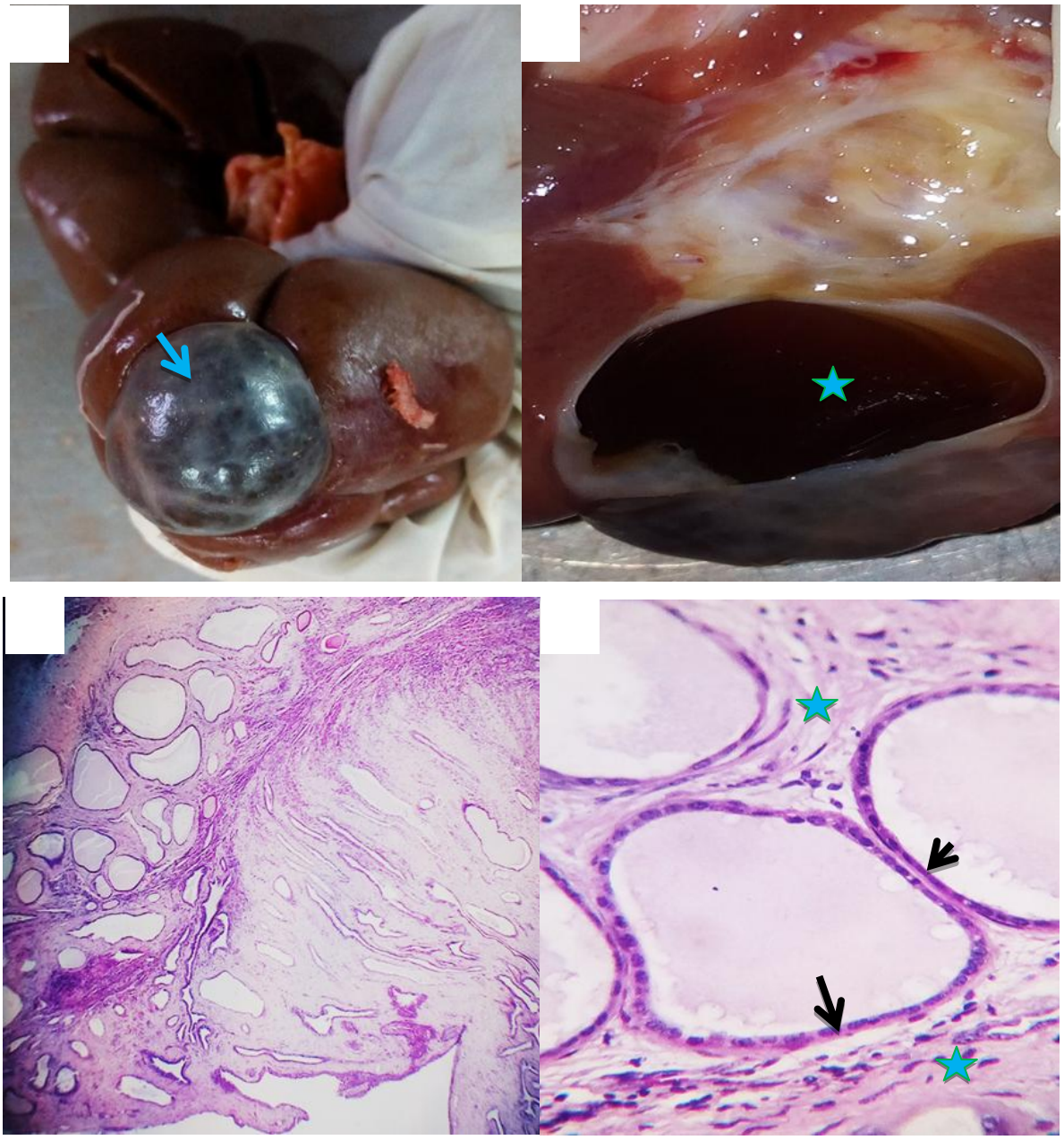


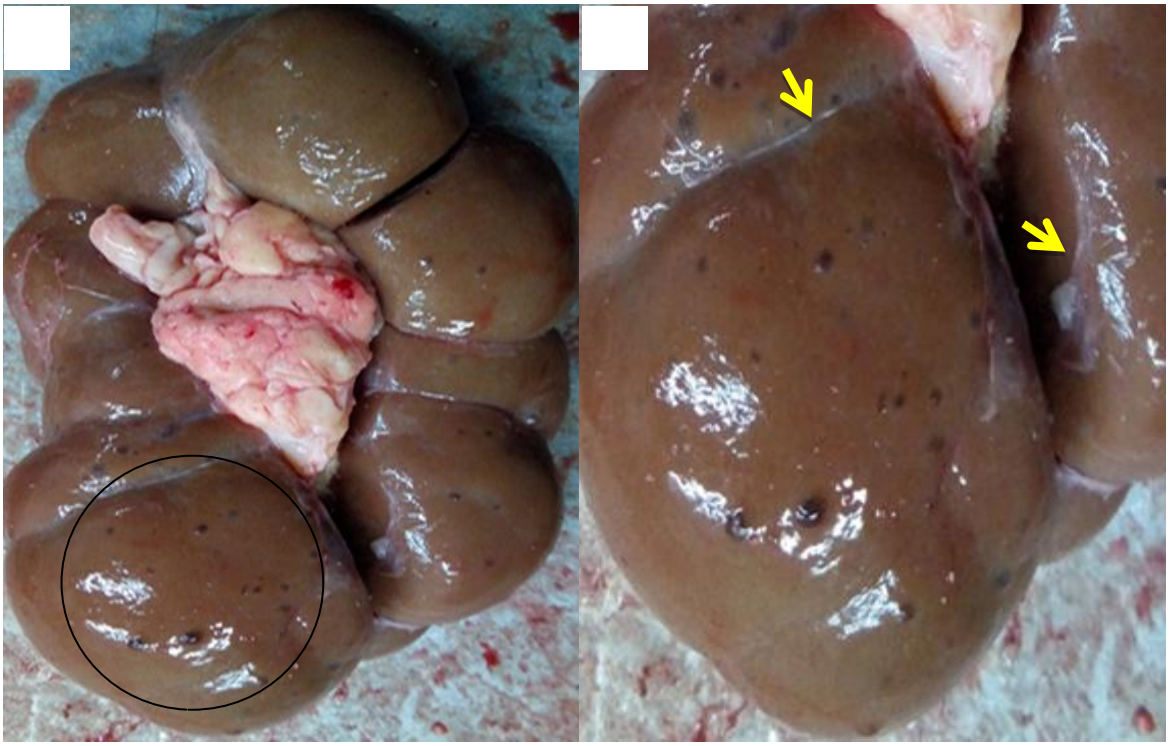
**Figure 26:** Collecting duct cyst cysts cattle, macroscopic and microscopic changes

(A) Rounded, blackish cyst fitted between the surrounding lobules of the kidney (arrow). (B) Brownish or coffee color fluids (star). (C) Circular to tubular arranged cysts on medullary area. (D) Cysts walls lined by cuboidal type of cells (arrow) and fibrosis of interstitial tissues of kidney (stars).

*4.5.2. Glomerular/cortical cysts*

The rate of occurrence of histologically confirmed glomerular/cortical cysts was 0.14% (1/685). Grossly, there were diffuse prominent variable size cysts on the cortex of the right kidney. The cysts were found on both dorsal and ventral surface of the kidney. The capsule was thickened, whitish and attached to the kidney and there was difficulty to separate capsule from the kidney (Figure 27 A and B).

Histologically, there were several cysts on the cortex of the kidney (Figure 27 C and D) which involved the glomerulus and the bowman's space were dilated and had rudimentary glomerular tufts. The cysts were lined by single cell layer of epithelium (Figure 27 E). There were glomerulitis, inflammation, necrosis and fibrosis of interstitial tissue with infiltration of inflammatory cells (Figure 27 F).



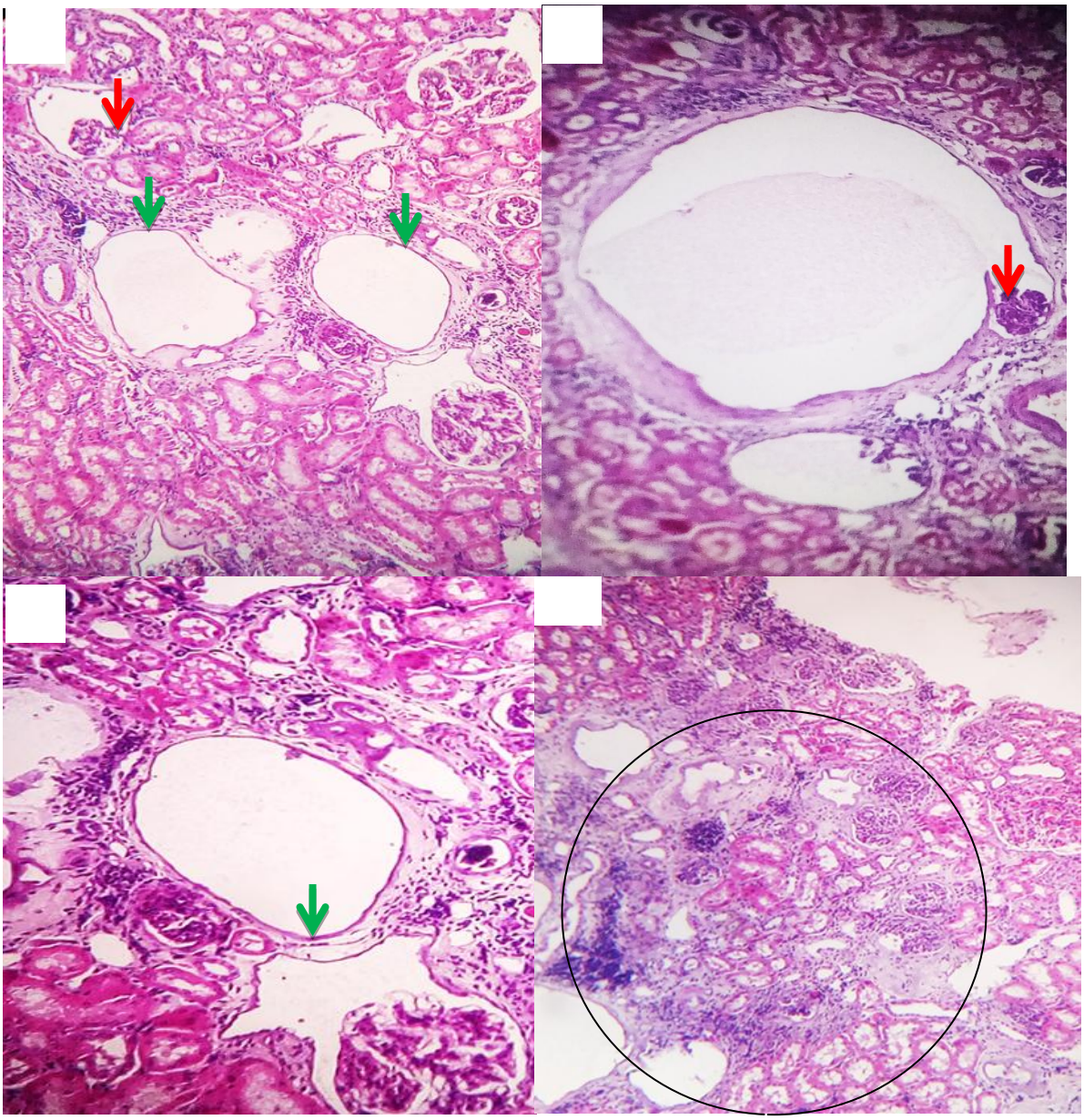


**Figure 27:** Cystic glomerulus cattle, macroscopic and microscopic changes

(A) Cysts on the cortex of the right kidney (circle). (B) Thick, whitish capsule attach to the kidney (arrow). (C) Cysts on the cortex of the kidney which involves the glomerulus (arrows). (D) Rudimentary glomerular tufts (red arrows) (E) Glomerulus lined by single cell layer of epithelium. (F) Inflamed and fibrosis interstitial tissue with infiltration of inflammatory cells (circle

*4.5.3. Tubulointerstitial nephritis*

Out of 685 kidneys 97 (14.1%) had nephritis, but the rate of occurrence of histologically confirmed tubulointerstitial nephritis was 0.14% (1/685). Grossly, there was small growth between the lobules of the kidney. The growth had underdeveloped lobular like structure and its some parts were attached to the adjacent lobules of the kidney while some of its parts were not attached. The size was about 2 cm by 1 cm (Figure: 28 A).

Histologically, some of renal tubules were hypertrophied while the others were atrophied. These tubules were contained eosinophilic fluid and resembled thyroid follicles and the interstitial tissues were necrotized and infiltrated with lymphocytes (Figure 28 B). There were few glomerular scleroses (Figure 28 C).

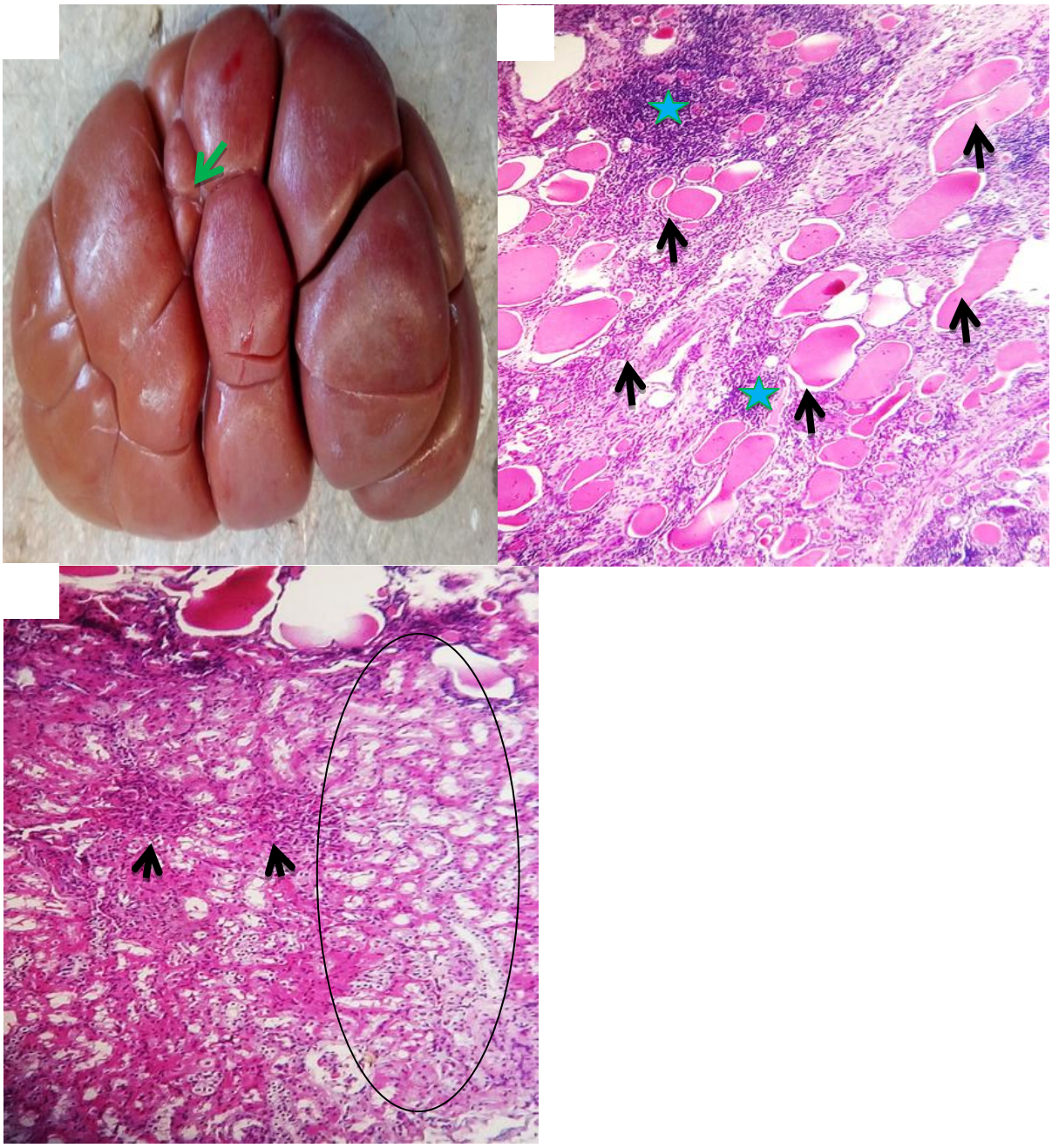


**Figure 28:** Tubulointerstitial nephritis cattle, macroscopic and microscopic changes

(A) Growth between the lobules of the kidney (arrow). (B) Hypertrophied and atrophied renal tubules filled with eosinophilic fluid (arrows) and interstitial tissues infiltrated with lymphocytes (stars). (C) Necrotized tubules (elliptic), glomerular scleroses (arrows).

*4.5.4. Atrophy*

The rate of occurrence of atrophy was 14.8% (102/685) but histologically confirmed atrophy was 0.14% (1/685). Grossly, the right kidney looked normal in structure, but the overall size was decreased and its dorsal surface was inflamed. The left kidney appeared normal in size and structure (Figure: 29 A).

Histologically, there were atrophy of the glomerulus and the tubules (Figure 29 B). There were reductions in bowmen capsule space and hemorrhagic area with total loss of the tubule (Figure 29 C). In other there was evidence of infarction marked by wedge shape of necrosis (Figure 2 D).

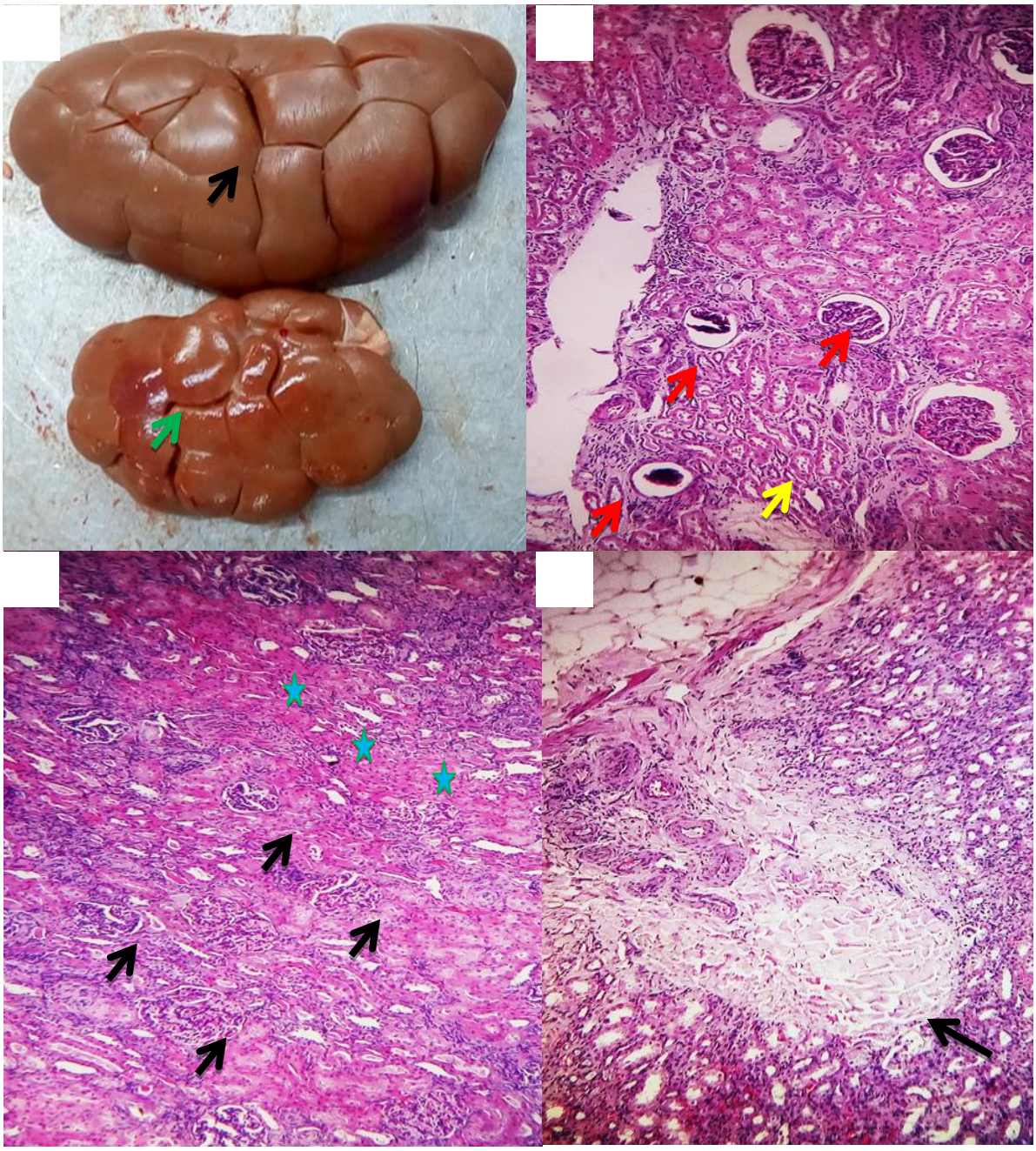


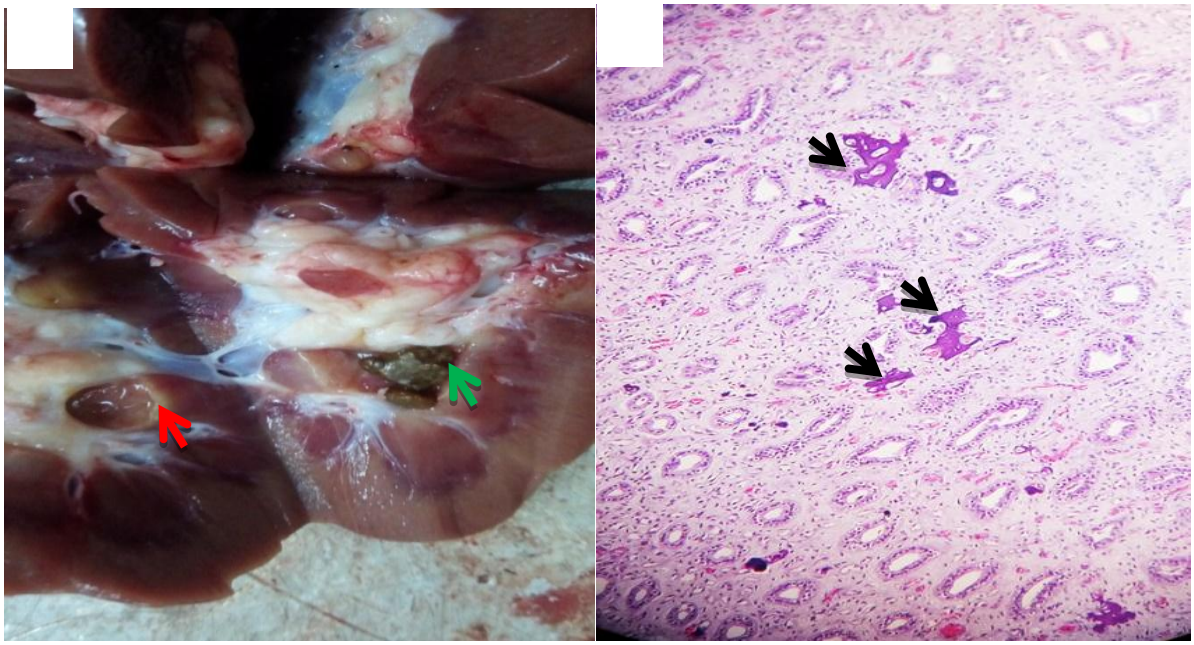
**Figure 29:** Atrophy of kidney cattle, macroscopic and microscopic changes

(A) Normal left kidney (black arrow) and small size inflamed right kidney (green arrow). (B) Atrophied glomerulus (Red arrow) and tubules (Yellow arrow). (C) Reduced bowmen capsule space (arrows) and hemorrhagic area with total loss of the tubule (stars). (D) Infarction marked by wedge shape of necrosis (arrows).

