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ABO and Rh Blood Groups Association with Preeclampsia Risk: Maternal Factors, Adverse Outcomes, and Hematological Profiles at Nekemte Hospital, Nekemte Town, Western Ethiopia

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Acronyms and Abbreviations

AAU----------Addis Ababa University

APGAR----------Appearance, Pulse, Grimace, Activity, Respiration

BMI----------Body Mass Index

EPI Data----------Epidemiological Data

HDP---------- Hypertensive Disorders of Pregnancy

ID---------- -- Identification

IRB---------- Institutional Review Board

IUGR---------- Intra Uterine Growth Restriction

MCH---------- Maternal and Child Health

MMR---------- Maternal Mortality Ratio

NICU----------Neonatal Intensive Care Unit

NSH----------Nekemte specialized hospital

PE ----------Preeclampsia

P1GF ---------- Placental Growth Factor

PIH----------Pregnancy-Induced Hypertension

sEng ----------Soluble endoglin

sFlt ---------- Soluble fms-like tyrosine kinase

SPSS----------Statistical Package for the Social Sciences

VEGF---------- Vascular Endothelial Growth Factor

WUCSH----------—Wallaga University Compressive Specialized Hospital

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Abstract

Background: Preeclampsia (PE) is a complex, multisystem disorder that causes significant maternal and fetal morbidity and mortality worldwide. Recent studies suggest an association between ABO/Rh blood groups and preeclampsia, but studies that examined these associations in Ethiopia remain shortcoming.

Objective*:* To investigate the association between ABO/Rh blood groups and preeclampsia risk, by assessing maternal factors, adverse outcomes, and hematological profiles among pregnant women at Nekemte Hospitals that is located in Wallaga, Western Ethiopia.

Methods: A hospital-based comparative prospective study was conducted in Nekemte town that included 208 pregnant women (104 with PE and 104 normotensives), chosen using a convenience sampling technique. Data were entered in Epi Info (version 3.1) and analyzed using SPSS® (version 27). Chi-square tests and logistic regression ($p<0.05$ significant) were employed and continuous variables were reported as mean ± SE.

Results: No significant association was found between PE and ABO/Rh blood group or BMI. However, PE was strongly associated with reduced anti-natal care follow-up (OR= 69.24, 95 % CI: 6.77 - 707.73) and proteinuria (OR= 477.75, 95% CI: 93.71 - 2435). Hematological analysis showed significantly lower hemoglobin (12.6 ±0.22 vs. 13.23±0.19g/dL, $p< 0.05$), Higher RDW (32.94±2.17 vs. 24.83±1.50%, $p< 0.05$)**,** and elevated neutrophil, lymphocyte, and monocyte counts among PE women compared to normotensive controls ($p < 0.05$). Regarding maternal outcomes, PE was linked to higher cesarean section (71.4%), and pregnancy termination rates (94.7%) ($p < 0.05$). Neonates of PE women had lower mean birth weights (2608.17±54.54 gm. vs. 3281.25± 46.01 gm. $p< 0.05$), reduced APGAR scores, higher preterm birth (83.3%), IUFD, and early neonatal death ($p < 0.05$). Most of PE cases (70.2%) occurred after 34 weeks of gestation. However, adverse neonatal outcomes such as low birth weight and reduced APGAR scores were significantly more common in pregnancies between 20-34 weeks ($p < 0.05$).

Conclusion: No association was found between PE and ABO/Rh blood group and BMI. PE was linked to poor ANC follow-up, proteinuria, altered hematological parameters, and adverse neonatal outcomes. This effect suggests the necessity for a better follow-up of pregnant women visiting Nekemte Hospital.

Keywords: Preeclampsia, hematological parameters, ABO and Rh blood groups, antenatal care, neonatal outcomes,

1. Introduction

1.1 Background

Maternal deaths are significantly impacted by complications, in that 75% of the deaths are attributed to severe bleeding, infection, hypertension, delivery delays, and unsafe abortions(Say et al., 2014). PE and gestational hypertension are the deadly triad of interrelated contributors to maternal morbidity and mortality, with population-level changes in risk factors over the last few decades(Wallis et al., 2008).These complications affect 4-5% of all pregnancies worldwide (Duley, 2009).

PE is characterized by new-onset hypertension (blood pressure ≥140/90 mmHg) and proteinuria (≥300 mg in a 24-hour urine sample) after the 20th week of gestation (Duley, 2009). The updated classification excludes proteinuria in the presence of other end-organ damage, and severe features include blood pressure ≥160/110 mmHg on two occasions (Lisonkova et al., 2014). The disease is divided into two subtypes: early onset (<34 weeks of gestation) and late onset (≥34 weeks) (Burton et al., 2019). Late-onset PE is more common than early-onset PE, with an incidence of 2.7 versus 0.3 percent in a population-based study(Lisonkova et al., 2014). PE is associated with factors such as primiparous status, previous maternal PE/eclampsia, family history, high maternal BMI, chronic hypertension, anemia, lack of antenatal care, and maternal blood group and rhesus incompatibility (Ferguson-Smith et al., 1976; Liumbruno & Franchini, 2013).

Karl Landsteiner discovered the ABO blood group in 1900, a genetic trait crucial in transfusion medicine (Hosoi, 2008). It consists of four major phenotypic groups, A, B, AB, and O, resulting from three major alleles of the ABO gene(Ferguson-Smith et al., 1976). The ABO antigens are highly expressed by some human cells and tissues including epithelia, platelets, vascular endothelial, RBC and neurons (Liumbruno and Franchini, 2013). There is a large body of evidence supporting the notion that ABO antigens are involved in the pathogenesis of various systemic multifactorial traits-diseases such as, cancers, infectious, neurological, cardiovascular disorders, and other disorders (Chen et al.., 2014; Wang et al.., 2014; Han et al.., 2020).

Inflammatory cytokines, such as VWF and factor VIII, are linked to increase circulating levels in non-O blood types, contributing to the association between the ABO blood group and arterial or venous thrombosis (Wang et al.., 2014; Han et al.., 2020). The exact mechanism of PE pathogenesis remains unknown, but it is believed to involve decreased placental circulation, increased maternal endothelial dysfunction, and PE clinical presentations (Thornton et al., 2013).

Despite global efforts, its profound impact on maternal and fetal health, the underlying pathophysiology remains largely unknown, and accurate prediction of at-risk populations is an ongoing challenge. Since there is no research has been done on the association between maternal blood group and Rh factor with PE in Ethiopia, this study aims to determine whether the ABO and Rh Blood groups have a significant influence on PE risk among pregnant women. Additionally, this study explores the magnitude of PE and maternal and neonatal outcomes of pregnancies complicated by PE. Addressing this gap is crucial for understanding population-specific risk factors and improving maternal health outcomes.

1.2 Statement of the Problem

PE is a severe and potentially life-threatening condition unique to pregnancy and postpartum, claiming the lives of thousands of mothers and their babies annually worldwide. Globally, PE complicates approximately 4–5% of all pregnancies, contributing to significant maternal and neonatal morbidity and mortality (Duley, 2009; Alemu et al., 2022).

In the developed world, the incidence of PE varies. According to a population-based study in Australia, the incidence of PE is 3.3 per 100 person-years (Thornton et al., 2013). It is 5.2 per 1,000 person-years in Yorkshire (Tuffnell et al., 2005). However, in the developing world, PE is the third most common cause of maternal death behind hemorrhage and infection, which accounts for the overwhelming majority of the estimated 50,000 annual maternal deaths from hypertensive disorders of pregnancy. In Africa, PE is estimated to account for 20-25% of maternal deaths (Duley, 2009).

According to a population-based study in South Africa, the incidence of PE was 12%. Other hospital-based studies showed that HDP was the commonest cause of maternal death, which contributed to 20.7% of maternal deaths in the country (Moodley, 2004). The incidence rate of PE

in developing countries varies from 1.8% to 16.7% (Erden et al., 2016; Wallis et al., 2018). In Ethiopia, it also varies from 1.2% (Hinkosa et al., 2020) to 19.1% (Mekonen et al., 2018).

Previous studies suggest that maternal blood type characteristics (ABO and Rh factor) may influence PE risk, but findings are inconsistent across different populations. For example, research among Chinese women indicates a higher prevalence of PE in those with non-O blood types (30.2% A, 36.3% B) (Jiang et al., 2023). In contrast, studies in Iranian and Nigerian populations have found no significant association between blood type, Rh factor, and PE risk (Burgess et al., 2019; Lee et al., 2012). This implies blood type's role depends on ethnic or environmental factors, making it an unreliable universal predictor. Further research should explore gene-environment interactions to clarify these discrepancies.

1.3 Significance of the Study

The significance of this study lies primarily for pregnant women through its revelation of the relationship between blood types and PE risk factors. Through the identification of the risk factors, the research can lead to improved outcomes of such patients. Additionally Health care workers in the Maternal and Child Health centers can use this information towards increased screening, observation, and control of high-risk patients. This study provides valuable evidence for local health bureaus and policymakers to design targeted interventions, such as early screening for high-risk blood types in susceptible populations and tailored health education on PE prevention. By incorporating these findings, public health strategies can be optimized to reduce preeclampsia incidence more effectively.

2. Literature Review

2.1 The Magnitude of Preeclampsia

Hypertensive disorders of pregnancy (HDP) are Major cause of maternal mortality. They account for nearly 18% of all maternal deaths worldwide, with an estimated 62,000 to 77,000 deaths per year (Khan et al., 2006).

A population-based study done on primiparas with singleton pregnancies from 1999 to 2017 in Sweden showed that 2.9% had mild to moderate PE and 1.4% had severe preeclampsia (Simpson, 2002). Another cross-sectional study conducted in India revealed that out of a total of 500 pregnant women, 31 developed preeclampsia; hence, the prevalence of preeclampsia was found to be 6.2% (Shandilya et al., 2023).

A retrospective descriptive cohort study was carried out at Mpilo Central Hospital, a tertiary teaching referral government hospital in a low-resource setting in Bulawayo, Zimbabwe. There were 9,086 deliveries at the institution during the period from January 1, 2016, to December 31, 2016. There were 121 cases of severe preeclampsia/eclampsia. The incidence of severe preeclampsia/eclampsia was 1.3% at Mpilo Central Hospital (Ngwenya, 2017)Retrospective case-control study from October 2010 to May 2011. The cases were all pregnant women admitted to the Jahun Hospital, Nigeria. A total of 1,257 women (44%) were recorded as having normal pregnancy, and 419 (16%) women had severe preeclampsia/eclampsia (175 with severe preeclampsia and 244 with eclampsia) (Guerrier et al., 2013).

An institution-based cross-sectional study was conducted in Debre Tabor Specialized Hospital among 261 women from January 1– 30, 2021. Overall 15.7% of women had preeclampsia (Alemu Degu Ayele and Zemenu Alemu Tilahun, 2022). A facility-based retrospective unmatched case-control study was conducted to identify risk factors associated with Hypertensive disorders of pregnancy in Nekemte Referral Hospital, just two years back from the study period July 1, 2015, to June 30, 2017. Among 6826 total delivery records from July 2015 to June 2017, 199 women developed hypertension during pregnancy. Among 199 women, 153(76.9%) were preeclampsia/eclampsia,28(14.1%) were gestational hypertension, 14(0.7%) were superimposed hypertension, and 4 (2.9%) were chronic hypertension (Hinkosa et al., 2020).

2.2 Risk Factors for Preeclampsia

The updated 2023 National Institute for Health and Care Excellence (NICE) guidelines (National Guideline Alliance (UK) Hypertension in Pregnancy, n.d.). Classify a woman at high risk of preeclampsia if there is a history of hypertensive disease during a previous pregnancy or a maternal disease, including chronic kidney disease, autoimmune diseases, diabetes, or chronic hypertension. Women are at moderate risk if they are nulliparous, ≥40 years of age, have a body mass index (BMI) ≥ 35 kg/m (National Guideline Alliance (UK) Hypertension in Pregnancy,2023).

