ADDIS ABABA UNIVERSITY

SCHOOL OF GRADUATE STUDIES

M. Sc RESEARCH THESIS

Sero-prevalence of HBV and HCV among chronic liver disease patients visiting OPD in public hospitals in Addis Ababa, Ethiopia

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May 2011

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A Thesis Submitted To the School of Graduate Studies, Addis Ababa University in Partial Fulfillment of the Requirements for the Degree of Masters of Science in Medical Microbiology

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May 2011

Acknowledgments

I express my sincere thanks to my advisor; Dr. Solomon Gebre-Silasse for his unreserved guidance, constructive suggestions and comments from the very conception, development and finalization of this thesis.

I would like to thank the Department of Medical Microbiology, AAU graduate school for granting fund for this research and ArbaMinch University for sponsoring me to pursue my masters' degree.

I would also like to acknowledge Tikur Anbessa Hospital, Zewditu Memorial Hospital and St. Paul Hospitals for granting permission to conduct the research.

I would take this opportunity to extend my thanks to all of the nurses, physicians and laboratory staffs for their help and encouragement through the research process.

I am so grateful to acknowledge the study participants who were willing to participate in the study.

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List of Abbreviations and Acronyms

AAU: - Addis Ababa University

Ab: - Antibody

Ag: - Antigen

CDC: - Center for Disease Control

CLD: - Chronic liver disease

ELISA: - Enzyme linked immune sorbent assay

DMIP: - Department of Microbiology, Immunology and Parasitology

FoM: - Faculty of Medicine

HBsAg: - Hepatitis B surface antigen

HBV: - Hepatitis B Virus

HCC: - Hepatocellular carcinoma

HCV: - Hepatitis C Virus

IDU: - Injection drug users

TBA: - Traditional birth attendant

OPD: - Out patient department

SOPS: -Standard operating procedures

USA: - United States of America

WHO: - World Health Organization

Abstract

***Background**: Hepatitis B and C viruses (HBV & HCV) are hepatotropic viruses causing viral hepatitis, chronic liver disease and hepatocellular carcinoma. Hepatitis B and C virus infections are still major public health problems around the globe. Both of these viruses are transmitted mainly through the parenteral route and therefore a dual infection of these viruses can occur and even persists in the same patient. They are prevalent in different parts of the world including Ethiopia. In view of this, the present study was designed to determine the prevalence of HBV and HCV infection and co-infection in clinically diagnosed chronic liver disease patients who visited public hospitals of Addis Ababa.*

***Objective:** The objective of this study was to determine the prevalence of HBV and HCV infection among chronic liver disease patients who visited public hospitals of Addis Ababa, Ethiopia.*

***Method:** Hospital based cross-sectional study was conducted in three public hospitals of Addis Ababa over a period of 7 months (Nov 2010- May 2011) on clinically diagnosed chronic liver disease patients. By using questionnaire brief history and risk factors were taken from each volunteering patient. Serum samples from each volunteering patients was screened for the presence of HBsAg and anti-HCV Ab by using qualitative rapid test kits.*

***Result:** A total of 120 participants who have chronic liver disease participated in the study, where 76 of them were males and the remaining 44 were females. The age distribution range form 18-80 years and the mean age was 40.99 years ±14 SD. The overall prevalence of HBsAg and HCV was 35.8% and 22.5% respectively. The prevalence of HBsAg and anti-HCV Ab was high in age below 50 years. 2.5% of the study participants had combined HBV/HCV infection which is possible because of their common modes of transmission.*

***Conclusion:** The prevalence of HBV and HCV in chronic liver disease patients is high and dental extraction at health facility was statistically associated with HCV infection.*

***Key words:** Hepatitis B virus, Hepatitis C virus, chronic liver disease, prevalence, co-infection*

1. Introduction

1.1 Background

Hepatitis is a general term meaning inflammation of the liver and can be caused by a variety of different viruses such as hepatitis A, B, C, D and E (WHO 2002). The awareness and understanding of viral hepatitis has risen dramatically over the past three decades though it is not a new problem. Descriptions of jaundice exist in literature as far back as several centuries BC (Sobia et al. 2007). Since the development of jaundice is a characteristic feature of liver disease, a correct diagnosis can only be made by testing patients' sera for the presence of specific anti-viral antigens or antibodies. Of the many viral causes of human hepatitis few are of greater global importance (WHO, 2002). Hepatitis B virus (HBV) and hepatitis C virus (HCV) infections are among the most frequent viral infections in humans, and represent a major global public health problem.

Hepatitis B and C virus are members of two different viral families, but both display a strong hepatotropism, whose underlying molecular mechanisms are not yet entirely understood. HBV belongs to the family of *hepadnaviridae,* a group of hepatotropic mammalian and avian DNA viruses, which replicate via reverse transcription of a genomic RNA intermediate. HBV contains a partially double stranded genomic DNA. HCV is member of the *flaviviridae,* where it forms its own genus, Hepacivirus. HCV is a small, enveloped positive-sense, single stranded RNA virus, and its life cycle is thought to be predominantly cytoplasmic (Birke, 2010).

Despite fundamental differences in genome structure, HBV and HCV viruses use many of the same strategies to achieve high-level replication and persistence of infection in hepatocytes. The limited genome size of these viruses necessitates an efficient use of host-cell machinery to carry out viral gene expression. This strong dependence on virus-host interaction restricts HBV- and HCV- cell tropism to the hepatocytes of higher primates and precludes their reliable and reproducible propagation in cell culture (Robert et al., 2001). The two viruses are responsible of multiple liver damages ranging from minor histological disorders to liver cirrhosis and HCC that are responsible for high rate of morbidity and mortality. Severe liver diseases are more frequent when patients are co-infected by the two viruses (Benvegnu et al., 1994). Combined HBV/HCV infection is possible because of common modes of viral transmission (Liu et al., 2006).

In the last few years, knowledge of the epidemiology and the natural history of HBV and HCV infection have markedly improved and considerable progress has been made in the efficacy of therapy. New drugs and new therapeutic strategies that are currently under evaluation could further improve the efficacy of therapy in the near future (Patrick, 2009).

WHO estimates that there are 350 million people with chronic HBV infection and 170 million people with chronic HCV infection worldwide (WHO, 2002). HBV is estimated to result in 563,000 deaths and HCV in 366,000 deaths annually (Perz et al., 2006). Many researchers done in Ethiopia have investigated prevalence rates of HBV and HCV infections in various groups. In Ethiopia as in other Sub-Saharan Africa, the prevalence of liver disease is high. It accounts for 12% of the hospital admissions and 31% of the mortality in medical wards of Ethiopian hospitals as it is reported by Tsega, 2000. To ensure the optimal clinical managements of CLD patients it is important to know the HBV and HCV status of these patients.

1.2. Statement of the problem

HBV and HCV contribute to a major health burden, affecting millions of people worldwide. WHO estimates that there are 350 million people with chronic HBV infection and 170 million people with chronic HCV infection worldwide (WHO, 2002). Chronic infection by these viruses leads to slow progressive liver disease that over a period of up to 30 years may result in cirrhosis, chronic liver failure and hepatocellular carcinoma (HCC) (Shimotohno, 2000). An infection with both viruses B and C is frequent particularly in areas where the two viruses are endemic and among populations at high risk for perinatal infections. Thus chronic viral hepatitis ranks as the fifth most common cause of death globally (Shaw-Stiffel, 2000). Several studies regarding the prevalence of Hepatitis viruses in the causation of CLD have documented the presence of co-infection of HCV and HBV besides single infection (Bukhtiar, 2003).

In terms of health policy, problems have been caused by hepatitis B and C, as they frequently follow a chronic course and involve a risk of transition to hepatocellular carcinoma (Lemon et al., 1995). Patients with acute hepatitis B and underlying chronic hepatitis C had a more severe course, including fulminant hepatitis in which HBV and HCV co-infection appears to be clearly associated with a higher rate of morbidity and mortality than either infection alone (Robinson, 1995).

In Ethiopia 12% of medical admission and 31% of mortality in medical wards is due to liver disease (Tsega, 2000). Many researchers have investigated prevalence rates of HBV and HCV infections in various groups (blood donors, health care workers, medical waste handlers, and others), however the studies conducted in Ethiopia on single and co-infection of HBV and HCV among chronic liver disease, are limited to a few case series (Tsega, 1991 and Tsega, 1995). Therefore this study was designed to determine the prevalence of HBV and HCV in chronic liver disease patients.

2. Literature review

2.1 Chronic liver disease

Chronic liver disease (CLD) is a disease of the liver resulting from an inflammatory, infiltrative, immunologic, mechanical or metabolic injury to the liver, which has persisted for six or more months without complete resolution. Chronic liver disease comprises of a spectrum of disease such as chronic hepatitis, liver cirrhosis and HCC (Parry, et al., 2004). Milder forms are non-progressive or only slowly progressive, while more severe forms may be associated with scarring and architectural reorganization, which, when advanced, lead ultimately to cirrhosis. Both the enterically transmitted forms of viral hepatitis, hepatitis A and E, are self-limited and do not cause chronic hepatitis. In contrast, the entire clinicopathologic spectrum of chronic hepatitis occurs in patients with chronic viral hepatitis B and C as well as in patients with chronic hepatitis D superimposed on chronic hepatitis B (Conjeevaram, 2003).

Chronic liver disease is responsible for over 1.4 million deaths annually (WHO, 1999) and is characterized by permanent inflammatory processes that predispose to liver cancer and in particular hepatocellular carcinoma (HCC). In healthy liver, inflammatory processes stimulate growth and repair and restore normal liver architecture. However, if liver inflammation becomes chronic, the balance of damage *versus* regeneration in the liver is disrupted and can lead to the formation of excessive scar tissue, termed fibrosis. In the long-term, an exacerbation of fibrosis will lead to cirrhosis, which is characterized by abnormal liver architecture and function and is associated with a significant reduction in overall health and wellbeing. At cirrhotic stages, the liver damage is often irreversible or difficult to treat. Cirrhosis leads frequently to death from liver failure or due to HCC (Parkin, et al., 2001).

2.2 Viral hepatitis

The word "**hepatitis**" means inflammation of the liver and also refers to a group of viral infections that affect the liver. Viral liver diseases are among the most important communicable disease worldwide (WHO, 2000). Different species of viruses, including Cytomegalovirus, Epstein-Barr, Herpes simplex, Adenovirus, Coxsackie virus, Mumps, Yellow fever, and other cause parenchymal hepatic inflammation, but the term viral hepatitis generally implies the five hepatotropic viruses: Hepatitis A, B, C, D and E virus (Tesga, 2004). Hepatitis B virus (HBV) and hepatitis C virus (HCV) infections are among the most frequent viral infections in humans, and represent a major global public health problem and are among the principal causes of severe liver disease, including hepatocellular carcinoma and cirrhosis-related end-stage liver disease. The World Health Organization (WHO) estimates that there are 350 million people with chronic HBV infection and 170 million people with chronic HCV infection worldwide (WHO, 2002). Hepatitis B is estimated to result in 563,000 deaths and hepatitis C in 366,000 deaths annually (Perz et al., 2006). The two viruses are responsible of multiple liver damages ranging from minor histological disorders to liver cirrhosis and hepatocellular carcinoma (HCC) that are responsible for high rate of morbidity and mortality. Severe liver diseases are more frequent when patients are co-infected by the two viruses (Benvegnu et al., 1994). Combined HBV/HCV infection is possible because of common modes of viral transmission (Liu et al., 2006).

Liver diseases are common in Africa and account for high morbidity and mortality. Hospital based analysis indicate that acute viral hepatitis, chronic hepatitis, cirrhosis and hepatocellular carcinoma are responsible for at least 12% of medical admissions and over 20% of hospital mortality in many parts of Africa (Tsega, 2004).

2.2.1 Hepatitis B Virus

Hepatitis B virus (HBV) is the main representative of the family of hepadnaviruses, Hepadnaviridae. The name of the family is an acronym of the disease caused by the virus and its genomic type. It causes a chronic form of liver inflammation (hepatitis) and its genome consists of partially double-stranded DNA (hepadnavirus= hepatitis DNA virus). The replication cycle of the HBV includes a transient RNA phase. The HBV possess an envelope made up of a cellular double lipid layer in which are integrated the hepatitis B surface (HBs) antigen, a 25 kDa polypeptide, and its precursor stages PreS1 (40 kDa) and PreS2 (33 kDa). This envelope encloses

the actual capsid, which consists of the hepatitis B core (HBc) antigen with 21 kDa and contains the genome together with the DNA polymerase (a reverse transcriptase). The complete, infectious virion, also known as a Dane particle after its discoverer, has a diameter of 42 nm, the inner structure 27 nm (Bienz, 2005)

The virus replicates in liver cells. The Dane particles and the HBs antigen, but not the HBc antigen, are released into the bloodstream, whereby the HBs antigen is present in two different forms, a filamentous particle approximately 22x 100 nm and a spherical form with a diameter of about 22 nm. A further viral protein is the HBe antigen, which represents a posttranslational, truncated form of the HBc antigen and is no longer capable of spontaneous capsid formation. It is also released from the hepatic cells into the blood (Bienz, 2005). The hepatitis B surface antigen (HbsAg) in serum is the first seromarker to indicate active HBV infection, either acute or chronic (Horvat et al., 2003). Detection of HBsAg in the serum of an individual indicates that the person is currently infected with the virus. Detection in the serum of hepatitis B e antigen (HBe Ag), a soluble protein in the inner core of the virus, correlates with the presence of virus in large amounts and is associated with greater infectivity (WHO, 2002).