*4.5.5. Urolithiasis*

The rate of occurrence of urolithiais was 11.3% (78/685) but histologically confirmed urolithiais was 0.14% (1/685). Grossly, the left kidney was large, up on incision the cortex and medulla of kidney were slightly pale and there was friable stone like structure in in the minor calyx. Minor calyx that occupied the stone like material was inflamed (Figure: 30 A).

Histologically, there were several depositions of minerals on the interstitial tissue of medulla (Figure 30 B). Some of the medullary tubules or collecting tubules were distended and hyperemic (Figure 30 C) and the other were filled with purple or basophilic materials (Figure 30). The medullary papillae was highly necrotized and infiltrated with inflammatory cells (Figure 30 E).



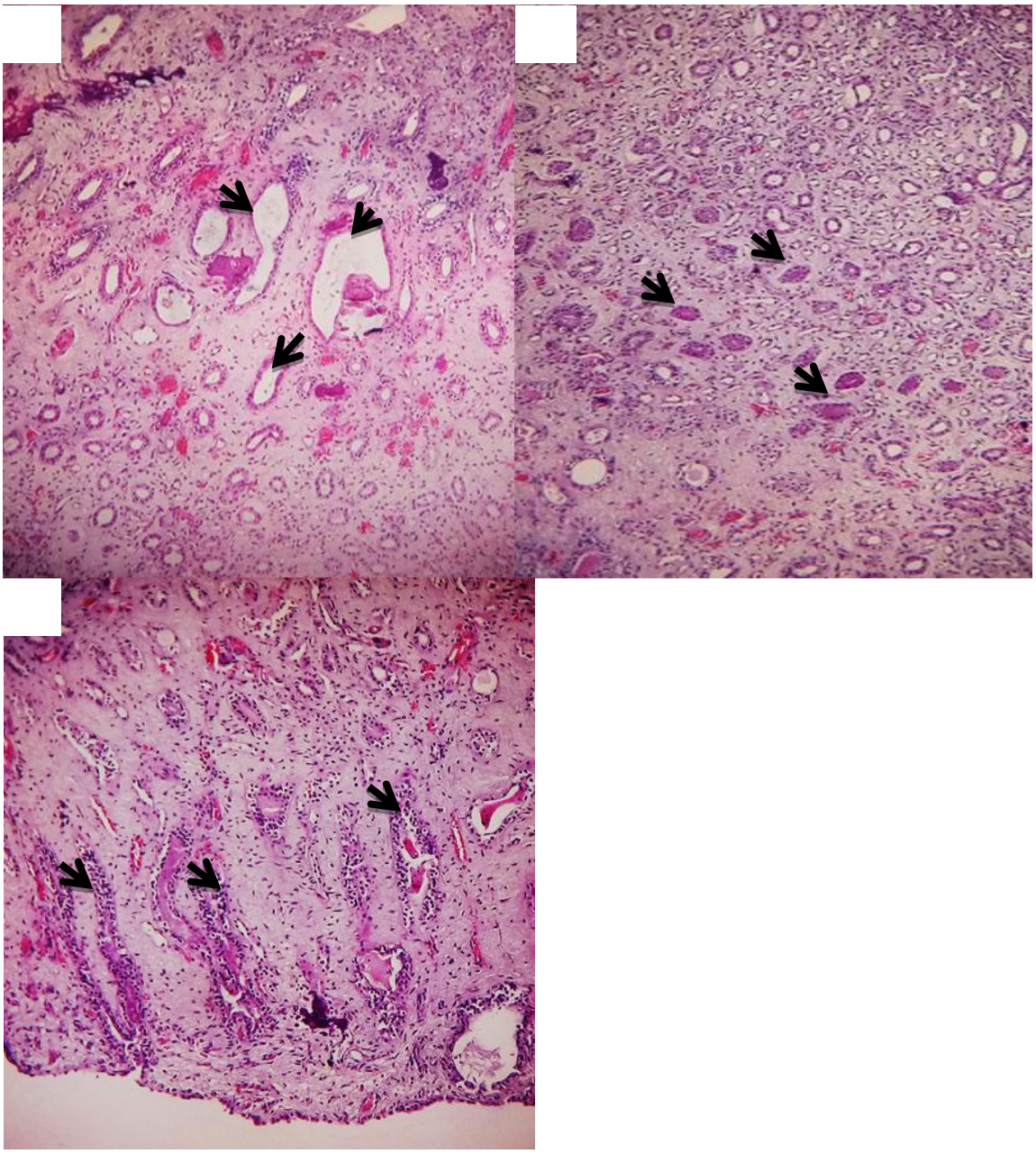


**Figure 30:** Urolithiais macroscopic and microscopic changes

(A) Friable stone in the minor calyx (arrow). (B) Minerals deposited on the interstitial spaces of medulla (arrows). (C) Distended and hyperemic collecting tubules (arrows). (D) Collecting tubules filled with minerals (arrows) (E**)** Necrotized medullary papillae.

### 4.6. Pathological Changes Encountered in Kidney of Camel

Out of 560 examined camels only two of them had pathological changes in their kidney. Of these 1/560 (50%) had abscess and 1/150 (50%) pyelonephritis were found.

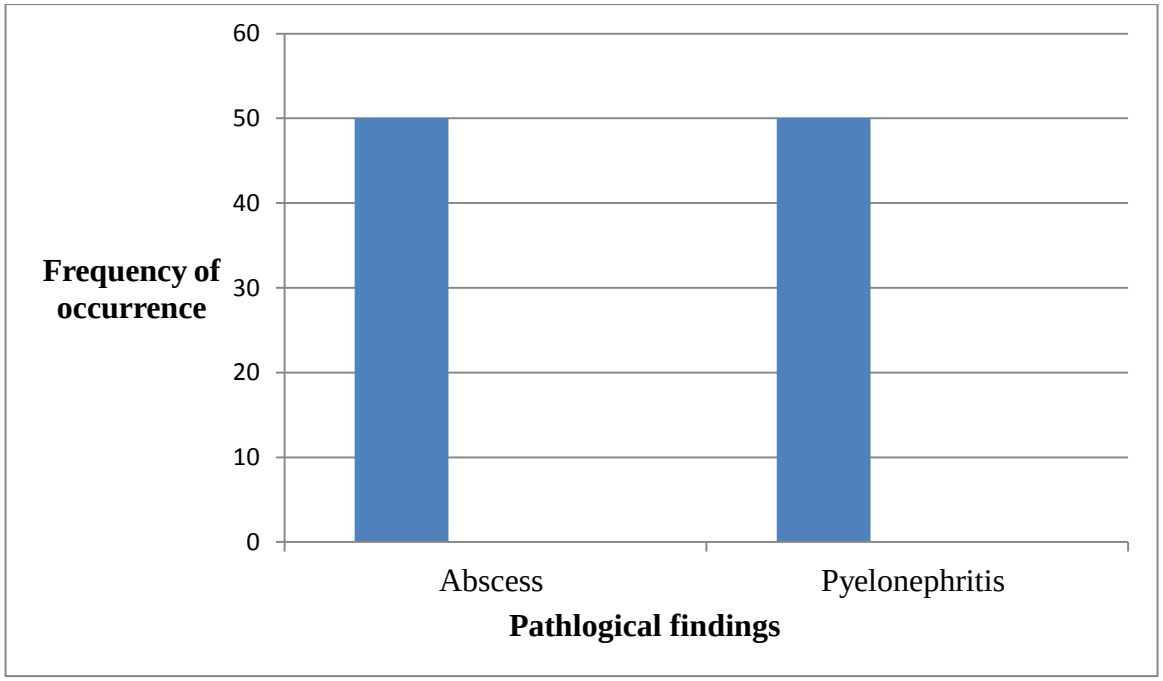



**Figure 31:** Frequency of pathological change in the kidney of camels

*4.6.1. Abscess*

The rate of occurrence of abscess was 50%. Grossly, the right kidney was attached to right lob of the liver; the right kidney was larger than the left kidney. Upon incision, pus was oozed from the medulla area (Figure 32 A). The capsule of the kidney was adhered to liver and on attachment area between the liver and kidney there was accumulation of pus. (Figure 32 B).

Histologically, there were diffuse infiltrations of few neutrophils and more macrophages throughout the kidney tissue. The cellular architecture of the kidney in the area of infiltration of inflammatory cells was totally damage (Figure 32 C, D and F) at different magnification power.

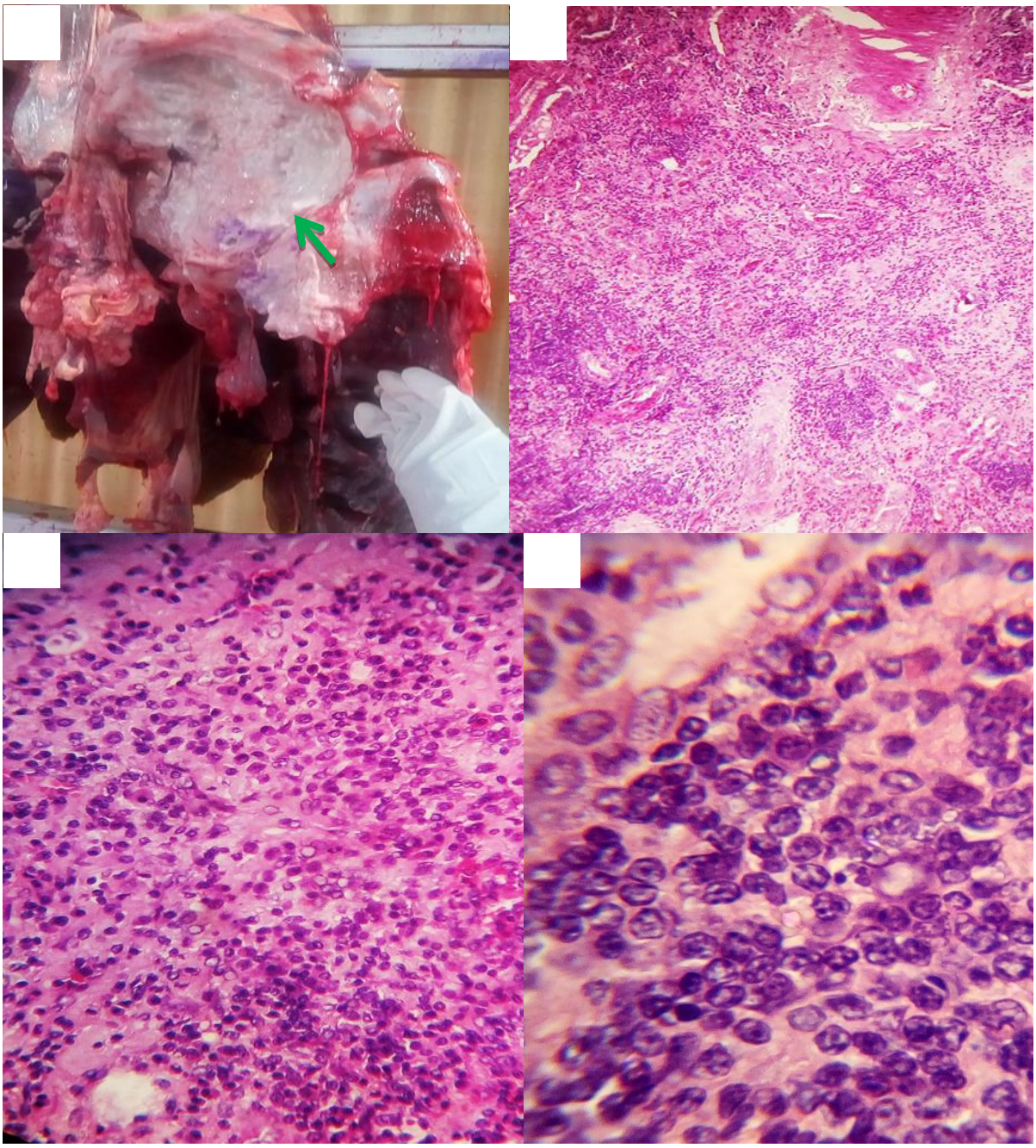


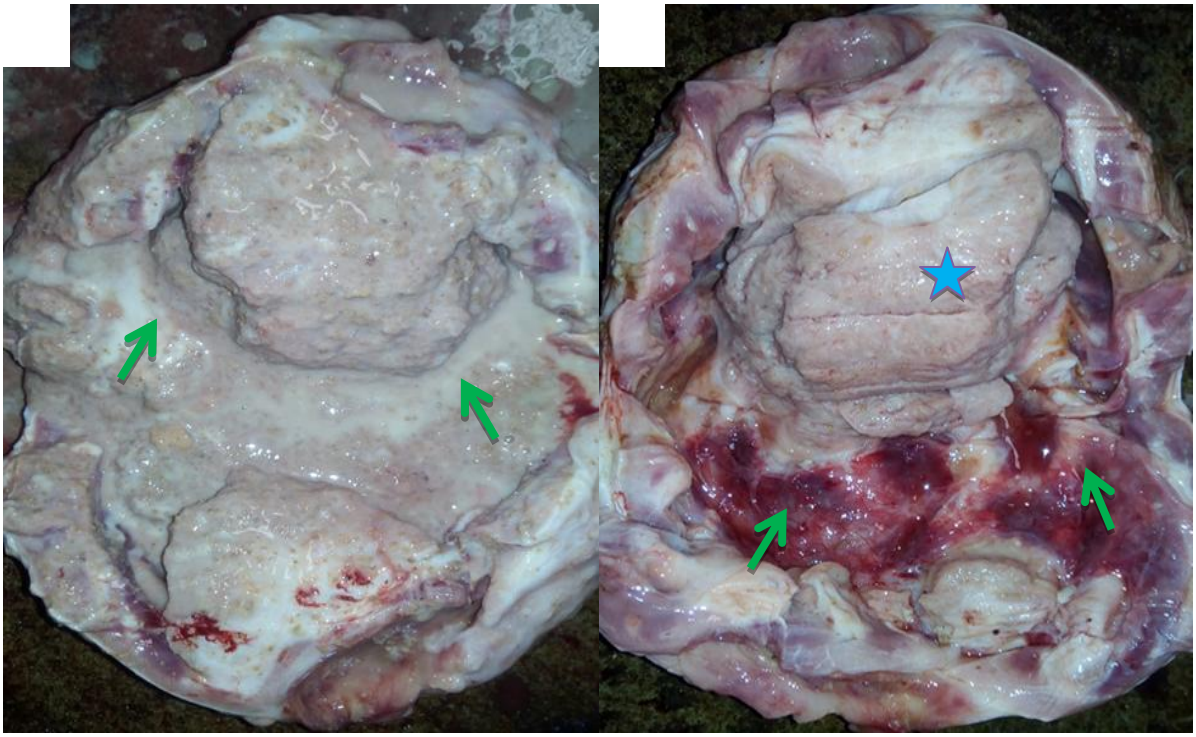
**Figure 32:** Abscess camel kidney, macroscopic and microscopic changes

(A) Pus oozed from the medulla area of right kidney (arrow). (B) and (C) Site of attachment between liver and kidney with accumulation of pus between them (arrows). (D) Damage cellular architecture of the kidney (E) cellular debris or dead neutrophils, few plasma cell and more macrophages. (F) X 100 inflammatory cells

*4.6.2. Chronic pyelonephritis*

The rate of occurrence of chronic pyelonephritis was 50%. Grossly, the left kidney was enlarged as compared to the right kidney. On cut, milky slightly thicken with sand like nature fluids/pus were freely came out from it and there was enlarged area of (Figure 33 A and B). On the other side of the kidney there was hemorrhagic area and it was very difficult to identify the cortex and the medulla (Figure 33 B). In contrast of this the left kidney looked normal with no gross lesions (Figure 33 C)

Histologically, epithelial of the tubule were totally sloughed, there several distended tubules contained hyaline cast (Figure 33 D), the interstitium was heavily infiltrated with lymphocytes and macrophages (Figure 33 E and F).



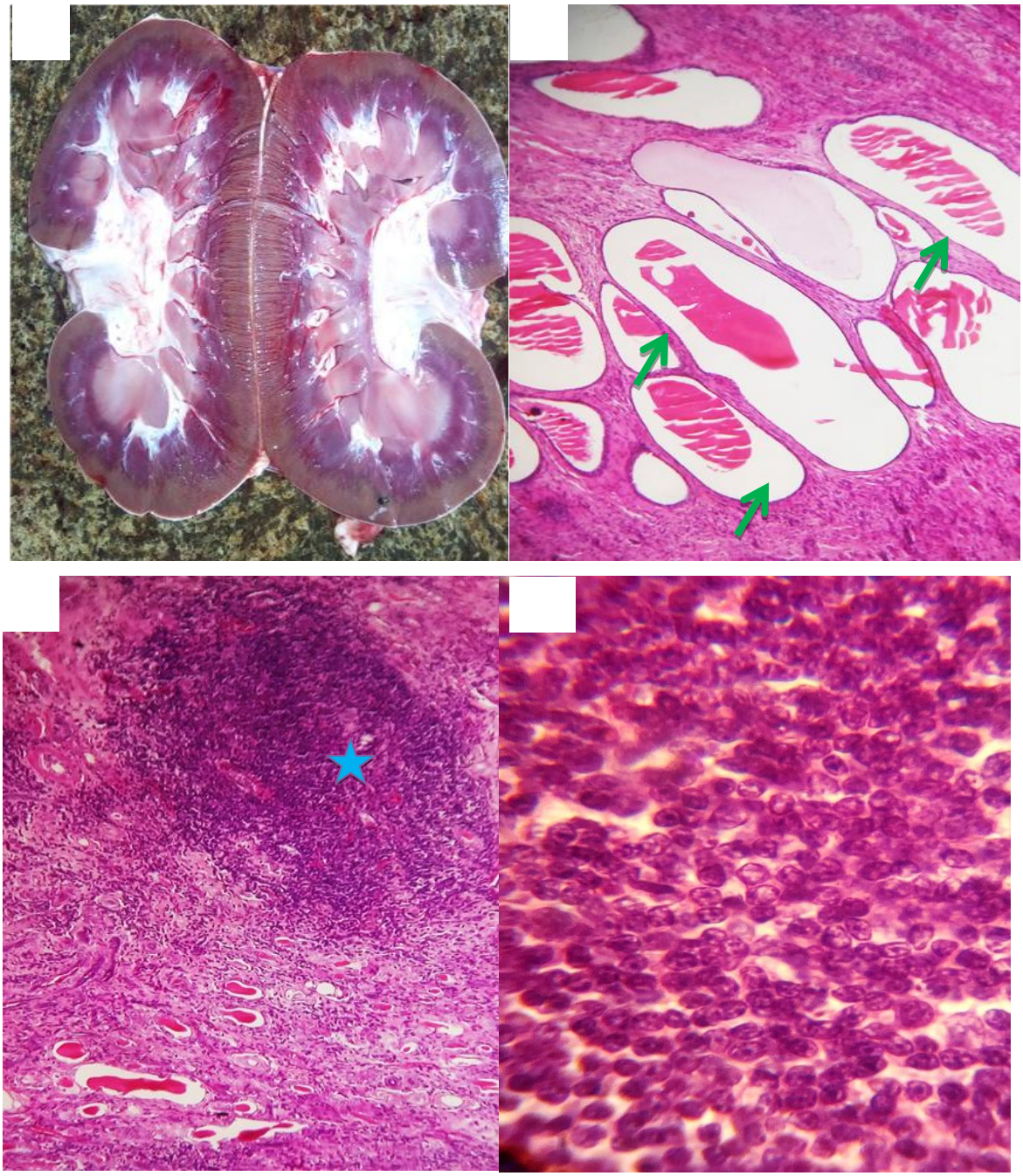


**Figure 33:** Chronic pyelonephritis camel kidney macroscopic and microscopic changes

(A) Milky slightly thicken fluids with fine granule nature (arrow). (B) Swelled area (star) with hemorrhagic area (arrow). (C) Grossly normal left kidney. (D) Distended tubules contained hyaline cast (arrows). (E) Heavily infiltrated interstitium (star). (F) infiltrated lymphocytes and macrophages

### 4.7. Pathological Changes Encountered in Liver of Cattle

Out of 3520 examined cattle 541(15.3%) had different pathological changes in their livers. Of these 1(0.18%) focal nodular hyperplasia, 1/541(0.18%) primary sclerosing cholangitis, 1/541(0.18%) solitary fibrous tumor, 1/541(0.18%) micro cirrhosis, 1/541(0.18%) biliary adenoma, 229/541(42.3%) hydatidosis, 1/541(0.18%) cholangiocarcinoma, 282/541(52.1%) fasciolosis, 23/541(4.2%) abscess and 1/541(0/18%) eosinophilic hepatitis. The most frequently occurred pathological change was fasciolosis followed by hydatidosis, abscess. Focal nodular hyperplasia, sclerosing cholangitis, solitary fibrous tumor, micro cirrhosis, biliary adenoma, cholangiocarcinoma and eosinophilic hepatitis were the least and occurred at equal rates.

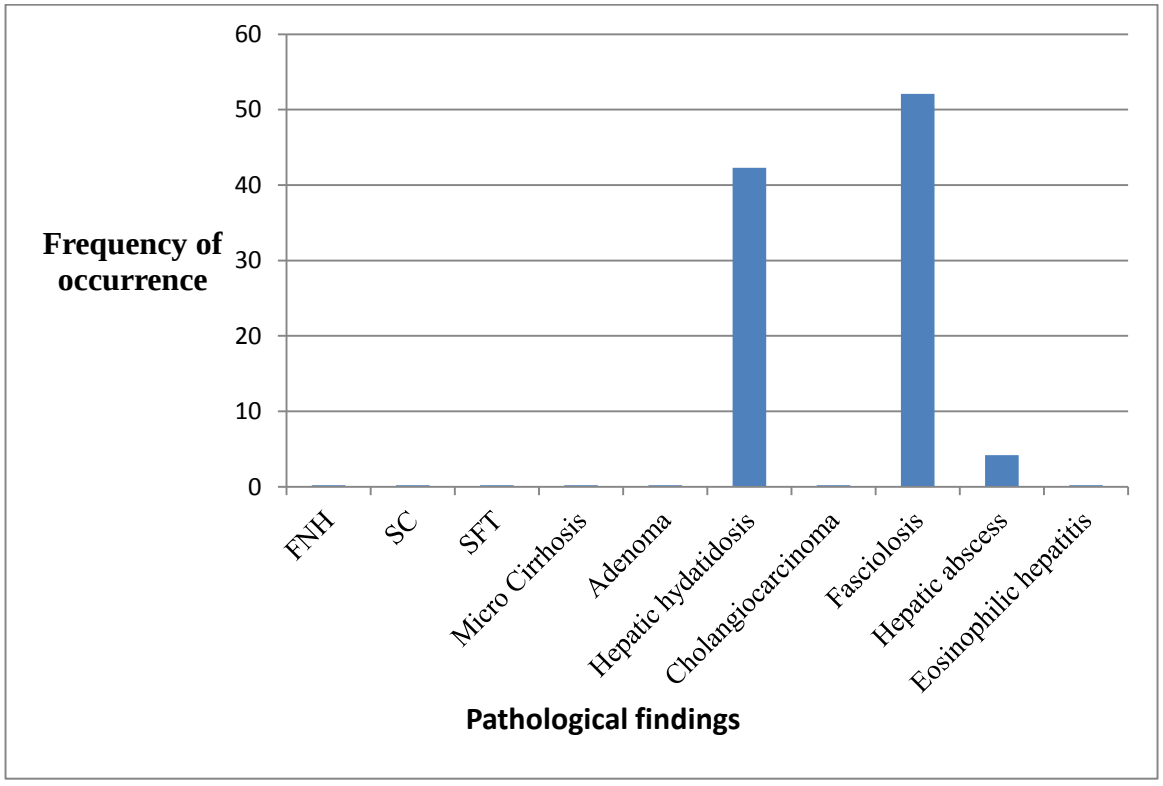



**Figure 34:** Frequency of pathological changes in the liver of cattle

*4.7.1. Focal nodular hyperplasia*

The rate of occurrence of focal nodular hyperplasia was 1/541(0.18%). Grossly, there were well circumscribed two small solitary nodular masses on parietal surface of liver one on left and other on right lob of liver and surrounded by falciform ligament. The size of these masses was about 3 cm by 5 cm. (Figure 35 A). The cut showed that the mass or the nodules were grown on surface of the liver (Figure 35 B).