Several factors significantly increase the risk of preeclampsia (PE), including family history of PE, multifetal pregnancies, and long inter pregnancy intervals (>10 years) (National Guideline Alliance (UK) Hypertension in Pregnancy, n.d.). Additional clinical risk factors include elevated mean arterial pressure before 15 weeks (North et al., 2011), polycystic ovarian syndrome(Bahri Khomami et al., 2019) Sleep disordered breathing(Pamidi et al., 2014), and various infections such as periodontal disease, urinary tract infections (Rustveld et al., 2008)Helicobacter pylori (Bellos et al., 2018). Obstetric complications like vaginal bleeding lasting ≥5 days during pregnancy also heighten PE risk (North et al., 2011)

The presence of one high-risk factor, or two or more moderate-risk factors, is used to help guide aspirin prophylaxis, which is effective in reducing the risk of PE if administered before 16 weeks of pregnancy ((Bellos et al., 2018); Askie LM et al.,2007).

2.3 ABO and Rh Blood group association with Preeclampsia

A Turkish study (2002–2012) of 250 pulmonary embolism (PE) patients from Kayseri Training and Research Hospital analyzed ABO/Rh blood group distribution. PE patients had a significantly higher proportion of blood group AB compared to controls (P=0.029), with group AB showing a greater PE risk than non-AB groups (P=0.006). Additionally, group O had a higher risk of hypertension (HT) after PE (P=0.004), particularly in O^{+ve} individuals (P=0.001). (Erden et al., 2016).

A case-control study in Northeastern Mexico (253 PE/eclampsia cases vs. 457 controls) found no significant association between ABO blood groups (O:60.6%, A:22%, B:12.4%, AB:5.1%) or Rh factor (97.7% positive) and PE risk, regardless of parity (multiparous p=0.152; nulliparous

p=0.223). Unadjusted ORs for multiparous women were: A=0.7 (95%CI 0.4-1.1), B=1.1 (95%CI 0.6-1.9), AB=0.4 (95%CI 0.1-1.1); nulliparous women showed slightly elevated but non-significant risks (A=1.5, B=1.5, AB=2.0). Adjusting for confounders did not alter these findings (Cordero-Franco et al., 2023b). Similarly, a Pennsylvania study (126 early-onset, 126 late-onset PE cases, 252 controls) with 80% power detected no significant ABO-PE association (OR=1.9, p=0.06) (Burgess et al., 2019).

A case-control study conducted in Shiraz, southwestern Iran, examined 331 pregnant women (121 with PE and 210 normotensive controls) to assess associations between ABO/Rh blood groups and PE risk. Using blood group O and Rh+ as references, logistic regression revealed no significant link between ABO phenotypes and PE. Specifically, group A (OR = 0.67, 95% CI = 0.39–1.17, p = 0.165), group B (OR = 0.86, 95% CI = 0.48–1.53, p = 0.615), and group AB (OR = 1.14, 95% CI = 0.37–3.45, p = 0.212) showed no elevated risk compared to group O. Similarly, while the Rh− phenotype was more frequent among PE cases (OR = 1.79, 95% CI = 0.69–4.65), this association was not statistically significant (p = 0.229). Thus, neither ABO blood groups nor Rh status demonstrated a meaningful relationship with PE risk in this population. (Aghasadeghi and Saadat, 2017).

A retrospective study in Turkey analyzed hospital records of 2,177 women who delivered between November 2005 and October 2006 to examine potential associations between ABO blood groups and preeclampsia (PE). The blood group distribution was: O (605, 27.8%), A (1,056, 48.5%), B (369, 16.9%), and AB (147, 6.8%). Among 220 potential cases identified through electronic records, only 167 mothers were confirmed to have PE. The study found no significant relationship between ABO blood groups and the occurrence of preeclampsia (Beyazıt et al., 2017).

A cross-sectional study conducted in the Department of Pathology, Navodaya Medical College, India, examined 100 preeclampsia cases and 100 controls. Using logistic regression with blood group O as reference, adjusted analysis revealed significant associations: group AB showed markedly higher preeclampsia risk (OR = 16.07, 95% CI = 6.14-42.07, p < 0.0001), while groups A (OR = 1.21, 95% CI = 0.48-3.99) and B (OR = 0.96, 95% CI = 0.42-2.21) demonstrated no significant association. These findings suggest AB blood group carries the highest risk for pregnancy-induced hypertension among ABO blood groups.(S. and K., 2015).

A cross-sectional diagnostic study conducted in the Department of Obstetrics and Gynecology at S.M.S. Medical College, Jaipur. Mothers who present with PE were selected as cases, comprising a total of pregnant women with Rh-positive blood group and matching the diagnostic criteria for preeclampsia and 250 women without any complications. Those available at that time having Rh positive blood group were selected as controls Among 100 mothers who had PE, 26.8% belonged to type O blood, 17.6% belong A blood, 27.6 % B Type, 28 % have AB while among control (healthy pregnant women) was O in35.2% A in 17.2%, B in 35.6%, and AB in 12%. Results: ABO blood groups distribution had a significant difference between patients of PE and control group (P = 0.00008) that showed the percent of AB group significantly higher in patients with PE than control group. The association between blood group and preeclampsia with the blood group O as reference group was determined by odds ratios (ORs) and 95% confidence intervals using logistic regression models. The results A (OR = 1.344, 95% CI = 0.7935-2.2763, P = 0.2715), B (OR = 1.0183, 95% CI = 0.6512-1.5922, P = 0.93) and AB (OR = 3.0647, 95% CI = 1.7988-5.2215, P = 0.0001) indicated that AB has the highest, and O has lowest risk for preeclampsia among the ABO blood groups (Mital et al., 2016).

A case-control study conducted in Thailand involving 5,320 participants, comprising 350 cases and 4,970 controls, revealed that women with A or AB blood types, excluding B, exhibited an elevated risk of preeclampsia compared to those with O type. The adjusted relative risks were 1.7 (95% confidence interval (CI), 1.3 to 2.3; P=0.001) for the A phenotype and 1.7 (95% CI, 1.1 to 2.6; P=0.01) for the AB phenotype(Phaloprakarn and Tangjitgamol, 2013)**.** In a case-control study conducted in Finland, Europe, 248 patients meeting rigorous PE criteria were evaluated, along with 679 controls. Blood type AB increased the incidence of PE overall (OR 2.1, 95% CI 1.3-3.5) (Hiltunen et al., 2009).

Retrospective cohort research conducted in Sweden from 1987-2002 assessed the risk of prenatal hypertension disorders, pre-eclampsia, and severe PE based on maternal ABO blood group and RhD status, using odds ratios. From 1987 to 2002, there were 641,926 singleton births in Sweden. Of them, 39,011 (6.1%) had gestational hypertensive disorders, 29,337 (4.6%) had pre-eclampsia, and 8477 (1.3%) had severe pre-eclampsia. Non-O blood types showed considerably greater chances of PEthan blood group O. Blood group AB had the greatest risk for PE(OR=1.10, 95% CI

1.04–1.16) and severe PE(OR=1.18, 95% CI 1.07-1.30). RhD-positive women had a slightly higher risk of PE(OR =1.07, 95% CI 1.03–1.10) (Lee et al., 2012).

Cross-sectional analytical research conducted in Nigeria involved a total of 147 participants, comprising 66 women with PE and 81 seemingly healthy women as controls, carried out at a tertiary health facility. 46 (69.7%) women with PE had blood group O, and 20 (30.3%) had a non-O blood group. 49 (60.5%) of the controls had blood group O, and 32 (39.5%) had a non-O blood group. The observed difference was not statistically significant (OR 1.50; 95% CI: 0.75–3.0; P = 0.26) (Okoye et al., 2020).

2.4 Pathophysiology of Preeclampsia

Pathogenesis of PE is influenced by abnormal placental development, endothelial dysfunction, and immunologic abnormalities, possibly due to genetic susceptibility. Clinical features include hypertension, proteinuria, renal dysfunction, neurological abnormalities, eclampsia, cardiac dysfunction, pulmonary edema, hepatic dysfunction, hematologic dysfunction, and fetal growth restriction. The uncertainty of its etiology and mechanisms makes it difficult to control morbidity and mortality, both maternal and perinatal (Karmia and Serudji, 2022).

The clinical syndrome begins with abnormal placentation with subsequent release of antiangiogenic markers, mediated primarily by soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng). High levels of sFlt-1 and sEng result in endothelial dysfunction, vasoconstriction, and immune dysregulation, which can negatively impact every maternal organ system and the fetus (Ives et al., 2020).

2.4.1 Hypertension

Growing evidence supports the concept that the placenta plays a central role in the pathogenesis of PE and that reduced uteroplacental perfusion, which develops as a result of abnormal cytotrophoblast invasion of spiral arterioles, triggers the cascade of events leading to the maternal disorder. Placental ischemia is thought to lead to widespread activation/dysfunction of the maternal vascular endothelium that results in enhanced formation of endothelin and superoxide, increased vascular sensitivity to angiotensin II, and decreased formation of vasodilators such as nitric oxide. These endothelial abnormalities, in turn, cause hypertension by impairing renal-pressure natriuresis and increasing total peripheral resistance (Palei et al., 2013).

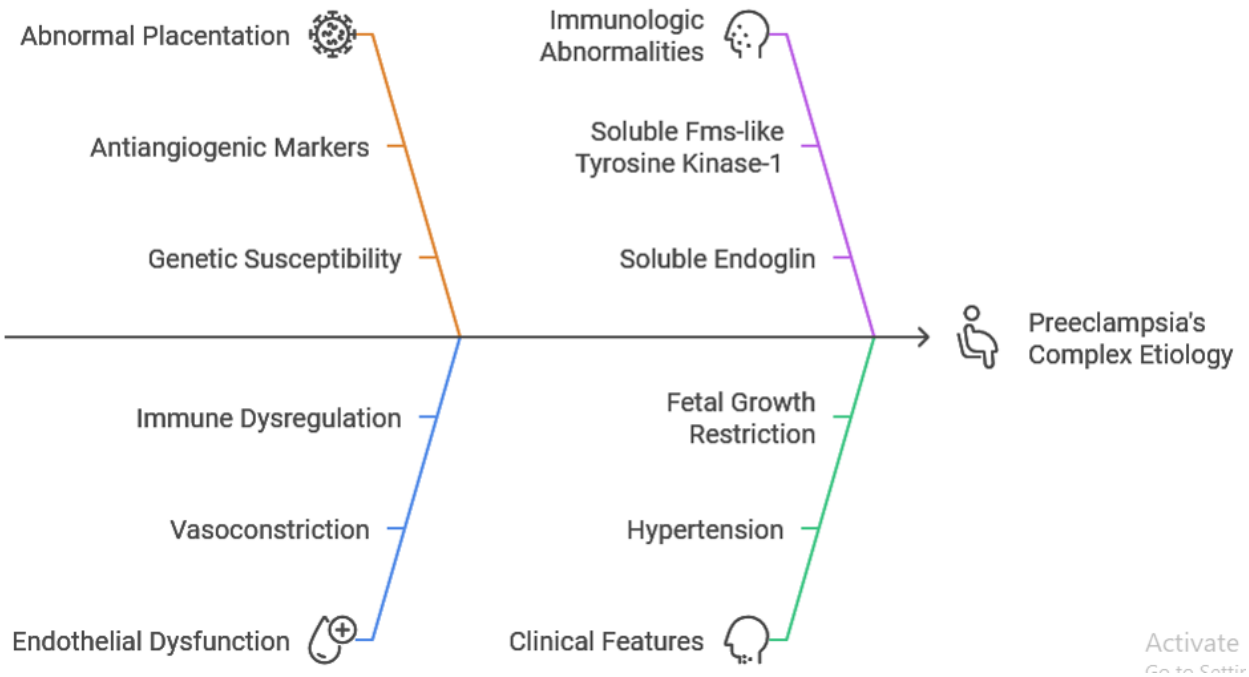

Figure 1: Understanding Preeclampsia Cause and Effect (Ives et al., 2020; Karmia and Serudji, 2022; Palei et al., 2013).