Table 1. Typical interpretation of serologic test results for hepatitis B virus infection (Adapted from CDC, 2008)

Serologic marker				
HBsAg	Total anti-HBc	IgM anti-HBc	Anti-HBsAg	Interpretation
-	-	-	-	Never infected and no evidence of immunization
+	+	-	-	Chronic infection
+	+	+	-	Acute infection
-	+	-	+	Recovered from past infection and immune
-	-	-	+	Immune (immunization or natural)

Sequencing of many HBV isolates has revealed eight genetic groups (genotypes A-H) and these human viruses are related to similar viruses in other primate species. The genetic groups are fairly restricted geographically, e.g. genotype A predominates in Northern Europe while genotype B and C are in Asia (John et al., 2007).

A hepatitis B virus (HBV) infection of the liver cells results in expression of viral antigen on the cell surface, followed by immunological cell damage with acute, possibly fulminant, chronic persistent or chronic aggressive hepatitis. The final stages can be liver cirrhosis or hepatocellular carcinoma. A concurrent or later super-infection by a defective, RNA-containing and HBV-dependent hepatitis D virus (HDV, delta agent) normally exacerbates the clinical course (Bienz, 2005).

The incubation period of hepatitis B is 4 to 12 weeks, followed by the acute infection phase, icteric, or anicteric course, once again with a variable duration of 2 to 12 weeks. The hepatic cell damage resulting for an HBV infection is not primarily due to cytopathic activity of the virus, but rather to a humoral and cellular immune response directed against the virus-induced membrane antigens (HBs, HBc) on the surface of the infected hepatocytes: 0.5-1% of those infected experience a fulminant, often lethal, hepatitis. In 80-90% of cases the infection runs a benign course with complete recovery and elimination of the HBV from the body. A chronic infection develops in 5-10% (Bienz, 2005).

A chronic infection can result in development of a carcinoma (hepatocellular carcinoma, HCC) or cirrhosis of the liver, with incidence varying widely from one geographic area to another. The delta agent appears to have an unfavorable influence on the clinical course, usually making the disease more aggressive, increasing the number of complications and worsening the prognosis (Bienz, 2005).

Transmission of the virus is parenteral, either with blood or body fluids containing HBV (unprotected sexual intercourse) that come into contact with mucosa, lesions, or microlesions in the skin, use of unsterile needles, and from an infected mother to her newborn during the delivery process. In transmission by blood, the tiniest amounts contaminating syringe needles, ear-piercing needles, tattooing instruments, etc. suffice to produce an infection (Bienz, 2005). The highest concentrations of the virus occur in blood and wound secretions with which either

skin puncture or mucous membrane contact with infected blood or other body fluids causes an infection. Moderate concentrations of HBV are found in semen and vaginal fluid, and lower concentrations occur in saliva. HBV is not spread by air, food or water (WHO, 2001). Hepatitis B infections from blood transfusions have been greatly reduced by thorough screening of blood donors for HBs antigens, despite which patients receiving multiple transfusions or dialysis remain a high-risk group (Bienz, 2005).

Epidemiology of HBV

More than two billion individuals alive today, about 1/3 of the world's population have been infected at some time in their lives with the HBV, of whom approximately 400 millions are chronically infected carriers worldwide. Annual death rate exceeds a million among infected individuals. More than three-quarters of HBV infections occur in Asia, the Middle East and Africa (Perz et al., 2006). It is estimated that about one to two million people die annually from HBV- related acute and chronic liver disease worldwide. The majority of chronic carriers of HBV are found in South East Asia and Sub-Saharan Africa. Twenty five per cent of carriers are at risk of chronic hepatitis and eventual death from liver cirrhosis and hepatocellular carcinoma. In endemic regions, HBV related diseases exert a heavy toll socio-economically by affecting mainly the young and economically active age group. The demands on health care resources are also enormous.

HBV infection prevalence varies markedly in different geographic areas of the world, as well as in different population subgroups. The prevalence of chronic HBV infection worldwide could be categorized as high, intermediate and low endemicity (Margolis et al., 1991). Approximately 45% of the world population lives in areas where chronic HBV infection is highly endemic (≥8% of the population are HBsAg-positive); 43% live in areas of intermediate endemicity (2-7% HBsAg-positive); and 12% live in areas of low endemicity (<2% HBsAg-positive) (Fig 1.)

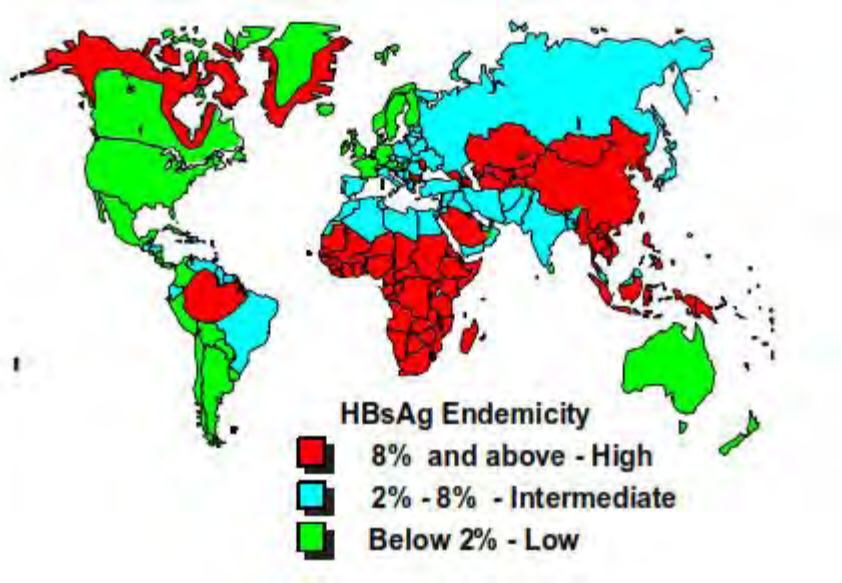

Figure 1. Estimated global prevalence of hepatitis B virus infection (adapted from WHO, 2002)

Hepatitis B is moderately endemic in part of Eastern and Southern Europe, the Middle East, Japan, and part of South America. Between 10–60% of the population have evidence of infection and 2-7% is chronic carriers. Acute disease related to HBV is common in these areas because many infections occur in adolescents and adults; however, the high rates of chronic infection are maintained mostly by infections occurring in infants and children. In these areas, mixed patterns of transmission exist, including infant, early childhood and adult transmission (Margolis et al., 1991, McMahon 2005).

Hepatitis B is highly endemic in developing regions with large population such as South East Asia, China, Sub-Saharan Africa and the Amazon Basin, where at least 8% of the population are HBV chronic carrier. In these areas, 70–95% of the population shows past or present serological evidence of HBV infection. Most infections occur during infancy or childhood particularly acquired from the carrier mothers at birth. Since most infections in children are asymptomatic, there is little evidence of acute disease related to HBV, but the rates of chronic liver disease and liver cancer in adults are high (McMahon 2005).

In Africa, infections with HBV play a major role in the etiology of most liver diseases. The WHO African region includes all of Sub-Saharan Africa and Algeria. By country, estimated HBsAg sero-prevalence ranges between 5% and 19%, and the total number of carriers may

approach 58 million with as many as 12.5 million likely to die prematurely due to hepatitis B-induced liver disease (Brian et al., 2004).

In Ethiopia as in other Sub-Saharan Africa, the prevalence of liver disease is high. It accounts for 12% of the hospital admissions and 31% of the mortality in medical wards of Ethiopian hospitals (Tsega, 2000). In a study, the health status of 239 Ethiopian refugees in the United States was evaluated; over 70 per cent were males 15-30 years old and Hepatitis B surface antigenemia was detected in 9.4 per cent (Parenti et al., 1987). Another study which tested sera of 200 recent Ethiopian immigrants (less than 1 year in Israel) for the presence of antibodies against hepatitis B and hepatitis C viruses has shown the following result: the prevalence of the various markers was: anti-HBc 52%, and HBsAg 11.5% (Flatau et al., 1993).

In a nationwide sero-epidemiological study of hepatitis B markers prevalence conducted in Ethiopia on 5,270 young males from all regions of the country the overall prevalence rates were 10.8% for HBsAg and 73.3% for "at least one marker positive"; a remarkable geographical and ethnic variability of marker prevalence was observed, reflecting the wide differences existing in Ethiopia in socio-cultural environment and activities such as tribal practices and traditional surgery. Sexual practices and medical exposure also play some role as determinants of hepatitis B marker prevalence in Ethiopia (Brian et al., 2004).

A community based sero-prevalence study in the capital city of Ethiopia; Addis Ababa has shown a 7% sero-prevalence of HBsAg, higher in males than females. In this study overall HBV sero-prevalence rose steadily to over 70% in 49 year olds. The age at which 50% had evidence of infection was around 20 years (Abebe et al., 2003). Another study done in Ethiopia has shown an overall HBsAg prevalence of 6.2% and 2.1:1 male to female ratio, infection occurring early in life and continuing to increase gradually without leveling off. Overall HBV marker prevalence (HBsAg, anti-HBc, anti-HBs) over 15 years of age was 76% (Tsega, 1991). Screening sera of all male donors appearing at the blood bank of a regional hospital in Northwest Ethiopia (Gondar) in 1994 (n=1022) and 1995 (n=1164), for HBsAg was carried out on 549 consecutive sera. The crude sero-prevalence of HBsAg was 14.4% (Rahlenbeck et al., 1997). One or more hepatitis B virus markers were also found in 86% of chronic hepatitis, 88% cirrhosis and 78% hepatocellular carcinoma patients studied in Addis Ababa (Sobia et al., 2007).

Prevention and treatment

Hepatitis B vaccine has been available for several decades, and is highly safe and effective in preventing HBV infection and the development of its serious consequences (WHO, 2002). Prevention of primary infection by vaccination is an important strategy to decrease the risk of chronic HBV infection and its subsequent complications. The first-generation hepatitis B vaccine, an inactive plasma-derived vaccine, became available in 1982. Consequently, the second generation of HB vaccine, a DNA recombinant HB vaccine was also available for general use in 1986. Both of the vaccines were proven to be safe and efficacious in preventing HBV infection (Jinlin et al., 2005). The extraordinary protective efficacy of HBV vaccines, combined with their ability to provide prolonged protection in most individuals, strongly suggests that, despite concerns about the emergence of mutant HBV strains, universal use of the vaccines will eventually markedly reduce, if not eliminate, this infection (Raymond et al., 2002). In 1991, the World Health Organization (WHO) recommended that hepatitis B vaccination should be included in national immunization system in all countries with a hepatitis B carrier prevalence (HBsAg) of 8% or greater by 1995 and in all countries by 1997. By May 2002, 154 countries had routine infant immunization with hepatitis B vaccine (Jinlin et al., 2005). Although routine infant immunization against HBV has been adopted in many nations, coverage is still limited, particularly in developing countries where the infection may be endemic (Raymond et al., 2002). By preventing HBV infection, hepatitis B vaccine also protects against HDV infection (WHO, 2002).

Alpha-interferon has been used for some years to treat HBV-infected persons. This treatment does not eliminate the infection, but it results in a significant reduction in viraemia in about 20-30 percent of cases. There is a price to pay for interferon treatment in the form of side-effects such as influenza-like symptoms and weight loss, which may necessitate reducing the dosage or even discontinuing the treatment. The drug lamivudine, a nucleoside analogue is also used to treat HBV infection. In many ways lamivudine is an improvement over α-interferon as it suppresses virus replication with a low incidence of side-effects, is administered by mouth rather than by injections and is cheaper. Long-term treatment, however, results in the appearance of lamivudien-resistant HBV mutants, though they appear to be susceptible to other nucleoside analogues such as adefovir. (John et al., 2007)

2.2.2 Hepatitis C Virus

Hepatitis C Virus (HCV) was first recognized as a separate disease entity in 1975 when the majority of transfusion related hepatitis were found not to be caused by the only two hepatitis viruses recognized at that time i.e. hepatitis A virus and hepatitis B virus. The disease at that time was called 'non-A non-B hepatitis.' The discovery of hepatitis C genome in 1989 has lead to the realization that this virus is a major health problem worldwide (Sobia et al., 2007).