Histologically, there were well circumscribed encapsulated areas composed of nodular of hepatocytes with lymphocytes infiltration. The nodules of the hepatocytes were separated by thick fibrosis septa which had markedly proliferated bile duct, dilated portal veins and fibrosis of tissue surrounding the portal track (Figure 35 C and D).

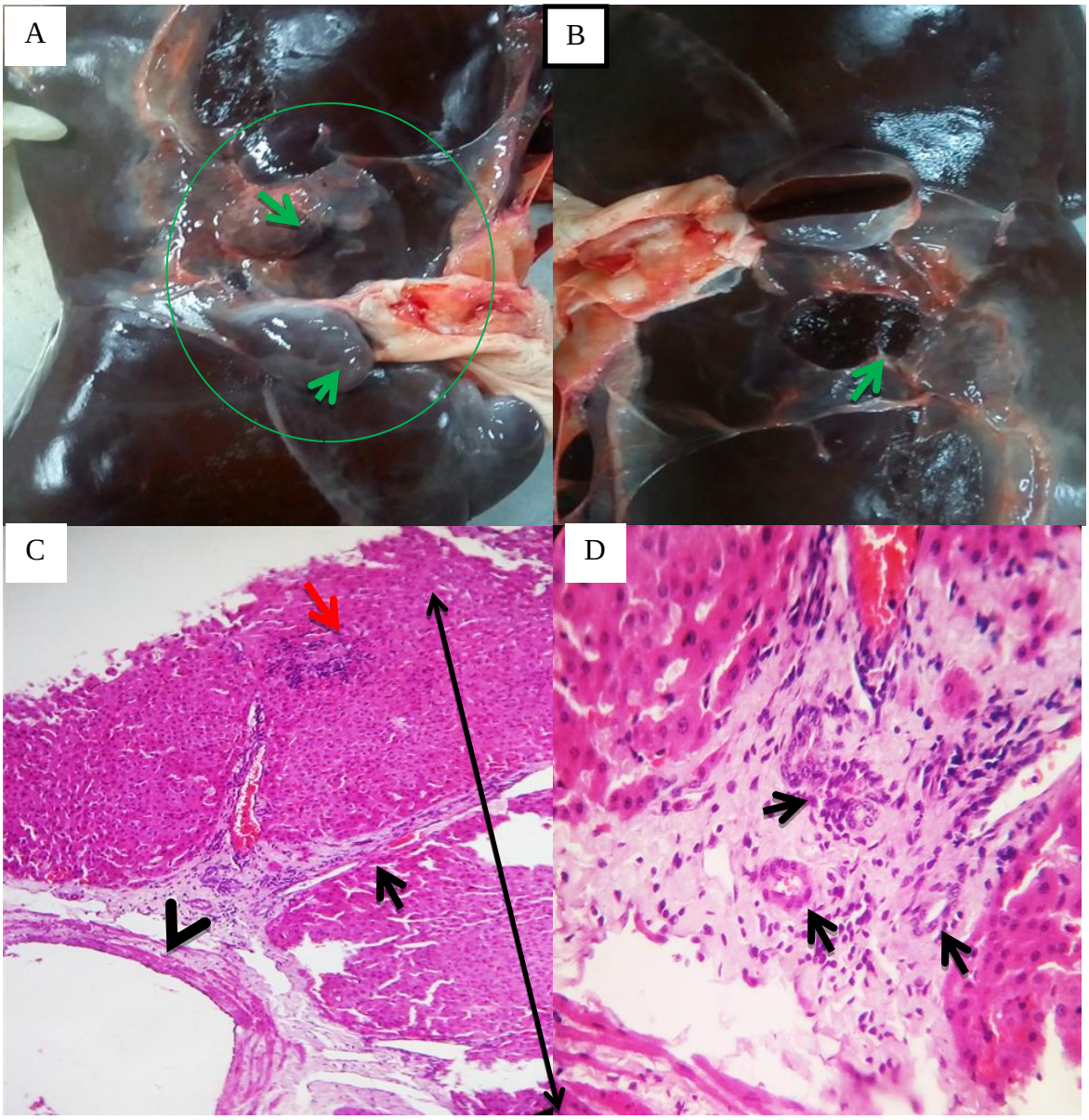



**Figure 35:** Focal nodular hyperplasia liver cattle, kidney macroscopic and microscopic changes

(A) Nodular masses (arrows) surrounded capsular structures (circle). (B) Cut, area show superficial growth of the nodules (arrow). (C) Nodular of hepatocytes infiltrated with lymphocytes (red arrow) separated by thick fibroses (arrow) and dilated portal vein (arrowhead). (D) Proliferated bile duct with fibrosis of tissue surrounding the portal track (arrows).

*4.7.2. Sclerosing cholangitis*

The rate of occurrence of sclerosing cholangitis was 1/541(0.18%) There was single mass on the quadrate lobe of liver. The mass was attached to the liver from one end. There was space between the mass and the liver that means the mass was slightly pendulous and had necrotized area at the attachment sites between quadrate lob of liver and the mass. The diaphragm were attach to the liver along the falciform ligament (Figure 36 A). Up on incision the bile ducts were slightly dilated (Figure 36 B).

There were multifocal distributions of intrahepatic bile duct which had onion skin fibrosis with bile plug inside it (Figure 36 C). There was few infiltration of inflammatory cells over it. Several proliferated capillaries occupied the fibrosis liver parenchyma (Figure 36 D).

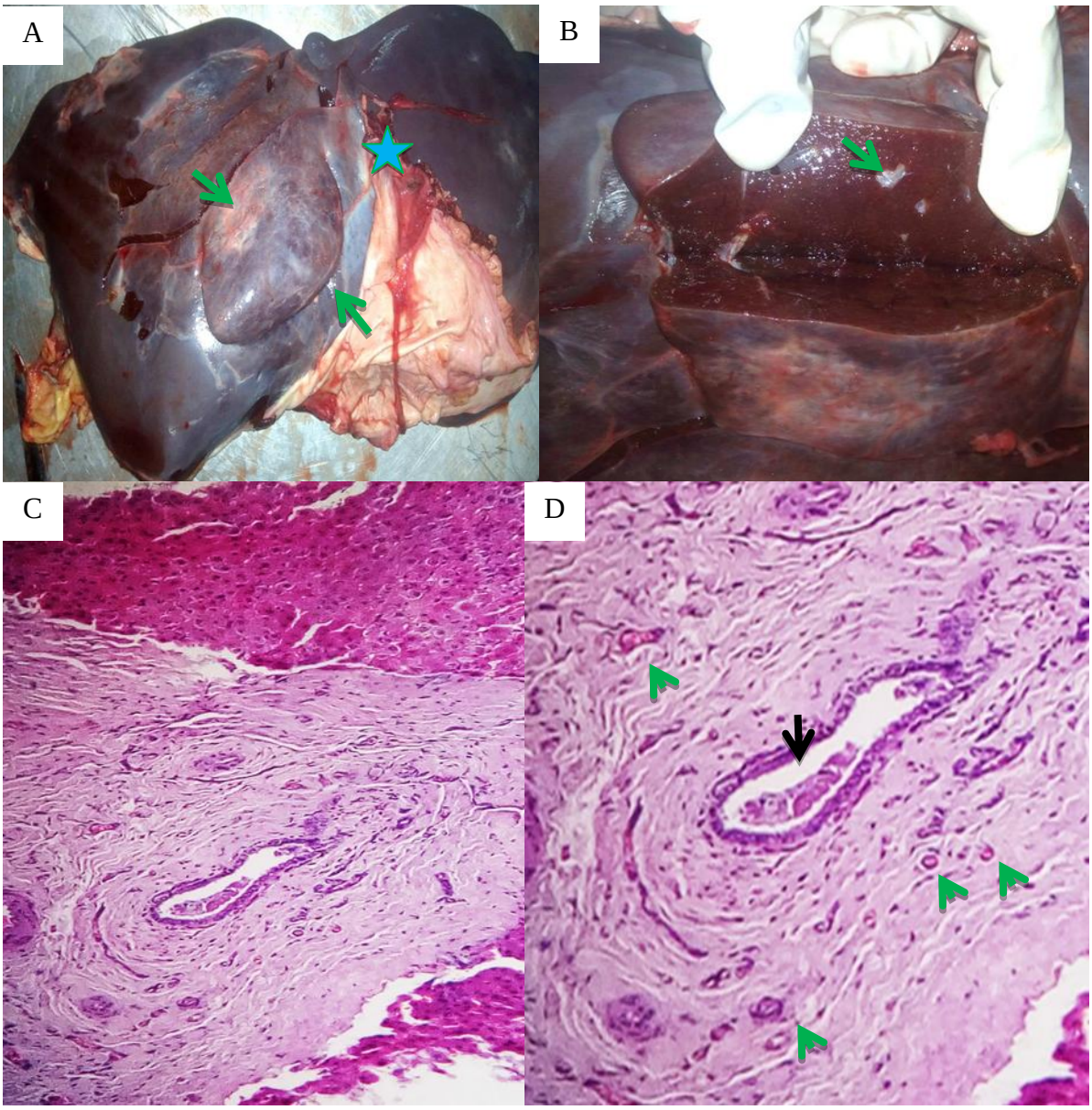



**Figure 36:** Primary sclerosing cholangitis cattle, macroscopic and microscopic findings

(A) Liver with single mass on the quadrate lob (arrow), necrotized area at site of attachment (arrowhead), attached diaphragm along falciform ligament (star). (B) Slightly dilated bile ducts (arrow). (C) Intrahepatic bile duct with onion skin nature fibrosis. (D) Proliferated capillaries (arrowheads) with few inflammatory cells and the lumen of bile plug with bile (arrow).

*4.7.3. Solitary fibrous tumor*

The rate of occurrence of solitary fibrous tumor was 1/541(0.18%). Grossly, the liver was enlarged, had rounded edge and lack proper lobation. There was mass on the left lob of visceral surface of liver. The capsule of the liver was thicken and cloudy. The diaphragm was attached to one side of the mass (Figure: 37 A). Up on incision, the mass had focal fibrosis in the center (Figure: 37 B).

Histologically, the tumor had heavily collagenized stroma background. The cell had ovoid to spindle shape with eosinophil cytoplasm. The arrangements of cells were in fascicular patterns. Degrees of cellularity vary from one area to another area and in less cellular area the intercellular collagen is thick (Figure C and D).

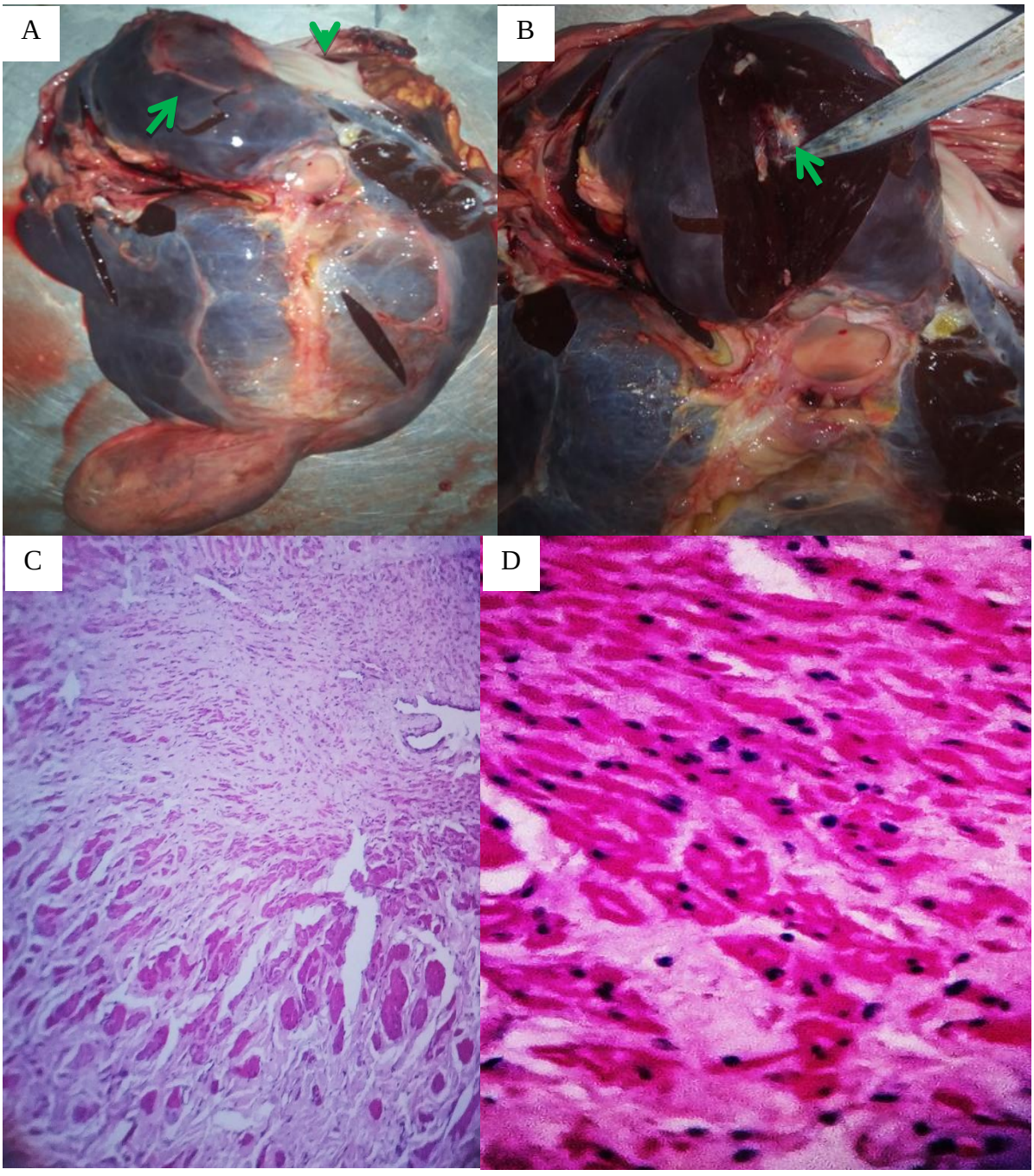


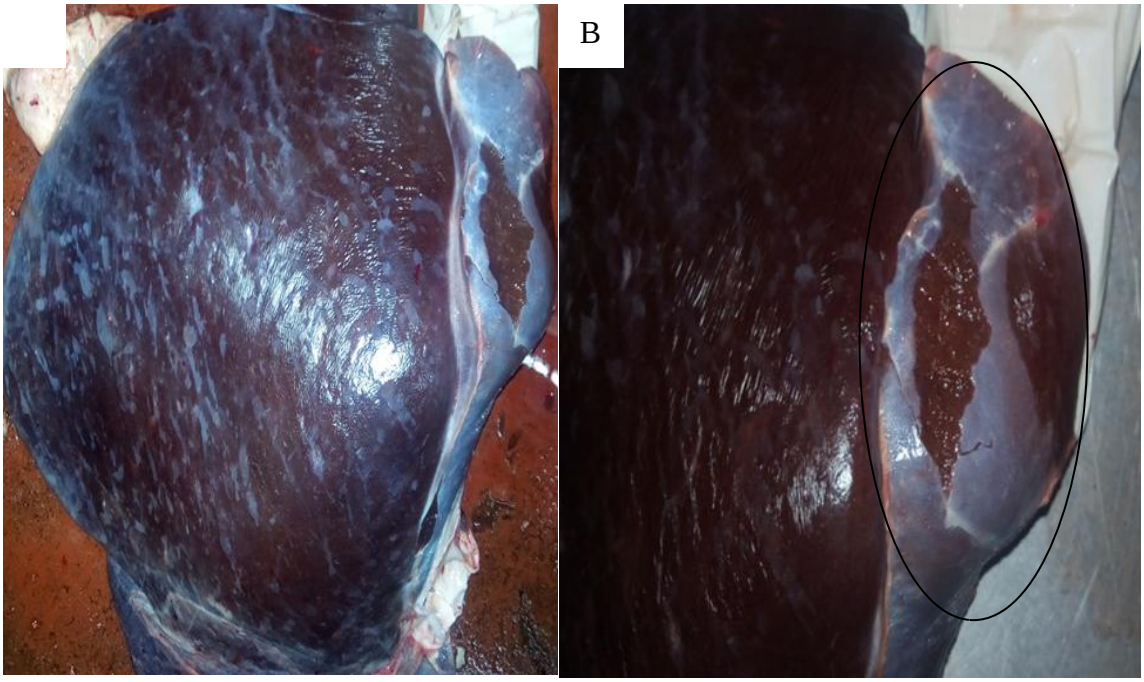

**Figure 37:** Solitary fibrous tumor liver cattle, macroscopic and microscopic findings

(A) Enlarged papillary lob. (B) Focal fibrosis in the center on cut area (arrow). X 10 (C) and X 40 (D) Collagenized stroma background with the cell that had ovoid to spindle shape with eosinophil cytoplasm and fascicular patterns

*4.7.4. Micro cirrhosis*

The rate of occurrence of micro cirrhosis was 1/541(0.18%) Grossly, the right lob of liver had rounded swelled area which had clear demarcation from the rest of the liver. The capsule surrounded this part of liver was tightly attached to liver and was dried and had fibrosis like nature (Figure: 38 A and B).

Histologically, the liver had multi focal nodular structure surrounded or separated by mild to severe dense band of collagenized fibrous scar (Figure 38 B and C). On collagenized fibrous scar there were ductular reactions, hyperplasia of bile duct and infiltration of inflammatory cells (Figure 38 D).



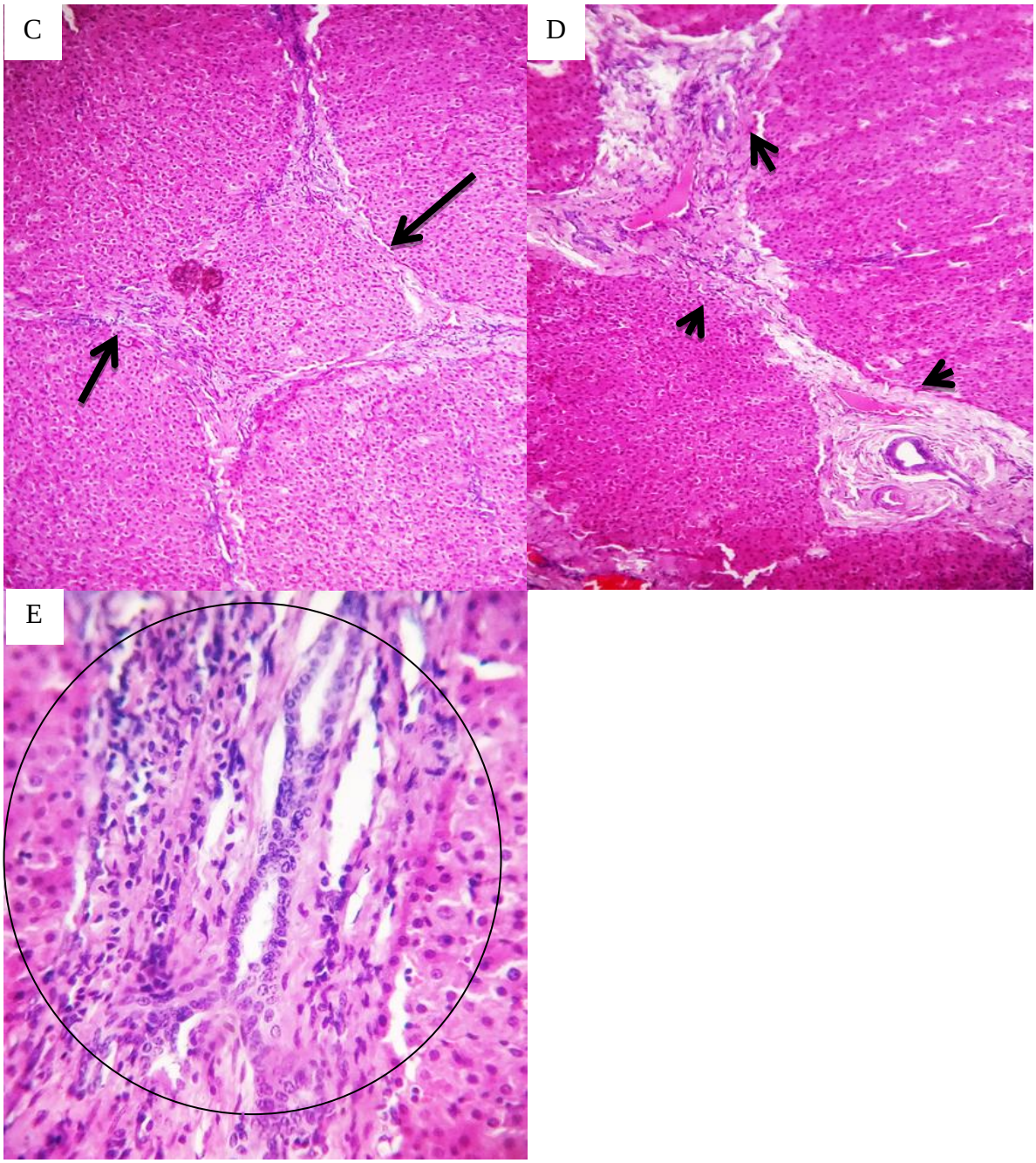



**Figure 38:** Micro cirrhosis liver cattle, macroscopic and microscopic findings

(A) Fibrosis capsule of the liver (B) swelled area surrounded by thick capsule (elliptic). (C) Nodular hepatocytes separated by band of collagenized fibrous scar (arrows). (D) Collagenized fibrous scar (arrows). (E) Ductular reactions

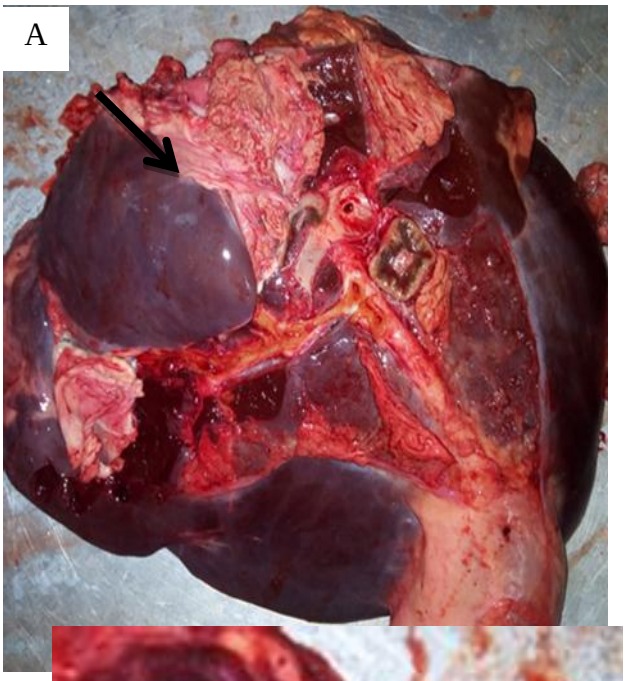
*4.7.5. Biliary adenoma*

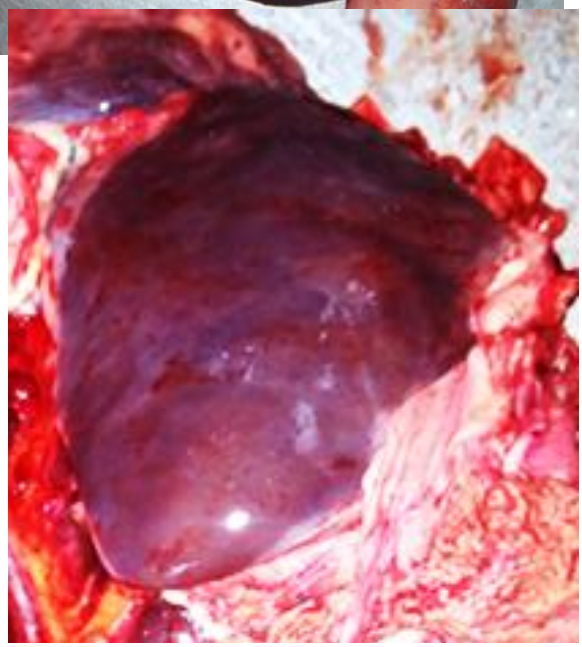
The rate of occurrence biliary adenoma was 1/541(0.18%). Grossly, papillary process of caudate lobe on visceral surface of liver was enlarged and had irregular raised structures over it (Figure 39 A and B). Up on incision there was whitish fibrous like structure inside the liver (Figure 39 C).