2.4.2 Proteinuria

PE is found to be associated with a distinctive glomerular appearance of endothelial vacuolization and hypertrophy of the cytoplasmic organelles. Women with PE have increased serum concentrations of sFlt-1 and soluble endoglin (sEng) and reduced concentrations of free VEGF and free placental growth factor (PlGF). High circulating sFlt-1 and decreased nitric oxide are both involved in mediating renal tubular injury in the setting of PE (Sircar et al., 2015). sFlt-1 inhibition of VEGF also causes glomerular endothelial injury, a process termed glomerular endotheliosis that is pathognomonic for PE (Tomimatsu et al., 2017). High sFlt-1 levels inhibit podocyte-specific VEGF, disturbing the glomerular filtration barrier and resulting in the formation of fenestrae, contributing to proteinuria (Moghaddas et al., 2019).

2.4.3 Dysfunction

Renal dysfunction in PE is defined as serum creatinine >1.1 mg/dl or a doubling of baseline creatinine (Wilkerson and Ogunbodede, 2019). Renal blood flow and glomerular filtration rate are

often decreased in PE (Mustafa et al., 2012). Biopsy changes in these patients include diffuse fibrin deposition, endothelial swelling, loss of podocytes, and loss of capillary space (glomerular endotheliosis) ((Palei et al., 2013; Sircar et al., 2015; Moghaddas Sani et al., 2019,)

2.4.4 Neurological Dysfunction

PE can cause neurological issues like headache, visual disturbances, seizure, posterior reversible encephalopathy syndrome, and hemorrhagic stroke. The classic PE headache is progressive, bilateral, and worsens with higher blood pressure and physical activity (Ives et al., 2020).
HDPs, most commonly PE , are the most frequent cause of secondary headache and become more common as gestational age increases (54,55). One theory of the pathophysiology of headache in PE is that blocking VEGF and TGF-β leads to loss of fenestrae on the choroid plexus, resulting in endothelial cell instability and periventricular edema. These changes may then precipitate seizures and posterior reversible encephalopathy syndrome, defined by neurological abnormalities with neuroimaging findings of vasogenic edema in the distribution of the posterior cerebral circulation (Jim and Karumanchi, 2017).

Visual disturbance in PE may be due to arise from hormonal fluctuations, such as progesterone level changes (Ramírez-Montero et al., 2020). Retinopathy, retinal detachment, or cortical blindness, which typically resolves following delivery (Adekomi et al., 2019). Central serous chorioretinopathy occurs as fluid accumulates behind the retina, leading to detachment and retinal microvascular damage ((Adekomi et al., 2019; Park YJ et al., 2017)

2.4.5 Eclampsia

Defined as new-onset tonic-clonic, focal, or multifocal seizures in the setting of HDP in the absence of other causes (Acog Committee On Obstetric Practice, 2002). Although progesterone raises the seizure threshold, estrogen lowers the seizure threshold via down-regulation of gamma-aminobutyric acid (Pankiewicz et al., 2019).

2.4.6 Hematologic Disturbance

The most common hematologic disturbances are thrombocytopenia and disseminated intravascular coagulation, a disruption of the clotting cascade leading to intravascular coagulation accompanied by secondary fibrinolysis (Pankiewicz et al., 2019). Thrombocytopenia (platelets <100,000) is likely due to increased platelet activation, aggregation, and consumption (Acog Committee On

Obstetric Practice, 2002). Pregnancy is a procoagulant state because of increases in fibrinogen and other clotting factors, and decreases in anticoagulants (Protein C and S) (Pankiewicz et al., 2019). An imbalance in angiogenic factors, specifically sFlt-1 and sEng, may also be involved (Jodkowska et al., 2015). Another theory is that sudden activation of the vascular endothelial cascade leads to the release of von Willebrand factor multimers that bind platelets, causing excessive platelet aggregation and subsequent thrombus formation in the microcirculation, resulting in consumptive thrombocytopenia, hemolytic anemia, and hepatic dysfunction (Jodkowska et al., 2015; Pankiewicz et al., 2019).

This retrospective case-control study included 186 pregnant women who gave birth at Hacettepe University Hospital between January 2012 and December 2017. A statistically significant difference in white blood cell (WBC) and neutrophil counts was observed among the three groups. The highest WBC and neutrophil counts were found in the early-onset PE (EOPE) group, followed by the late-onset PE (LOPE) group, which had higher levels than the control group. The diagnostic performance of WBC and neutrophil counts in distinguishing between the EOPE and control groups was evaluated using receiver operating characteristic (ROC) analysis. The area under the curve (AUC) was 0.758 (95% CI: 0.633–0.866) for WBCs and 0.749 (95% CI: 0.639–0.877) for neutrophils, with p-values < 0.05 for both parameters (Örgül et al., 2019).

In a study conducted at the Central Laboratory of Omdurman Hospital, eighty women with PE were enrolled, along with eighty normotensive pregnant women who served as the control group. The results showed that hemoglobin (Hb) levels and neutrophil counts were significantly lower in the PE group ($P < 0.01$), while red cell distribution width (RDW), packed cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and lymphocyte counts were significantly higher compared to the control group ($P < 0.01$) (Elgari et al., 2019).

A retrospective, descriptive, cross-sectional study was conducted at Göztepe Training and Research Hospital, Istanbul Medeniyet University, Istanbul, Turkey, involving 186 pregnant women. The study included 72 healthy pregnant women and 114 women diagnosed with PE, categorized into two groups: those with severe PE ($n = 41$) and those with mild PE ($n = 73$), defined as PE without severe clinical features. The median leukocyte count was significantly higher in

both mild and severe PE groups compared to healthy pregnant women (9450/μL), with the highest levels observed in the severe PE group (12,100/μL), followed by the mild PE group (11,450/μL). Additionally, median neutrophil and lymphocyte counts were significantly elevated in the severe PE group compared to the control group. Leukocyte count at hospital admission was found to be significantly associated with the presence of severe preeclampsia (OR: 1.0002, 95% CI: 1.0001–1.0003; p = 0.0001). However, neutrophil count, mean platelet volume (MPV), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) demonstrated poor predictive value for severe preeclampsia (Bozdağ et al., 2018).

This retrospective case–control study was conducted at Dr. Sami Ulus Women's Health Education and Research Hospital, a tertiary care center in Ankara, Turkey. Medical records of all pregnant women managed at the obstetrics clinic between January 2013 and January 2015 were systematically reviewed. A total of 118 pregnant women diagnosed with PE and 120 women with uncomplicated pregnancies were included in the study. Complete blood count (CBC) parameters, including red cell distribution width (RDW), were analyzed. The mean RDW value was significantly higher in the PE group compared to the control group (15.23 ± 1.96 vs. 14.48 ± 1.70; $p < 0.05$) (Yılmaz et al., 2016).

This observational longitudinal study was conducted at the antenatal clinic (ANC) and maternity ward of Zagazig University Hospital (ZUH), Zagazig, Egypt, between 2nd June 2014 and 28th May 2015. The study observed dynamic changes in platelet indices among pregnant women as PE progressed. A decreasing trend was noted in platelet count (PC), while both mean platelet volume (MPV) and platelet distribution width (PDW) showed increasing trends. Receiver operating characteristic (ROC) curve analysis revealed that PDW had the largest area under the curve (AUC = 0.980; 95% CI: 0.964–1.000), identifying it as the most accurate marker for predicting the development of PE. Furthermore, PDW demonstrated the strongest and most statistically significant correlation with mean arterial pressure (MAP) ($r = 0.902$, $p = 0.000$), suggesting its potential as the best marker for predicting the severity of hypertension in preeclamptic patients (Nooh and Abdeldayem, 2015).

Adnan Menderes University Faculty of Medicine, where the study was conducted between January 2013-2015, and 98 gestational age-matched control participants. The study population consisted

of 102 pregnant women diagnosed with PE (49 with mild and 53 with severe PE) and 98 normotensive pregnant controls. In the PE group, the median red cell distribution width (RDW) was significantly higher at 15% (range: 13.8–16.57), compared to 13.9% (range: 13–15.6) in the control group ($p < 0.01$). Conversely, the mean corpuscular volume (MCV) was significantly lower in the preeclampsia group (80.42 ± 7.86 fL) than in the control group (83.88 ± 2.31 fL; $p = 0.003$). The mean corpuscular hemoglobin concentration (MCHC) was higher in the preeclampsia group (33.66 ± 1.71 g/dL) compared to controls (33.09 ± 1.48 g/dL; $p = 0.012$). However, no statistically significant differences were observed in mean corpuscular hemoglobin (MCH) and red blood cell (RBC) count between the two groups ($p > 0.05$) (Avcıoğlu et al., 2015).

A comparative cross-sectional study was conducted on a total of 126 pregnant women at the Gondar University Comprehensive Specialized Hospital, using a convenient sampling technique. The eosinophil count of PE pregnant women was significantly lower than that of normotensive (NT) pregnant women (median (IQR): 50 (10—200) vs. 120 (60 – 270); $p = 0.002$). The eosinophil count ≤ 55 cells/μL had an AUC of 0.66 (95% CI: 0.56—0.75) for diagnosis of PE with a sensitivity of 50.8%, specificity of 77.8%, and positive and negative predictive value of 69.6% and 61.3%, respectively (Gelaw et al., 2022).

A cross-sectional study was carried out in 2015 at the University of Gondar hospital. Thirty-three mild PE, 30 severe PE cases, and 63 healthy pregnant women were enrolled in the study. The means of white blood cells (WBC), absolute Neutrophil count (ANC), Absolute middle cell count (AMC), mean Platelet count (PTC), Platelet distribution width (PDW), neutrophil-to-lymphocyte ratio (NLR), and median of platelet-to-large cell ratio (P-LCR) were significantly increased; while Absolute lymphocyte count (ALC) and platelet count (PTC) were significantly decreased in PE groups. WBC, ANC, MPV, PDW, P-LCR, and NLR showed statistically significant positive correlations, whereas PTC displayed a statistically significant negative correlation with a MAP in the PE group (Sitotaw et al., 2018).

An institution-based comparative cross-sectional study was done from July 22 to October 30, 2021, at Arba Minch General Hospital. A total of 136 pregnant women were included in the study (46 with preeclampsia and 90 without preeclampsia). The complete blood count analysis showed that there were mean differences in Red Cell Distribution (RDW) ($p < 0.036$), neutrophil-to-

lymphocyte ratio (NLR) ($p < 0.016$), and relative lymphocyte count (Lymp%) ($p < 0.047$). The ROC analysis of the AUC for RDW, NLR, and Lymp% resulted in 0.607, 0.609, and 0.600, respectively (Kassahun et al., 2024).

This study was conducted at Al-Hussein University Hospital and included a total of 150 pregnant women. Participants were divided into two groups: Group 1 consisted of 34 pregnant women diagnosed with preeclampsia, and Group 2 included 116 healthy pregnant women with no medical disorders revealed no statistically significant difference in mean platelet volume (MPV) values between the preeclampsia group and the control group ($p > 0.05$), indicating a non-significant association (El-garhey et al., 2018).

A systematic search was conducted to identify relevant articles published in English between January 10, 2011, and January 10, 2021, using PubMed, Web of Science, and African Journals Online. A total of 25 studies were included in this systematic review and meta-analysis. Of these, 23 studies contributed data to the analyses of platelet count (PC) and mean platelet volume (MPV). The overall pooled weighted mean difference (WMD) between the preeclampsia (PE) and normotensive (NT) groups was -41.45×10^9/L for PC (95% CI: −51.8 to −31.0) and +0.98 fL for MPV (95% CI: 0.8 to 1.1). These findings indicate a significant decrease in platelet count and a significant increase in MPV in women with preeclampsia compared to normotensive pregnant women (Walle et al., 2022).