Hepatitis C virus is an enveloped RNA virus with a diameter of about 50 nm, classified as a separate genus (Hepacivirus) within the Flaviviridae family. The genomic organization and sequence of HCV resembles that of the pestiviruses and flaviviruses (EASL, 1999). The reservoir of HCV is man, but the virus has been transmitted experimentally to chimpanzees (Purcell, 1999). The genome of HCV is highly mutable. Because HCV is an RNA virus and lacks efficient proofreading ability as it replicates, virions infecting humans undergo evolution with time, giving rise to the notion that HCV persists as a collection of virus **quasispecies**. By constant mutation, HCV may be able to escape host immunologic detection and elimination (Walker, 1999). Although its means of transmission are well documented, the hepatitis C virus itself still remains an enigma. Very little is known about the replication cycle of HCV, because there is no in vitro cell culture system that is permissive for virus replication (Purcell, 1999) even though progress has been made (WHO, 2000).

HCV infection causes an acute or chronic necroinflammatory disease of the liver. Only relatively small fractions of HCV infections are symptomatic. Most infected individuals remain asymptomatic and presumably, undiagnosed (Alter et al., 1994). Chronic HCV infection has multiple manifestations, the most common of which is chronic progressive liver disease associated with inflammation and fibrosis that in some patients may progress to cirrhosis. Cirrhosis or end–stage liver disease is associated with multiple complications including variceal bleeding, ascites, encephalopathy, and hepatocellular carcinoma. These conditions are common causes of morbidity and mortality in HCV infected patients (Weinstock, 1999). Major clinical manifestation is progressive hepatic fibrosis, which leads to cirrhosis and an increased risk of hepatocellular carcinoma (Feinsone et al., 1975).

A primary infection with HCV leads to persistent viremia in approximately 85% of patients with development of chronic liver disease in >60% of cases. Approximately 20% of individuals with chronic hepatitis C eventually develop medically significant sequel including cirrhosis, end stage liver disease or hepatic cellular carcinoma (Wolfgang et al., 2000). The chronic phase of HCV can last many decades and can ultimately lead to end stage liver disease. In retrospective studies in individuals with chronic HCV infections, cirrhosis of the liver occurred in 17-55%, hepatocellular carcinoma (HCC) developed in 1-23%, and liver related death occurred in 4-15% (Ali, 2009).

The liver is usually presumed to be the primary source of virus present in the blood. HCV is most efficiently transmitted through transfusion of infected blood, transplantation of infected organs, and sharing injection drug equipment.

Prevention and treatment

Currently there is no vaccination against hepatitis C. One reason being that the virus comes in many forms and constantly mutates leading to "swarms" of closely related viral genomic sequences (referred to as quasispecies) (WHO, 2002). Neither passive immunization with conventional or HCV hyperimmune globulin preparations nor active immunization with an HCV vaccine are currently recommended or available. Immunity to HCV infection is thought to be weak, and neutralizing, protective antibodies can be detected in only a small proportion of infected individuals, and seem to be isolate-specific. One consequence of this limited response is that super-infection by another HCV genotype may be identified in some patients chronically infected with one HCV genotype. The high frequency of mutations, in that portion of the viral genome encoding the HCV envelope proteins, may be responsible for the escape of the virus form antibody-mediated clearance (Raymond, 2002). The rationales for treatment of chronic hepatitis are to reduce inflammation, to prevent progression to fibrosis, cirrhosis, and HCC through the eradication of the virus in chronically infected patients, and to decrease infectivity and control the spread of the disease. Combination therapy results in better treatment responses rates have been achieved with pegylated interferon in combination with ribavirin. Genotype determinations influence treatment decisions (WHO, 2002).

Epidemiology

Hepatitis C virus infection is a major global public health problem in both developed and developing countries. More than 170 million people are chronically infected with HCV worldwide, and the infection results in the development of chronic liver diseases, including liver cirrhosis and hepatocellular carcinoma (Guy-Roger et al., 2008). There are, however, considerable temporal and geographical variations in the incidence and prevalence of infection (WHO, 1999). The prevalence of chronic hepatitis C ranges from 0.1 to 5% in different countries (Lee et al., 2001, Lavezzo et al., 1999). It is estimated that there are 4 million HCV chronic carriers in the US and 5 million HCV chronic carriers in Western Europe. The prevalence seems to be higher in Eastern Europe than in Western Europe (Figure 2). In Europe, the general prevalence of antibodies to HCV (anti-HCV) is about 1% (Touzet et al., 2000) but varies between countries (0.87% in Belgium (Van Damme et al., 2002), 3.2% in Northern Italy (Bellentani et al., 2001), 1.3% in France (Pradat, 2001, Theodore and Mazen, 2006). In Central and South America, the prevalence varies between 1.2% (Puerto Rico) and 6.3% (Perez et al., 2005) (Mexico). Intermediate rates of HCV infection have been reported across Asia (0.49% in Japan (Tanaka et al., 2004), 1% in China (Wang et al., 1994, Zhang et al., 1992) with higher rates in some areas (Hubei province – 30.1%, Inner Mongolia Autonomous Region – 31.9% (Tang 1993). The recently estimated prevalence in Australia is 2.3% (Amin et al., 2004). A study conducted in West Bangal, India showed of 1481 chronic liver disease patients 375 (25%) of them were anti-HCV positive (Chaudhuri et al., 2005). In industrialized countries, HCV accounts for 20% of cases of acute hepatitis, 70% of cases of chronic hepatitis, 40% of cases of end-stage cirrhosis, 60% of cases of HCC and 30% of liver transplants (Manns et al., 1999, Mansour, 2002). The prevalence rates in healthy blood donors is estimated 0.01-0.02% in the United Kingdom and Northern Europe, 1-1.5% in Southern Europe, 1.8% in US and 6.5% in parts of Equatorial Africa.

Areas of higher prevalence include countries in the Far East, Mediterranean countries and certain areas in Africa and Eastern Europe (Hsu et al., 1994, WHO, 1999). There have been fewer studies in Africa; however, the region is reported to have the highest HCV prevalence rate (5.3%) (WHO, 1999) with differences between regions (Madhava et al., 2002). Prevalence is generally high in Central Africa (6.0%) (Madhava et al., 2002) with the maximum in Cameroon (13.8%) although lower rates have been reported (1.9% in pregnant women). (Njouom et al.,

2003) Nevertheless, much higher rates (20%) have been found in Egypt (Darwish 2001, Mohamed MK. et al., 2006). In West Africa, the overall weighted average prevalence is 2.4%. The highest HCV prevalence for this region is in Guinea (5.5%) (Madhava et al., 2002).

Prevalence rates as high as 22% are reported in Egypt and are attributed to the use of parenteral antischistosomal therapy (Pellicano et al., 2004). In retrospective studies in individuals with chronic HCV infections, cirrhosis of the liver occurred in 17-55%, hepato-cellular carcinoma (HCC) developed in 1-23%, and liver related death occurred in 4-15%. In prospective studies, cirrhosis developed in 7-16% of chronically infected individuals, HCC occurred in 0.7% - 16%, and liver related death in 1.3-3.7% HCC, by itself, is the third leading cause of cancer related deaths worldwide with 40.1% of patients with HCC being anti-HCV positive. Interestingly in the absence of liver disease, chronic infection with the hepatitis C virus (HCV) compromises health related quality of life with profound negative impacts on both physical and mental well being, similar to other chronic conditions. In a case control study conducted in Nigeria the prevalence of anti-HCV was 14.4% in study group and 2.4% in control group, which was significantly higher in patients with CLD than in the control ($P<0.05$) (Olokova et al., 2008).

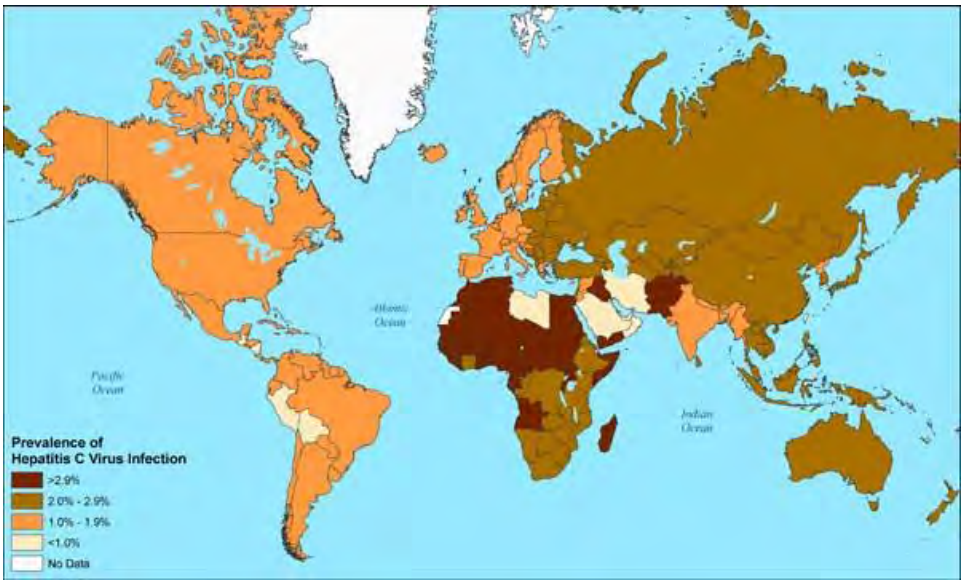

Figure 2. Estimated global prevalence of hepatitis C virus infection (adapted from WHO, 2004)

Sera of 200 recent Ethiopian immigrants (less than 1 year in Israel) were tested for the presence of antibodies to hepatitis C viruses. The prevalence rate was found to be (3%) (Flatau et al., 1991). Another study which aims to assess the prevalence and possible aetiological association of HCV with chronic liver disease and hepatocellular carcinoma HCC, anti-HCV were determined by enzyme-linked immunosorbent and recombinant immunoblot assays in 500 healthy volunteer blood donors, 14 patients with chronic hepatitis, 156 cirrhotics and 68 cases of HCC in Ethiopia. The prevalence of anti-HCV was 1.4%, 21%, 36% and 46%, respectively (Tsega et al., 1995). Another study done in Ethiopia on various group of subjects has demonstrated 2% sero-prevalence (Frommel et al., 1993) and a population based sero-prevalence study revealed 0.9% prevalence (Ayele et al., 2002). Recent study done in Gondar on blood donors estimated the prevalence of HCV to be 0.7%. Previous studies estimated the prevalence of HCV to be 1.4%. As per WHO, the prevalence of HCV in Ethiopia is estimated to be within a range of 2-2.9.

2.2.3 HBV/HCV co-infection

Due to shared routes of transmission, co-infection with HBV and HCV is not uncommon among individuals in HBV endemic areas who also have a high risk of parenteral infections, such as injection drug users (Pallas, 1999), patients on hemodialysis (Reddy, 2005), patients undergoing organ transplantation (Aroldi, 2005) and HIV-positive individuals (Zhou, 2007). Dual infection with HBV and HCV in the same host ranges from 9% to 30%, depending on the geographic region (Zarski, 1998; Liaw, 1995). These numbers may underestimate the true number of people with HBV/HCV co-infection as there is a well-known entity of occult HBV infection (i.e., patients with negative hepatitis B surface antigen (HBsAg) but detectable serum HBV DNA) in patients with chronic hepatitis C (Cacciola, 1999).

Patients with both HBV and HCV infections may show a large spectrum of virologic profiles. HCV infection can suppress HBV replication and it has been shown that HBV/HCV co-infected patients have lower HBV DNA levels, decreased activity of HBV DNA polymerase, and decreased expression of HBsAg and hepatitis B core antigen in the liver (Chu, 1998). Moreover, patients with chronic HBV infection who become superinfected with HCV can undergo sero-conversion of HBsAg (Liaw, 1994; Liaw, 1991). Several authors have reported that HBV can reciprocally inhibit HCV replication as well (Sato 1994). Specifically, HBV DNA replication has been shown to correlate with decreased HCV RNA levels in co-infected patients (Zarski, 1998). Furthermore, co-infected patients have been shown to have lower levels of both HBV DNA and HCV RNA than corresponding monoinfected controls, indicating that simultaneous suppression of both viruses by the other can also occur (Jardi, 2001). Thus, HBV or HCV can play the dominant role, HBV and HCV can inhibit each other simultaneously and they can alternate their dominance (Liaw, 1995). Both viruses have the ability to induce sero-conversion of the other. The chronology of infection may have a role in determining the dominant virus. However, the overall effect appears to be HCV suppression of HBV (Liaw, 2001).

3. Significance of the study

Representative sero-prevalence data is essential to estimate the true burden and distribution of viral hepatitis in chronic liver disease patients. However information on the prevalence of HBV and/or HCV in CLD in Ethiopia is sparse and no recent evaluation of HBV and HCV infection has been carried out. These viral infections still poses a major health problem in Ethiopia. It is against this background that the study was set to evaluate the presence of HBV surface antigen and antibodies to HCV in patients clinically diagnosed with CLD visiting public hospitals in Addis Ababa, Ethiopia. Association of age, gender and marital status with prevalence of these viruses in clinically diagnosed for CLD patients was investigated. The study outcome will be vital for appropriate preventive strategies, particularly for the implementation and evaluation of vaccination strategies for hepatitis B. Therefore, markers for the two viral hepatitis infections was determined in serum samples provided by voluntary clinically diagnosed chronic liver disease participants in public hospitals of Addis Ababa, Ethiopia.