Histologically, the tumor was well circumscribed and the surrounded by fibrosis tissue. There were non uniform large to small proliferated bile ducts lined by cuboidal cells combined with inflammatory cells infiltration (Figure 39 D and E).

B

A





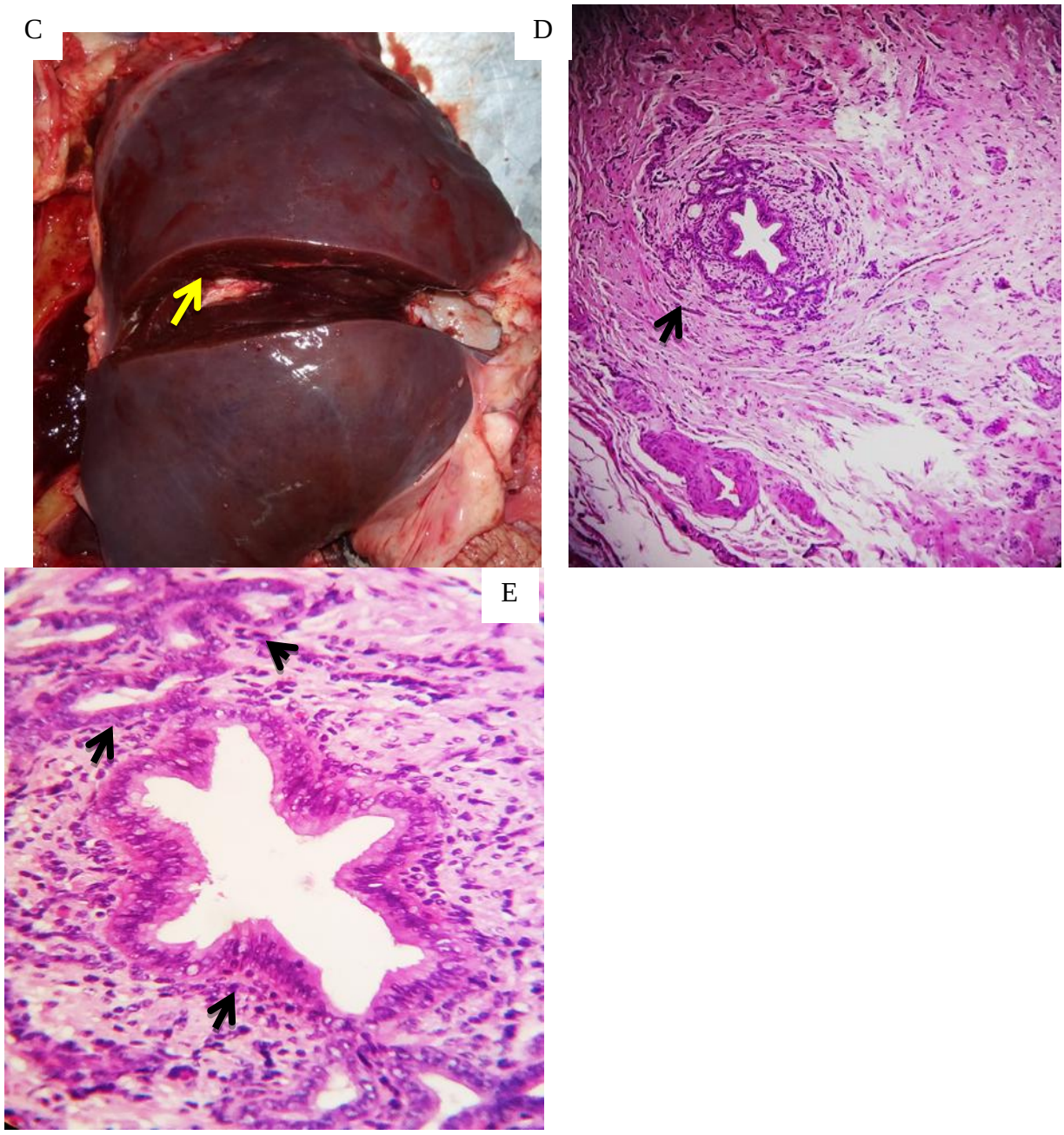


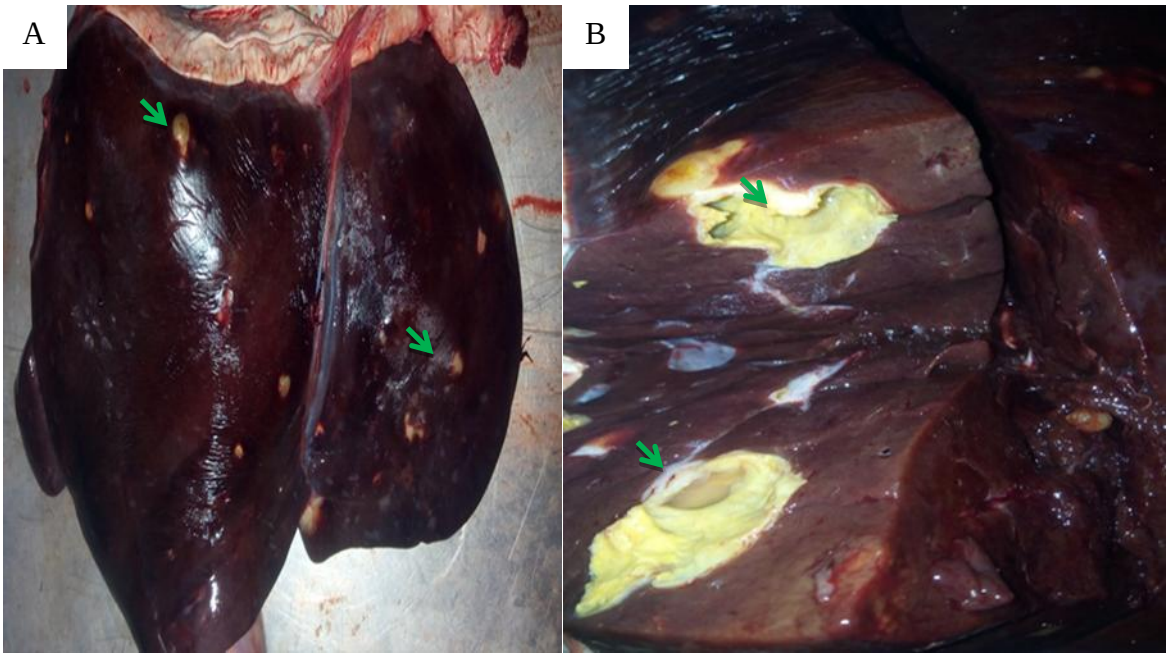

**Figure 39:** Biliary adenoma cattle, macroscopic and microscopic changes

(A) Enlarged papillary lob. (B) Irregular raised structures on papillary lob. (C) Whitish fibrous like structure (arrow). (D Tumor surrounded by fibrosis tissue. (E) Proliferated bile ducts lined by cuboidal cells and infiltrated with inflammatory cells.

*4.7.6. Hepatic hydatidosis*

The rate of occurrence of hepatic hydatidosis was 229/541(42.3%) Grossly, there were multifocal different sizes, clear fluid filled cysts on both visceral and parietal surface of the liver (Figure 40 A). Upon incision there were cystic cavities which were varying in size from small to large and thickness of capsule. Some of the cyst had yellowish and thick fibrous capsule with calcified material inside them other had thin and white to slightly yellowish capsule (Figure 40 B).

Histologically, there was cyst in the tissue of liver (Figure 40 C). The parenchyma of the liver surrounded the cyst wall were edematous, highly fibrosised and infiltrated with inflammatory cells and there were depositions of calcified materials (Figure 40 D). Higher magnification of the parenchyma of the liver surrounding the cyst wall showed infiltration with macrophages, lymphocytes and eosinophils (Figure 40 E and F).



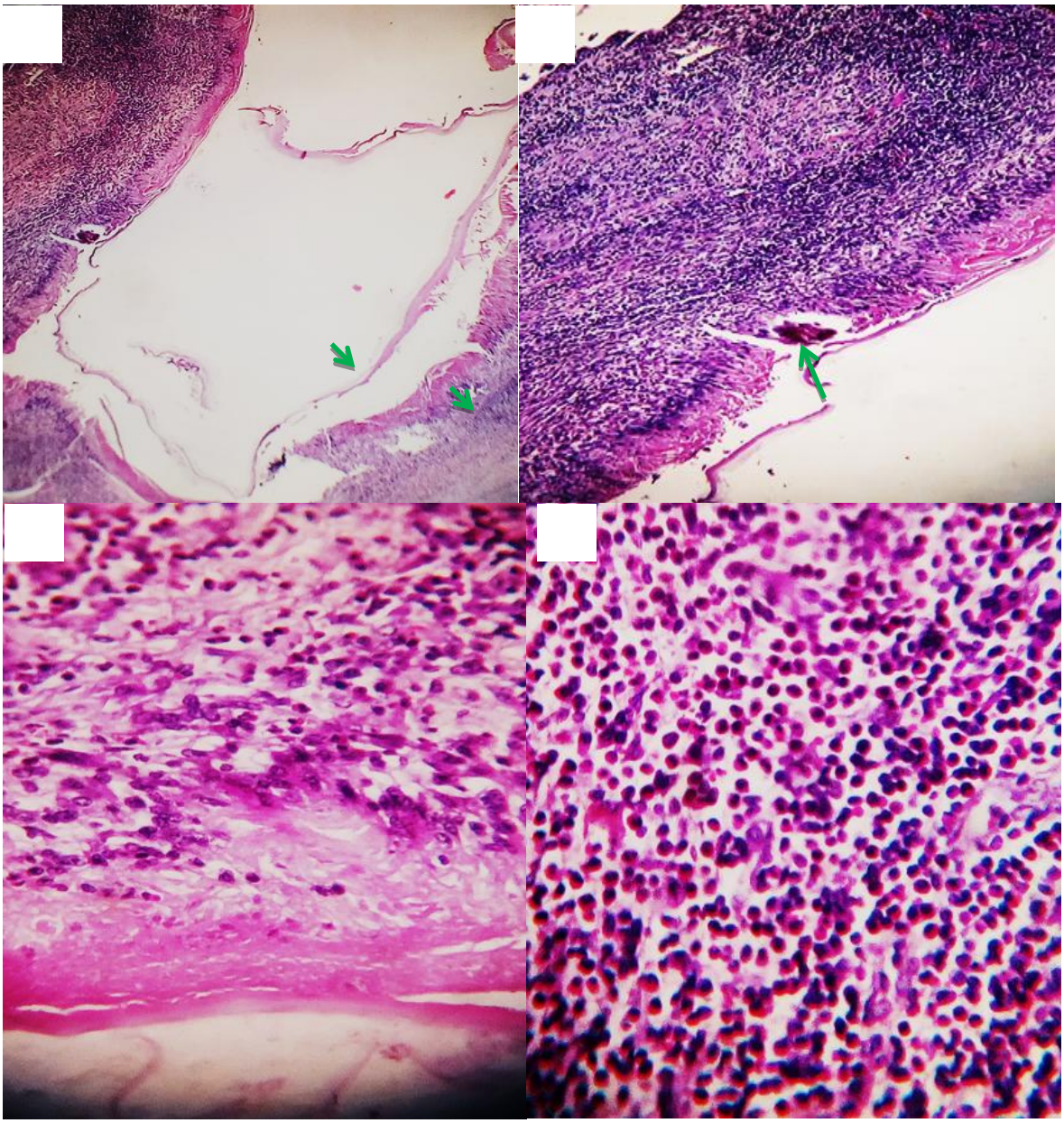


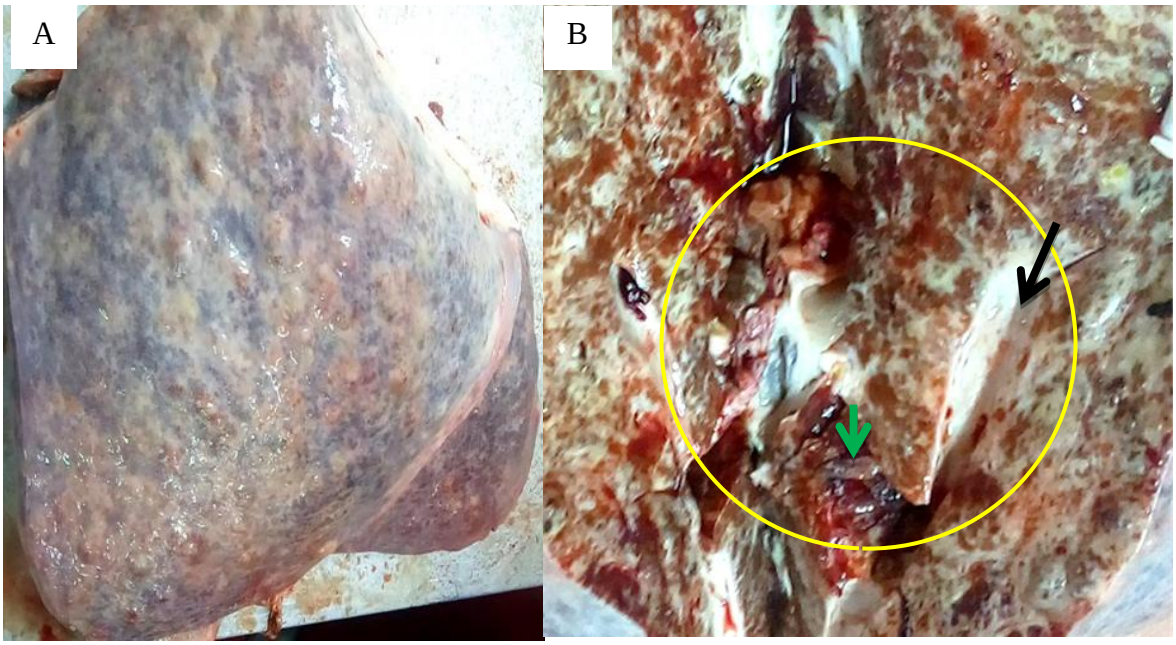
**Figure 40:** Hepatic hydatidosis cattle, macroscopic and microscopic changes

(A) Liver shows different sizes cystic surface (arrows). (B) Yellowish and thick capsule cystic cavities (arrows). (C) Cyst in the tissue (arrow). (D) Infiltration of inflammatory cells with focal calcified materials (arrow). (E) Infiltrated macrophages, lymphocytes and few Eosinophils. (F) Infiltrations of eosinophils.

*4.7.7. Cholangiocarcinoma*

The rate of occurrence cholangiocarcinoma was 1/541(0.18%). Grossly, the liver was enlarged, heavy, had rounded edge, the capsules were fibrosised and difficult to be detached; there were diffuse variable size of white raised nodules like structure on the surface of the liver (Figure 41 A). Upon incision, the cut surfaces had gray to reddish area with pale parenchyma of the liver, much widened bile duct and the liver also had infestation of fasciola (Figure 41 B).

Histologically, the liver parenchyma were infiltrated with tubular to papillary glandular like structure of bile duct lined by cuboidal type of epithelial cells with variable fibrous stroma. There was also hepatocellular adenoma (Figure 41 C and D) which had scattered small nest of tumor cells (Figure 41 E). There was fasciola parasite in the tissue could be the possible causes of cholangiocarcinoma in this animals (Figure 41 F).



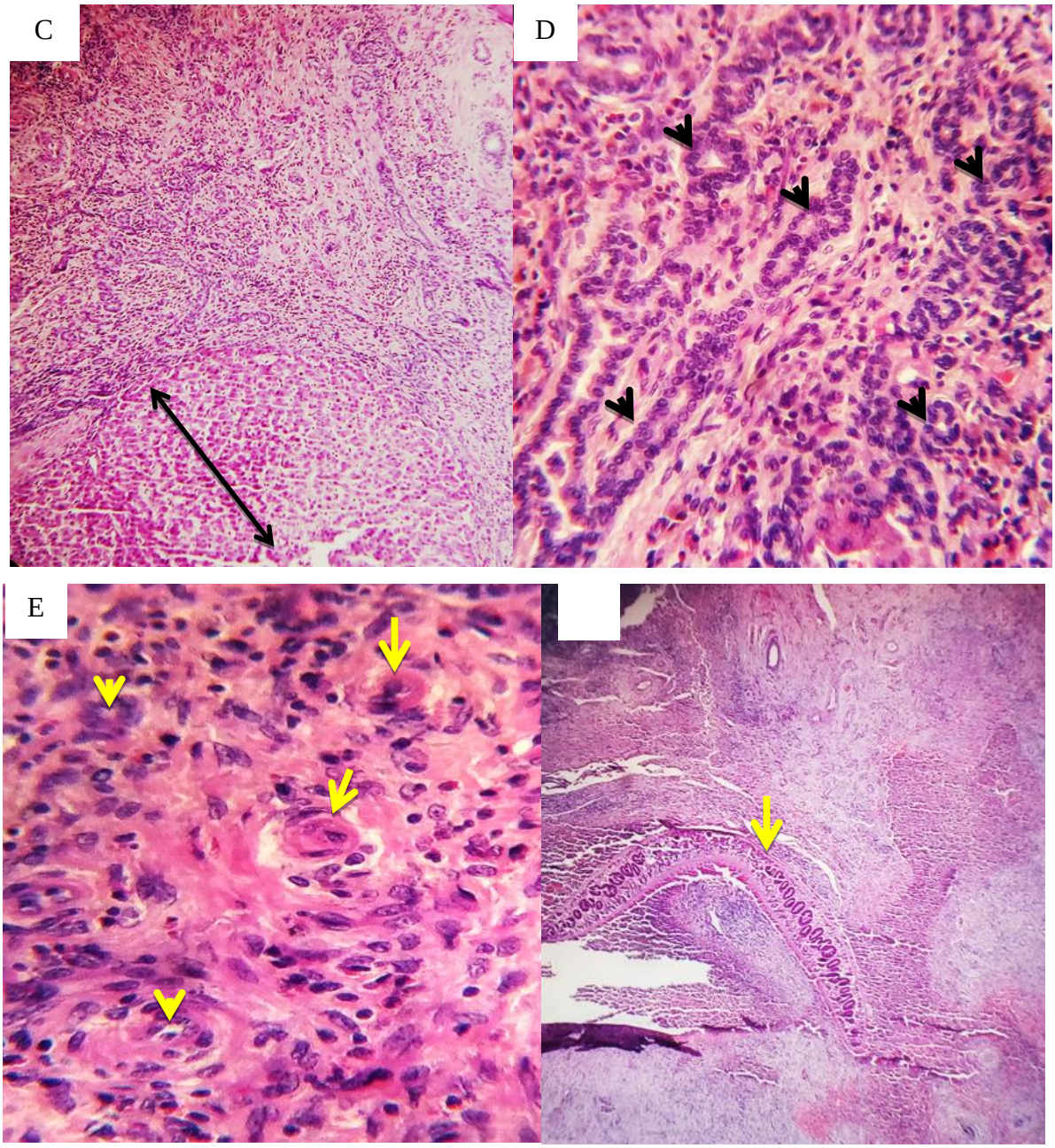


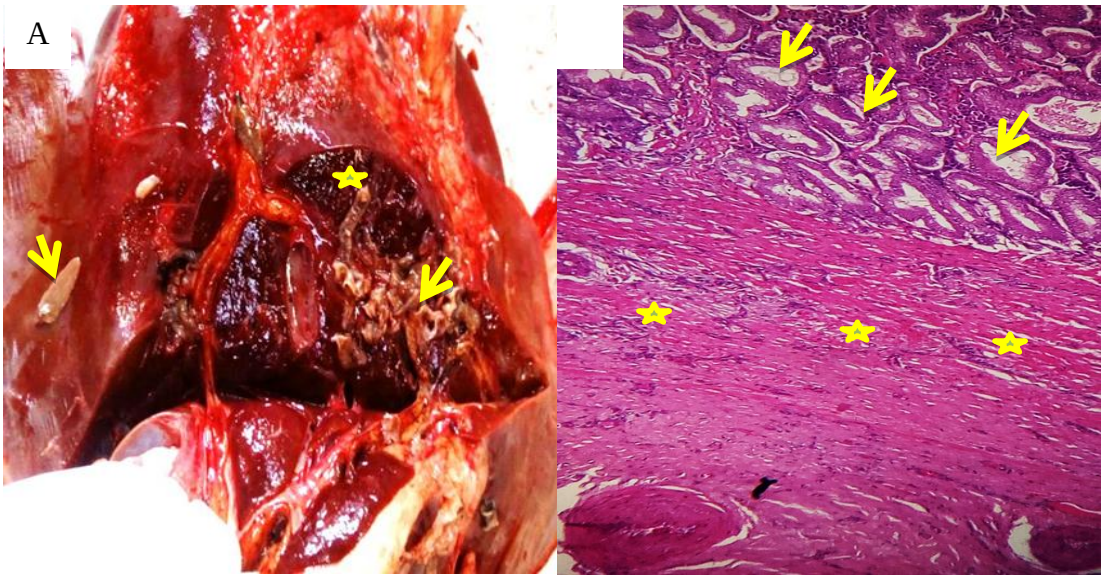

**Figure 41**: Cholangiocarcinoma liver cattle, macroscopic and microscopic changes

(A) Enlarged, rounded edge, fibrosis capsules with variable size of white raised nodules. (B) Gray to reddish parenchyma of the liver (circle), dilated bile duct(black arrow) and infestation of Fasciola (arrow). (C) X 10 and (D) X 40 papillary to glandular like structure of bile (arrows) and hepatocellular adenoma (arrow). (E) Scattered small nest of tumor cells (arrows). (F) Adult fasciola (arrow).

*4.7.8. Fasciolosis*

The rate of occurrence fasciolosis was 282/541(52.1%). Grossly, the liver was enlarged, on cut it was covered with rust colored materials, there were large number of immature and mature fasciola in bile duct. The fasciola caused extensive destruction of liver parenchyma marked with haemorrhages. The bile ducts were more prominent, dilated, thickened and had liver flukes inside them (Figure: 42 A).

Histologically, the portal tracks of the liver were extensively fibrosis, hemorrhagic and infiltrated with inflammatory cells mainly eosinophils. The bile ducts were highly proliferated and metastasized in to mucus producing like cells (Figure 42 B and C). There were extensive proliferation of fibrous connective tissue around the intra-hepatic bile ducts and (Figure 42 D). The adult parasites were seen in tissue section with its distinctive structure including tegumental syncytium (A), tegumental cell bodies (B), undifferentiated vitelline cells (C), tegmental spines (D), mature vitelline cells (E), intermediate vitelline cells (F) and gut (G), (Figure 42 E). Infiltration of eosinophils (Figure 42 F).