2.4.7 Fetal Growth Restriction/Fetal Implications

PE leads to uterine and placental dysfunction, which causes fetal growth restriction, defined as an estimated fetal weight <10th percentile for gestational age ("ACOG Practice Bulletin No. 204," 2019). As spiral arteries fail to develop appropriately and lead to incomplete pseudo-vasculogenesis, placental vascular insults such as placental infarcts occur (Jim and Karumanchi, 2017; Mustafa et al., 2012). Incomplete spiral artery remodeling leads to atherosclerosis of maternal radial arteries (lipid-laden macrophages in the lumen, fibrinoid necrosis in the wall, and mononuclear perivascular infiltrate) (Rana et al., 2019). Decidual vasculopathy, including loose, edematous endothelium, hypertrophy of the vessel media, loss of smooth muscle modifications, and up-regulation of hypoxia-inducible transcription factor-1α, results from these changes (Rana et al., 2019). Structural changes of the glycocalyx and hyaluronic acid are also seen (El-Sayed,

2017). Inhibition of TGF-β by sEng also leads to impaired endothelial vasodilation (Jim and Karumanchi, 2017). These changes cause impaired diastolic placental flow on ultrasound and placental ischemia (El-Sayed, 2017; Rana et al., 2019). The resultant ischemia can lead to decidual and placental endoplasmic reticulum stress and further oxidative stress (Rana et al., 2019). Taken together, these factors contribute to fetal growth restriction that commonly occurs in pregnancies complicated by preeclampsia.

2.4.8 Neonatal Outcomes

The pathogenesis of PE is complex and not fully understood; however, it is known to involve Poor early placentation, systemic inflammation, and oxidative stress (Phipps et al., 2019a; Steegers et al., 2010). Abnormal placentation occurs due to failure of appropriate remodeling of the spiral arteries, resulting in higher resistance to placental blood flow and hypoperfusion of the placenta. This causes chronic placental ischemia and reduced blood flow to the developing fetus (Steegers et al., 2010). These maladaptive processes can precipitate fetal hypoxia and adverse outcomes, including IUGR, preterm birth (both spontaneous and iatrogenic), oligohydramnios, placental abruption, fetal distress, and fetal death in utero (Haddad et al., 2004; Madazli et al., 2014). The frequency of fetal complications differs depending on the onset of PE. Early onset of preeclampsia has been associated with significantly higher rates of adverse outcomes for the fetus, including IUGR, oligohydramnios, and fetal death (Haddad et al., 2004; Madazli et al., 2014).

A Swedish Medical Birth Register study found that PE in primiparas with singleton pregnancies increased the risk of complications for newborns. Neonates born to mothers with PEhad the risk of being small for gestational age (aOR 5.3, CI: 5.1–5.5) and needing resuscitation (aOR 2.6, CI: 2.4–2.7) were increased. The risk of a low Apgar score and convulsions/hypoxic ischemic encephalopathy was increased at 32–41 weeks of gestation. Moreover, the overall risk of sepsis (aOR 1.9. CI: 1.8–2.1) and perinatal death (aOR 1.2, CI: 1.1–1.5) was also increased. Severe preeclampsia is a significant risk factor for intrauterine fetal demise, with a stillbirth rate of 21 per 1000. However, in cases of mild PE, the risk of fetal demise is over 50% less. PE accounts for over 40% of premature deliveries and significantly increases the risk of low birth weight (Simpson, 2002).

A prospective follow-up study was conducted in the Obstetrics & Gynecology Department (OGD) of Al-Zahraa Maternity and Pediatric Hospital (ZMPH) in Al-Muqdadia District in Diyala

province from the period 1st of February 2017 to 31st of January 2018. The study sample comprised 60 pregnant women with PE and 60 healthy pregnant women as controls. There was a highly significant association between high cesarean section rates and PE women. A significant association was observed between neonates of PE women and low birth weight preterm birth, low Apgar score at 1st minute. Low Apgar score at 5th minutes and admission to the neonatal intensive care unit (Ali et al., 2018).

A cross-sectional study was carried out at two selected public hospitals in Addis Ababa, among 348 mothers between January 1, 2023, and July 1, 2023. The overall prevalence of unfavorable perinatal outcomes was 59.2% (95% CI: 54.0–63.8). Among the complications, low birth weight, prematurity, NICU admission, and a low fifth-minute APGAR score, encompass 48.9%, 39.4%, 20.4%, and 14.7%, respectively (Tadese et al., 2024).

3. Conceptual Framework

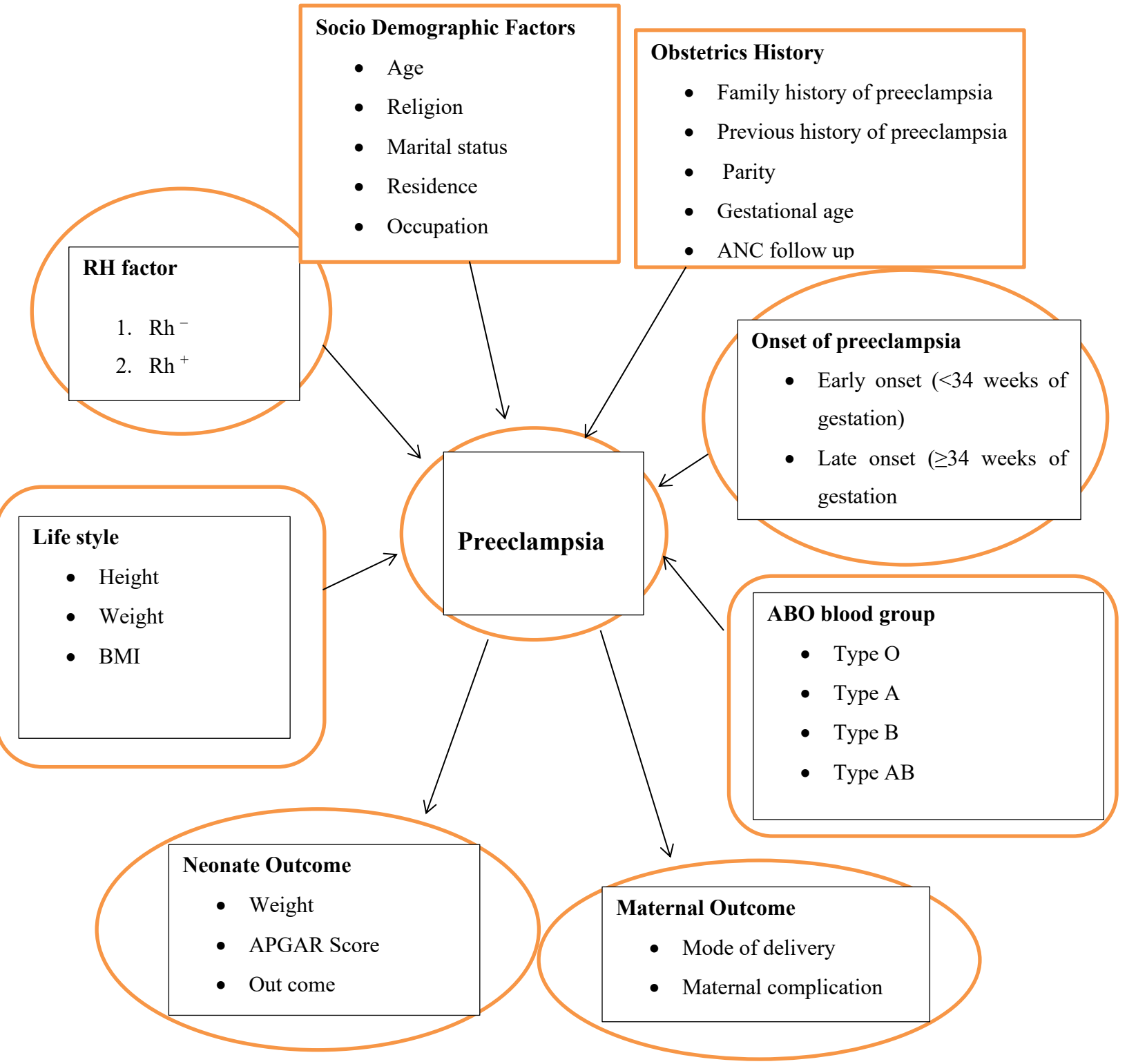

Figure 2: Conceptual framework of the study (Developed from literature review)

4. Objectives

4.1 General Objective

To investigate the association between ABO/Rh blood groups and preeclampsia risk, while assessing maternal factors, adverse outcomes, and hematological profiles among pregnant women at Nekemte hospitals that is located in Wallaga, Western Ethiopia

4.2 Specific Objectives

1. To determine distribution and association of ABO and Rh blood groups preeclampsia
2. To identify the maternal factors associated with preeclampsia.
3. To identify preeclampsia maternal and neonatal adverse outcome.
4. To compare hematological parameters between preeclamptic and normotensive pregnant women.
5. To determine the prevalence of early and late-onset preeclampsia

5. Materials and Methods

5.1 Study Area and Period

The research was conducted at Nekemte Specialized Hospital (NSH) and Wallaga University Comprehensive Specialized Hospital (WUCSH) located in Nekemte. Nekemte is the administrative center of the East Wollega Zone in the Oromia Region, Ethiopia. It is located about 331 kilometers west of Addis Ababa, the capital city of Ethiopia, and is one of the major urban centers in western Ethiopia. The city has an estimated population of 120,000-150,000 people and serves as a critical hub for healthcare services for both urban and rural residents of the surrounding areas. The town has four main exits: to Amahara Region (Nekemte-Bure-Bahirdar), to Benishangul Gumuz (Nekemte-Gimbi-Asosa), to Gambela Region (Nekemte- Mettu- Gambela) and to Addis Ababa (Nekemte-Ambo-Addis Ababa). There is an active neighborhood linkage with Jimma, Dambidolo, Shambu, Gimbi and Bedele.

These are the only Governmental Hospitals found in Nekemte town and act as the main referral centers for medical services within the region by providing a full range of healthcare services to more than 2.1 million people living in East Wollega Zone, parts of West Wollega Zone, Horroo guduru Wollega Zone, and West Shoa Zone. These services also include obstetrics and gynecology, emergency care, specialized maternal health, and WUCSH offers services as a teaching hospital.

These hospitals serve as the principal healthcare providers for several pregnant women in Nekemte and surrounding areas. It is also a critical point of care for women referred from rural health centers and health posts, where maternal health services may not be comprehensive. The study involved the outpatients and the inpatients of these hospitals. The study was conducted from December 2024 to June 2025.

5.2 Study Design and Population

This hospital-based prospective cohort study compared 104 preeclamptic women with 104 normotensive pregnant controls (total N=208), recruited from obstetrics/gynecology outpatient/inpatient departments and high-risk units via convenience sampling. Participants were followed longitudinally until delivery to assess outcomes

5.3 Population

5.3.1 Source Population

All pregnant women who were attending the antenatal clinic and delivery units at Nekemte Hospitals during the study period.

5.3.2 Study Population

Pregnant women at Nekemte Hospital, including those diagnosed with PE and those with normal pregnancies.

5.3.3 Study Unit

Each individual pregnant woman diagnosed with PE or belongs to the control group, who underwent fresh ABO/Rh blood typing using standard laboratory methods for this study.

5.4 Eligibility Criteria

5.4.1 Inclusion Criteria

The study included pregnant women aged between 18 and 45 years and who completed the written informed consent. Pregnant women with PE, which was diagnosed after 20 weeks of gestation, were included in the case group, and normotensive pregnant women without PE, proteinuria, or any other related complications constituted the control group. All the participants were free from pre-existing medical conditions to minimize confounding.

5.4.2 Exclusion Criteria

Pregnant women with gestational age of less than 20 weeks or were carrying multiple pregnancies were excluded from the study. Women with a history of chronic hypertension, inflammatory diseases, asthma, diabetes mellitus, gestational HTN or non-pregnancy-induced hypertension were also excluded from the study. In addition, pregnant women diagnosed with Eclampsia were not eligible to participate in the study.

5.5 Sample Size Determination

The minimum sample size (n) for this study was calculated using the formula for comparison of proportions according to Kirkwood(Kirkwood and Sterne, 2010). Assuming a 28% increase in the incidence of preeclampsia among blood group AB women compared to the general population (Mital et al., 2016).

$$\left(\frac{Z_{1-\alpha/2}\sqrt{2P(1-P)} + Z1-\beta\sqrt{p1(1-p1)+p2(1-p2)}}{\boldsymbol{p1-p2}} \right)^{\mathbf{2}}$$

Where

$Z\,1-\alpha/_2$ =1.96 for a 95% confidence level

$Z1-\beta$ = 0.84 for 80% power.