4. Objective of the study

4.1. General Objective

- To determine the prevalence of HBV and HCV infection among clinically diagnosed chronic liver disease patients visiting OPD of public hospitals of Addis Ababa, Ethiopia.

4.2. Specific Objective

- To determine HBV sero- prevalence among clinically diagnosed chronic liver disease patients
- To determine HCV sero- prevalence among clinically diagnosed chronic liver disease patients
- To determine prevalence of HBV and HCV co-infection among chronic liver disease patients
- To describe HBV and HCV infection with respect to socio demographic variables
- To determine the risk factors for HBV and HCV infection in chronic liver disease patients

5. Materials and Methods

5.1. Study design and study period

A hospital based cross sectional study was conducted in Addis Ababa public hospitals; St. Paul, Tikur Anbessa, and Zewditu Memorial hospitals, from November 2010 to May 2011.

5.2. Study Area

The study was conducted in Addis Ababa; the capital city of Ethiopia, which lies at an altitude of 7,546 feet (2,300 meters) with a grassland biome. The city lies at the foot of Mount Entoto. From its lowest point, around Bole International Airport, at 2,326 meters (7,631 ft) above sea level in the southern periphery, the city rises to over 3,000 meters (9,800 ft) in the Entoto Mountains to the north. According to the preliminary 2007 census result Addis Ababa has a total population of 2,738,248, consisting of 1,304,518 men and 1,433,730 women. The city contains 22.9% of all urban dwellers in Ethiopia. With an estimated area of 53,014 square kilometers, the city has an estimated density of 5,165.1 inhabitants per square kilometer (13,378/sq. m).

5.3. Study Variables

Dependent variables: - Hepatitis B and Hepatitis C sero- status

Independent variables: - Age, sex, marital status, occupational status, risk factors for HBV and HCV (blood transfusion history, abortion, history of surgical procedure, uvuloctomy, ear/nose piercing, circumcision, contact with jaundiced patients, dental extraction, tattooing, shaving by barbers)

5.4. Sample size estimation and Sampling technique

Convenient sampling method was used in which selection of the sample was based on the easy accessibility of the patient during the study period. Sample size for the study was calculated using a single population proportion (p).

$$ni = \frac{Z^2 \alpha_{/2} P(1-P)}{d^2}$$

Where ni= sample population for n>10,000

P = prevalence of HBV/HCV antigen

d = marginal error (0.05)

Z (α/2) = the reliability coefficient of 95% i.e. 1.96

By using this formula the calculated sample size was 126.

5.5. Source population

All patients who were visiting Addis Ababa public hospitals during the data collection period were the source population.

5.6. Study population

Clinically diagnosed CLD patients who were visiting Addis Ababa public hospitals; St. Paul, Tikur Anbessa and Zewditu memorial hospitals, during the study period were the study population. The clinical diagnostic criteria for grouping patients as chronic liver disease patients were based on the presence of patient history of either:-

a) Clinical signs like ascites, hepatomegaly, splenomegaly with other clinical features including jaundice, palmar erythema, clubbing, edema, axillary and pubic hair loss, Spider nevi, flapping tremors, drowsiness, confusion and coma.

b) Ultra sound showing coarse hepatic texture, changes in liver size, increased portal vein diameter, and splenomegaly.

c) Impaired liver function tests including raised level of alanine aminotransferase (ALT).

5.7. Eligibility criteria

5.7.1. Inclusion criteria

- All voluntary adult patients (age 18 and above) with clinically recognizable chronic liver disease in the three public hospitals during the study period were eligible.

5.7.2. Exclusion criteria

- Patients clinically diagnosed with chronic liver disease; who were not willing to participate in the study and age below 18 were excluded.

5.8. Data collection and processing

Clinically diagnosed chronic liver disease patients were sorted by the clinical history on their patient history sheet. All patients, whose clinical history report they are chronic liver disease patients were contacted. Then study participants who fulfill eligibility criteria were interviewed by the investigator to gather socio demographic and other risk factor data by using pre-designed questionnaire after consent was obtained. 5ml venous blood was collected at spot by the

investigator and was left for 30 minutes to facilitate clotting. Then the clotted blood was centrifuged to separate the serum from blood. The serum was divided in two aliquots. One of the aliquot was used for HBsAg screening as per manufacturer instruction. The other aliquot was used for Anti-HCV antibody screening as per manufacturer instruction.

5.8.1. Serological Test Principle

5.8.1.1. Instant HBsAg:

One Step test for HBsAg (INSTANT HBsAg one step test for HBsAg Device, TULIP DIAGNOSTICS (P) LTD., INDIA) utilizes the principle of immunochromatorgraphy, a unique two site immunoassay on a membrane. As the test sample flows through the membrane assembly of the dipstick, the colored anti-HBsAg colloidal gold conjugate complexes with HBsAg in the sample. This complex moves further on the membrane to the test region where it is immobilized by the anti-HBsAg coated on the membrane leading to formation of a pink-purple colored band which confirms a positive test result. Absence of this colored band in the test region indicates a negative test result. The unreacted conjugate and unbound complex, if any move further on the membrane and are subsequently immobilized by the anti-rabbit antibodies coated on the membrane at the control region, forming a pink-purple colored band. This control band serves to validate the test result.

5.8.1.2. Rapid Anti-HCV test

Rapid Anti-HCV test (one step HCV serum/plasma test strip, Biocare TM DIAGNOSTICS, CHINA) is a rapid immunochromatographic direct binding test for the visual detection of hepatitis C virus antibodies in serum/plasma samples in the diagnosis of hepatitis C infection. This HCV test adopts double antigen sandwich method. When the test device is immersed into the specimen, the specimen is absorbed into the device by capillary action, mixes with the antigen-dye conjugate, and flows across the pre-coated membrane.

When the HCV antibody levels are at or above the target cutoff, HCV antibodies in the specimen binds to the antigen-dye conjugate and are captured by antigen immobilized in the Test region (T) of the device. This produces a colored test band and indicates a positive result.

When the HCV antibody levels are zero or below the target cutoff (the detection sensitivity of the test) there is not a visible colored band in the test region (T) of the device. This indicates a negative result.

5.9. Quality Control

- Each questionnaire was checked for completeness.
- Standard operation procedures (SOPS) was followed during laboratory analysis

5.10. Data analysis and interpretation

The collected data were entered in Epi Info 3.5.1 and analyzed using SPSS 17.0 statistical software. They were organized and summarized in terms of frequencies and the results of the study were presented in a descriptive measure such as tables and graphs. The chi-square (x2) test was utilized in assessing statistical significance of association that could exist between measured variables and a *p-value* of less than 0.05 was considered as significant. Odds ratio (OR) and 95% confidence interval (CI) were used a measure of the strength of association.

5.11. Ethical Considerations

The study protocol together with consent form was submitted to DMIP scientific and ethical committee and to Addis Ababa Health Bureau before the study started. After getting ethical clearance, letter of support was written to public hospitals found in Addis Ababa by DMIP and Addis Ababa health Bureau. Consent was taken from each patient whenever it was appropriate to collect the 5ml venous blood from patients. Participation in the study was on voluntary basis and after informed consent has been obtained. The patients were free to withdraw from the study before the collection of blood without losing any of the benefits they were supposed to obtain from the hospital.

While collecting venous blood the patients might experience pain therefore maximum effort was taken to minimize the pain and/or associated complications. All samples were collected using disposable tubes, syringes and needles.

6. Results

6.1. Study participants

A total of 120 study participants who were clinically diagnosed for CLD were included in the study for the detection of HBV and HCV infections from November 2010 to May 2011 in three public hospitals (Tikur Anbassa, Zewditu Memorial and St. Paul Hospitals), Addis Ababa, Ethiopia. The gender profile of these 120 study participants showed that 63.3% (76/120) were males and 36.7% (44/120) were females resulting in overall male to female ratio of about 1.73:1. According to gender distribution, males took the higher proportion in all age groups. A higher; sex specific proportion of female participants was observed both in age 28-37 and 38-47 years relative to the other age groups. The mean age of the participants was 40.99 years ± 14 SD, with range from 18 to 80 years. About ninety-eight percent (98.3%) of the study participants were between the age 18 and 67 years. 75.8% were below the age 50.

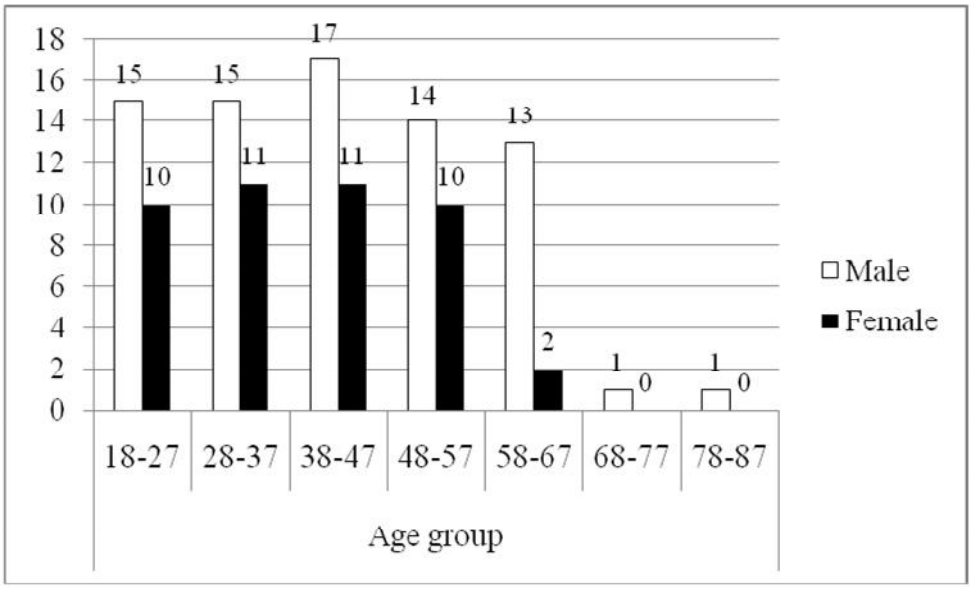

Figure 3. Age and sex distribution of chronic liver disease patients in three public hospitals of Addis Ababa, Nov 2010– May 2011.

In relation to marital status 55.8% (67/120) of the study participants were married, 26.7% (32/120) were single and 9.2% (11/120) divorced, whereas the remaining 8.3% (10/120) were widow with no statistical difference.

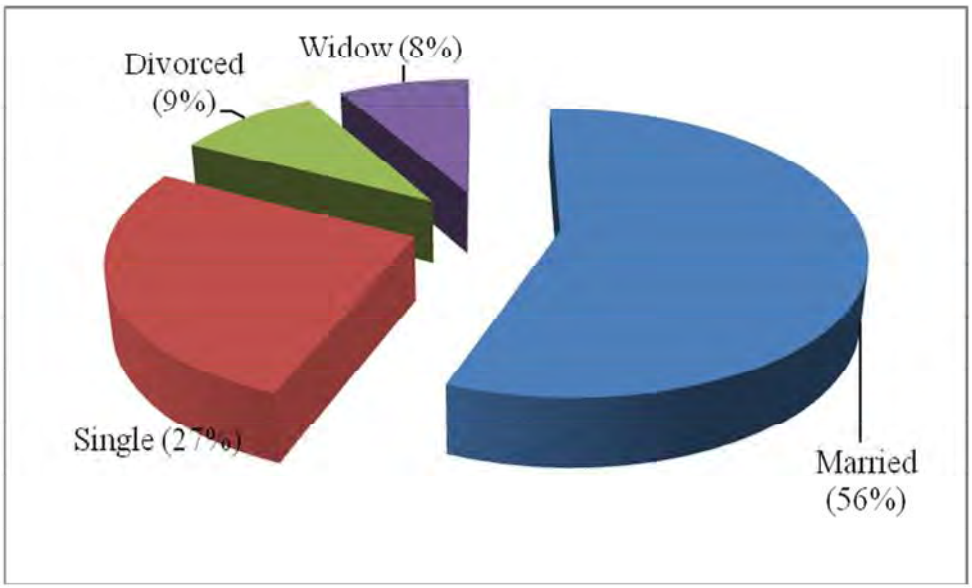

Figure 4. Marital status of chronic liver disease patients in three public hospitals of Addis Ababa, Nov 2010 – May 2011.

Concerning ethnicity majority 47.5% (57/120) of chronic liver disease patients were Amhara followed by Oromo 20.8% (25/120) and Gurage 20.0% (24/120). There was no significant difference between being Amhara, Oromo or other ethnic group in relation to HBV and HCV infection. The same was true concerning religion with which 39% (39/100) and 24% (24/100) among Christians were sero-positive for HBV and HCV respectively which took the higher proportion than Muslims accounting 20% (4/20) and 15% (3/20) respectively for both virus markers. In relation to living area, 75.8% (91/120) of the study participants were urban dwellers and the remaining 24.2% (29/120) were rural dwellers.