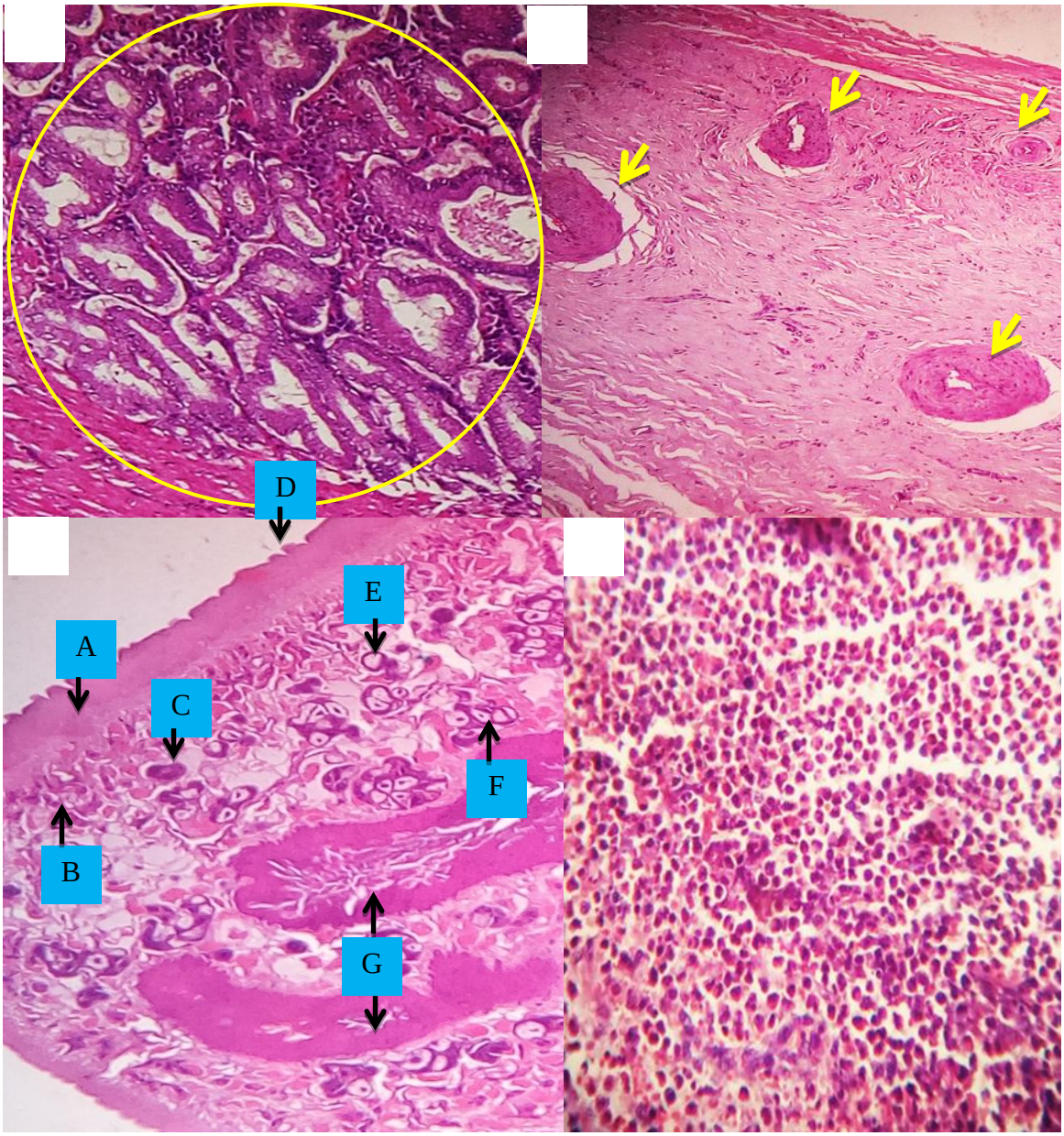



**Figure 42:** Fasciolosis cattle, macroscopic and microscopic changes

(A) Liver showing rust colored materials (star) fasciola (arrows). (B) Portal tracks of the liver showed fibrosis, hemorrhagic and infiltrated with inflammatory cells (stars) and metastasis bile duct in to mucus producing like cells (arrows). (C) X 40 metastasis bile ducts. (D) Proliferation of fibrous connective tissue. (E) Adult fasciola (F) eosinophils infiltration

*4.7.9. Hepatic abscess*

The rate of occurrence abscess was 23/541(4.2%). Grossly, there was encapsulated swelled area on the left lobe of the liver. The swelled area was covered by the diaphragm. The capsule that surrounded the swelled area was made by parts of the diaphragm. Up on incision thick slightly yellowish pus was flow from it and there was large cavity inside it (Figure: 43 A and B).

Histologically, there were multifocal infiltration of inflammatory cells with neutrophils domination and few lymphocytes (Figure 43 C).

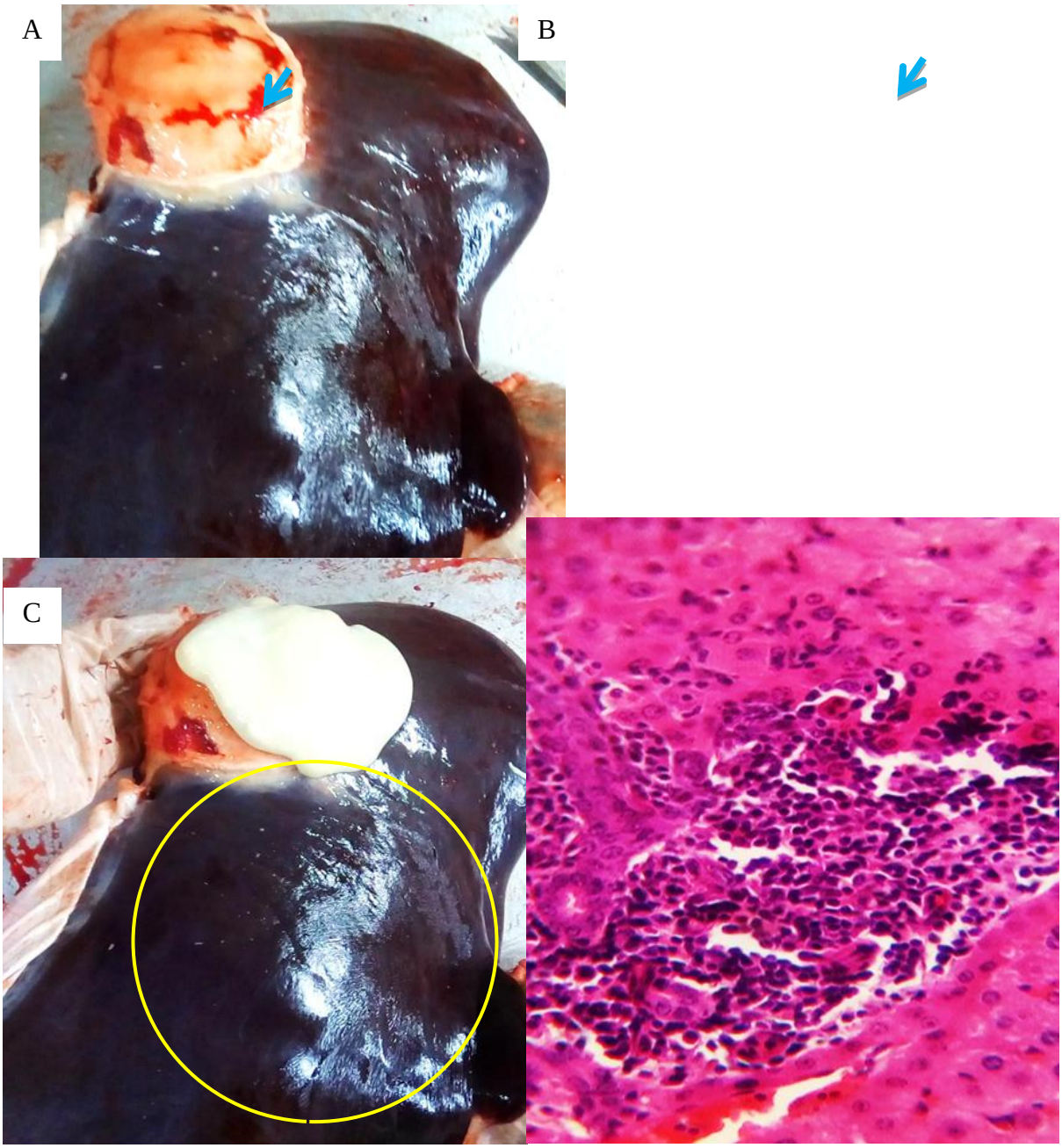



**Figure 43:** Hepatic abscess macroscopic and microscopic changes

(A) Encapsulated swelled area of liver (arrow). (B) Pus oozing from swelled encapsulated area (arrow). (C) Infiltration of inflammatory cells dominated by neutrophils (Circle).

*4.7.10. Eosinophilic hepatitis*

The rate of occurrence eosinophilic hepatitis was 1/541(0/18%). There was small size, multiple pale diffuse areas of necrosis on the left lob of the liver. The liver also had dark hemorrhagic area throughout parenchyma. The capsule was very thin and was unable to detach from the liver easily (Figure: 44 A).

Histologically, all most all hepatocytes were replaced by infiltrated eosinophils (Figure 44 B)

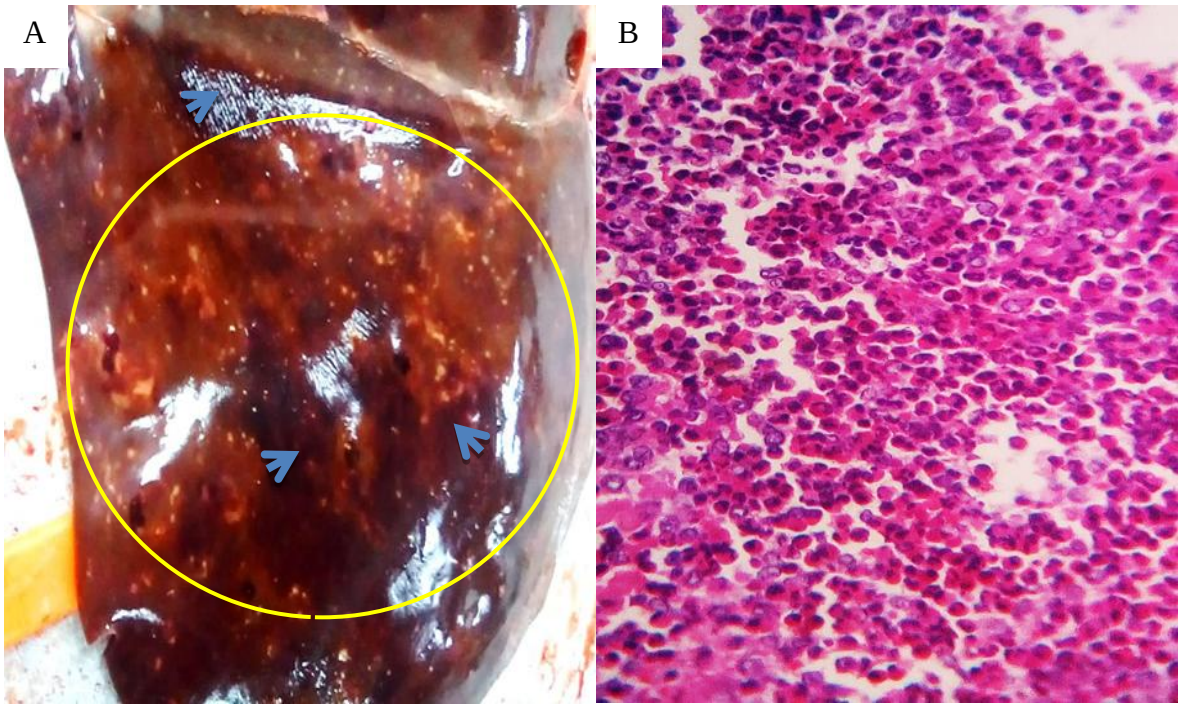



**Figure 44:** Eosinophilic hepatitis cattle, macroscopic and microscopic changes

(A) Multiple pale focal areas on the left lob of the liver (circle) and dark hemorrhagic area throughout the liver parenchyma (arrows). (B) Eosinophils infiltration on liver parenchyma

### 4.8. Pathological Changes Encountered in Liver of Camel

Out of 560 examined camels, only 5(0.89%) had abnormalities in their livers. Of these had 2/560 (40%) calcification and 3/560 (60%) had cyst with fatty change..

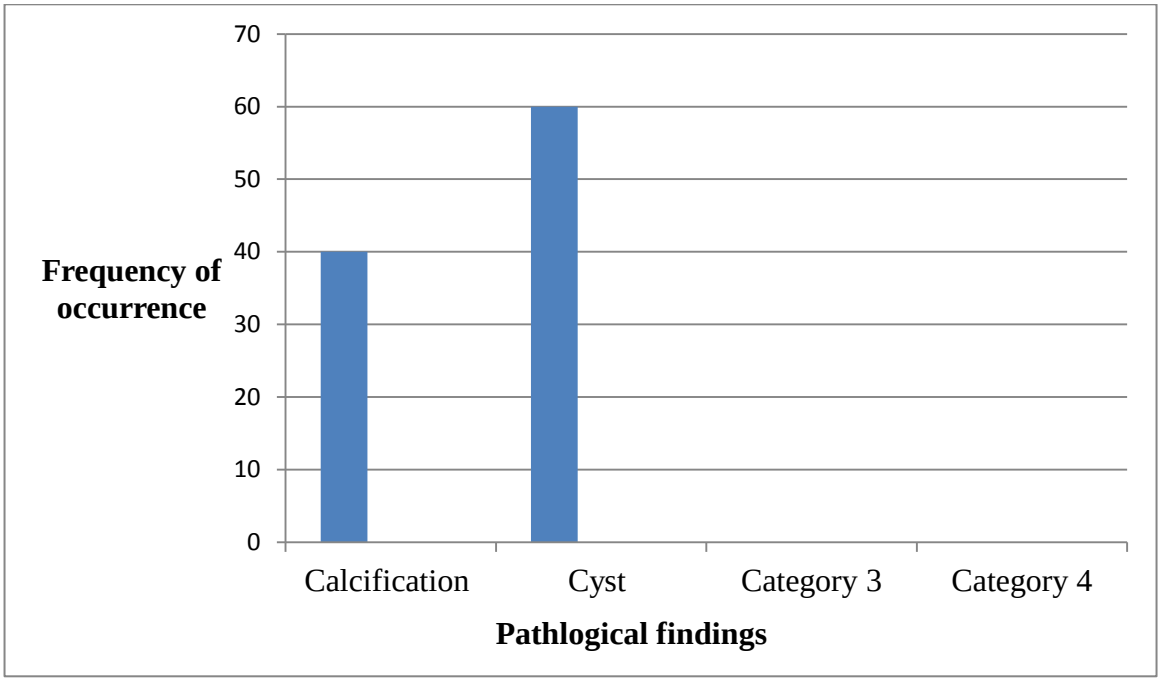
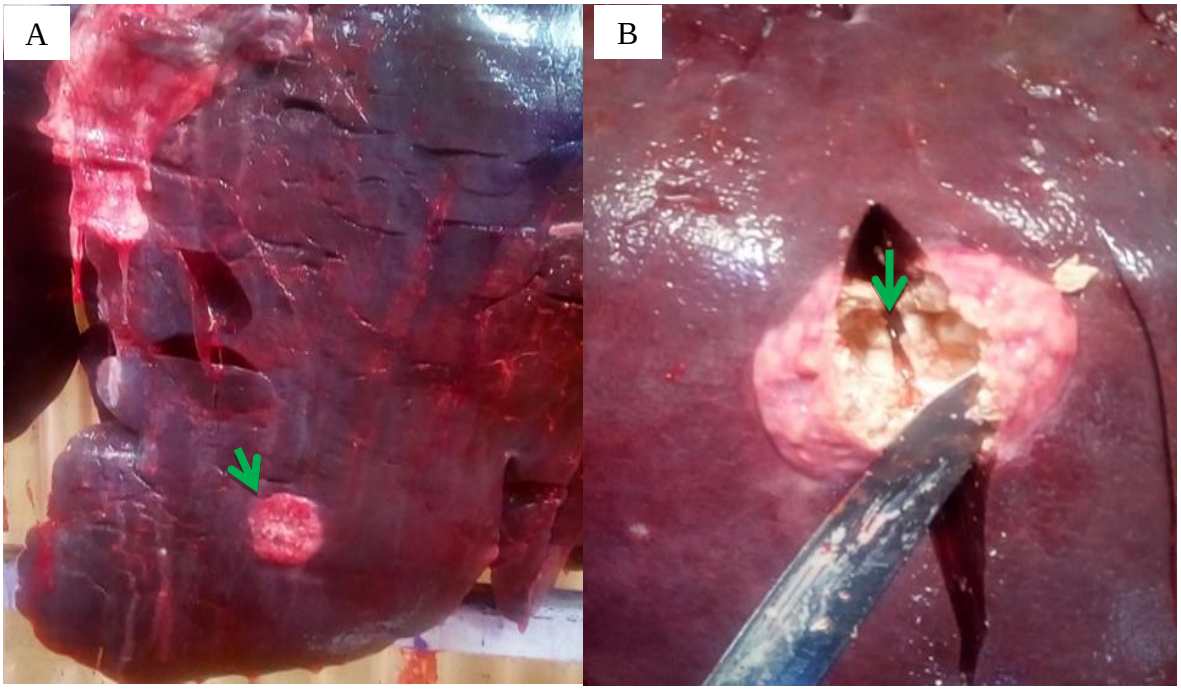



**Figure 45:** Frequency of liver pathological findings in camels

*4.8.1. Calcification*

The rate of occurrence calcification was 2/560 (40%). Grossly, there was about 2 cm in diameter hard, well circumscribed, slightly raised, yellowish, rough area on the quadrate process of the liver (Figure 46 A). Upon incision of this area, there were small sized yellowish calcified materials with turbid fluid inside it (Figure: 46 B)

Histologically, there was deposition of dark purple calcified material in cavity like structure on the parenchyma of the liver (Figure 46 C). Its surrounding tissue and the portal track undergo necrosis with proliferation of bile duct, the capillary were distended and filled with the blood (Figure 46 D). In other part the liver parenchyma there were vacuolation and individualization of the hepatocytes and these parts were separated by thick fibrous septa (Figure 46 E and F).



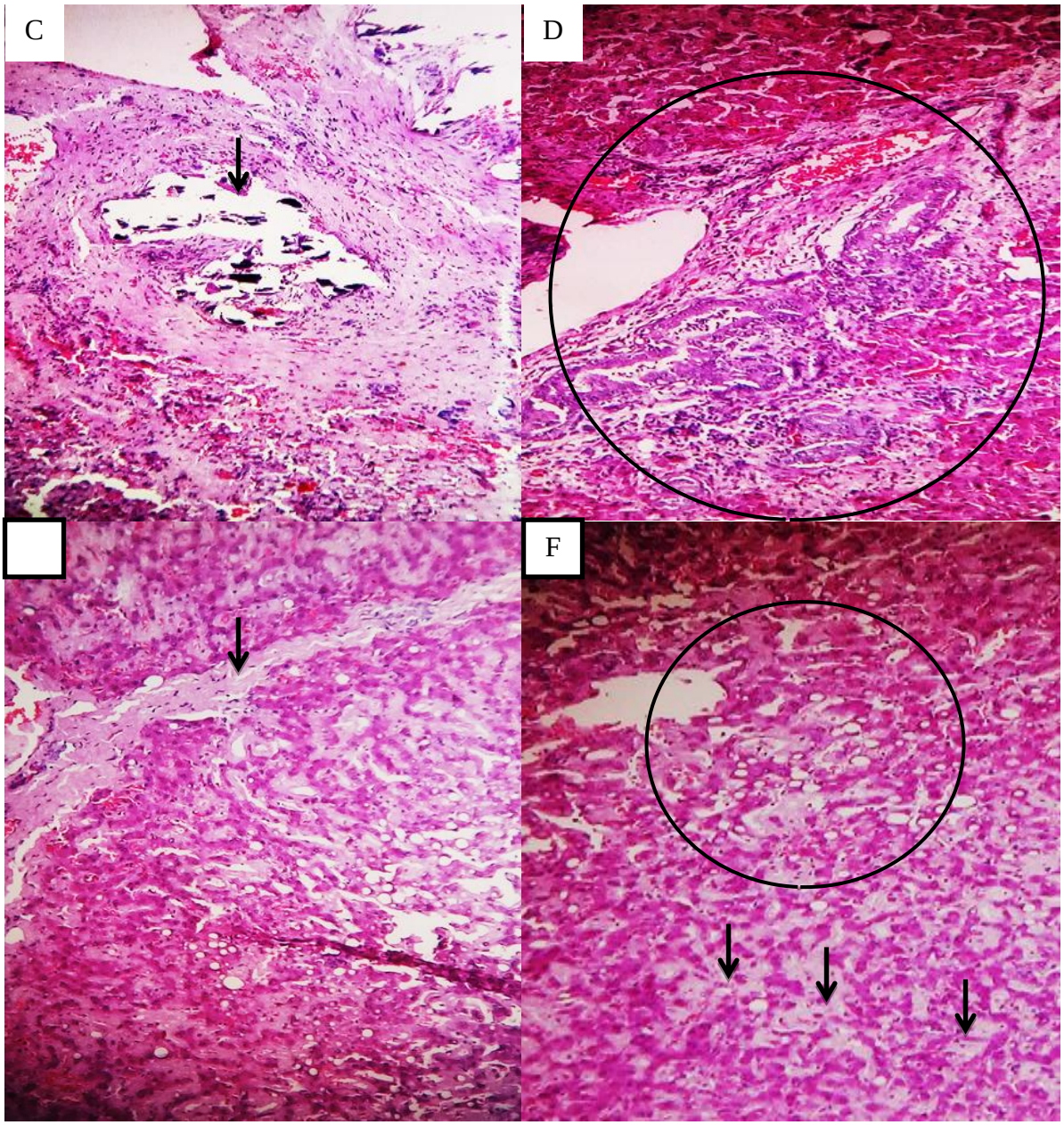


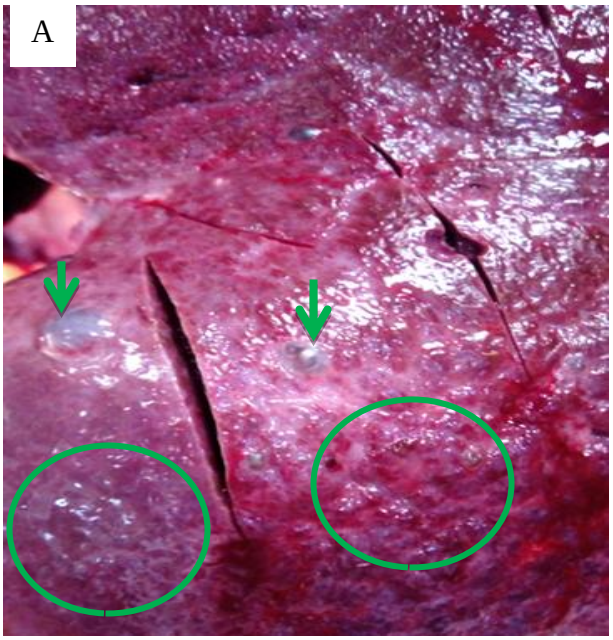

**Figure 46:** Calcification liver camel, macroscopic and microscopic changes

(A) Yellowish, rough area (arrow). (B) Yellowish calcified materials with turbid fluid (arrow). (C) Calcification (arrow). (D) Necrotized area with proliferation of bile duct (arrow). (E) The lobules of liver separated by fibrous septa (arrow). (F) Vacuolation (circle) and individualization of the hepatocytes (arrows).