P1= Proportion of cases with the exposure = 0.28 proportion of AB group in preeclampsia case

P2= Proportion of controls with the exposure. =0.12 proportion of AB group in control

P= average proportion

$$\boldsymbol{P}=\frac{P1+p2}{2}=\frac{0.28+0.12}{2}=\frac{0.4}{2}=0.2$$

$$\left(\frac{\mathbf{1.96}\sqrt{\mathbf{2\times 0.2(1-0.2)}} + \mathbf{0.8}\sqrt{\mathbf{0.28(1-0.28)+0.12(1-0.12)}}}{\mathbf{0.28-0.12}} \right)^{\mathbf{2}}$$

$$\left(\frac{\mathbf{1.11+0.44}}{\mathbf{0.16}} \right)^{\mathbf{2}}$$

$$\left(\frac{\mathbf{1.55}}{\mathbf{0.16}} \right)^{\mathbf{2}} = \mathbf{9.6875^2} = \mathbf{94}$$

10% non-response rate = 9.4

Totally = 94 + 9.4 = 103.4 ≈ 104

= 104 participants per group (i.e. preeclampsia case and control)

= Total sample size is 208

5.6 Sampling Technique and Procedure

This hospital-based study recruited 208 pregnant women (104 PE cases and 104 controls) from Nekemte Hospitals using consecutive convenience sampling technique. Balanced allocation was used as NSH (1,180 ANC contact/month) and WUSCH (1,240 ANC contact/month) have nearly equal pregnant women flow for ANC contact. Not anticipated to miss any cases in a row until the required sample size was reached. Cases were diagnosed in accordance with established criteria (BP ≥140/90 mmHg and proteinuria after 20 weeks) ("Hypertension in Pregnancy," 2013). Controls were selected from women attending routine third-trimester contacts (≥28 weeks), who were confirmed normotensive at delivery. All participants had prospective ABO/Rh typing using standard tube agglutination, without pre-selection for study based previous knowledge (or awareness) of blood group types.

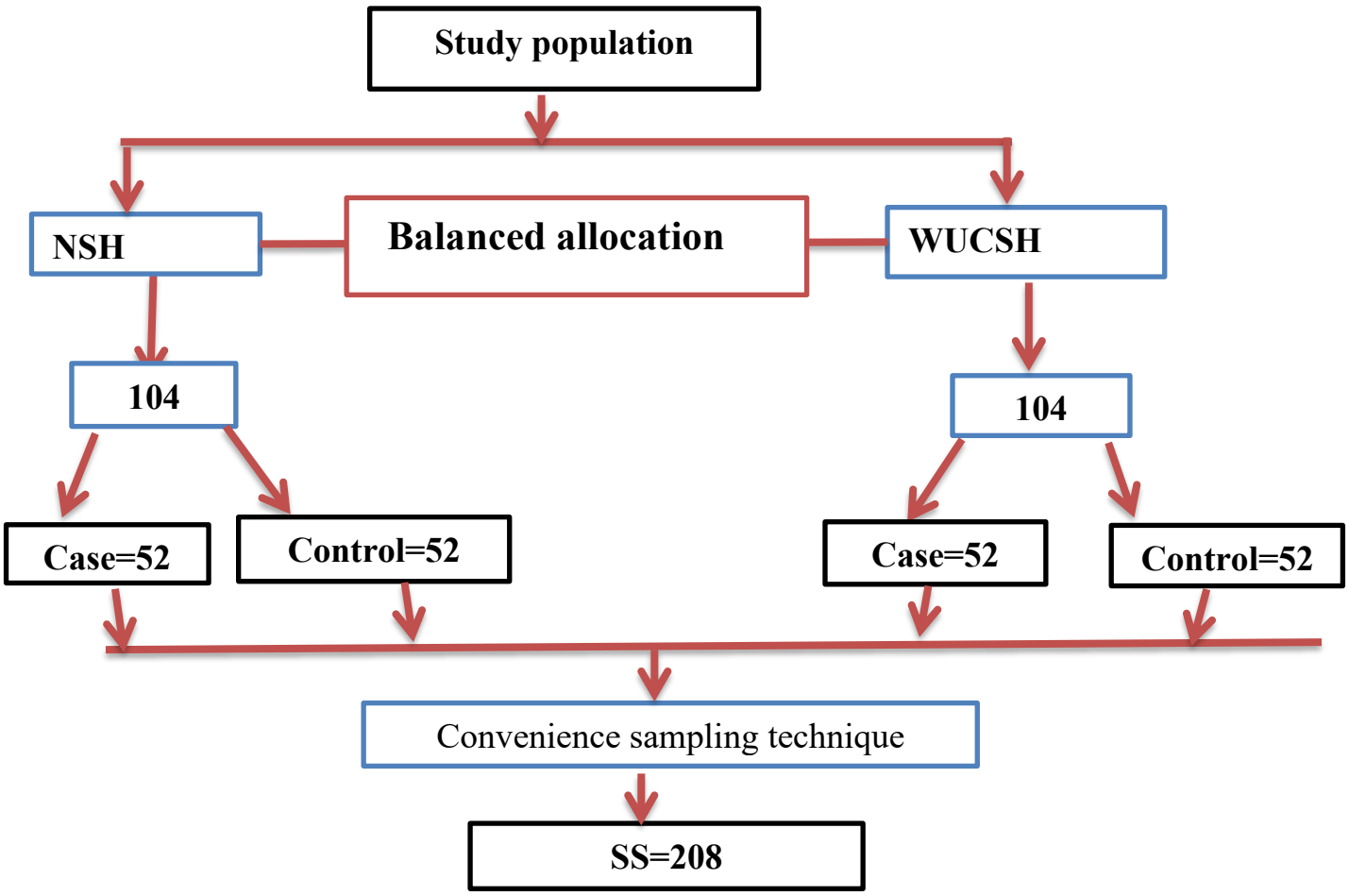

Figure 3: Sampling Procedure

5.7 Study Variables

5.7.1 Dependent Variable:

The dependent variables in this study include PE, birth weight, APGAR score, neonatal outcome, and maternal outcomes such as mode of delivery, maternal complications, and overall maternal outcome.

5.7.2 Independent Variables:

The independent variables are categorized into socio-demographic, obstetric history, and biological factors. The socio-demographic variables consist of age, marital status, residence, occupation, educational status, and socioeconomic status. The obstetric history variables include a history of PE in previous pregnancies, parity, gestational age at diagnosis of PE, and ANC follow-up. Additionally, the biological variables examined are ABO blood groups (A, B, AB, and O) and Rh factor (positive or negative).

5.8 Data Collection Techniques and Instruments

Socio-demographic characteristics, obstetric characteristics, assessment criteria for diagnosing PE, lifestyle, maternal and neonatal outcome was collected using structured questionnaire while ABO and Rh Blood Group Distribution, ABO incompatibility and Rh incompatibility data was determined by blood collected from the participant. Performed ABO/Rh testing on maternal and cord blood, including Direct Coombs Test (DAT). The questionnaire was translated into the local language and back to English for data entry and analysis. Data was collected by trained nurses, midwives and by obstetrics and Gynecology residents. The questionnaire was piloted and checked by experts to accommodate corrections.

5.8.1 Blood pressure measurement: Blood pressure was measured by using an aneroid sphygmomanometer and a stethoscope from the upper right arm for consistency. A trained nurse/midwife working at ANC, patients took SBP and DBP measurements after 10 minutes rest. The participants sit comfortably, with their back supported, legs uncrossed, feet on the ground, and their upper arms positioned at heart level during blood pressure measurement.

5.8.2 Anthropometric Data Collection

Weight and height were measured by using a digital weighing machine and a height scale, respectively. BMI was calculated as weight in kg divided by height squared in meters (BMI= Kg/mt^2). During the height measurement, the study participant's shoes and any hats or hair

ornaments were removed. With the subject looking straight ahead, the projection was placed at the crown of the head, and with the reader's eye at the level of the headpiece. Then the height was measured in meter (Eaton–Evans, 2005).

5.8.3 Blood Sample Collection

A total of 5mL venous blood was collected from patients diagnosed with PE and the control group at the antenatal clinic (ANC). This was to perform ABO and Rh blood grouping, CBC analysis. The CBC was analyzed using the DIMIND DH36 hematology analyzer. While blood grouping was performed using the serological method in which known anti bodies (anti-A, anti-B, anti-D) were used to detect unknown antigen on the surface of red blood cell.

5.9 Operational Definitions

Adverse perinatal outcome: pregnancy outcome including stillbirth, growth restriction (IUGR), respiratory distress syndrome (RDS), low birth weight, <7 APGAR score at 1st and 5th minute of a life , neonatal death (Yahaya et al., 2024).

Blood group: set of serologically defined variations or polymorphisms in red cell surface antigens. The specificity of blood group antigens is determined either by oligosaccharide epitopes (eg, ABO antigens) or by the amino acid sequence (eg, Rh, Kell, and Duffy antigens) (Quraishy & Sapatnekar, 2016).

Case Group: A subset of the study population diagnosed with preeclampsia based on the inclusion criteria

Control Group: Individuals without preeclampsia, matched for demographic and clinical factors, such as gestational age and parity, but who do not exhibit hypertension or proteinuria during pregnancy.

Complications of Preeclampsia: Eclampsia (seizures), HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count), organ damage (kidneys, liver, brain), placental abruption, preterm birth, fetal growth restriction, maternal and fetal death (WHO systematic analysis, Lancet Glob Health. 2025).

Eclampsia: Convulsions and coma occurring in a pregnant or puerperal woman and associated with preeclampsia, that is a condition in pregnancy manifested by hypertension, edema and/or proteinuria (Cunningham et al., 2018).

Gestational age: The duration of the pregnancy measured from the first day of the last normal menstrual period (Cunningham et al., 2018).

Hypertensive Disorders of Pregnancy: Refers to either systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg measured twice on separate occasions at least 4 hours apart. Conditions classified as Preeclampsia, Gestational hypertension, Chronic hypertension, Superimposed, and Eclampsia ("Hypertension in Pregnancy," 2013)**.**

Maternal mortality ratio: Defined as the number of maternal deaths per 100,000 live births during a given period (Say et al., 2014).

Normal pregnancy: Pregnancy without any complications

Parity: Refers to the number of pregnancies that have resulted in the birth of one or more live children (Cunningham et al., 2018).

Preeclampsia: Is a hypertensive disorder in pregnancy characterized by new-onset hypertension (systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg) after 20 weeks of gestation, in combination with proteinuria (≥300 mg in a 24-hour urine sample) or other signs of organ dysfunction (e.g., thrombocytopenia, renal impairment, liver involvement, or pulmonary edema) ("Hypertension in Pregnancy," 2013).

Rh factor: Refers to the presence or absence of the Rh antigen (specifically RhD) on red blood cells. If the antigen is present, the individual is Rh-positive; if absent, the individual is Rh-negative (Liumbruno and Franchini, 2013).

Stillbirth: Fetus born with no sign of life after 28 weeks of gestational age (Belay Tolu et al., 2020).

5.10 Data Quality Assurance and Management

The English version of the questionnaire was translated to Afaan Oromoo and Amharic by experts during data collection. During data entry, the questionnaire was translated back to English to ensure consistency and accuracy. A pretest on 5% of the participants for each group was conducted to check for clarity, comprehensibility, and feasibility of the questions.

Data collectors and supervisors were trained for two days before the data collection period on study objectives, ethical considerations, appropriate administration of the structured questionnaire, process procedures for collecting data, and sensitivity regarding sensitive or incomplete data. Regular feedback during this process was important to address different challenges.

Depending on the level and type of data, we performed data analysis by applying appropriate statistical techniques. This included chi-square tests for categorical data and logistic regression to

find associations between blood groups and preeclampsia. The data was first cleaned, coded, and checked for inconsistencies before analysis. The findings were interpreted and presented in the final report.