Regarding educational status the study revealed two extremes; in which 27% (33/120) of the study participants had above grade 12 education and 20.0% (24/120) of the study participants were not educated/ had not received formal education. But there was no statistically significant relation between illiterates and the educated in acquisition of HBV and HCV infection ($P > 0.05$). The prevalence of HBsAg and anti-HCV Ab in relation with socio-demographic characteristics are shown in Table 2.

Table 2 The prevalence of HBsAg and anti-HCV Ab in relation with socio-demographic characteristics in Tikur Anbessa, Zewditu Memorial and St. Paul hospitals, Nov 2010–May 2011

Socio-demographic characteristics		Sero-status for HBV		Sero-status for HCV	
		Positive No. (%)	Negative No. (%)	Positive No. (%)	Negative No. (%)
Gender	Male	29 (38.2)	47 (61.8)	14 (18.4)	62(81.6)
	Female	14 (31.8)	30 (68.2)	13 (29.5)	31(70.5)
Marital status	Married	23 (34.3)	44 (65.7)	18 (26.9)	49 (73.1)
	Single	15 (46.9)	17 (53.1)	1 (3.1)	31 (96.9)
	Divorced	3 (27.3)	8 (72.7)	3 (27.3)	8 (72.7)
	Widow	2 (20.0)	8 (80.0)	5 (50.0)	5 (50.0)
Residence	Urban	34 (37.4)	57 (62.6)	22 (24.2)	69 (75.8)
	Rural	9 (31.0)	20 (69.0)	5 (17.2)	24 (82.8)
Religion	Christian	39 (39.0)	61 (60.0)	24 (24.0)	76 (76.0)
	Muslim	4(20.0)	16(80.0)	3(15.0)	17(85.0)
Educational status	Illiterate	6(26.1)	17(73.9)	6(26.1)	17(73.9)
	Can write and read	1(10.0)	9(90.0)	2(20.0)	8(80.0)
	Grade 1-4	2(50.0)	2(50.0)	0(0.0)	4(100.0)
	Grade 5-8	10(50.0)	10(50.0)	3(15.0)	17(85.0)
	Grade 9-10	5(33.3)	10(66.7)	5(33.3)	10(66.7)
	Grade 11-12	7(46.7)	8(53.3)	4(26.7)	11(73.3)
	>12	13(39.4)	20(60.6)	6(18.2)	27(81.8)
Ethnicity	Oromo	10 (40.0)	15 (60.0)	3(12.0)	22(88.0)
	Amhara	24(42.1)	33(57.9)	12(21.1)	45(78.9)
	Tigrae	0(0.0)	7(100.0)	3(42.9)	4(57.1)
	Gurage	5(20.8)	19(79.2)	8(33.3)	16(66.7)
	Wolayta	4(57.1)	3(42.9)	1 (14.3)	6 (85.7)
Current occupation	Driver	1(20.0)	4(80.0)	2(40.0)	3(60.0)
	Jobless	4 (30.8)	9(69.2)	3(23.1)	10 (76.9)
	Daily laborer / house servant	3(42.9)	4(57.1)	0(0.0)	7(100.0)
	Commercial	2(28.6)	5(71.4)	3(42.9)	4(57.1)
	Student	4(50.0)	4(50.0)	0(0.0)	8(100.0)
	Government / Private employee	21 (45.7)	25 (54.3)	11 (23.9)	35 (76.1)
	Farmer	7 (41.2)	10(58.8)	4 (23.5)	13 (76.5)
	House wife	1(7.1)	13(92.9)	3(21.4)	11(78.6)
	Others	1(33.3)	2 (66.7)	0(0.0)	3 (100.0)

6.2. HBV and HCV

6.2.1. Magnitude and Distribution of Hepatitis B infection by age, sex, religion and marital status

The assessment of hepatitis B prevalence among chronic liver disease patients was made by detecting HBsAg in serum. The presence of this marker was taken as measure of infection. The prevalence of HBsAg in chronic liver disease was 35.8% (43/120). The sex specific prevalence rate was higher in males 38.2 % (29/76) than females 31.8% (14/44) but the difference did not reach statistical significance (COR= 1.322; 95% CI: 0.603-2.900; P=0.556). The prevalence of HBV by specific age group was highest 61% (16/26) in the age group of 28-37 and was lowest (0%) in both the age groups of 68-77 and 78-87 (Table 2). (Fig. 5)

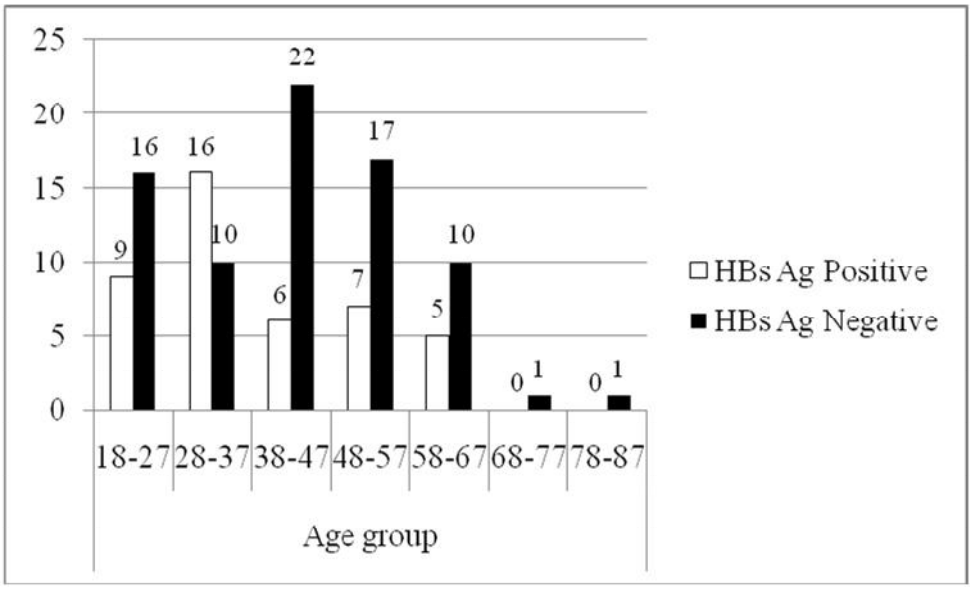

Figure 5. The distribution of HBsAg by age among CLD patients at Tikur Anbessa, Zewditu Memorial, and St. Paul hospitals Nov 2010 – May 2011

Concerning religion distributions of HBsAg positivity was higher among Christians accounting 39% (39/100) and it was only 20% (4/20) among Muslims showing no statistical difference (P>0.05). With regard to educational status highest percent was found in study participants with an education level above grade twelve with a frequency of 13/120 (10.8%) followed by grade 5-8 (7.5%) and least frequency was found with study participants who can only read and write.

In relation to living area 28.3% (34/120) HBsAg tested positive study participants were urban dwellers and 7.5% (9/120) were rural dwellers. With regard to marital status the highest percent of positivity was taken by married with a prevalence of 19.2% (23/120) followed by singles 12.5% (15/120) and a least prevalence was observed in widows 1.7% (3/120).

Table 3. HBV and HCV prevalence by age and sex among CLD patients in Tikur Anbessa, Zewditu Memorial and St. Paul hospitals, Nov 2010–May 2011

	HBsAg positive (%)		Anti-HCV Ab positive (%)	
Age group (years)	Male	Female	Male	Female
18-27	6/15(40.0)	3/10(30.0)	1/15(6.7)	1/10(10.0)
28-37	9/15(60.0)	7/11(63.6)	1/15(6.7)	3/11(27.3)
38-47	5/17(29.4)	1/11(9.1)	4/17(23.5)	3/11(27.3)
48-57	5/14(35.7)	2/10(20.0)	5/14(35.7)	6/10(60.0)
58-67	4/13(30.8)	1/2(50.0)	3/13(23.1)	0/2(0.0)
68-77	0/1(0.0)	-	0/1(0.0)	-
78-87	0/1(0.0)	-	0/1(0.0)	-
Total	29/76(38.2)	14/44(31.8)	14/76(18.4)	13/44(29.5)

6.2.2. Magnitude and Distribution of Hepatitis C infection by age, sex, religion and marital status

The assessment of hepatitis C prevalence among chronic liver disease patients was made by detecting anti-HCV Ab. The presence of this marker was taken as measure of infection. Twenty seven 27/120 (22.5 %) of the study participants were positive for anti-HCV Ab. The sex specific prevalence rate was higher among females 29.5% (13/44) than males (18.4%) (14/76) (but the difference did not reach statistical significance (COR=0.538; 95% CI: 0.226-1.284; P= 0.179). Majority 10% (12/120) of anti-HCV Ab positive CLD patients were in the ethnic group Amhara (7 males and 5 females) followed by Guragae 6.7% (8/120) where 50% of them were males and 50% females..

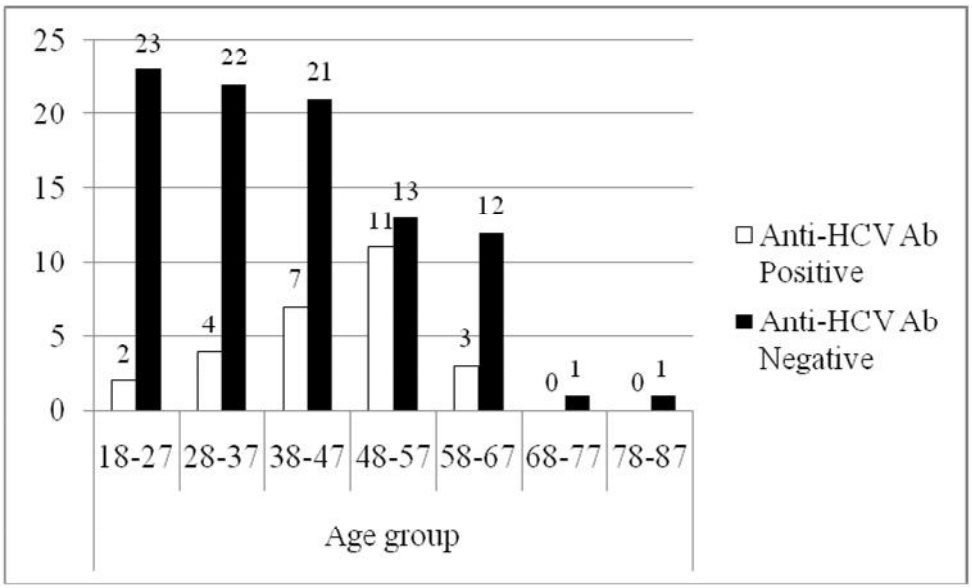

Figure 6. The distribution of anti-HCV Ab by age among CLD patients at Tikur Anbessa, Zewditu Memorial, and St. Paul hospitals Nov 2010 –May 2011

The magnitude of HCV by specific age group was highest in the age group of 48-57 years and was lowest above 68 years of age (Fig 6). The prevalence of anti-HCV progressively increases from 1.67% in the age group 18-28 to reach a peak in the age group 48-57 years (9.16%) then progressively decline to 0% in the age group >68 years (Table 3).

With respect to marital status sero-positivity for anti-HCV Ab was highest among widow 50% (5/5) followed by divorced 37.5% (3/8). Concerning religion and ethnicity, similar to HBV positivity, the ethnic group Amhara and Christians took the highest portion for anti-HCV Ab positivity with figure of 20% (24/120) and 10% (12/120) respectively.

6.2.3. Prevalence of HBV and HCV co-infection

The detection of co-infection with both HBV and HCV in CLD patients was based on the presence of combination of HBsAg and anti-HCV Ab. Thus the current study has revealed 2.5% (3/120) of the study participants screened for both HBsAg and anti-HCV Ab were positive for both sero-markers. The gender profile for these participants, who have both HBV and HCV infections, showed all were females aged <58 years and two of them were Amhara and one of them was Guragae. In an interesting manner all the females who were positive for both sero-

markers have history of circumcision and 2 (66.7%) of them have histories of home delivery with traditional birth attendants even though there was no significant association found.

Table 4. The prevalence of HBV/HCV co-infection among CLD patients in Tikur Anbessa, Zewditu Memorial and St. Paul hospitals, Nov 2010–May 2011

Sero-status for HBV	Sero-status for HCV		Total
	Positive	Negative	
Positive	3	40	43
Negative	24	53	77

6.3 Risk factors and distribution of HBV and HCV infection

Among 120 CLD patients 70 (58.3%) reported that they had a history of hospital admission within which 28.6% (20/70) and 22.9% (16/70) were found to be positive for HBsAg and anti-HCV Ab respectively; and 30% (21/70) of them had a history of either a minor or major surgery for which 4.28% (3/70) and 7.14% (5/70) were found to be positive for HBsAg and anti-HCV Ab respectively. Even though 70 (58.3 %) of the patients had reported they had history of hospital admission only 35.7% (25/70) of them had history of blood transfusion showing majority of the study participants with history of hospitalization (64.3%) had no history of blood transfusion. However, no statistical significance was found ($P>0.05$) in history of hospital admission, surgical procedure and blood transfusion in relation with HBV and HCV infections.