*4.8.2. Cystic liver with fatty change*

The rate of occurrence cyst with fatty change was 3/560 (60%). Grossly, the liver had variable size of cysts on left lateral lob. The liver had pale and slightly elevated nodular like structure especially on the left lob that had cysts (Figure: 47 A).

Histologically, there were multiple cystic spaces with empty space while others were filled with pinkish fluids and lined by single layer of epithelium supported by variable amount of connective tissue. (Figure 47 B and C). Von Meyenburg complex were on some area of the liver (Figure 47 D). Both macro and micro vesicular
r type of fatty change were seen on liver. On micro vesicular fatty change, the nucleus of the hepatocytes were in the center, but surrounded by fatty droplets. In macro vesicular fatty change the nucleus of the hepatocyte were pushed aside and the vesicles were empty. The fatty lobules were separated by fibrous septal (Figure 47 E).



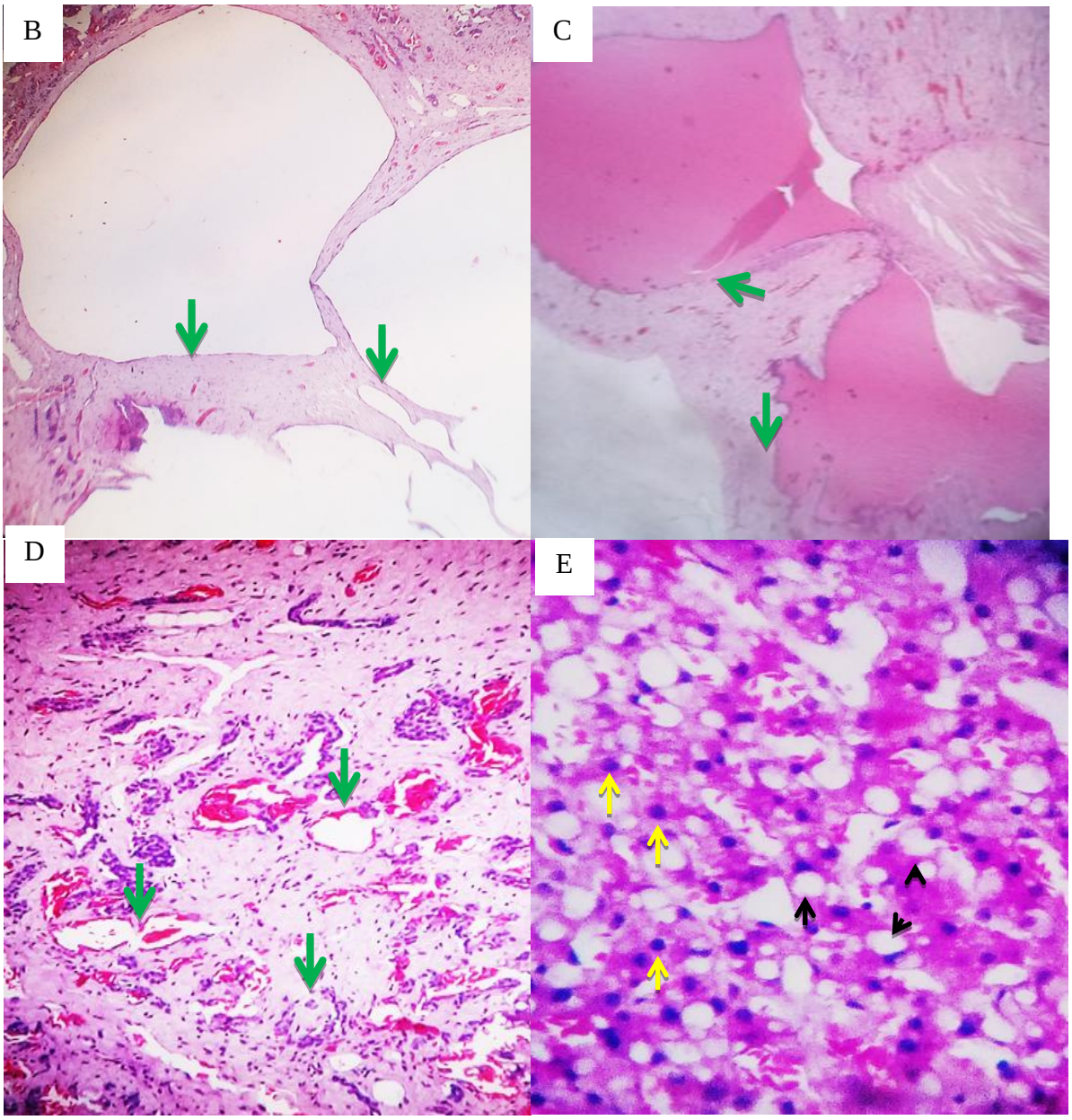



**Figure 47:** Cystic liver with fatty change camel macroscopic and microscopic findings

(A) Variable size of cysts (arrows) and pale and slightly elevated nodular like structure (circle). (B) and (C) Cystic spaces lined by single layer of epithelium (arrows). (D) Von Meyenburg complex (arrows). (E) Micro vesicular (arrow) and Macro vesicular fatty change (arrowhead).

### 4.9 Financial Loss Assessment

During study period out of 3520 cattle examined 820 lungs, 12 hearts, 685 kidneys and 541 were condemned due to varieties of grossly observed lesions. Out of 560 camels examined 1 heart, 2 kidneys and 5 livers were condemned. 322 lungs out of 560 lungs from camels were found to be abnormal, but we did not assess the financial loss from lung of camels because whether the lungs are normal or abnormal, they will be condemned.

The average number of animals slaughtered at Akaki abattoir from 2007 to 2010 Ethiopian calendar was 19,918 for cattle and 2,698 for camels

**Table 1:** Organ condemnation rates and the corresponding annual financial loss in cattle

| **Organs type** | **Amount of condemn-ed organs** | **Average price of each organs** | **Condemnation rate %** | **Annual financial loss in ETB** |
|---|---|---|---|---|
| Lung | 820 | 50 | 23.2 | 152,372.7 |
| Heart | 14 | 50 | 0.39 | 3,386.06 |
| Kidney | 685 | 60 | 19.4 | 112,337.52 |
| Liver | 541 | 200 | 15.3 | 609,490.8 |

**Table 2:** Organ condemnation rates and the corresponding annual financial loss in camel

| Organs type | Amount of condemn-ed organs | Average price of each organs | Condemnation rate % | Annual financial loss in ETB |
|---|---|---|---|---|
| Heart | 1 | 50 | 0.17 | 229.33 |
| Kidney | 2 | 100 | 0.35 | 944.3 |
| Liver | 5 | 700 | 8.9 | 168,085.4 |

The annual financial losses due to organ condemnation from cattle and camels slaughtered at Addis Ababa Akaki abattoir was 877,587.08 ETB and 169309.03 ETB respectively.

## 5. DISCUSSION

The present study revealed the incidence of different abnormality in lungs, hearts, kidneys and livers of cattle and camels.

Out of 3520 examined cattle 23.2% (820/3520) had different pathological changes in their lung

In the current study both cavitary and miliary types of tuberculosis were seen in cattle. In the case of cavitary tuberculosis, caseous materials were seen in the mediastinal lymphnode and in the lung parenchyma with in cavitary structure and histologically there were caseous central necroses accompanied by infiltration of inflammatory cells. According to Timoney *et al.,* 1992; Jones; Hunt; King, 1997), disseminated granulomatous bTB lesions have also been described in the pleural and peritoneal surfaces of bovines. Though, the lesions were more frequently observed in the bronchial, mediastinal, submaxillary and retropharyngeal lymph nodes than in the lung or liver. According to Gupta *et al.* (2016) who reported tuberculosis in cattle, the cut surface revealed caseation, consolidation, and miliary nodules in 10 case each, cavity in 5 cases, and dilated bronchi. The histological evaluation of lung sections revealed necrotizing granulomas and caseous necrosis with few Langhans giant cells. Their findings supported this study.

In this study histologically confirmed granulomatous pneumonia was only found in cattle at rate of 0.12%. Grossly the lesion was characterized by dark red area of streak with dilation of interlobular septa by mucoid like exudates and occlusion of bronchioles with garish to yellowish caseous materials and the mediastinal lymphnode was replaced by caseous to calcified materials. Microscopically granulomatous lesions around the bronchioles were necrotized, edematous and infiltrated with inflammatory cells and there were necrotized and mineralized mediastinal lymphnode. The finding of this study was comparable with the study by Elsiddig (2000) who reported granulomatous pneumonia in cattle characterized by

granulomatous inflammation with central area of necrosis and a zone of cellular infiltration including epithelioid and giant cells and with necrotized lymphnode.

In the current study, both lungs of camels and cattle were found to be affected by hydatidosis The gross findings of hydatidosis in cattle characterized by variable size of fluid filled cysts and the cyst were protruded to the surface of the lung or deep in the lung parenchyma resulting gross enlargement was comparable with the study by Singh *et al.* (2014) who found numerous cysts embedded at different depths of lungs surrounded by outer fibrous layer over the inner germinal layer and filled with clear hydatid fluid. Its microscopic finding was characterized by the presence of three layers of the cyst and infiltrated with macrophage, lymphocytes and eosinophil. These findings were similar with the study reported by Ibrahim and Gameel (2014) who found laminated membranes surrounded by a zone of cellular infiltration in sections of cysts from infected lungs of cattle. In this study microscopic finding of hydatidosis in camel were fibrosised cyst walls and infiltration with mononuclear cells and very similar to those in cattle expect finding of migratory oncosphere in the alveoli duct. This result was comparable with study by Khadidja *et al.* (2014) who reported variable size of hydatid cysts in camel which was characterized by presence of cyst wall and infiltration of inflammatory cells.

In this study CBPP was identified only in one cattle and was manifested macroscopically by fibrinous pleuritis and marbling appearance of the lung and microscopically by markedly expanded interlobular septa with abundant edema, infiltrations of inflammatory cells and cuffs of round cells around arteries. The finding of this study was agreed with results reported by Karam *et al.* (2017) who studied CBPP in Khartoum state during their study they found hepatized lung tissue, widened interlobular septa, extensive loss of airspace, hyperemia of alveolar wall capillaries and infiltration of few fibrin and inflammatory cells.

In this study emphysema of different types were reported both in cattle and camels. Among them bullous/alveolar emphysema and interlobular emphysema were histologically confirmed. In camel bullous emphysemas was characterized by cone shaped air filled area of lung. According to Nourani and Rohani (2009) who reported bullous emphysema in camel they found two foci of air accumulations in pulmonary parenchyma and distained alveoli. Their

findings were similar to the current study. In this study interlobular emphysema was characterized both in cattle and camel by distended interlobular septa. In cattle the distention was due to accumulation of air where as in cattle the distention was due to accumulation of gelatinous fluids in the lung. The finding of the current study agreed with study by Costa *et al.* (2018) studied acute and chronic bovine pulmonary edema and emphysema in Uruguay. According to their study they found lungs which were diffusely distended, dark red color with glistening surface and subpleural emphysema were distended with air blebs distributed throughout the pleural surface. The lobules and interlobular spaces were distended by abundant light yellow and gelatinous material and air blebs.

In this study pulmonary edema was observed only in cattle at rate of 7.8%. According to Hananeh and Ismail (2018) during their study on occurrence of acute bovine pulmonary edema, they found moderately thickened alveolar walls by prominent fibro musculature, edema, and a few inflammatory cells. In this study the pulmonary edema was characterized grossly by prominent interlobular septa with slack tissue because of accumulation of fluids in interlobular septa and microscopically by thickened edematous alveolar wall with infiltration of inflammatory cells and fibrosis and collapsed bronchioles. The current findings were quite similar with their findings.

Different types of hemorrhage were seen in the lung of cattle and camels, but histologically confirmed hemorrhages were two. Alveolar hemorrhages in cattle and Intra-alveolar hemorrhages in camel. The alveolar hemorrhage in cattle in this study grossly characterized by pin pointed diffuse dark spots on all lobs of the lung and microscopically by markedly thickened, distended unbroken alveolar septa. In some area the alveolar septa were broken and alveoli were filled with blood. This finding was supported by Birhan *et al.* (2016) who reported intra alveolar hemorrhage in cattle in Gondar. In their report they found petechial type of haemorrhage on the surface of all part emphysematous lung and blood filled intra alveolar space. In camel the alveolar septal hemorrhage was characterized macroscopically by petechial type of hemorrhage on the apical lobe of the lung and microscopically by widened alveolar septa filled with blood. The current finding supported by the study Bayou *et al.* (2015) who reported intra-alveolar hemorrhage in camel characterized by multifocal and

petechial type of hemorrhage on emphysematous cranial lob of lung and hemorrhage in the intra alveolar space at Akaki abattoir.

In camel the tuberculosis was accompanied by sever suppurative bronchopneumonia in which the lumen of the bronchi filled with inflammatory cells. Grossly the lung was hepatized and had pus and histologically, there were focal area of granulomatous lesions with infiltration of inflammatory cells. According to Ahmed *et al.* (2017) who reported tuberculosis in camel in Sudan they found proliferative granulomatous reaction which was characterized by focal fibrosis and infiltration of mononuclear cells resembled to tuberculous lesion Similarly to Jenberie *et al.* (2016) who reported suppurative bronchopneumonia in camel, we found suppurative bronchopneumonia in camel in which the lumen of the bronchioles were infiltrated with neutrophils and debris. All the above studies by different authors support the current finding of this study.

In this study the pulmonary cryptococcosis was found only in a camel. According to McGill *et al.* (2009) showed evidence of cryptococcosis in cats, 57 dogs, 20 horses, three alpacas, two ferrets and a sheep in their retrospective study in Australia. Another study by Akange *et al.* (2013) showed evidence of cryptococcosis in cattle in Nigeria using serological study. To the best of author knowledge there is no published data on pathological study on pulmonary cryptococcosis of camel dromedarius. However, the study on pulmonary cryptococcosis in sheep reported by Lemos *et al.* (2007) found destruction of the columnar respiratory epithelium architecture associated to suppuration, and the growth of spherical yeast buddings sometimes involved by clear halo produced by unstained capsular substance. Another report by Park *et al.* (2018) who studied cryptococcosis in rats model reported different lesions of lungs in different group of rats which includes infiltrations by inflammatory and yeast cells in damaged alveoli, damaged interalveolar septum and infiltrated by yeast cells in the alveoli and alveolar saccules and thickening interalveolar septum around terminal bronchioles which was caused by inflammation and the infiltration of yeast cells. Their finding were somehow in support of the current study which was characterized distended; rupture alveolar septa contained the cryptococci or the yeast with soapy bubble appearance of cryptococci which had large mucoid or mucinous capsule around bluish round nucleus..

Aspiration pneumonia was only observed in camels at rate of 4.6% with zero occurrences in cattle. Macroscopically the lung was elevated and incision along the trachea revealed the presence of ingesta along the bronchi. Microscopically, the bronchus was edematous, infiltrated with inflammatory cells and food particles were seen in the lumen of bronchi and alveoli. In contrast of this study Jenberie *et al.* (2016) did not find aspiration pneumonia in camel at Addis Ababa Akaki branch instead of other types of pneumonia. According to Abubakar *et al.* (2011) who studied influence of pulmonary lesions on some hematological parameters of camels reported collapsed alveoli and respiratory bronchiole in case of atelectasis. Their findings were similar to current findings.

During this study period atelectasis was only found in camels and grossly was characterized by dark red, smaller and depressed lung, diffused soft raised structure over the surface of the lung with hepatized nature of lung and microscopically, the bronchi and bronchioles were collapsed and filled with blood and surrounded by inflammatory cells and hyperplastic and slightly collapsed pulmonary arteries, collapsed alveoli and thickened and haemorrhagic alveolar septa. Similar findings were reported in camel by Abubakar *et al.* (2011) who found narrow respiratory bronchiole, thickened interaveolar septae, congested capillaries, inflammatory exudates and cellular infiltration.

Out of Out of 3520 cattle that we examined 0.39% (14/3520) cattle had with different heart abnormalities. The findings were serous cysts, calcification, sarcocystis and pericarditis during our study period.

In this study serous cysts were found only in cattle. They were characterized grossly by rounded cyst that had thin wall filled with serious fluid and microscopically by edematous cystic wall which were infiltrated with plasma cells, lymphocytes and macrophages. In contrast of this study Shekarforoush *et al.* (2006) and Marcato *et al.* (1996) reported blood cyst in cattle, but the microscopic and macroscopic characterizations of their finding on serous cysts were in support of this study findings. Their findings were characterized grossly by roundish or oval cyst protruding above the wall of heart with thin cyst wall.

In this study yellowish calcified materials were seen on lung. Histologically, these calcified materials were seen as purple on the endocardial area. According to Ferrans and Vleet (1985) myocardial lesions in selenium and vitamin E deficient animals were seen most frequently in calves, lambs, pigs, turkey poults, and ducklings. The lesions show hyaline necrosis, with or without calcific deposits, macrophagic invasion, and eventual stromal collapse and fibrosis. According to Capen *et al.* (1966) experimental hypervitaminosis D in cow produced sever mineralization in cardiac muscle and other component of heart. According to Abunna and Hordofa (2013) and Tilahun (2017) calcification of heart were seen in cattle during postmortem examination. All the above studies support the current study.

In the present study sarcocystis in the heart were only found in cattle. Grossly the heart had glistening, small sized, yellowish, semi soft consistency, rounded structure on the surface of right ventricle of the heart and microscopically characterized by oval shaped sarcocyst with basophilic bodies surrounded by thin outer wall were seen on myocardium of the heart. The result was supported by study Choi *et al.* (2018) who reported sarcocystis in the cardiac tissue of the Korean native cattle. The cysts had oval to spherical shape. The sarcoplasms of the myocardia presented with numerous bradyzoites enclosed in the thin and smooth host cell wall. Valinezhad *et al.* (2008) reported sarcocystis in camel which was characterized by thin-walled type and infiltrated with different type of inflammatory cells. In contrast of current study, sarcocystis were not found in camel this may be due to limited sample of hearts from camels.

Pericarditis was seen both in camel and cattle, but in camel the pericarditis were accompanied by cysticercosis. Grossly, the cysticercosis were characterized by circular, small size, white hard nodular like structure and, microscopically by the presence of cyst in the myocardium and egg within reproductive track of female nematode and the cyst wall was infiltrated with inflammatory cells. According to Calla *et al.* (2016) who study cysticercosis on pig they found mild to moderate degree of inflammation composed of eosinophils, macrophages, lymphocytes, plasmocytes, and fibroblasts with connective tissue in the surrounding parenchyma. According to Schuster *et al.* (2015) who study cysticercosis in dromedary calf

liver they found the metacestode surrounded by a host-derived capsule. Bothe studies were support the current study to some extent.

Out of 3520 kidney of cattle examine, 19.4% (685/3520) cattle had different kidneys abnormalities. The abnormalities found during the study were cysts hemorrhage, atrophy, nephritis, urolithiais and hypertrophy. The highest findings were cysts.

In this study histologically confirmed collecting duct and medullary cysts were seen in cattle. Grossly collecting duct cyst fitted between the surrounding lobules of the kidney and had very thin wall, filled with brownish fluid and microscopically, cysts were found on medullary area and the walls were lined by cuboidal type of cells and Grossly glomerular cysts were found on the cortex characterized by multiple cysts involved the glomerulus and microscopically the cystic glomerulus were lined by single cell layer of epithelium and the interstitial tissue were fibrosised and infiltrated with inflammatory cells. Tavassoly (2003) reported kidney cyst in bovine which was characterized by variably sized spherical, thin-walled cysts and lined by flattened epithelium, and contained clear watery fluid. Another study in human being by Gupta *et al.* (2007) who reported dilated bowman's space with rudimentary glomerular capillary tufts and the cyst was lined by flattened epithelial cells in their study on glomerulocystic kidney disease. According to Ibrahim, *et al.* (2008) in their study, kidney of camel showed cystic dilatation of the renal tubules, proliferation of the fibrous connective tissue. All these studies supported the current findings.

In this study histologically confirmed tubulointerstitial nephritis was found in one cattle. In camel the tubulointerstitial nephrites was seen with pyelonephritis. Grossly, the tubulointerstitial nephritis was characterized by hypertrophied and atrophied tubule filled with eosinophilic fluid. The interstitial tissues were necrotized and infiltrated with lymphocytes and there were few glomerular scleroses. This study supported by Mahouz *et al.* (2105) who study renal disease in sheep and cattle they found infiltration of eosinophils and lymphocytes in the interstitium, presence of small and inflamed glomerulus and necrotized tubules.

Urolithiasis were found only in cattle, but not seen in camel. The gross findings of urolithiais in this study were accumulation of stone like material the minor calyx of kidney and microscopically by dispersed minerals depositions on the interstitial spaces of medulla. Distended and hyperemic medullary tubules filled with basophilic minerals in their lumen. According to Oryan *et al.* (2015) who studied nephrolithiasis among slaughtered cow in Iran they found numerous, soft, yellow sand-like calculi and multiple, gray calculi with hard consistency localized in the renal pelvis. Site of finding of the stone in in their study and current study was different this may be due to formation of stone begin as depositions of calcium phosphate in the interstitium, grow outwards reaching the renal papillary surface (Khan *et al.,* 2016). Study by Ozmen (2004) who studied Non-obstructive Urolithiasis in cattle found stones usually localized in the calyx and pelvis renalis. These stone were either solitary or multiple. Microscopically there were basophilic calcium deposits in the lumen of the tubules. Cystic tubules were commonly observed in this study. Both study supported the current study with minor difference.

Unlike kidney of cattle, kidneys abscesses were only found in camel. In contrast of this study by Mohammed (2010) reported abscess in the lung, heart and lymphnodes of camel, but not in kidney. Another study by Tenaw *et al.* (2015) at same study area with this study did not report abscess in kidney of camel this may be due to rareness of occurrence of kidney abscess in camel. According to Tharwat *et al.* (2018) renal abscess are common renal diseases in ruminants, especially in cattle but rarely reported in camels. In this study the kidney with abscesses macroscopically was characterized by pus on medullary region and microscopically the interstitium of the kidney were infiltrated with neutrophils, plasma cell and macrophage.

Pyelonephritis was found only in camel in which grossly it was characterized by enlarged kidney due to hump like swelling inside the kidney and accumulation fluid that had sandy and milky with nature and microscopically the tubules had cystic nature contained hyaline cast. The interstitium were heavily infiltrated with lymphocyte and macrophages. According to Tharwat *et al.* (2018) who reported pyelonephritis in camel found the kidney that showed, pus evacuated from the affected left kidney. According to Karimi *et al*. (2006) there was neutrophil infiltration in the renal pelvis, urinary tubules and interstitial connective tissue with

tubular epithelial cells necrosis and tubular destruction. Both study were support the current findings.

Out of 3520 cattle examined 541(15.3%) cattle had different pathological changes in their livers These pathological changes were fasciolosis, hydatidosis, abscess, focal nodular hyperplasia, primary sclerosing cholangitis, solitary fibrous tumor, micro cirrhosis, biliary adenoma, cholangiocarcinoma and eosinophilic hepatitis.

Focal nodular hyperplasia is a benign condition of the liver (Hsee *et al.,* 2005). In this study grossly the liver with FNH had two solitary nodular masses on parietal surface of liver and microscopically there were nodular of hepatocytes infiltrated with lymphocytes. The nodules of the hepatocytes were separated by thick fibroses septa which had markedly proliferated bile duct, dilated portal veins and fibrosis of tissue surrounding the portal track. To the best of the author knowledge there is no published data on FNH in cattle, but there are few researches on dogs and macaques. There are several studies in humans. However, the findings of the current study can be comparable to some extent with the finding of Xavier *et al.* (1992) who studied hepatocellular adenoma and focal nodular hyperplasia of the liver: differential diagnosis in dogs. In their study they found biliary ducts proliferation and vacuolar degeneration in the hepatocytes.