5.11 Data entry and analysis

Data were entered into Epi Info (version 3.1), transferred, and analyzed using the Software Package for the Social Sciences (SPSS version 27.0). To analyze the continuous and categorical variable differences between cases and controls, independent t and chi-square tests were used, respectively. Bivariate and multivariate logistic regressions were also used to determine the association between independent variables and the dependent variable in the preeclamptic women. In the bivariate analysis, variables having an association with a p-value<0.25 were taken into multivariate logistic regressions. Mean, standard error (SE), and percentage were used as descriptive statistics, and a **p-value < 0.05** was considered statistically significant.

5.12 Ethical Considerations

Ethical approval was obtained from the Department of Medical Physiology, School of Biomedical and Laboratory Sciences, CHS, AAU. Letters from the department were written to the hospitals where this study was conducted. Permission was obtained from the clinical directors of NSH and WUCSH and heads of the respective departments to make sure authorization the actual study.

Training was given to the data collectors on ethical principles: purpose of the study, the risks, and benefits. After the purpose of the study was disclosed to the study participants, and able to withdraw at any time and it will not affect them, written informed consent was obtained from each participant. The confidentiality of participant data was ensured by assigning unique ID numbers to each questionnaire, and personal identifying information was not entered into the computer either for analysis or reporting.

5.13 Dissemination Plan

The results of this study will be presented to the Department of Medical Physiology, School of Biomedical and Laboratory Sciences, CHS, Addis Ababa University. Furthermore, the findings will be disseminated to NSH & WUCSH and the AAU postgraduate library, and the manuscript of this research will be prepared and submitted to appropriate journals for publication.

6. Results

6.1 Socio-Demographic Characteristics

A total of 208 participants aged between 18 and 48 years (mean age 29 ± 6 years) were included in this study. Age distribution showed significant variation between groups (δ^2 (2) =12.675, p<0.02), with PE higher among younger women aged 15–24 (35 (62.5%) vs. 21 (37.5%)) and lower in older women aged 35–48 (6 (21.4%) vs. 22 (78.6%)).

Religious affiliation showed not significant variation between groups (δ^2 (2) =4.909, p=0.086). Rural residence was higher among participants in the PE group than control group (47 (52.8%) vs. 42(47.2%) but, difference was not significant ((δ^2 (2) =4.909, p=0.086). Majority of participants in both PE (104 (50.5%)) and control (102(49.5%)) groups were married and the difference was not significant ((δ^2 (1) =2.019, p=0.155).

Occupation demonstrated highly significant associations (δ^2 (3) =26.420, p<0.05). Compared to the control group housewives were higher among PE cases, (85 (61.6%) vs. 53(38.4%)). while employed women were higher in controls (48(75%) vs. 16(25%)). Few participants were students, mostly controls 3(75%) vs. 1(25%).

Additionally, Educational attainment showed significant differences (δ^2 (4) =14.658, p<0.001). Higher PE cases lacked formal education (12(54.5%) vs. 40(45.1%)). while higher controls had attained higher education, (33(70.2%) vs.14 (29.8%)). Attending primary education was higher in the PE group, (13(61.9%) vs. 8(38.1%)) **(Table 1).**

Table 1. Socio-demographic Characteristics of the Study Participants

Variables	Categories	Mother condition(N=208)		Total N (%)	chi-square test(δ^2)	p-value
		Preeclampsia N (%)	Control N N (%)			
Age	15-24	35(62.5)	21(37.5)	56(100)	12.675	0.02
	25-34	63(50.8)	61(49.2)	124(100)		
	35-48	6(21.4)	22(78.6)	28(100)		
Religion	Orthodox	23(54.8)	19(45.2)	42(100)	4.909	0.086
	Protestant	68(45.9)	80(54.1)	148(100)		
	Muslim	13(72.2)	5(27)	18(100)		
Residence	Urban	57(47.9)	62(52.1)	119(100)	0.491	0.483
	Rural	47(52.8)	42(47.2)	89(100)		
Marital status	Married	104(50.5)	102(49.5)	206(100)	2.019	0.155
	Unmarried	0(0)	2(100)	2(100)		
Occupation	House wife	85(61.6)	53(38.4)	138(100)	26.420	0.001
	Employee	16(25)	48(75)	64(100)		
	Student	1(25)	3(75)	4(100)		
	Others	2(100)	0(0.0)	2(100)		
Educational status	Not attend formal education	12(54.5)	10(45.1)	22(100)	14.658	0.05
	Write and read	37(48.1)	40(51.9)	77(100)		
	Complete primary school	13(61.9)	8(38.1)	21(100)		
	High school	28(68.3)	13(31.7)	41(100)		
	Higher level education	14(29.8)	33(70.2)	47(100)		

6.2 Association between Body Mass Index (BMI) and Preeclampsia

No significant difference in BMI was observed between the groups (23.575±0.1297 vs. 23.7221±0.1297, 95% CI: -0.2781 to -0.5752; p=0.496) **(Table2).**

Table2: Association between Body Mass Index (BMI) and Preeclampsia

Variable	Categories	Mean ±SE	95% CI difference		p-value
			Lower	Upper	
Women condition	Preeclampsia	23.775±0.1297	-0.2781	-0.5752	0.496
	Normal pregnancy	23.3221±0.1297			

6.3 Logistic Regression Analysis results

In bivariate analysis, age, religion, educational status, gravidity, parity, number of ANC follow-ups, proteinuria, ABO blood group were significantly associated with PE (P<0.25). However, multivariate logistic regression analysis revealed that the number of ANC follow-ups (OR= 69.24, 95 % CI: 6.77 to 707.73) and proteinuria (OR= 477.75, 95% CI: 93.71 to 2435) were significantly associated with PE (**Table 3**).

Table 3. Multivariable Logistic Regression Analysis Results

Variable	Categories	Mother condition(N=208)			CoR	AoR
		Normal N (%)	Preeclampsia N (%)	Total N (%)		
Age	18-24	21(37.5)	35(62.5)	56(100)	1	1
	25-34	61(49.2)	63(50.8)	124(100)	1.614(0.846-3.077)	0.172(0.008-3.555)
	35-48	22(78.6)	6(21.4)	28(100)	6.111(2.133-17.05)	2.3×10^{9}(0.000-∞)
Religion	Orthodox	19(45.2)	23(54.8)	42(100)		1
	Protestant	80(54.1)	68(45.9)	148(100)	1.424(0.716-2.834)	2.703(0.187-38.984)
	Muslim	5(27)	13(72.2)	18(100)	0.466(0.414-1.541)	0.036(0.001-2.344)
Education	No Formal education	10(45.1)	12(54.5)	22(100)		1
	Write and read	40(51.9)	37(48.1)	77(100)	1.297(0.501-3.357)	0.838(0.014-51.566)
	Completed primary school (1-8)	8(38.1)	13(61.9)	21(100)	0.738(0.219-2.493)	3.333(0.019-573.106)
	High school (9-12)	13(31.7)	28(68.3)	41(100)	0.557(0.192-1.618)	0.340(0.002-51.718)
	Higher level education	33(70.2)	14(29.8)	47(100)	2.829(0.993-8.054)	0.212(0.003-14.829)
Gravidity	Once	19(29.7)	45(70.3)	64(100)		1
	Two or more	81(62.3)	49(37.7)	130(100)	3.915(2.058-7.447)	1.67×10^{7}(0.000-∞)
	more than five	4(28.6)	10(71.4)	14(100)	0.947(0.26-3.399)	1.14×10^{7}(0.000-∞)
Parity	Once	20(45.5)	24(54.5)	44(100)	1	1
	Two or more	62(66)	32(34)	94(100)	1.833(0.828-4.058)	0.41(0.021-4.866)
	more than five	20(31.3)	4(66.77)	64(100)	4.262(2.161-8.402)	2.65×10^{9}(0.000-∞)
	Zero	19(29.7)	45(70.3)	64(100)	1.100(0.186-6.508)	4.5×10^{7}(0.000-∞)
ANC contact	.<4x	1(2.3)	42(97.7)	43(100)		1
	>4x	103(62.4)	62(37.6)	165(100)	69.77(9.367-519.721)	69.24(6.77-707.73) *
Proteinuria	Yes	2(2.1)	94(97.9)	96(100)		1
	No	91.1	10(8.9)	112(100)	479.4(102.382-2244.78)	477.75(93.71-2435.7) *
Blood Group	O	33(38.4)	53(61.6)	86(100)		1
	A	26(53.1)	23(46.9)	49(100)	1.816(0.893-3.692)	1.958(0.773-4.961)
	B	26(60.5)	17(39.5)	43(100)	2.456(1.160-5.200)	2.344(0.82-6.695)
	AB	19(63.3)	11(36.7)	30(100)	2.774(1.173-6.558)	4.691(1.394-15.789)

6.4 Association of Preeclampsia with Maternal Outcomes

Pregnancy termination was significantly higher in PE women than in the control group (90(94.7%) vs. 5 (5.3%); δ^2 (1) = 139.991, p < 0.001). Mode of delivery showed significant associations (δ^2 (3) =26.420, p<0.001). Caesarean section was higher in PE women (40(71.4%) vs. 16(28.6%)), while spontaneous vaginal delivery was lower (56(40%) vs. 84(60%)), Breech and instrumental deliveries were also more common in the PE group (4(66.7) vs. 2(33.3). No significant difference in maternal complications was observed between the groups (p = 0.186) **(Table 4)**.

Table 4. Association of Preeclampsia with Maternal Outcomes

Variables	Categories	Mother condition(N=208)		Total N (%)	chi-square test(δ^2)	p-value
		Preeclampsia N (%)	Control N (%)			
pregnancy termination	Yes	90(94.7)	5(5.3)	95(100)	δ^2=139.991	0.001
	No	14(12.4)	99(87.6)	104(100)		
Mode of delivery	SVD	56(40)	84(60)	140(100)	17.219	0.001
	Breech D.	4(66.7)	2(33.3)	6(100)		
	Instrumental	4(66.7)	2(33.3)	6(100)		
	C/S	40(71.4)	16(28.6)	56(100)		
Maternal complication	No complication	97(48.5)	103(51.5)	200(100)	6.180	0.186
	Placenta abruption	2(100)	0(0)	2(100)		
	Thrombocytopenia	1(100)	0(0)	1(100)		
	Eclampsia	3(100)	0(0)	3(100)		
	PPH	1(50)	1(50)	2(100)		
Maternal outcome	Improved and discharged	103(49.8)	104(50.2)	207(100)	1.005	0.316
	Referred	1(100)	0(0)	1(100)		

6.5 Neonatal Outcomes

Preterm births were observed significantly higher in the PE women than the control group, 40 (83.3% vs. 8(16.7%)); δ^2 (3) = 36.710, p< 0.001)). Intrauterine fetal death (IUFD) 2(100%) and early neonatal death 4(100%) were reported exclusively among PE women. (**Table 5**).

Table 5. Association of Preeclampsia with Neonatal Outcomes (categorical variables)

Variables	Categories	Mother condition(N=208)		Total N (%)	chi-square test(δ^2)	p-value
		Preeclampsia N (%)	Normal N (%)			
Neonatal outcome	Preterm	40(83.3)	8(16.7)	48(100)	36.710	0.001
	IUFD	2(100)	0(0)	2(100)		
	Early neonatal death	4(100)	0(0)	4(100)		

The birth weight of the neonate from PE women was significantly less than in the control group (2608.17±54.537 vs. 3281.25±46.009, 95% CI: -813.751 to -532.402, p< 0.001). The 1st and the 5th minutes APGAR scores of the neonates from PE women were also significantly less than in the neonates from the control group (6.66±0.166, 95% CI: -1.110 to-0.352, p < 0.001; 7.66±0.197 vs. 8.55±0.095, 95%CI: -1.316 to 0.453, p < 0.001, respectively) **(Table 6).**

Table 6. Association of Preeclampsia with Neonatal Outcomes (continuous variable)

Variable	Categories	Group	mean ±SE	95% CI of the difference		p-value
				Lower	upper	
Neonate outcome	Weight(gram)	Preeclampsia	2608.17±54.537	-813.751	-532.402	0.001
		Control	3281.25±46.009			
	1st min APGAR	Preeclampsia	6.66±0.166	-1.110	-0.352	0.001
		Control	7.39±0.097			
	5st min APGAR	Preeclampsia	7.66±0.197	-1.316	0.453	0.001
		Control	8.55±0.095			

6.6 Hematological Indices

No significant differences were observed in red blood cell count (4.79±0.108 million cells /mm^3 vs. 4.73±0.118 million cells /mm^3, p = 0.726), hematocrit (36.75±0.624% vs. 36.29±0.450 %, p < 0.549), MCV (85.42±1.352 fL vs. 85.52±0.674 fL, p = 0.947), MCH (31.68±0.532 pg vs. 33.27±0.761 pg, p = 0.088), and MCHC (35.44±0.597 g/dL vs. 35.85±0.610 g/dL, p = 0.627) between PE and control groups.