Concerning dental extraction 42.5% (51/120) of CLD patients reported that they had history of dental extraction at health facilities and 34.2% (41/120) had history of dental extraction at home. About seven percent 8/120 (6.7%) of the study participants have also reported that they had history of dental extraction at home and health facilities. From those having dental extraction at health facility 35.2% (18/51) were positive for HBsAg (COR= 0.960; 95% CI: 0.451-2.044, P= 1.00) and 33.3% (17/51) were positive for anti-HCV Ab (COR= 2.95; 95% CI= 1.21-7.17, P= 0.015). Those who had dental extraction at health facilities were 2.95 time more likely to have infection with HCV than their counter parts and the difference was statistically significant ($P<0.05$).

Participants who had contact with jaundiced person account 20.8% (25/120) and those who didn't have account 79.2% (95/120). From the participants with a history of contact with jaundiced person 44% (11/25) were positive for HBsAg and 24% (6/25) were positive for anti-HCV Ab showing no significant difference (AOR= 0.461; 95% CI: 0.150-1.422, P= 0.178) and (AOR= 0.342; 95% CI: 0.197- 3.17=64, P= 0.739) respectively.

Table 5. Risk factors distribution among CLD patients with respect to sero-status of HBV in three public hospitals of Addis Ababa, Nov 2010 – May 2011

Risk factors	HBsAg (%)			OR (95%, CI)	*p value*
	Positive	Negative	Total		
Community acquired					
Tattooing on gum	8(40.0)	12(60.0)	20	1.24(0.46-3.31)	0.67
Tattooing on body	5(38.5)	8(61.5)	13	1.14 (0.35-3.71)	0.53
Venous or body piercing	8(42.1)	11(57.9)	19	1.37(0.51-3.72)	0.53
Ear piercing	16(31.4)	35(68.6)	51	0.711(0.33-1.53)	0.38
Uvuloctomy	14 (27.5)	37 (72.5)	51	0.52(0.24-1.14)	0.09
Shaving in barbershop ***	22(42.3)	30(57.7)	52	1.78(0.63-5.03)	0.27
Contact with jaundiced patient	11(44.0)	14(56.0)	25	1.55(0.63-3.79)	0.34
Circumcision	43(36.4)	75(63.6)	118	NA	0.41
Dental extraction at home	15(36.6)	26(63.4)	41	1.05(0.48-2.30)	0.90
Hospital acquired					
Hospitalization	20(28.6)	50(71.4)	70	0.47(0.22-1.00)	0.05
Blood transfusion	6(22.2)	21(77.8)	27	0.43(0.16-1.17)	0.09
Dental extraction	18(35.3)	33(64.7)	51	0.96(0.45-2.04)	0.92
Surgical procedure	4(17.4)	19(82.6)	23	0.31(0.09-0.99)	0.04
Behavioral acquired					
Alcohol consumption	13(37.1)	22(62.9)	35	1.08(0.48-2.45)	0.85
Delivery by TBA**	6(31.6)	13(68.4)	19	0.98(0.27-3.53)	0.98
Abortion**	3(25.0)	9(75.0)	12	0.64(0.14-2.84)	0.42

= concerning female *= concerning male NA= not applicable

Table 6. Risk factors distribution among CLD patients with respect to sero-status of HCV in three public hospitals of Addis Ababa, Nov 2010 – May 2011

Risk factors	Anti-HCV-antibody (%)			OR (95%, CI)	*p value*
	Positive	Negative	Total		
Community acquired					
Tattooing on gum	5(25.0)	15(75.0)	20	1.18(0.39-3.61)	0.48
Tattooing on body	1(7.7)	12(92.3)	13	0.26(0.03-2.09)	0.16
Venous or body piercing	3(15.8)	16(84.2)	19	0.60(0.16-2.24)	0.33
Ear piercing	15(29.4)	36(70.6)	51	1.98(0.83-4.71)	0.12
Uvuloctomy	15 (29.4)	36 (70.6)	51	1.98(0.83-4.71)	0.12
Shaving in barbershop***	8(15.4)	44(84.6)	52	0.55(0.17-1.79)	0.24
Contact with jaundiced patient	6(24.0)	19(76.0)	25	1.11(0.39-3.14)	0.84
Circumcision	27(22.9)	91(77.1)	118	NA	0.59
Dental extraction at home	8(19.5)	33(80.5)	41	0.77(0.30-1.94)	0.57
Hospital acquired					
Hospitalization	16(22.9)	54(77.1)	70	1.05(0.44-2.51)	0.91
Blood transfusion	8(29.6)	19(70.4)	27	1.64(0.62-4.32)	0.31
Dental extraction	17(33.3)	34(66.7)	51	2.95(1.21-7.17)	0.02*
Surgical procedure	5(21.7)	18(78.3)	23	0.95(0.32-2.84)	0.92
Behavioral acquired					
Alcohol consumption	11(31.4)	24(68.6)	35	1.98(0.81-4.85)	0.13
Delivery by TBA**	7(36.8)	12(63.2)	19	1.85(0.49-6.83)	0.36
Abortion**	5(41.7)	7(58.3)	12	2.14(0.52-8.68)	0.24

*= having statistical association **= concerning female ***= concerning male NA= not applicable

In summary with regard to HBV and HCV risk factors, which include ear piercing, tattoo, tooth extraction at home, hospital admission, history of transfusion, contact with jaundiced person, history surgical procedure, venous or body piercing for treatment, abortion, history of home delivery with traditional birth attendants, uvuloctomy and circumcision; none of these were significantly associated with HBV and HCV sero-positivity in chronic liver disease patients ($p > 0.05$) except history of dental extraction at health facility with respect to HCV infection (Table 5 and 6).

7. Discussion

Hepatitis B and Hepatitis C viral infections are among the most prevalent infectious diseases in humans worldwide. Both infections are associated with a broad range of clinical presentations ranging from acute or fulminant hepatitis to chronic infection that may be clinically asymptomatic or may progress to chronic hepatitis and liver cirrhosis (Sebastion et al., 1990). Hepatitis B and C virus infections are important community health problems in Ethiopia as different studies have examined HBV and HCV infection in different study population (Tsega 1991, Tsega et al., 1995, Baye and Yohannes, 2008 and Belay et al., 2010). The current study was set to examine the prevalence of HBV and HCV infections among chronic liver disease patients.

A total of 120 chronic liver disease patients participated in the study. In this study, patients presenting with chronic liver disease 35.8% tested positive for HBsAg and 22.5% tested positive for anti-HCV Ab. However, it should be noted that the kits used were not confirmatory. A similar study conducted in North India has reported high prevalence of HBV (55%) and HCV (25.75%) in chronic liver disease patients (Chakravarti and Verma, 2005). Moreover other studies from different parts reported high prevalence of HBV and HCV infection in chronic liver disease patients. For HCV prevalence rates of 26%, 46% and 75.5% in cases of chronic liver disease have been reported from India (Issar et al., 1995), Ethiopia (Tsega et al., 1995) and Egypt (Waked et al., 1995) respectively and also higher HBV prevalence reports were 22% from Sudan in HCC patients (Hatim, 2008) and 30.35% from Pakistan in chronic liver disease patients (Taher and Farhat, 2000). Our results reflect high prevalence for both viruses in this patient population; therefore we can say HBV and HCV are important, if not the primary, causes of chronic liver disease.

Age distribution of CLD in our study population showed that 75.8% of CLD cases were before fifty years of age. 7.5% of patients were up to 20 years of age, about 22.5% in second and 23.3% in third decade and 22.5% in fourth decade of life. A comparable high age distribution (42%) of CLD cases age <50 years was observed by Bukhtiari et al., 2003. A high prevalence of HBV was recorded in the age group 28-37 with a prevalence of 61.5% (16/26) and for HCV it was 45.8% (11/24) in the age group 48-57. The reason could be an early childhood infection with the

hepatitis viruses. The chronic infection with hepatitis viruses leads to slow progressive liver disease. It may end up in cirrhosis, chronic liver failure and hepatocellular carcinoma over a period of up to 30 years (Shimotohno, 2000). The low prevalence of hepatitis virus infection in the older age group may reflect loss of carrier status due to sero-conversion with age.

HBV and HCV share a common route of transmission and can coexist with each other. Co-infection with evidence of chronic HBV and HCV seems to result in more severe liver disease than either infection alone (Sato et al., 1994), with an increase risk of liver cancer (Zarik et al., 1998) and probably an increase risk of fulminant hepatitis when superinfection with HCV occurs on the background of chronic HBV (Liaw, 1995). In our study the detection of co-infection with both HBV and HCV in CLD patients was based on the presence of combination of HBsAg and anti-HCV Ab. Out of 120 chronic liver disease patients dual infection was observed in 3 patients (2.5%). The gender profile for these participants, who have both HBV and HCV infections, showed all were females aged <58 years. Nearly similar result was reported in a study on the prevalence of HBV and HCV dual infection in patients on haemodialysis with a prevalence of 3.5% (Reddy et al., 2005). In addition, a comparatively higher co-infection of HBV and HCV is frequently found in patients undergoing organ transplantation (8%) (Aroldi, 2005), in injection drug users (IDU, 42.5%) (Pallas, 1999). However, these numbers may underestimate the true number of people with HBV/HCV co-infection as there is a well-known entity of occult HBV infection (i.e., patients with negative hepatitis B surface antigen (HBsAg) but detectable serum HBV DNA) in patients with chronic hepatitis C (Cacciola, 1999). In addition the difference in the magnitude of co-infection among these studies and our study could be attributable to difference in the study population, geographical variation and difference in methodology.

In our study, cases of HCV associated CLD was much less common than the HBV associated cases and this higher HBV to HCV pattern is well recognized in Ethiopia for the last few years as many studies have shown on different study populations, in Gondar HBV to HCV was 4.1%/0.7% (Belay et al., 2010), in Amhara and Tigray regional state 6.2%/1.7% (Baye and Yohannes, 2008). Similarly studies carried out in different countries on different study populations reported results which are in line with our study outcome; in Vietnam which is a developing country, CLD due to HBV was 47% while 23% of these cases were HCV sero-positive (Kakum et al., 1998) and also higher HBV prevalence reports were 22% from Sudan in

HCC patients (Hatim, 2008) and 30.35% from Pakistan in CLD patients (Taher and Farhat, 2000). Contrary to the above reports different studies reported higher prevalence of HCV to HBV in different study populations; in Pakistan (64.9%/24.7%) (Bukhtiari et al., 2003) in Czech Republic (31%/26%) (Strhnsl et al., 1997). Higher HCV prevalence (73.5%) was reported among chronic liver disease in a study conducted in Egypt (Waked et al., 1995). The higher prevalence of HCV to HBV infection in the other studies could be explained by geographical variation and with the fact that Hepatitis C virus has a higher propensity for causing liver disease than Hepatitis B virus (Kumar and Contran, 1999).

The sex specific frequency of HBV and chronic liver disease was more among males than females while the frequency of HCV was higher among females than males. The greater frequency of chronic liver disease and HBV infection in males as compared to females could be a reflection of more males coming for treatment in our setting. Besides it could be due to more social mobility in males than females and thus greater vulnerability to be infected. However, there was no significant differences in HBV sero-prevalence among sex (p>0.05). Thus this finding can suggest that gender may not enhance susceptibility to HBV infection due to differences in risk-behavior. Comparable results were reported in a number of studies concerning HBV infection, Ethiopia [4.9% (273/5592) in males and 3.3 % (25/769) in females] (Belay et al., 2010), Pakistan [72% (26/36) in males and 28% (10/36) in females] (Ahmad et al., 2006).

In contrast to the results of HBV, the prevalence of HCV in sex was higher in females (29.5%) than males (18.4%) as our result revealed. Our result is consistent with a study done by Ahmad et al., 2006 on “Frequency of Hepatitis B and Hepatitis C among cataract patients” which reported a frequency of 45% (13/29) in males and 55% (16/29) in females. In addition a study conducted in Madagascar on prevalence of HCV reported higher prevalence of anti-HCV in females than male (Charles et al., 2008). However, not any known risk exposure that could be an explanation for the difference observed by gender was inferred from this data.

The prevalence of HBV and HCV among chronic liver disease patients who had history of either minor or major surgery was 17.4% (4/23) and 21.7% (5/23) respectively. However, history of either minor or major surgery could not be inferred from this study as a cause of infection either by HBV or HCV. In comparison a comparatively low prevalence was reported in a study aimed to determine the sero-prevalence of HBV and HCV viral infection in patients undergoing

elective eye surgery, out of 1418 study participants only 1.8% and 1.2% were sero-positive for HBV and HCV respectively (Akhatar et al., 2007). Similarly a study on the frequency of hepatitis B and hepatitis C among cataract patients reported a high prevalence, even though it was lower when compared to our finding, of HBsAg 3.18% and anti-HCV 2.57% in patients presenting for surgery (Ahmad et al., 2006). In addition a result which was contrary to our finding was reported in a local study by Pasquini et al., 1988. The study revealed exposure to three or more tribal practices, such as ritual scarring or traditional surgery, is associated with an increased risk of hepatitis B infection (AOR=1.7(1.1-2.6)).