In this study sclerosing cholangitis occurred in cattle but not in camel. To best of the author knowledge there is no work done in cattle regarding to SC. Several researches have been done on human about SC. The characteristic finding SC is concentric "onion-skin" fibrosis surrounding the bile ducts (Worthington and Chapman 2006). In this study there were multifocal distributions of intrahepatic bile duct which had onion skin fibrosis with bile plug inside the lumen of bile ducts and infiltrated with few inflammatory cells.

Solitary fibrous tumor extensively studied in human beings but not in other animals. Histologically features of the lesion showed the tumor was composed of small spindle cells, variably admixed with fiber texture (Liu *et al.,* 2013). In this study grossly, the liver was enlarged, had rounded edge and lack proper lobation. There was mass near to the papillary lob on visceral surface of liver. Histologically, the tumor had heavily collagenized stroma background. The cell had ovoid to spindle shape with eosinophil cytoplasm. Degrees of cellularity vary from on area to another area and in less cellular area the intercellular collagen is thick. To best of author knowledge there was not research on solitary fibrous tumor in any of animals, but according to Dey *et al.* (2016) who study solitary fibrous tumor in human, reported the tumor that had low to moderate cellularity comprised of spindle to fibroblast like cells in a collagenous background The current findings were closely related to their findings.

Bile duct adenomas are benign proliferations of intrahepatic bile ducts. These lesions are usually found under the liver capsule (Johannesen *et al.,* 2014). In this study bile duct adenoma was found in cattle and grossly it was characterized by enlarged papillary lob with irregular protruding structures to the capsule of liver and microscopically by proliferated bile duct with well circumscribed area by fibrosis tissue of liver. There were large to small proliferated bile ducts with cuboidal cells combined with inflammatory cells infiltration. Bile duct adenomas were extensively studied in human being (Johannesen *et al.,* 2014; Chen *et al.,* 2014; Chuy *et al.,* 2018), but not in any of farm, pet or wild animals.

Despite the absence of hepatic hydatidosis in camels in this study, there were several hydatidosis in cattle. In this study grossly it was characterized by different sizes, clear fluid filled cysts. Some of the cyst had yellowish and thick fibrous capsule with calcified material inside them other had thin and white to slightly yellowish capsule. Histologically the parenchyma of the liver surround the cyst wall were infiltrated with eosinophil and mononuclear cells and on some parts of the liver, there were depositions of calcified materials According to Se´adawy and AlKaled (2012) who reported hydatidosis in liver of cattle, the liver had thickened fibrous layer and sever necrosis with eosinophilia and extensive calcification along hepatic tissue. The current study comparable with their finding except the extent of calcification was not sever as their finding.

Histologically confirmed cholangiocarcinoma during this period of study found only in cattle, but not in camel this may be due to absence of sufficient moisture for the aquatic intermediate host (mollusc) to live. According to Sisay and Awoke (2015) majority of camels in Ethiopia are found in the drier areas of the Eastern part of the country. In human imaging suggesting cholangiocarcinoma was associated with fasciola and able to extracted F. hepatica with endoscopic retrograde cholangiography (Alizadeh *et al.,* 2011). Bovine and ovine cholangiocarcinoma occurred in animals suffering from chronic fasciolosis (Anderso and Sandison 1968). In this study CC were characterized grossly by gray to reddish area with pale parenchyma of the liver, widened bile duct and mild infestation of fasciola. Microscopically it was characterized by infiltration of liver parenchyma with tubular to papillary glandular like structure of bile duct lined by cuboidal type of epithelial cells with variable fibrous stroma. There were scattered small nest of tumor cells. According to Ohfuji (2012) they found adenocarcinomatous tubular structures of CC surrounded by an abundant fibrous connective tissue stroma in liver of cow. Another study by (Azizi *et al.,* 2016) found irregular, gland-like, or tubular pattern of neoplastic epithelial cells embedded in abundant connective tissue stroma in liver of a 6-year-old female Holstein cow from the slaughterhouse. The findings from both studies were similar to our findings.

Fasciolosis was the highest infection in this study in which half of the cattle had fasciolosis, but camels did not affected by fasciolosis. In contrast of this study, study on the prevalence and pathology of fasciola *spp*. in dromedaries of Iran, by Eslami *et al.* (2003) examined the livers of 409 slaughtered camels and found that 5.3% of animal harbored *F. hepatica.* Most of the camels in Ethiopia are from arid area. This difference may be due to absence of sufficient moisture in arid area for the aquatic intermediate host (mollusc) to live. Because the presence of streams, wetlands and pastures on farms showed a significant association with the presence of fasciolosis in cattle herds (Olsen *et al.,* 2015). In this study the livers with fasciolosis were enlarged, up on cut the liver had rust colored materials and the bile duct harbored immature and mature fasciola. Histologically, there were extensively hemorrhagic, fibrosis portal track infiltrated with inflammatory cells, mild calcification, metastasized of bile duct in to mucus producing like cells with presence of adult parasites. This finding of this study supported by

several studies on fasciolosis in different part of the world that reported similar findings with this study (Adrien *et al.*, 3013; Kalu *et al.,* 2015; Quevedo *et al*., 2018).

Out of 560 camels' liver examined, 8.9% (5/560) had different abnormalities on their liver. These were calcification and cyst with fatty changes.

Histologically confirm calcification was only found in liver of camel and grossly it was characterized by well circumscribed and slightly raised area on the quadrate process of the liver and had turbid fluid inside it and microscopically purple color calcified material were seen in cavity like structure on liver. There was necrotized area and capillary was distended with blood. There was also vacuolation and individualization of the hepatocytes. According to Tenaw *et al.* (2015) about 1.5 % of liver were condemned in camel at same study area with this study was due to calcification.

Cystic liver with fatty change were seen in one camel. Histologically this liver was characterized by multiple cystic spaces lined by single layer of epithelium. Von Meyenburg complex were on some area of the liver. The micro vesicular fatty change was characterized by presence of nucleus of the hepatocytes in the center, but surrounded by fatty droplets. Whereas macro vesicular fatty change the nucleus of the hepatocyte were pushed aside and the vesicles were empty. According to Jamshidi and Zahedi (2014) they found micro vesicular fatty liver in 2.5% of slaughtered camel in Iran and were characterized by Intracellular vaccules formation and centrally located nuclei.

In this study 877,587.08 ETB were lost as the result of condemnation of livers, lungs, kidneys and hearts from cattle. According to Belina and Melese (2017) on their study at Aleta Wondo abattoir the financial loss as the result of organs condemnation in cattle was 840,649.84 ETB. Their result was comparable to this study. In camel 169,309.03 ETB lost due to condemnation of heart, kidneys and livers. According to Berhanu *et al.* (2015) who studied pathological causes and financial loss of camel organ at Akaki abattoir the financial loss was 320,760 ETB from 269 camels. Another study by Lemma *et al.* (2016) the financial loss was 399,060 ETB from 305 camels. Both study showed greater amount of financial loss than this study. The

reason for this may be due to the inclusion of assessment of financial loss of lungs in Berhanu *et al.,* (2015) which were not include in this study and according to the worker at Akaki abattoir the number of condemn organs from camel is decreasing.

## 6. CONCLUSION AND RECOMMENDATIONS

Different pathological findings were observed in lung, heart, kidney and liver of cattle and camels regardless of their age, sex, breed and body conditions. Comparing to other organs the lung of cattle and camels were found to be affected by common abnormalities. The liver, kidney and heart of these two species had completely different abnormalities. Tuberculosis, emphysema, pulmonary hydatidosis, bronchopneumonia and hemorrhage were found in the lung of both species. While granulomatous pneumonia, edema and CBPP found only in cattle and atelectasis, cryptococcosis and aspiration pneumonia only found in camel. Pericarditis was found in both species, but serous cyst, calcification and sarcocystis found only in cattle. On another hand cysticercosis was observed only in camel's heart. Among kidney lesions, cyst, atrophy, hypertrophy, nephritis, urolithiais and hemorrhage were seen in cattle. These lesions were not detected in kidney of camels. Abscess and pyelonephritis were the only abnormalities seen in kidney of camel. Focal nodular hyperplasia, sclerosing cholangitis, solitary fibrous tumor, micro cirrhosis, biliary adenoma, hydatidosis, cholangiocarcinoma, fasciolosis, abscess, and eosinophilic hepatitis were seen in the liver of cattle however calcification and cystic liver with fatty change were found in camel. In this study we did not find any similarities in affection of liver between these two species. The total financial loss calculated in this study due to organ condemnation was 1,046,896.11 ETB per year.

Based on the above conclusion the following recommendations are forwarded

- ✓ Further study should be conducted by including risk factors
- ✓ Etiological causes of the lesions on the organs observed at the abattoir should be identified
- ✓ Investigation on the epidemiology of the lesions causing diseases should further be conducted

## 7. REFERENCES


Abdelaal A., Gouda S., Abegaz S. and Mohammode A. (2018): Crossectional Study on the Prevalence and Economic Significance of Hydatidosis in Slaughtered Ruminants. *Biomed J Sci & Tech Res* **3(3):** 3273-3281

Abera Z., Mengistu D., Batu G. and Wakgari M. (2016): Review on Contagious Bovine Pleuropneumonia and its Economic impacts. *Academic Journal of Animal Diseases* **5(1):** 01-15.

Abubakar M.S., Fatihu M.Y., Ibrahim N.D.G. and Oladele S.B. (2011): Influence of Pulmonary lesions on Some Haematological Parameters of Camels (Camelus dromedarius) in Northwestern Nigeria Sokoto Journal of Veterinary Sciences **9(1):**1-6.

Abubakar M.S., Fatihu M.Y., Ibrahim N.D.G., Oladele S.B. and Abubakar M.B. (2010): Camel pneumonia in Nigeria: Epidemiology and bacterial flora in normal and diseased lung *African Journal of Microbiology Research* **4(23).** 2479-2483.

Abunna F. and Hordofa D. (2013): Major Causes of Organ Condemnation for Cattle and its Financial Impact at Wolaita Soddo Municipality Abattoir, Southern Ethiopia Global Veterinaria **11 (6):** 730-734

Addis Ababa Chamber of Commerce and Sectoral Associations (AACCSA) BCaD Consulting Management PLC 2015

Admassu B., Shite A. and Molla W. (2015): Contagious Bovine Pleuropneumonia in Ethiopia (Review Article) Academic Journal of Animal Diseases **4(2):** 87-103, 2015

Adrien M.L., Schild A.L., Pereira C.M., Fiss L., Ruas J.L., Grecco F.B. and Raffi M.B. (2015): Acute fasciolosis in cattle in southern Brazil1 *Brazilian journal of veterinary research.* **33(6):**705-709

Ahmad I., Kudi C., Magaji A., Yakubu Y., Salisu M., Shuaibu S. and Daninna Z. (2019): Disseminated tuberculosis in a cow and a dromedary bull-camel in Zamfara State in Nigeria. *Veterinary Medicine and Science* **5(1):** 93-98

Ahmed E.T., Hassan A.B. and Abakar A.D. (2017): Pathology of Tuberculosis in Camels (Camelus dromedaries) in the Sudan Journal of Tuberculosis Research 0**5(01):** 77-80

Akange E.N., Kwanashie C.N., Bisalla M., Useh N.M. Ngbede E.O . (2013): Evidence of Cryptococcosis in cattle in Zaria Kaduna state, Nigeria *Vet World* **6(2):** 64-67

Al Se´adawy M.A.H. and AlKaled M. J.A. (2015): Gross and histological comparison of hydatid cyst infection in livers of sheep and cows *Journal of Vet.Med.Sci*. **11(2):** 94-101

Alizadhi A.H.M., Roshani M., Lahmi F., Davoodi N.A., Nejad M.R., Seyyedmajidi M.R. and Zali M.R. (2011): cholangiocarcinoma in magnetic resonance cholangiopancreato-graphy and Fascioliasis in Endoscopic Ultrasonography Case Rep Gastroenterol **5(3):** 569 -577

Alsari A., Hassanin A., Elshiekh A.A., Altayb A.I., Mohammed I.I., Mohammed M.A. and Jomaa O.F. (2017): A Pathological Study of Lesions in Sheep Liver in Nasr Aldeen. A Dissertation submitted for partial fulfillment of the requirements of B.V.M. Honors Degree in Veterinary Medicine

Ameni A., Vordermeier M., Firdessa R., Aseffa A., Hewinson G., Gordon S. and Berg S. (2011): Mycobacterium tuberculosis infection in grazing cattle in central Ethiopia. *The Veterinary Journal* **188 (2011):** 359–361

Anderso L. and Sandison A.T. (1968): Tumors Of The Liver In Cattle, Sheep And Pigs **21(2):** 289-301

Arjmand J., Hajipour N., Khojasteh S., Baran A., Mirshekar F., Ghashghaei O., Samiei K. and Faramarzpour A. (2015): Prevalence, Macroscopic and Microscopic Lesions of Bovine Fasciolosis at Ahvaz Abattoir, Khozestan Province, Iran. *Biological Forum - An International Journal* **7(1):** 539-545

Aslan O., Cakir L., Bekdik, I.K., Dogan, Z. and Tunc, A.S. (2014): Cholangiocarcinoma of intrahepatic bile ducts with disseminated metastases in a Siamese cat: a case report *Veterinarni Medicina* **59(7):** 359–367

Athar H., Parra J.D., Moulvi B.A., Singh M. and Dedmari F.H. (2012): Pericarditis in Bovines- A Review *International Journal of Advanced Veterinary Science and Technology*, **1(1):**19-27

Azizi S., Kheirandiah R. and Sami M. (2016): Slaughterhouse report of intrahepatic cholangiocarcinoma in a Holstein *cow Comp Clin Pathol* **25(6):** 1321–1324

Bayou K., Dawo F., Demessie T., Abie G., Almaw G. and Feyisa F. (2016): Pathological and Bacteriological Studies with Viral Detection from Pulmonary Lesions of Dromedary Camels Slaughtered at Akaki Abattoir, Ethiopia *Global Veterinaria* **17(4):** 325-334

Belina D. and Melese M. (2017): Pathological lesion survey and financial losses associated with organs and carcass condemnation in cattle slaughtered at selected abattoirs in Ethiopia. *Indian J. Vet. Pathol*., **41(1) :** 1-11.

Belina D., Giro B., Ashenafi H., Demissie T. and Muktar Y. (2015): Review on camel liver pathology and its major diagnostic approaches *Global Journal of Veterinary Medicine and Research* **3(1):** 068-079

Berhanu N., Tefera M., Sibhat B. and Kebede N. (2015): Pathological Causes and Financial Loss of Camel Organ andCarcass Condemnation at Addis Ababa Abattoirs Enterprise, Ethiopia *European Journal of Biological Sciences* **7 (2):** 78-84 2015

Birhan G, Mersha C, Tesfaye S, and Yesuf M. (2016). Study on the Pathological Lesions and Bacterial Isolates of Bovine Lungs in Gondar Elfora Abattoir, North West Ethiopia 96p.

Böttner A., Charleston W.A.G. and Hopcroft D. (1987): The structure and identity of macroscopically visible Sarcocystis cysts in cattle. *Vet. Parasitol*. **24:** 35-45.

Braun U. (2009): Traumatic pericarditis in cattle: clinical, radiographic and ultrasonographic findings. *Vet J* **182 (2):** 176-86.

Britton A.P. and Zabek E.N. (2012): Bronchopneumonia in two dairy calves associated with Mannheimia species cluster V infection *Journal of Veterinary Diagnostic Investigation* **24(6):** 1043–1046

Brown C., 2012. A Field Manual for Collection of Specimens to Enhance Diagnosis of Animal Diseases University of Georgia Department of Veterinary Pathology Boca Publications Group Inc, Canada

Buczinski S., Francoz D., Fecteau G. and DiFruscia R. (2010): Heart disease in cattle with clinical signs of heart failure: 59 cases *Vet J* **51:**1123–1129

Buczinski S., Rezakhani A. and Boerboom D. (2010): Heart disease in cattle: Diagnosis, therapeutic approaches and prognosis *The Veterinary Journal* **184 (3):** 258–263

Calla AV., Puerta L.A.G., Calcina J., Viera O.S., Gavidia C., Urbina M.T.L., Garcia H.H. Gonzalez A.E. (2016): Evaluation of activity of triclabendazole against Taenia solium

metacestode in naturally infected pigs *Asian Pacific Journal of Tropical Medicine* **9(1):** 23–26

Capen C.C, Cole C.R. and Hibbs J.W. (1966): The Pathology of Hypervitaminosis D in Cattle *Path. vet*. **3(4):** 350-378

Chakraborty S., Kuma A., Rahal A., Malik Y., Dhama K., Pal A. and Prasad M. (2014): Advances in Diagnosis of Respiratory Diseases of Small Ruminants. *Veterinary Medicine International* 1-16

Che L., Xu M.Y., and Feng Chen F. (2014): Bile duct adenoma: a case report and literature review *World Journal of Surgical Oncology* **12(125):** 2-7

Choi D.K.T., Hong E.J., Ryu S.I., Sim C., Chae J.S., Kim H.C., Park J., Choi K.S., Yu D.H., Yoo J.G. and Park G.K. (2018): Detection and Identification of Sarcocystis cruzi (Protozoa: Apicomplexa) by Molecular and Ultrastructural Studies in Naturally Infected Korean Cattle (Bos taurus coreanae) *Korean J Parasitol* **56(2):** 121-127

Chuy J.A., Garg I., Graham R.P., VanBuren W.M. and Venkatesh S.K. (2018): Imaging features of bile duct adenoma: case series and review of literature. *Diagn Interv Radiol* **24(5):** 249-254.

Costa R.A., Schild C., Silveira C.S., Melissa Macías-Rioseco M.M., Mirazo S., Maya L., Clariget J. and Correa F.R (2018): Acute and chronic bovine pulmonary edema and emphysema in Uruguay *Pesq. Vet. Bras*. **38(10):**1929-1934

Cullen J.M. and Winkle T.J.V. (2007): Pathology of the liver: What's new and what's still true. The California Davis Foundation and the American College of Veterinary Pathologists Present

Dar J.S., Tak I.U.R., Ganai B.A., Shahardar R.A. and Gazanfar K. (2018): Gross pathological and Histopathological changes in theliver and Bile duct of Sheep with acute and chronic fasciolosis. International journal of advnce research in science and engineering **7(4):** 2031-2044

Dey B., Gochhait D., Kaushal G., Barwad A. and Pottakkat B. (2016): Solitary fibrous tumor of the liver: a rare tumor in a rarer location **8(3):**137-138

Dore E., Fecteau G., He´lie P. and Francoz D. (2007): Liver Abscesses in Holstein Dairy Cattle. *J Vet Intern Med.* **21(4):**853-856

Duggan M. and Kavanagh B.P. (2005): Pulmonary atelectasis: a pathogenic perioperative entity. **102(4):**838-54.

Eaton J.E., Talwalkar J.A., Lazaridis K.N., Gores G.J. and Lindor K.D. Gastroenterology (2013): Pathogenesis of Primary Sclerosing Cholangitis and Advances in Diagnosis and Management **2014(145)** 521–536

Elhiblu M.A., Dua K., Mohindroo J., Mahajan S.K., Sood N.K and Dhaliwal P.S. (2015): Clinico-hemato-biochemical profile of dogs with liver cirrhosis *Veterinary World* ***8(4):****487-491*

Waithanji, E., Mtimet, N. and Muindi, P. (2015): Delivery of the Contagious Bovine Pleuropneumonia (CBPP) vaccine in northeastern Kenya. International Livestock Research Institute Discussion paper 31. Nairobi, Kenya

El-mahdy M.M., Bakeer A.M. and Al Jumah M.M. (2013): Pathological Studies on the Respiratory System Affections in Saudi Arabia *Camels Egypt. J. Comp. Path &Clinic Path* **26 (1):** 101- 129

Elsiddig A.M. (2000). Pathological Changes in the Lungs of Slaughtered Sheep and Cattle in Khartoum State Abattoir Athesis submitted to the University of Khartoum in partial fulfillment for the requirements of the degree of M.Sc. of Veterinary Medicine

Elsiddig I. and Elsiddig A.M. (2003): Pathological Changes in the Lungs of Slaughtered Sheep and Cattle in Khartoum State Abattoirs A thesis submitted to the University of Khartoum in partial fulfillment for the requirements of the degree of M.Sc. of Veterinary Medicine

Erbet K., Zewde G. and Kumsa B. (2010): Hydatidosis of sheep and goats slaughtered at Addis Ababa Abattoir: Prevalence and risk factors. *Trop. Anim. Health Prod*. **42(5):**803-805

Ertan O.R.U. (2006). The Pathologic and Bacteriologic Comparison of Pneumonia in Lambs *Turkish Journal of Veterinary and Animal Science,* **30(6):**593-599

Eslami A., Bahadori S.B., Eskandari A. and Sedaghat R. (2003): Study on the prevalence and pathology of Fasciola in camels (Camelus dromedarius) of Iran. *Journal of Faculty of Veterinary Medicine University of Tehran,* **58(2):** 97–100,

Esposito I. and Schirmacher P. (2008): Pathological aspects of cholangiocarcinoma. Review article *Hepato Pancreato Biliary associatiom* **10(2):** 83–86

FADPReP (The Foreign Animal Disease Preparedness and Response Plan). (2017). Contagious bovine pleuropneumonia standard operating procedures: overview of etiology and ecology. Standard Operating Procedures (SOPs) provide operational guidance for responding to an animal health emergency in the United States.

Fairchild J. (2019): Hepatic Abscesses in Feedlot Cattle. Winter 2000 Newsletter https://www.addl.purdue.edu/newsletters/2000/winter/hafc.shtml

FAO, (Food and Agriculture Organization of the United Nations). (2007): "Manual on meat inspection for developing countries," in Animal Health and Production Papers, 27–31,

Farruggia P., Alaggio R., Cardella F., Tropia S., Trizzino A., Ferrara F. and D'Angelo P. (2010): Focal nodular hyperplasia of the liver: an unusual association with diabetes mellitus in a child and review of literature *Italian Journal of Pediatrics* **36(41):**1-4

Fehrenbach H. (2006). Animal models of pulmonary emphysema: a stereologist's perspective. *European respiratory review* **15(101):** 136–147

Ferrans V.J. and Vleet J.F.V. (1985): Cardiac lesions of selenium-vitamin E deficiency in animals **1(s1):** 294-297

Fulton R.W. (2009). Bovine respiratory disease research (1983–2009) *Animal Health Research Reviews,* **10(2):**131-139

Gabinaitiene A., Siugzdaite J., Zilinskas H., Siugzda R. and Petkevicius S. (2011): Mycoplasma bovis and bacterial pathogens in the bovine respiratory tract. *Veterinarni Medicina journal.* **75(56):***29-35*

Geller S.A. and deCamposc F.P.F. (2014): Focal nodular hyperplasia of the liver Autopsy Case Rep. **4(4):** 5-8.

Getachew H., Guadu T., Fentahun T. and Chanie M. (2012): Small Ruminant Hydatidosis: Occurrence and Economic Importance in Addis Ababa Abattoir. *Global Veterinaria*. **8(2):**160-167

Godfray H., Donnelly C., Kao R., Macdonald D., McDonald R., Petrokofsky J., Woodroffe R., Young D. and McLean A. (2013): A restatement of the natural science evidence base relevant to the control of bovine tuberculosis in Great Britain. *Proc R Soc B* ***280:*** *20131634*. 1-9.