However, hemoglobin level was significantly less in the PE women as compared with the control group (12.6 ±0.219 g/dL vs. 13.23±0.191g/dL, p< 0.031, 95% CI: -1.202 to -0.057). RDW was significantly higher in the PE group than in the control group (32.94±2.173 % vs. 24.83±1.504%, p< 0.02, 95% CI: 2.90 to 13.33)**.**

Neutrophil (11.97±1.861 cells/µL vs. 6.89±0.742 cells/µL, p < 0.012, 95% CI: 1.12 - 9.03), lymphocyte (4.55±0.749 cells/µL vs. 2.65±0.416 cells/µL, p < 0.028, 95% CI: 0.20-3.59), and monocyte counts (1.07±0.146 cells/µL vs. 0.6±0.081 cells/µL, p < 0.006, 95% CI: 0.13 - 0.79) were significantly higher among the PE women. Eosinophil (0.26±0.066 cells/µL vs. 0.48±0.1302 cells/µL, p = 0.139, 95% CI: -0.506- 0.0714) and basophil (0.25±0.079 cells/µL vs. 0.24±0.081 cells/µL, p = 0.932, 95% CI: -0.2136- 0.2329) counts showed no significant differences between groups.

Though the difference is not significant, the platelet count in the PE women is lower than in the control women (196.22±5.475/microliter vs. 210.50
±6.479/microliter, P = 0.094, 95% CI: -30.99-2.45). Significance difference was not observed between the groups in MPV (14.907±1.732 fLvs. 11.872±1.234 fL, p = 0.155, 95% CI: -1.1605-7.2297) **(Table 7).**

Table 7: Association of preeclampsia with hematological indices.

Variable	Categories	Group	mean ±SE	95% CI of the difference		p-value
				lower	upper	
Red blood cell Indices	RBC (million cells /mm^3)	Preeclampsia	4.79±0.108	-0.2589	0.3709	0.726
		Control	4.73±0.118			
	Hg (g/dL)	Preeclampsia	12.6 ±0.219	-1.2021	-0.0573	0.031
		Control	13.23±0.191			
	HCT (%)	Preeclampsia	36.75±0.624	-1.06	1.9789	0.549
		Control	36.29±0.450			
	MCV (fL)	Preeclampsia	85.42±1.352	-3.085	2.885	0.947
		Control	85.52±0.674			
	MCH (pg)	Preeclampsia	31.68±0.532	-3.421	0.2388	0.088
		Control	33.27±0.761			
	MCHC (g/dL)	Preeclampsia	35.44±0.597	-2.088	1.2622	0.627
		Control	35.85±0.610			
	RDW (%)	Preeclampsia	32.94±2.173	2.9004	13.3285	0.002
		Control	24.83±1.504			
White blood cell indices	Neutrophil (cells/µL)	Preeclampsia	11.97±1.861	1.115	9.026	0.012
		Control	6.89±0.742			
	Lymphocyte (cells/µL)	Preeclampsia	4.55±0.749	0.2046	3.5890	0.028
		Control	2.65±0.416			
	Monocyte (cells/µL)	Preeclampsia	1.07±0.146	0.1344	0.7933	0.006
		Control	0.6±0.081			
	Eosinophil (cells/µL)	Preeclampsia	0.26±0.066	-0.506	0.0714	0.139
		Control	0.48±0.1302			
	Basophil (cells/µL)	Preeclampsia	0.25±0.079	-0.2136	0.2329	0.932
		Control	0.24±0.081			
Platelet indices	Platelet (count /microliter)	Preeclampsia	196.22±5.475	-30.997	2.4508	0.094
		Control	210.50±6.479			
	MPV((fL)	Preeclampsia	14.907±1.732	-1.1605	7.2297	0.155
		Control	11.872±1.234			

6.7 Association between ABO blood group and feto-maternal outcomes among preeclamptic women.

No differences in the modes of delivery were in PE women with different blood groups (δ^2 (9) = 4.783, p = 0.853).Cesarean sections were highest in PE women with blood group O (60%, n = 24 of 40 cases), followed by group A (22.5%, n = 9 of 40), group B (10%, n = 4 of 40), and group AB (7.5%, n = 3 of 40). SVD rates were highest in group O (44.6%, n = 25 of 56 delivery), with similar rates for the other groups (A: 21.4%, 12 of 56; B: 19.6%, 11 of 56; AB: 14.3%, 8 of 56). Breech and instrumental deliveries were rare, both with 4 cases and without any blood group differences. Maternal outcomes were consistently positive, and 103 patients (51.5% in group O, 53 of 103; 22.3% in group A, 23 of 103; 15.5% in group B, 16 of 103; 10.7% in group AB, 11 of 103) recovered and were discharged, showing no notable differences (δ^2 (3) = 5.167, p= 0.16). Adverse neonatal results—preterm labor (60% in group O, 24 of 40 cases), intrauterine fetal death (IUFD; 50% each in groups O and B, 1of 2 cases), and early neonatal death (50% in group A, 2of 4 cases) did not differ significantly (δ^2 (9) = 9.86, p = 0.362) **(Table 8).**

Table 8: Association between ABO Blood group and Feto-maternal Outcomes among preeclamptic women.

		ABO Blood group (n (%))				Total N (%)	chi-square test(δ^2)	p-value
Variables	Categories	O	A	B	AB			
Pregnancy termination	Yes	48(53.3)	18(20)	14(15.6)	10(11.1)	90(100)	2.527	0.47
	No	5(35.7)	5(35.7)	3(21.4)	1(7.1)	14(100)		
Mode of delivery	SVD	25(44.6)	12(21.4)	11(19.6)	8(14.3)	56(100)	4.783	0.853
	Breech	2(50)	1(25)	1(25)	0(0)	4(100)		
	Instrumental	2(50)	1(25)	1(25)	0(0)	4(100)		
	C/S	24(60)	9(22.5)	4(10)	3(7.5)	40(100)		
Maternal Outcomes	Improved and discharged	53(51.5)	23(22.3)	16(15.5)	11(10.7)	103(100)	5.167	0.16
	referred	0(0)	0(0)	1(100)	0(100)	1(100)		
Neonatal outcomes	preterm	24(60)	4(10)	7(17.5)	5(12.5)	40(100)	9.86	0.362
	IUFD	1(50)	0(0)	1(50)	0(0)	2(100)		
	Early neonatal death	1(25)	2(50)	1(25)	0(0)	4(100)		

There was no statically significant association between Rh blood group and pregnancy termination (δ^2 (1) = 7.506, p = 0.12), mode of delivery (δ^2 (3) = 0.353, p = 0.95), maternal outcome (δ^2 (4) = 0.223, p = 0.994), and neonatal outcome (δ^2 (3) = 0.265, p = 0.966), among PE women. **(Table 9)**

Table 9: Association between Rh Blood group and fetal-maternal outcomes among preeclamptic women.

Variables	Categories	Rh Blood group (n (%))		Total N (%)	chi-square test(δ^2)	p-value
		Rh $^{+ve}$	Rh $^{-ve}$			
Pregnancy termination	Yes	89(98.9)	1(1.1)	90(100)	7.506	0.12
	No	12(85.7)	2(14.3)	14(100)		
Mode of delivery	SVD	54(96.1)	2(3.6)	56(100)	0.353	0.95
	Breech	4(100)	0(0)	4(100)		
	Instrumental	4(100)	0(0)	4(100)		
	C/S	39(97.5)	1(2.5)	40(100)		
Maternal Outcomes	Improved and discharged	100(97.1)	3(2.9)	103(100)	0.223	0.994
	Referred	1(100)	0(0)	1(100)		
Neonatal outcomes	Preterm	39(97.5)	1(2.5)	40(100)	0.265	0.966
	IUFD	2(100)	0(0)	2(100)		
	Early neonatal death	4(100)	0(0)	4(100)		

Additionally, the results showed no significant differences in birth weight among neonates of different blood groups (O, A, B, AB), with mean weights ranging from 2589.91±86.01gm (O group) to 2704±139.554gm (AB group) (p = 0.936). Similarly, APGAR scores at 1st and 5th minutes did not vary significantly across blood groups (p = 0.367 vs. 0.648, respectively), When comparing Rh^{+ve} and Rh^{-ve} neonates, Rh^{-ve} neonates had slightly higher mean APGAR scores, (7.63±0.202 vs. 8.67±0.333, 6.63±0.17vs. 7.67±0.33) in 1st and 5th minute of life respectively. However, the difference was not statistically significant in APGAR scores at both the 1st (p = 0.3) and 5th minute (p = 0.384) and birth weight (p = 0.217) (Table 10)

Table 10: Association between ABO/Rh Blood group and neonatal Outcomes (continuous variable) among preeclamptic women.

variables	BG	number	Mean	95% CI of the difference		P value
				Lower	upper	
weight	O	53	2589.91±86.01gm.	2412.31	2757.5	0.936
	A	23	2606.52±96.59 gm.	2406.19	2806.85	
	B	17	2620.59±128.27 gm.	2348.67	2892.51	
	AB	11	2704±139.554 gm.	2393.60	3015.49	
1st minute	O	53	6.77±0.228	6.32	7.23	0.367
	A	23	6.87±0.269	6.31	7.43	
	B	17	6.00±0.569	4.79	7.21	
	AB	11	6.73±0.407	5.82	7.63	
5th minute	O	53	7.72±0.272	7.17	8.26	0.648
	A	23	7.78±0.412	6.93	8.64	
	B	17	7.12±0.6	5.85	8.39	
	AB	11	8.00±0.381	7.15	8.85	
weight	$+^{ve}$	101	2596.13±55.3 gm.	2486.82	2706.25	0.217
	$-^{ve}$	3	3000±288.67 gm.	1757.13	4242.07	
1st minute	$+^{ve}$	101	6.63±0.17	6.3	6.97	0.3
	$-^{ve}$	3	7.67±0.33	6.23	9.10	
5th minute	$+^{ve}$	101	7.63±0.202	7.23	8.04	0.384
	$-^{ve}$	3	8.67±0.333	7.23	10.1	

6.8 Association between Onset of Preeclampsia and Neonatal Outcome

Most preeclampsia cases occurred after 34 weeks (73(70.2%) vs. 31(29.8%); $p<0.001$). Birth weight showed an inverse relationship with gestational age (δ^2 (24) =57.878, $p<0.001$). Reduced weights were higher at 20-34 weeks (<1000g: 1(100%) vs. 0(0%); 1000-1499g: 2(100%) vs. 0(0%); 1500-2499g: 19(63.3%) vs. 11(36.7%)), while increased weights (2500-3999g) were higher after 34 weeks (61(87.1%) vs. 9(12.9%)). One baby weighed >4000g (>34 weeks).