The overall prevalence of HBV and HCV infection in those study participants who had history of blood transfusion was 22.2% and 29.6% respectively. Local studies on Hepatitis B and C in blood donors have reported (4.7% and 0.7%) in Gondor (Belay et al., 2010) and (6.2% and 1.7%) in Amhara and Tigray regional states (Baye and Yohannes, 2008). In comparison to our study a study in Kosovo by Fejza and Telaku (2009) has reported a much lower prevalence (4.2% and 0.27%) of HBV and HCV among blood donors. With regard to history of blood transfusion no association was found in acquisition of HBV and HCV infection. This might be explained by the currently implemented screening for potential blood borne pathogens in blood banks.

Univariate analysis showed a strong association between history of dental extraction at health facility and HCV infection. The overall prevalence of HCV among chronic liver disease patients who had history of dental extraction at health facility was 33.3% (17/51) (P= 0.015). Those who had history of dental extraction at health facilities were 2.95 time more likely to have infection with HCV than their counter parts and the difference was statistically significant. This might be explained by poor sterilization procedures for reusable materials in dental clinics. Correspondingly, this finding agreed with a study conducted in Madagascar (Charles et al., 2008) and a very strong association of liver disease was found with a history of dental treatment (38% HCV with an Odd ratio 2.3; 95% CI:1.8-3) (Huma et al., 2009). However, no other apparent risk factor that caused HCV infection was inferred from our study.

A study by (Ahmed et al., 2006) revealed the frequency of Hepatitis B and Hepatitis C amongst urban and rural population as 45% and 55% which is completely different from our finding. Our finding showed greater frequency in urban than rural population. A coherent higher frequency in urban dwellers was also observed in a local study on the prevalence of HBV in Aris, Ethiopia

(12.6%/8.7%) by Pasquini et al., 1988. This greater frequency of infection in urban population may be explained by the nature of living; in which urban areas are densely populated as compared to rural areas and this might expose urban dwellers for different risk factors and hence infection.

Regarding the various occupational groups the distribution of HBV infection was, one (20%) out of 5 drivers, 3 (42.9%) out of 7 daily laborer/house servant, 2 (28.6%) out of 7 merchants, 21 (45.7%) out of 46 office workers, 7 (41.2%) out of 17 farmers and 1 (7.1%). No significant relationship was found between occupation and HBV infection ($p>0.05$). In the present study a higher prevalence of HBV was observed in daily laborer/house servant followed by office workers; however, because of their small numbers statistically conclusive inferences may be difficult to make. Yet, this higher rate of HBV infection in daily laborer/house servant and office workers might raise concern regarding need for implementation of more effective public education. Similarly the distribution of HCV infection in the various occupational groups was, 2 (40%) out of 5 drivers, 3 (23.1%) out of 13 who have no job, 0 (0%) out of 7 daily laborer/house servant, 3 (42.9%) out of 7 merchants, 0 (0%) out of 8 students, 11 (23.9%) out of 46 office workers, 4 (23.5%) out of 17 farmers and 3 (21.4%) out of 14 house wives. For HCV the higher sero-positivity was recorded among merchants even though it was not significantly associated with none of the occupational groups.

In summary this study has revealed high prevalence of HBV and HCV in chronic liver disease patients. However, neither associated risk factors nor associated socio-demographic characteristics have been significantly associated with acquisition of infection except history of dental extraction at health facility with respect to HCV infection.

Limitation of the study

- When human serum samples are tested for HBsAg and anti HCV antibodies using different commercial rapid test kits, discrepant results often occur. Usually confirmatory test is commonly used to evaluate positive rapid test results. Due to resource constraints this study was unable to conduct confirmatory tests for those positive results. Thus some false positive results might be expected.

- Chronic liver disease is a spectrum of diseases such as chronic hepatitis, liver cirrhosis and HCC. Our study was only set and able to measure the prevalence of HBV and HCV in clinically diagnosed chronic liver disease patients (in general term) and therefore this study didn't measure the prevalence of these viruses in the different sub-groups of chronic liver disease classification.

Conclusion and Recommendation

Conclusion

This study has found higher prevalence of hepatitis B and hepatitis C infection among chronic liver disease patients visiting Tikur Anbessa, Zewditu Memorial and St. Paul hospitals. There was no statistical difference in acquisition of HBV and HCV infection with respect to socio-demographic characteristics and risk factors except dental extraction at health facility for HCV infection. Hence, no apparent risk factor that caused HBV infection was inferred from this study.

Therefore we conclude HBV and HCV infection is high in chronic liver disease patients and those CLD patients who had history of dental extraction at health facility have 2.95 time higher risk of acquisition of HCV infection than those who do not have history of dental extraction at health facility.

Recommendations

Due to lack of education and awareness of this disease, HBV and HCV infections are rapidly spreading and becoming a major health problem due to its immediate and long-term effects. Proper measures must be adopted to educate the community and for application of preventive measures to prevent its spread in the community. We conclude that HBV and HCV are major health problem in chronic liver disease patients and therefore the following recommendations must be carried out for its prevention.

- All clinically diagnosed CLD patients should be tested for HBV and HCV sero status.
- Proper sterilization of dental and surgical instruments must be carried out.
- To prevent the spread of HBV and HCV, people must be educated about these infections.
- The good progress in improving immunization coverage in Ethiopia should be enhanced to full coverage.
- WHO guideline of universal immunization of adolescents for the preventable HBV must be strictly followed as it's for all infants.

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9. Annexes

Annexe I

Patient information and Consent form

[To be translated to Amharic]

Purpose

We have planned to conduct a study with objective of investigating the magnitude of HBV and HCV infection in Chronic liver. The knowledge gained from this work is believed to help the management and control chronic liver disease.

Participation

We are asking you and others to voluntarily participate in this study. What is expected from everyone is to be examined for HCV and be asked to answer few questions in relation to risk factor. The laboratory examination involves collection of 5 ml venous blood. All samples are collected using sterile and disposable equipments: tubes, syringes and needles.

Risks and discomforts associated

Taking 5ml of blood doesn't have any harm to your health except minor needle brick injury pain which lasts only for micro second. However if you have any discomfort you will be seen by physician.

Benefits

If there is any positive finding in laboratory investigation the result will be communicated to your physician and prescription of treatment and advice will be effected

Confidentiality

Any information that we collect about you during this research will be kept confidential. Information about your identity will be put away after recording you file; and kept in a secured place. Only the principal investigators will be able to link your identity with the code number, should this become necessary to assist you medically.

Sharing the result

At the end of this study we write a report about the results of the study through publication or any other means. The reports won't bear any information relevant to your personality e.g. your

name or identity. We assure you the confidentiality of such information. Thus we also need your permission to use the test results for writing a report.

Right to refuse

Since participation in this study is entirely voluntary. You can refuse to participate in this research at any time. Your refusal to participate in this study will not affect any of the benefits you are supposed to get from the center.

Contact Address

If you have any further question and in case of urgency you can contact the principal investigator at any time using the following address:-

Name: - Abel Girma (Principal investigator)

Address: - Addis Ababa University Faculty of Medicine Department of Microbiology, Immunology and Parasitology.

City: - Addis Ababa

Telephone (mobile):- +251911375584

P.O. Box: - 180278 A.A Ethiopia

Email: - jabel.girma@gmail.com

Consent form

I, the undersigned, confirm that, as I give consent to participate in the study, it is with a clear understanding of the objectives and conditions of the study and with recognition of my right to withdraw from the study if I change my mind.

I……do herby give consent to Dr/Mr./Mrs/Miss…….to include me in the proposed research. I have been given the necessary information about the research. I have also been assured that I can withdraw my consent at any time without penalty or loss of benefits. The proposal has been explained to me in the language I understand.

Name of the patient: - _____

Patient's signature: - _____

Name of Dr/Mr./Mrs./Miss: -_____

Dr/Mr./Mrs./Miss signature: -_____

Date: - _____

Witness: - _____

Annex II

Questionnaire

Addis Ababa University

Faculty of Medicine

Department of Microbiology, Parasitology and Immunology

(To be translated to Amharic)

For data collectors: please write on the space provided or circle on the answer among choices accordingly.

1. Identification
   - 1.1. Code _____
   - 1.2. Sex A. Male B. Female
   - 1.3. Age _____
2. Back ground information
   - 2.1. Residence A. Urban B. Rural
   - 2.2. Current occupational status

     A. Driver D. Commercial F. Government/private employee

     B. Jobless E. Student G. Farmer

     C. Daily laborer/house servant H. Housewife

     I. Other specify
   - 2.3. Religion

     A. Muslim B. Christian

     C. Does not have D. Other (Specify)
   - 2.4. Marital status

     A. Married B. Single

     C. Divorced D. Widow
   - 2.4. Ethnicity

     A. Oromo B. Amhara C. Tigrae

     D. Gurage E. Wolita F. Others (specify)
   - 2.5. Educational Status

     A. Illiterate B. Can wire and read C. Grade 1-4

     D. Grade 5-8 E. Grade 9-10 F. Grade 11-12 G. 12+

3. **Questions related to HBV and HCV risk factors**

Have you have or ever practiced the following?

3.1.	Ear piercing	A. yes	B. No	
3.2.	Nose piercing	A. yes	B. No	
3.3.	Uvuloctomy	A. yes	B. No	
3.4.	Tattooing on body	A. yes	B. No	
3.5.	Tattooing on gum	A. yes	B. No	
3.6.	Dental extraction at home	A. yes	B. No	
3.7.	Dental extraction at health facility	A. yes	B. No	
3.8.	Circumcision	A. yes	B. No	
3.9.	Shaving	A. yes	B. No	
3.10.	Delivery by TBA	A. yes	B. No	
3.11.	Abortion	A. yes	B. No	
3.12.	Hospital admission	A. yes	B. No	If yes why?_____
3.13.	Surgical procedure	A. yes	B. No	
3.14.	Blood transfusion	A. yes	B. No	
3.15.	Contact with jaundiced patient	A. yes	B. No	
3.16.	Frequent alcohol consumption	A. yes	B. No	
3.17.	Venous or body piercing for treatment	A. yes	B. No	
		(Traditional treatment check the scar)		

አዲስ አበባ ዩኒቨርስቲ

የ ህክምና ፋኩልቲ

የ ማይክሮ ባይሎጂ ፓራስቶሎጂ እና ኢሚኖሎጂ ትምህርት ክፍል

ለመረጃ ስብሳቢዎች፤ ጥያቄውን ከጠየቃችሁበኃላ መልሱን በተሰጠዉ ክፍት ቦታ ወይም ከተሰጡት አማራጮች አንዱን በማክበብ ይጻፉ፡ ፡

1. ጠቅላላ ጥያቄ

1.1. ኮድ_____

1.2. ፆታ ሀ. ወንድ ለ. ሴት

1.3. እድሜ_____

1.4. መኖሪያ አካባቢ ሀ. ገጠር ለ. ከተማ

1.5. ሥራ ሀ. ሹፌር ለ. ነጋዴ

ሐ. የመንግስት ወይም መንግስታዊ ያልሆነ ድርጅት ተቀጣሪ

መ. ሥራ የሌለዉ ሠ. ተማሪ ረ. ገበሬ

ሸ. የቀን ሰራተኛ/የቤት ሰራተኛ ቀ. የቤት እመቤት

በ. ሌላ (ይገለጽ)

1.6. ሃይማኖት ሀ. መስሊም ለ. ክርስቲያን ሐ. የለም መ. ሌላ (ይገለጽ)

1.7. የጋብቻ ሁኔታ ሀ. ያገባ/ች ለ. ያላገባ/ች ሐ. የፈታ/ች መ. የሞተበት/ባት

1.8. ብሔር ሀ. አሮሞ ለ. አማራ ሐ. ትግሬ

መ. ጉራጌ ሠ. ወላይታ ረ. ሌላ (ይገለጽ)

1.9. የትምህርት ደረጃ

ሀ. ያልተማረ ለ. መፃፍና ማንበብ የሚችል

ሐ. ከ 1ኛ እስከ 4ኛ መ. ከ 9ኛ እስከ 10ኛ ሠ. ከ 11ኛ እስከ 12ኛ

ረ. ከ 12ኛ በላይ

2. ሄፓታይተስ “ቢ” እና “ሲ”ን በተመለከተ ጥያቄዎች

ከዚህ በታች ያሉትን በህይወትህ/ሽ አድርገህ/ሽ ታዉቃለህ/ቂያለሽ?