Goodwin K.A., Jackson R., Brown C., Davies P.R., Morris R.S. and Perkins N.R. (2005): Pneumonic lesions in lambs in New Zealand: patterns of prevalence and effects on production. *New Zealand Veterinary Journal* **52(4):** 175-179

Gupta M., D Lobo F., Adiga, D. and Gupta A. (2016): A Histomorphological Pattern Analysis of Pulmonary Tuberculosis in Lung Autopsy and Surgically Resected Specimens. *Pathology Research International* 1-8

Hananeh W.M. and Ismail Z.B. (2018): Concurrent occurrence of acute bovine pulmonary edema and emphysema and endocardial fibroelastosis in cattle: A case history and literature review, *Veterinary World*, **11(7):** 971-976.

Headley S. (2002): Systemic bovine tuberculosis. *Semina: Ciências Agrárias, Londrina.* **23(1):**75-79

Heller M., Gicheru N., Zaire G., Muriuki C., Yu M., Botelho A., Naessens J., Jores J. and Liljander A. (2016): Contagious Bovine Pleuropneumonia. *Journal of Clinical Microbiology* **54(6):** 1557–1565.

Hersi F.K. (2017). Effects of organ condemnation and the direct financial loss attributed to condemned organs in cattle *Global Journal of Veterinary Medicine and Research* **5(6):** 141-147,

Hsee L.C., McCall J.L. and Koea J.B. (2005): Focal nodular hyperplasia: what are the indications for resection? **7(4):** 298–302

Ibrahem M., Ibrahem W., Abdorrahem M. and Ibrahem K. (2016): Livestock hydatid disease (cystic hydatidosis) in Libya. *American Journal of Animal and Veterinary Sciences.* **11 (2):** 70.84

Ibrahim E.M., Soliman A.S. and Gobran R.A. (2008): Bacteriological and pathological studies on kidney affections of slaughtered buffaloes at Kaluobia Governorate Egypt. *J. Comp. Path. & Clinic. Path* **2(3):** 263 - 275.

Ibrahim H.M.M. and Gomaa N.A. (2016): Traumatic Pericarditis in Cattle: Risk Factors, Clinical Features and Ultrasonographic Findings. *J Vet Sci Med Diagn* **5(3): 1-6**

Ibrahim S.A. and Gameel A. (2014): Pathological Histochemical and Immunohistochemical Studies of Lungs and Livers of Cattle and Sheep Infected with Hydatid Disease. The 5th Annual Conference - Agricultural and Veterinary Research , Khartoum, Sudan, Conference Proceedings – Volume Two

Jamshidi K. and Zahedi A. (2014): An Abbattoir-Based Study on Relative Prevalence of Histopathologic Patterns of Hepatic Lessions in One-Humped Camels (Camelus deromedarius), Semnan, Iran Camel*: Int. Journal of Veterinary Science: **2( 2):*** 1-10

Jemal A. (2016). Review on Zoonotic Importance of Bovine Tuberculosis and Its Control. *Open Access Library Journal*. **3(3):** 1-13

Jenberie, S., Awol N., Ayelet G., Gelaye E., Negussie H. and Abie G. (2012): Gross and histopathological studies on pulmonary lesions of camel (Camelus dromedarius) slaughtered at Addis Ababa abattoir, Ethiopia *Trop Anim Health Prod* **44:** 849–854

Johannesen E.J., Zihao W.U. and Holly J.S. (2004): Bile Duct Adenoma with Oncocytic Features Hindawi Publishing Corporation Case Reports in Pathology 1-3

Jones, T.C.; Hunt, R.D.; King, N. W. (1997): Veterinary pathology. 6.ed. Baltimore: Lippincott Williams & Wilkins.

Jubb E.V.F., Kennedy P.C. and palmer N. (1970): Pathology of Domestic Animals 4th Edeition Volume 1 edited by Guelph, Ontario, Canada

Jubb K. and palmers N. (2016): Pathology of domestic animals, 6th edition Edited by: Grant. M. Elsevier, Inc

Jubb K.V.F. Kennedy P.C. and Palmer N. (2005): Pathology of Domestic Animals 5th Edn Academic Press, San Diego, USA

Kadim T. O., Mahgoub, and Purchas R.W. (2005): A review of the growth, and of the carcass and meat quality characteristics of the one-humped camel (Camelus dromedaries). *Meat Science* **80:** 555–569

Kalu E., Akpabio U. and Gloria D.I. (2015): A Case of Chronic Fascioliasis in a Cattle Slaughtered at Ubakala Abattoir *J. Vet. Adv.* **5(6):** 1017-1022

Karam I., Zaki A. and Elshalalli A.A. (2017): A Study on Contagious Bovine Pleuropneumonia in Khartoum State, *Sudan Journal of Veterinary Healthcare* **1(1):**13-20.

Karimi I., Shahgholian M., Ebrahimi A. and Mahzounieh M. R. (2006): Abattoir survey of bovine pyelonephritis Iranian Journal of Veterinary Research, University of Shiraz **7(1):** 14-59

Kerr L.A. and Linnabary R. (1969): A review of interstitial pneumonia in cattle *Vet Hum Toxicol.* **131(3):**247-254

Khadidja H., Achour Y., Houcin B. and Vasile C. (2014): Histological Appearance of Echinococcus Granulosus in the Camel Species in Algeria Bulletin UASVM Veterinary Medicine **71 (1):** 79-84

Khan SR., Pearle M.S., Robertson W.G., Gambaro G., Canales B.K., Doizi6 ., Traxer O. and Tiselius H.G. (2016): Kidney stones

Kimball T. (2011). Livestock Production Systems and their Environmental Implications in Ethiopia. Environ. Policy Rev. Key Issues Ethiop. Handbook 8-13.

Kirovski D. and Sladojevic Z. (2017): Prediction and Diagnosis of Fatty Liver in Dairy *Cows Journal of Gastroenterology & Hepatology* **3(1):** 1-7

Kojouri G., Nourani H., Sadeghian S., Imani H. and Rais A. (2014): Pathological findings of slaughtered camels'(Camelus dromedaris) kidneys in Najaf-Abad, Iran. *Veterinary Research Forum.* **5(3):**231-235

Kok D.J., Papapoulos S.E. and Bijvoet O.L. (1990): Crystal agglomeration is a major element in calcium oxalate urinary stone formation **37(1):** 51–56

Kumar A., Rahal S., Chakraborty A., Verma K. and Dhama K. (2016): Mycoplasma agalactiae, an etiological agent of contagious agalactia in small ruminants-a Review. *Veterinary Medicine International*. 1-13

Laak E.A.T. (1992): Contagious bovine pleuropneumonia a review, *Veterinary Quarterly*, **14(3):** 104-110

Lemma A., Beyi A.F. Shibbiru T. (2016): Causes of Organ Condemnation and Estimated Financial Loss in Dromedary Camels Slaughtered at Akaki Abattoir, Ethiopia Food Science and Quality Management **55:** ISSN 2224-6088

Lemos L.S., Santos A.S Motta O.V., Gina Nunes Texeira G.N. and Carvalho E.C.Q. (2007): Pulmonary cryptococcosis in slaughtered sheep: *Anatomopathology and culture Veterinary Microbiology* **125:** 350–354

López A. (2010): The Respiratory System, Mediastinum and Pleura: In, Pathological Basis of Veterinary Disease, 5th Edition. McGavin, Zachary Eds. Mosby.

Maas J. (2008). University of California Davis vet views California cattlemen's magazine Treating Pneumonia in Beef Cattle School of Veterinary Medicine University of California, Davis

Magaji A., Ibrahim K., Salihu N., Saulawa M., Mohammed A. and Musawa A. (2014.): Prevalence of Fascioliasis in Cattle Slaughtered in Sokoto Metropolitan Abattoir, Sokoto, Nigeria. *Advances in Epidemiology* 1-5

Malamsha N.J.M. (2009): Contagious Bovine Pleuropneumonia (CBPP) in the Maasai south-western Kenya: Evaluation of seroprevalence, risk factors and vaccine safety and efficacy. A thesis submitted for the degree of Doctor of Philosophy College of Medicine and Veterinary Medicine the University of Edinburgh

Malhotra K.K. (2008): Medical aspects of renal stones: review. *Journal of Indian Academy of Clinical Medicine* **9(4):** 282–286.

Mamo M., Bayleyegn G., Tessema T., Legesse M., Medhin G., Bjune F. and Amen G. (2011): Pathology of Camel Tuberculosis and Molecular Characterization of Its Causative Agents in Pastoral Regions of Ethiopia. *PLoS ONE **6(1):** e15862. doi:10.1371/journal.pone.0015862.*

Marcato PS., benazz C., Bettini G., .Masi L., Sa Lda G., Sarli G., Vecchi G. and Poli A. (1996): Blood and Serous Cysts in the Atrioventricular Valves of the Bovine Heart *Vet Pathol* **33(1):** 14-21

Marik P. (2001). Aspiration pneumonitis and aspiration *pneumonia New England Journal of Medicine* **344(9):**665–671

McGavin M.D. and Zachary J.F. (2010): Pathologic Basis of Veterinary Diseases 4th Edition, Mosby Elsevier

McGavin M.D. and Zachary J.F. (2012): Pathologic Basis of Veterinary Disease, 5th Edition, Mosby Elsevier Ltd

McGill S., Malik R., Saul N., Beetson S., Secombe C., Robertson I. . Irwin P. Cryptococcosis in domestic animals in Western Australia: a retrospective study from 1995–2006 (3009): *Medical Mycology*. **47(6):** 625–639

McGuirk S.M. (1991). Treatment of Cardiovascular Disease in Cattle Veterinary Clinics of North America: *Food Animal Practice* **7(3):**729-746.

Mekuriaw E., Walelign B. and Awukew A. (2016): Prevalence of gross pathological lesion in lung and liver of cattle slaughtered at Gondar Elfora Abattoir, North West, Ethiopia. *International Journal of Applied Research* **2(7):** 475-480

Mellau L.S.B., Nonga H.E. and Karimuribo E.D, (2010): A Slaughterhouse Survey of Liver Lesions in Slaughtered Cattle, Sheep and Goats at Arusha, Tanzania. Research Journal of Veterinary Sciences, 3**(3):** 179-188

Millar M., Foster A., Bradshaw J., Turner A., Blowey R., Evans N. and Hateley G. (2017): Embolic pneumonia in adult dairy cattle associated with udder cleft dermatitis **180(8):**205-206

Borai M.G.E., Nagi A.R.A, Allah M.S.G., El-Mashad A.B.I. and Moustafa S.A. (2013): Comparative Pathological Studies on Bacterial Affections of Liver in Farm Animals . *Benha veterinary medical journal* 1-12

Muhammad G., Saqib M. and Naureen A. (2010): Acute pulmonary emphysema cum pulmonary edema apparently associated with feeding of Brassica juncea in a dairy buffalo. *Turkish Journal of Veterinary and Animal Sciences*, **34(3):** 299-301.

Mummed Y. and Webb E. (2015): Causes of Beef Carcass and Organ Condemnations in Ethiopia. *Asian Journal of Animal and Veterinary Advances*. **10(4):** 147-160

Mustafa I. (1987). Bacterial diseases of dromedaries and bactrian camels. *Rev. sci. tech. Off. int. Epiz* **6 (2):** 391-405.

Nagaraja T. and Chengappa M. (1998): Liver Abscesses in Feedlot Cattle. *Journal of Animal Science.* **76(1):**287-298

Nishikawa H. and Osaki Y. (2015): Review Article Liver Cirrhosis: Evaluation, Nutritional Status, and Prognosis Mediators of Inflammation 1-9

Nourani H. and Rohani R. (2009): Pathological Study on Lungs of Dromedary Camels Slaughtered In the Central Part of Iran **16(1):** 51-54

O'Donoghue J.G. (1960). Bovine pulmonary emphysema *Can Vet Jour* **1(11):**

Ogunrinade A, Ogunrinade B.I (1980): Economic importance of bovinefasiolosis in Nigeria. *Trop. Anim. Health Prod*.**12(3):** 155-160.

Ohfuji S. (2012). Cholangiocarcinoma of possibly hepatic progenitor cell origin in a cow, *Veterinary Quarterly* **32(3):** 183-186

Okaiyeto S., Salami O., Samaila D., Isreal O. and Lusharkiah A. (2012): Clinical, Gross and Histopathological Changes Associated With Chronic Fasciolosis Infection in a Dairy Farm. *J. Vet. Adv.* **2(12):** 557-567

Olsen A., Frankena K., Bødker R., Toft N., Thamsborg A.M., Enemark H.L. and Halasa T. (2015): Prevalence, risk factors and spatial analysis of liver fluke infections in Danish cattle herds **8(160):** 2-10

Oryan A., Azizi S., Kheirandish R. and Hajimirzaei M.R. (2015): Nephrolithiasis among Slaughtered Cow in Iran: Pathology Findings and Mineral Compositions. *J Vet Sci Med Diagn* **4(1):**

Ozmen O. (2014).Kidney Pathology in Non-obstructive Urolithiasis in Cattle. *J. Vet. Med.* **51:** 405–408

Padma N.P., Sahadeb D. and Subash C.J. (2016): Review Article Urolithiasis: Critical Analysis of Mechanism of Renal Stone Formation and Use of Medicinal Plants as Antiurolithiatic Agents **11(1):** 9-16, *Asian Journal of Animal and Veterinary Advances*

Park G.M., Kim S.Y., Kim H.R., Jung B.K., Jun A. D., Hong S.B. and Chang K.S. (2018): Comparison of Virulence between Five Strains of Cryptococcus Species Complex in a Rat Model *Biomedical Science Letters* **24(3):** 183-195

Parmar M.S. (2004). Kidney stones *British Medical Journal*, **328 (7453)** 1420–1424

Perkins S.L., Magdesian K.G., Thomas W.P. and Spier S.J. (2004): Pericarditis and pleuritis caused by Corynebacterium pseudotuberculosis in a horse. *J Am Vet Med Assoc*. **224(7):**1133-1138

Provvido A., Teodoro G., Muuka G., Marruchella G. and Scacchia M. (2017): Lung lesion score system in cattle: proposal for contagious bovine pleuropneumonia. Trop *Anim Health Prod* **50(1):**223-228

Quevedo L.S., Bruhn F.R.P., Teixeira J.L.R., Alberti T.S., Scheid H.V., Raffi M.B., Sallis E.S.V. and Schild A.L. (2018):Epidemiological and clinical-pathological aspects of fasciolosis in livers of cattle slaughtered in southern *Brazilian journal of veterinary research* **38(9):**1761-1766

Radostits O.M., Gay C.C., Blood D.C. and Hinchliff K.W. (2000): A text book of the diseases of cattle, sheep, pigs, goats and horses. 9th ed. London: WB Saunders

Radostits O.M., Gay C.C., Hinchcliff K.W. and Constable P.D. (2006): Veterinary Medicine A textbook of the diseases of cattle, horses, sheep, pigs and goats 10th ed

Rashid M.M., Ferdoush M.J., Dipti M., Roy P.M., Rahman M., Hossain M.I. and Hossain M.M. (2013): Bacteriological and Pathological Investigation of Goat Lungs in

Mymensingh and Determination of Antibiotic Sensitivity *Bangladesh Journal of Veterinary Medicine,* **11(2):** 159-166.

Refai M., El-Hariri M., and Alarousy R. (2014): Monograph on Cryptococcus and Cryptococcosis

Robbins and Cotran (2005): Pathologic Basis of Disease, 7th ed.

Runnels R., Monlux W. and Monlux A. (1965): Principles of Veterinary Pathology 7th ed. Scientific Book Agency, Calcutta.

Salem S.I. and Hassan A.H.M (2011): Clinicopathological, Cytological and Histopathological Studies on Liver and Kidney Affections in Camels *Global Veterinaria* **7 (6):** 557-571, 2011

Schroeder P. (2005). Bovine Pulmonary Physiology and Emphysema 1-2

Schuster R.K., Kinne J., Boufana B. and Varcasia A. (2015): A Case of Hepatic Cysticercosis in a *Dromedary Calf Journal of Camel Practice and Research* **22(2):** 1-5

Severo C.B. Gazzoni A.F. and Severo L.C. (2009): Continuing education course mycoses chapter 3 pulmonary cryptococcosis *J. bras. pneumol* **35 (11)**

Shakespeare, A.S., 2012, 'Aspiration lung disorders in bovines: A case report andreview', *Journal of the South African Veterinary Association* **83(1)**

Sharafkhaneh A., Hanania NA. and Kim V. (2007): Pathogenesis of Emphysema from the *Bench to the Bedside Proceedings of the American thoracic society* **5(4):** 475–477

Shekarforoush S.S., Rezakhani A. And Katannejad A. (2006): The prevalence of blood and serous cysts in the atrioventricular valves of the heart of cattle *Revue Méd. Vét.*

Singh B., Sharma R., Mahajan J. and Gil J. (2016): Histopathological changes associated with E. granulosus echinococcosis in food producing animals in Punjab (India*).* **40(3):** *997–1000 J. Parasit. Dis.*

Sisay F. and Awoke K. (2015): Review on Production, Quality and Use of Camel Milk in Ethiopia. J Fisheries Livest Prod **3(3):** 2-4

Skidmore, J. A. (2005): Reproduction in dromedary camels: An update. *Animal Reproduction*, **2:** 161–171.

Slaoui M. and Fiette L. (2011): Histopathology Procedures: From Tissue Sampling to Histopathological Evaluation 70-81

Smith B.P. (2002): Large Animal Internal Medline 5th ed Mosby

Sohair IB., Nasr EM. (2009): Histopathological and bacteriological studies on livers affected with fascioliasis in cattle. Egypt. *J. Comp. Path. And Clinic. Path*. **22 (1);** 19 – 45.

Sorden S.D., Kerr R.W. and Janzen E.D. (2000): Interstitial pneumonia in feedlot cattle: concurrent lesions and lack of immunohistochemical evidence for bovine respiratory syncytial virus infection, *J. Vet. Diagnostic* **12(6):**510–517

Souza A.J.S., Silva C.A.M., Soares M.C.P., Cogliati B. and de Sá L.R.M. (2018): Focal Nodular Hyperplasia of the Liver in a Free-Ranging Opossum (Didelphis marsupialis) *Acta Scientiae Veterinariae.* **46(Suppl 1):** 307

Stoupis C., Taylor H.M., Paley M.R., Buetow P.C., Marre S., Baer H.U., Vock P. and Ros P.R. (1998): The Rocky Liver: Radiologic-Pathologic Correlation of Calcifled Hepatic Masses *Scientific Exhibit* **18(3):**675-85

Talkhan O.F.A., Salama S.E., Kholy M.M., Mosallam S.A. and Atwa E.I. (2009): Bacterial Agent of Respiratory Manifestation in Cattle and the Associated Biochemical Alterations in Menoufiea Governorate, *Nature and Science,* **7(9):** 26-30

Tavassoly A. (2003): Pathological Study of Renal Lesions in the Condemned Bovine Kidneys in Ziaran Slaughter-house **55:** 97-104 Short Communication

Tavassoly A. (2005). Pathological Study of Renal Lesions in the Condemned Bovine Kidneys in Ziaran Slaughter-house Short Communication *Arch. Raz; Ins*. ***55:*** 97-104

Teixeira MS., Faleiros FTV., Griva BL., Kiy Y., Moriguchi SM., Teixeira AS., Kategawa, B., Coelho KIR. and Coelho CAR. (2007): Focal nodular hyperplasia of the liver: a case report and review of the literature Radiol Bras **40 (4)**:

Tenaw M, Feyera T and Abera B. (2015): Major causes of organ condemnation in camels slaughtered at Akaki Abattoir, Addis Ababa, Ethiopia. J. Anim. Health Prod. **3(1):** 14-20.

Tharwat M., Sadan M., El-Shafaey ES., Saeed EH. and Al-Hawas A. (2018): Bilateral renal abscessation and chronic active pyelonephritis in a male camel (Camelus dromedarius) caused by Escherichia coli. *J. Vet. Med. Sci.* **80(5):** 778–783

Tilahun S., Fekadu A. and Mekibib B. (2017): Major Causes of Total Organ Condemnation and their Direct Financial Impact in Cattle Slaughtered at Hawassa Municipality Abattoir, Southern Ethiopia. J Vet Sci Technol **8(5):** 2-5

Timoney J.F., Gillespie J.A., Scott F.W. and. Barlough J.E. (1992): Hagan and Bruner's Microbiology and Infectious diseases of domestic animals.

Tonamo A. (2015): A review on cattle husbandry practices in Ethiopia. *International Journal of Livestock Production* **7(2):** 5-11

Ueli B. (2009): Traumatic pericarditis in cattle: clinical, radiographic and ultrasonographic findings. *Veterinary Journal,* **182(2):**176-186.

Worthington J. and Chapman R. (2006): Primary sclerosing cholangitis Orphanet *Journal of Rare Diseases* **1(1):** 1-7

Xavier J.G., Dagli M.L.Z., Vianna M.R. and Malucelli B.E. (1992): Hepatocellular adenoma and focal nodular hyperplasia of the liver: differential diagnosis in dogs. *Braz. J. vet. Res. arum. Sci., SSo Paulo* **29(1):** 77-82.

Zidan K.H., Mazloum K., Saran M.A. and Hatem M.E. (2013): Abscesses in dromedary camels, sheep and goats etiology and Pathology 1st International Scientific conference of Pathology Department, Faculty of Veterinary Medicine 47-59

Zubair R., Khan A.M.Z. and Sabri M.A .(2004): Pathology of Camel Lungs. *Journal of Camel Science,* **1:**103-106.

## 8. ANNEX

**Annex 1:** procedure for histopathology slide making

Histopathology slide masking procedures with some modification (Slaoui and Fiette, 2011)

### Fixation

Fix the tissue in 10% buffered formalin
The samples were trimmed at 5mm thickness

### Trimming

Trim the tissue at 5 microns
Placed trimmed tissue in tissue cassettes
Immersed the cassettes in 10% buffered formalin (2 times) for two hours each

### Dehydrating

70% ethanol for 1 hour
95% ethanol for 1 hour
100% ethanol for 1 hour
100% ethanol I for 2 hours
100% ethanol II for 2 hours

### Clearing

Xylene I 1 and 1/2 hours
Xylene II 1 and 1/2 hours

Xylene III 2 and ½ hours

Impregnation

Wax I for 2 hours

Paraffin wax II for 2 hours

**Embedding**

The cassettes from the last tissue processor bath were removed from it

Transferred the cassette onto the hot plate

Pour melted paraffin from the paraffin dispenser in to mold

Paraffin-infiltrated tissues transfer the into the mold

Transferred the mold onto the cool plate

Refilled the molds with paraffin to above the upper edge of the cassette

Transferred the molds and cassette onto the cool plate

Snap off the molds

Store paraffin blocks in the refrigerator

**Sectioning**

Coarse cut the block at 15 microns until the whole surface of the embedded tissue can be cut

Sectioned the blocks at thickness of 5 microns to produce ribbon

Float the ribbon on a water bath maintained at 45°C

Removed the ribbon from the water bath

Dried in oven at 37°C for overnight

**Staining**

Dewax the paraffin sections in xylene 2 changes for 5 min each.

Rehydrate in 100% ethanol 2 changes for 5 min each

Rehydrate in 95% ethanol 2 changes for 5 min each.

Wash in running tap water for 3 min.

Stain for 3 min in hematoxylin

Wash in running tap water for 3 min.

Decolorize briefly in acid alcohol for 2 seconds.

Wash and blue the sections in running tap water for 3 min.

Stain for 3 min in 0.1% aqueous eosin Y.

Rinse in tap water for 30 seconds.

Dehydrate in 95% ethanol two times for 2 minutes each.

Dehydrate in 100% ethanol two times for 2 minutes each.

Clear sections in xylene two times for 2 minutes each

The slides remained in xylene agent until cover slipping.

**Mounting**

Wipe the surface under the slide while keeping the tissue section

Apply drops of mixture of distyrene, plasticizer and xylene (DPX).

Place a cover slip on the slide by avoiding the formation of bubbles and pressed gently with forceps to remove any bubble.

Dry the slides overnight at room temperature on a flat surface within the fume hood