2.1.	ጆሮ መበሳት	ሀ. አዎ	ለ. አይደልም
2.2.	አፍን ጫመበሳት	ሀ. አዎ	ለ. አይደልም
2.3.	እንጥል ማስቆረጥ	ሀ. አዎ	ለ. አይደልም
2.4.	ሰዉነት መነቀስ	ሀ. አዎ	ለ. አይደልም
2.5.	ድድ መነቀስ	ሀ. አዎ	ለ. አይደልም
2.6.	ቤት ዉስጥ ጥርስ ማስነቀል	ሀ. አዎ	ለ. አይደልም
2.7.	ጤና ድርጅት ጥርስ ማስነቀል	ሀ. አዎ	ለ. አይደልም
2.8.	ግርዛት	ሀ. አዎ	ለ. አይደልም
2.9.	ፀጉር ቤት ፂም መላጨት	ሀ. አዎ	ለ. አይደልም
2.10.	በልምድ አዋላጅ መዉለድ	ሀ. አዎ	ለ. አይደልም
2.11.	ማስወረድ	ሀ. አዎ	ለ. አይደልም
2.12.	ሆስፒታል መተኛት	ሀ. አዎ (ለ ምን)	ለ. አይደልም
2.13.	ማንኛዉም አይነት ቀዶ ጥገና	ሀ. አዎ	ለ. አይደልም
2.14.	ደም መቀበል	ሀ. አዎ	ለ. አይደልም
2.15.	ወፍ ከያዘዉሰዉጋር ንክኪ	ሀ. አዎ	ለ. አይደልም
2.16.	መጠጥ መጠጣት	ሀ. አዎ	ለ. አይደልም
2.17.	ሞኝ ባገኝ መቆረጥ	ሀ. አዎ	ለ. አይደልም

(አዎ ከሆነ መልሳቸዉ ጠባሳ ይመለከቱ)

ለ ጥ ና ቱ መ ረ ጃ ና ተ ሳ ታ ፊ ነ ት መ ግ ለ ጫ ቅ ጽ

የ ጥ ና ቱ ዓ ላ ማ

ሄ ፓ ታ ይ ተ ስ ቢ; እ ና ሲ; ቫ ይ ረ ሶ ች በ ጉ በ ት ህ መ ማ ን መ ካ ከ ል ያ ለ ዉ ን ስ ር ጭ ት ለ ማ ጥ ና ት የ ታ ቀ ደ ነ ዉ። ።

በ ጥ ና ቱ ስ ለ መ ሳ ተ ፍ

በ ዚ ህ ጥ ና ት መ ሳ ተ ፍ በ መ ሉ ፈ ቃ ደ ኝ ነ ት ላ ይ የ ተ መ ሰ ረ ተ ነ ዉ። ። ስ ለ ሆ ነ ም በ ጥ ና ቱ እ ን ዲ ሳ ተ ፉ ፈ ቃ ደ ኛ ነ ት ዎ ን እ ን ጠ ይ ቃ ለ ን ። ። ለ መ ሳ ተ ፍ ከ ፈ ቀ ዱ 5 ሚ ሊ ሊ ት ር የ ደ ም ና መ ና መ ና ከ ክ ን ድ ዎ ተ ወ ስ ዶ የ ላ ቦ ራ ቶ ሪ ም ር መ ራ ይ ደ ረ ግ ሎ ታ ል ። ። የ ላ ቦ ራ ቶ ሪ ም ር መ ራ ዉ ም ሄ ፓ ታ ይ ተ ስ “ቢ” እ ና “ሲ” ቫ ይ ረ ስ ን በ ደ ሞ ዉ ስ ጥ መ ኖ ር ና አ ለ መ ኖ ር ማ ረ ጋ ገ ጥ ይ ሆ ና ል ። ። የ ደ ም ና መ ና ዉ ም የ ሚ ወ ሰ ደ ዉ ን ጽ ህ ና ዉ በ ተ ጠ በ ቀ አ ዲ ስ እ ና በ ታ ሸ ገ መ ር ፌ ና ስ ሪ ን ጅ ነ ዉ። ።

በ ጥ ና ቱ ሊ ከ ሰ ቱ የ ሚ ች ሉ ተ ያ ያ ዥ ች ግ ሮ ች

5 ሚ ሊ ሊ ት ር የ ደ ም ና መ ና ዉ ን ለ መ ዉ ሰ ድ መ ር ፌ ሲ ተ ባ ከ ሚ ፈ ጥ ረ ዉ የ ቅ ጽ በ ት የ ህ መ ም ስ ሜ ት በ ስ ተ ቀ ር የ ጎ ላ ች ግ ር አ ያ መ ጣ ም ነ ገ ር ግ ን ም ኞ ት ካ ል ተ ሰ ማ ዎ ት ህ ኪ ም እ ን ዲ ያ ይ ዎ ት ይ ደ ረ ጋ ል ። ።

በ ጥ ና ቱ በ መ ሳ ተ ፍ የ ሚ ገ ኝ ጥ ቅ ም

የ ደ ም ና መ ና የ ላ ብ ራ ቶ ሪ ዉ ጤ ት ም ን ም አ ይ ነ ት ች ግ ር ካ ሳ የ የ መ ድ ሃ ኒ ት ት እ ዛ ዝ ና የ ባ ለ ሙ ያ ም ክ ር ይ ሰ ጥ ዎ ታ ል ። ።

የ ጥ ና ቱ መ ረ ጃ ዎ ች ሚ ስ ጥ ራ ዊ ነ ት

በ ጥ ና ቱ ዉ ስ ጥ የ ተ ሰ በ ሰ ቡ ማ ና ቸ ዉ ም ግ ላ ዊ መ ረ ጃ ዎ ች ሚ ስ ጥ ራ ዊ ነ ታ ቸ ዉ የ ተ ጠ በ ቀ ይ ሆ ና ል ። ። ከ ማ ን ነ ት ዎ ጋ ር በ ቀ ጥ ታ ተ ያ ያ ዥ ነ ት ያ ላ ቸ ዉ መ ረ ጃ ዎ ች በ መ ሉ በ ዋ ና ተ መ ራ ማ ሪ ዉ ሚ ስ ጥ ራ ዊ በ ሆ ነ የ መ ረ ጃ ጥ ን ቅ ር ዘ ዴ ከ ተ ቀ የ ሩ በ ጎ ላ ብ ቻ ለ ም ር ም ር ሂ ደ ቱ የ ሚ ዉ ሉ ይ ሆ ና ሉ። ።

የ ጥ ና ቱ ን ዉ ጤ ት ስ ለ ማ ሳ ወ ቅ

የ ዚ ህ ጥ ና ት ዉ ጤ ት በ ተ ለ ያ ዩ የ ህ ት መ ት ወ ጤ ቶ ች የ ሚ ቀ ር ብ ሲ ሆ ን ይ ህ ከ ማ ን ነ ት ዎ ጋ ር የ ተ ያ ያ ዘ ም ን ም አ ይ ነ ተ መ ረ ጃ ን አ ያ ካ ት ት ም። ። ስ ለ ዚ ህ ም የ ጥ ና ቱ ን ዉ ጤ ት በ ሪ ፖ ር ት እ ና ቀ ር በ ዉ ዘ ን ድ ፈ ቃ ዶ ን እ ነ ጠ ይ ቃ ለ ን ። ።

ከ ጥ ና ቱ ስ ለ መ ዉ ጣ ት ና ስ ለ ማ ቋ ረ ጥ

ይህ ጥ ና ት በ ፈ ቀ ደ ኝ ነ ት ላ ይ የ ተ መ ሰ ረ ተ እ ን ደ መ ሆ ኑ መ ጠ ን በ ማ ን ኛ ዉ ም ወ ቅ ት በ ፈ ቃ ድ ዎ ከ ጥ ና ቱ መ ዉ ጣ ት ይ ች ላ ሉ ፡ ፡ ከ ጥ ና ቱ ቢ ወ ጠ ም እ ን ኳ ን የ ተ ለ መ ደ ዉ ን የ ህ ክ ም ና እ ር ዳ ታ በ ጤ ና ተ ቋ ሙ ዉ ስ ጥ በ ማ ን ኛ ዉ ም ጊ ዜ የ ማ ግ ኘ ት መ ብ ት አ ል ዎ ት ፡ ፡

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እ ኔ ስ ሜ ከ ታ ች የ ተ ገ ለ ፀ ዉ የ ጥ ና ቱ ተ ሳ ታ ፊ ለ መ ሆ ን ስ ወ ስ ን የ ጥ ና ቱ ን አ ላ ማ ዎ ች አ ሰ ራ ሮ ች ና ቅ ድ መ ሁ ኔ ታ ዎ ች በ ግ ል ጽ በ መ ረ ዳ ት ና ከ ጥ ና ቱ ተ ሳ ታ ፊ ነ ት ፈ ቃ ደ ኛ ነ ቴ ን በ ማ ን ኛ ዉ ም ደ ረ ጃ የ ማ ን ሳ ት መ ብ ቴ ን በ ማ ረ ጋ ጥ ነ ዉ ፡ ፡

እ ኔ ---------- በ ጥ ና ቱ ተ ሳ ታ ፊ መ ሆ ኔ ን በ ፊ ር ማ ዬ እ ያ ረ ጋ ገ ጥ ሁ ይ ህ ን ን ስ ወ ስ ን በ ጥ ና ቱ ሳ ቢ ያ ሊ ከ ሰ ቱ የ ሚ ች ሉ አ ደ ጋ ዎ ች በ ሚ ገ ባ የ ተ ረ ዳ ሁ ና ከ ጥ ና ቱ በ ማ ን ኛ ዉ ም ደ ረ ጃ እ ራ ሴ ን ለ መ ሰ ረ ዝ ብ ወ ስ ን ተ ገ ቢ የ ሆ ኑ ህ ክ ም ና ዎ ች ና እ ገ ዛ ዎ ች ሁ ሉ እ ነ ደ ማ ይ ነ ፍ ጉ ኝ በ ማ መ ን ነ ዉ ፡ ፡ እ ነ ዚ ህ መ ረ ጃ ዎ ች ሁ ሉ በ ሚ ገ ባ በ ም ረ ዳ ዉ ቋ ን ቋ የ ተ ገ ለ ጸ ል ኝ መ ሆ ኑ ን በ ፊ ር ማ ዬ አ ረ ጋ ግ ጣ ለ ሁ ፡ ፡

የ በ ሽ ተ ኛ ዉ መ ሉ ስ ም---------- ፊ ር ማ----------

የ ተ መ ራ ማ ሪ ዉ መ ሉ ስ ም፤ ዶ /ር & አ ቶ & ወ /ሮ & ወ /ት ----------

ፊ ር ማ----------

የ ምስ ክ ር መ ሉ ስ ም ---------- ፊ ር ማ----------

Annex III

Blood sample collection procedure

1. Introduce yourself and identify the patient (explain the procedure)
2. Wash hands and wear gloves
3. Prepare the equipment (needles, tubes, etc.)
4. Prepare the patient
5. Apply tourniquet (do not leave it on for more than 1 minute)
6. Choose a vein
7. Disinfect the draw site
8. Obtain the required blood
9. Draw Vacutainer tubes in correct order and invert (mix) correctly according to manufacturer instructions
10. Exit the vein and apply pressure
11. Discard the needle (in appropriate biohazard container)
12. Label the specimen before leaving the patient
13. Check the patient and apply a bandage if necessary
14. Allow the specimen for 30 minute to facilitate clotting
15. Centrifuge with medium speed for 5 minute
16. Separate serum from the blood by Micro-pipette
17. Perform lab tests and store the remaining serum at 20^{0}_{C}.

Annex IV

Laboratory data collection format

Code No.	Laboratory result for							
	HBsAg				Anti-HCV Ab			
001.	Positive		Negative		Positive		Negative	
002.	Positive		Negative		Positive		Negative	
003.	Positive		Negative		Positive		Negative	
004.	Positive		Negative		Positive		Negative	
005.	Positive		Negative		Positive		Negative	
006.	Positive		Negative		Positive		Negative	
007.	Positive		Negative		Positive		Negative	
008.	Positive		Negative		Positive		Negative	
009.	Positive		Negative		Positive		Negative	
010.	Positive		Negative		Positive		Negative	
011.	Positive		Negative		Positive		Negative	
012.	Positive		Negative		Positive		Negative	
013.	Positive		Negative		Positive		Negative	
014.	Positive		Negative		Positive		Negative	
015.	Positive		Negative		Positive		Negative	
016.	Positive		Negative		Positive		Negative	
017.	Positive		Negative		Positive		Negative	
018.	Positive		Negative		Positive		Negative	
019.	Positive		Negative		Positive		Negative	
020.	Positive		Negative		Positive		Negative	
021.	Positive		Negative		Positive		Negative	
022.	Positive		Negative		Positive		Negative	
023.	Positive		Negative		Positive		Negative	
024.	Positive		Negative		Positive		Negative	

Make an “X” mark for the results

Declaration

I, the undersigned, declare that this thesis is my own original work which has not been presented as thesis work for a degree in any university, college or other institution for similar degree or other purpose and that all sources of material used for the thesis have been duly acknowledged.

Investigator

Name of investigator: Abel Girma Ayele

Date: _____ Signature: _____

Advisor

Name of the advisor: Dr. Solomon G/Selassie Teklegiorgis

Date: _____ Signature: _____

Place and date of submission

Addis Ababa University, Faculty of Medicine, Department of Microbiology, Immunology and Parasitology, May 2011