Both 1st minute ($\delta^2(7)=36.499$, $p<0.05$) and 5th minute Apgar scores ($\delta^2(7)=37.838$, $p<0.001$) showed lower values at 20-34 weeks (1st: 0-3=3(100%) vs. 0(0%); 4-6=22(55%) vs. 18(45%); 5th: 0-3=5(100%) vs. 0(0%); 4-6=10(71%) vs. 4(29%)), with increased scores (7-10) were higher after 34 weeks (1st: 55(90.1%); 5th: 69(81.2%)). Gestational age significantly affected survival (δ^2 (3) = 30.614, $p<0.001$), with worse outcomes at 20-34 weeks (IUFD: 2(100%) vs. 0(0%); neonatal death: 4(100%) vs. 0(0%)), though preterm deliveries were similar (19(47.5%) vs. 21(52.5%)) (**Table 10).**

Table10: Association between Onset of Preeclampsia and neonatal outcome

Variable	Categories	Onset of Preeclampsia		Chi square value(δ^2)	p-value
		20-34 week N=31(29.8 %)	>34 week N=73(70.2 %)		
Neonatal weight	<1000gm	1(100%)	0(0%)	57.878	0.001
	1000-1499gm	2(100%)	0(0%)		
	1500-2499gm	19(63.3%)	11(36.7%)		
	2500-3999gm	9(12.9%)	61(87.1%)		
	>4000gm	0(0%)	1(100%)		
1st minute APGAR score	0-3	3(100%)	0(0%)	36.499	0.001
	4-6	22(55%)	18(45%)		
	7-10	6(9.9%)	55(90.1%)		
5th minute APGAR score	0-3	5(100%)	0(0%)	37.838	0.001
	4-6	10(71%)	4(29%)		
	7-10	16(18.8%)	69(81.2%)		
Neonatal outcome	Preterm	19(47.5%)	21(52.5%)	30.614	0.001
	IUFD	2(100%)	0(0%)		
	Early neonatal death	4(100%)	0(0%)		

7. Discussion

This prospective cohort study found that preeclampsia (PE) was significantly associated with fewer antenatal care (ANC) contacts **(Table 3).** This finding aligns with established diagnostic criteria (National Guideline Alliance (UK) Hypertension in Pregnancy, 2023; North et al., 2011), suggesting that reduced antenatal care (ANC) contacts hinder hypertension screening (National Antenatal Care Guideline, 2022), delaying PE detection and management (Vasconcelos et al., 2022). This is particularly dangerous for high-risk women with obesity or excessive weight gain (Shao et al., 2017; Sole et al., 2021), who often miss early warning signs (Carter et al., 2021). Fewer visits also lower aspirin prophylaxis use (Sium et al., 2024), while silent PE progression (Lamarca, 2012), irregular monitoring allows severe complications to develop undetected.

Additionally, the study showed that PE was associated with proteinuria, a diagnostic criterion for PE consistent with findings (Karmia & Serudji, 2022; Palei et al., 2013), suggesting proteinuria is a diagnostic, not predictive, marker for PE. Inadequate cytotrophoblast invasion of spiral arterioles (Phipps et al., 2019b) causes placental ischemia (Maynard & Karumanchi, 2011), triggering hypoxia-induced anti-angiogenic factors characterized by elevated sFlt-1 and endoglin (Zouganeli et al., 2025), that impair VEGF signaling (Ferrara et al., 2003; Melincovici et al., 2018). VEGF deficiency damages podocytes (Moghaddas et al., 2019b), while impaired natriuresis exacerbates the hypertensive state (Alexander et al., 2001), ultimately manifesting as the multisystem disorder we recognize clinically as preeclampsia.

Although several study have reported a significant relationship between BMI and PE risk (Motedayen et al., 2019; Nekkanti et al., 2023; Tessema et al., 2021), this study found no significant difference in BMI between the groups **(Table 2).** This discrepancy may stem from Key methodological variations: unlike prior research focused on obese populations, this study included a general pregnancy BMI distribution. Furthermore, while previous studies relied on pre-pregnancy BMI measurements, this study assessed BMI during pregnancy- a period marked by gestational weight changes. Differences in population characteristics (e.g. lifestyle, diet, and genetic factors) may also contribute to these divergent findings.

A higher number pregnancy termination (94.7%) and caesarean rate (71.4%) was observed in PE women **(Table 4).** These findings are consistent with studies (Ali et al., 2018; Simpson, 2002 ;

Alemu et al., 2022; Shandilya et al., 2006), confirming that PE consistently leads to higher rates of pregnancy termination and caesarean deliveries. The elevated intervention rates reflect clinical responses to maternal-fetal compromise in PE, which often requires early delivery to prevent maternal complications (e.g., eclampsia, HELLP syndrome) and fetal risks (e.g., growth restriction, placental abruption) (ACOG., 2019; Magee et al., 2024). The high cesarean rate stems from the sudden worsening that demands immediate delivery (Varnier et al., 2018), unfavorable cervical conditions in preterm cases (Awoyesuku et al., 2024; Justus Hofmeyr, 2003), and institutional protocols that prioritize quick delivery in severe situations (Tita et al., 2022; Wu & Zhang, 2021).

This study found significantly reduced birth weights and higher preterm birth rates in PE cases **(Table 5),** aligning with prior research (Ali et al., 2018; Simpson, 2002; Tadese et al., 2024), suggesting PE was associated with reduced birth weights and increased preterm deliveries. These findings align with global evidence linking PE to placental insufficiency and intrauterine growth restriction (Phipps et al., 2019 ; Steegers et al., 2010;Haddad et al., 2004; Madazli et al., 2014). Furthermore, mirroring results from earlier studies (Ali et al., 2018; Simpson, 2002), this study observed decreased 1st and 5th minute Apgar score in neonates born to PE women (**Table 6**). This suggests placental insufficiency as the likely cause: when the placenta fails to provide adequate oxygen and nutrients (Wardinger& Ambati, 2025), it affects the neonate's condition at birth.

There was no significant association found between ABO/Rh blood groups and PE, contrasting with reports from (S. & K., 2015 ; Mital et al., 2016 ; Hiltunen et al., 2009; Lee et al., 2012), that identified higher risks associated with the AB blood group. This discrepancy may be attributed to key population and methodological differences. Notably, in this study predominantly blood group O and Rh+ with fewer AB individuals **(Table 8)**, differed from populations showing thrombogenic risks in AB groups. The smaller sample size (N=208) in this prospective study vs. larger retrospective analyses (N>5000) may also affect risk detection. These results suggest blood type's role in preeclampsia varies by population, while placental pathology remains the primary disease mechanism.

Similarly, no significant association was found between ABO/Rh blood groups and adverse maternal/ neonatal outcomes **(Table 8),** aligning with findings by (Cordero-Franco et al., 2023a;

Okoye et al., 2020), confirming that blood type does not independently influence adverse maternal/neonatal outcomes. Although ABO blood groups may modestly affect hemostasis and inflammation (Dusse et al., 2011), placental dysfunction specifically abnormal trophoblastic invasion and anti-angiogenic factor release (Michalczyk et al., 2020), appears to be the primary pathological mechanism. Moreover, standard treatments ($MgSO_4$, antihypertensives, NICU care) were equally effective across all blood groups.

Additionally, pregnant women with PE exhibited significantly reduced hemoglobin levels alongside elevated RDW, neutrophils, lymphocytes, and monocytes **(Table 7).** The reduced hemoglobin levels observed in this study are supported by studies (Elgari et al., 2019; Ali et al., 2011; Ahmed et al,.2025), suggesting anemia was common among PE women. This phenomenon likely results from both pregnancy-induced hemodilution (Morton, 2021) and inflammation-mediated suppression of erythropoiesis (Kashiwagi et al., 2002; Weiss et al., 2019). The observed RDW elevation in PE women **(Table 7),** consistent with studies (Avcıoğlu et al., 2015; Kassahun et al., 2024), suggesting elevated RDW levels in PE that may reflect underlying inflammation and oxidative stress (Yılmaz et al., 2021).

In contrast to a study conducted in India (Smita et al., 2024), which reported higher MCV and MCHC in pregnant women with preeclampsia, our findings showed no such correlation. The discrepancy may reflect methodological variations between studies. Specifically, in this study, analysis included a larger cohort (208) with balanced case-control groups (104 PE patients/104 controls) compared to their smaller (151), unequally distributed sample (82 cases/70 controls). Additionally, this study enrolled participants at an earlier gestational age (>20 weeks) versus their >28-week inclusion criterion, which could influence hematological parameters.

Consistent with (Örgül et al., 2019), there were no significant differences in RBC count, HCT, or erythrocyte indices (MCV, MCH, MCHC) between groups. However, we found elevated lymphocyte, neutrophils and monocytes counts **(Table 7),** aligning with findings by (Örgül et al., 2019; Bozdağ et al.,2018 ; Liao et al.,2022) suggesting elevated leukocytes. These hematologic changes demonstrate PE's inflammatory nature, characterized by leukocyte activation (Faas et al., 2014), oxidative stress (Al-Qahtani et al., 2024) and endothelial dysfunction (Baddam & Burns, 2025), confirming systemic inflammation's central role in pathogenesis.

This study found no significant differences in platelet indices between PE and normotensive pregnant women **(Table 7),** contrasting with reports from (Nooh & Abdeldayem, 2015; Walle et al., 2022), who observed the lower platelet levels and an elevated MPV in PE cases. This discrepancy may be attributed to PE severity variation, as this study included milder cases and blood sampling occurred at earlier gestational ages while previous studies focused exclusively on severe PE. Additionally, methodological variations likely contributed to these divergent findings: this study employed a prospective design, whereas previous study used retrospective approaches.

Late-onset PE was significantly more common than early-onset cases. Additionally, late-onset PE correlated with better outcomes (higher neonatal weight, APGAR scores, and survival) compared to early-onset PE. **(Table 10).** This aligns with studies (Burton et al., 2019 ; Simpson, 2002) , who reported a higher prevalence of late-onset PE with comparatively lower maternal-fetal risk. This Suggest late-onset PE often linked to maternal metabolic/vascular stress, while early-onset PE involves placental dysfunction and higher fetal risks.

8. Strengths and limitations of the study

8.1. Strength

This study offers several important methodological advantages over previous work in this area. The prospective collection of primary clinical data provides robust, real-time measurements of disease progression. Standardized laboratory protocols, including complete blood counts and proteinuria assessments, help ensure consistent and reliable data collection. Inclusion of a matched control group strengthens the validity of observational comparisons. Unlike many prior studies that focused solely on maternal outcomes, this research also systematically evaluates neonatal outcomes, providing a more comprehensive understanding of preeclampsia's effects. These design features address several limitations present in earlier retrospective studies while offering new insights into the hematologic and clinical manifestations of the condition.

8.2 Limitations

This hospital-based study has several important limitations. The findings may have limited generalizability due to the sampling approach and relatively small cohort size. Additionally, unable to account for all potential confounders, including variations in preeclampsia severity and timing of onset. Inconsistent timing of laboratory measurements may also have affected biomarker reliability.

9. Conclusion

This study showed the presence of significant associations between reduced antenatal care contact, proteinuria and PE. While blood group O was most common in PE, no association was observed between ABO/Rh blood groups and PE. Maternal blood types were also not associated with neonatal adverse outcomes in preeclamptic women. However, PE was significantly associated with adverse neonatal outcomes. While hemoglobin level was reduced, RDW, neutrophils, lymphocyte, and monocytes were higher in the preeclamptic women as compared with the control group. Additionally, there were significant correlations between preeclampsia onset time and neonatal weight, APGAR scores, and neonatal survival

10.Recommendation

For the better management of PE, ANC protocols must be targeted towards early booking, regular visits, and routine screening for proteinuria, blood pressure, and hematological markers. RDW and leukocyte indices can be used as inexpensive inflammatory markers for assessing risk at an early point, especially in resource-poor settings. Policy changes, such as expanding ANC coverage through community health workers and expanding EmONC facilities, are a priority for poor regions.

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Annex-1 Questionnaire

Addis Ababa University, institute of health, medical faculty, Department of medical physiology Checklist format on Blood group and Rh association with preeclampsia at NRH, Nekemte Town, Ethiopia, and facility based comparative cross sectional study from Jan 2017-July 2017.

Consent form:

My name is _____. I am working with Debissa Kefiyalew, who is doing research on blood group and Rh association with preeclampsia in NRH.

The purpose of this study is to understand better why this happened so that we can help improve care for mothers in this hospital in the future. By addressing these issues, we hope to better inform policymakers, health providers, and women about key findings that will contribute towards devising interventions that will improve maternal health in the country. In order to assess what might have been reasons for the preeclampsia, we will interview you using a structured questionnaire. We would like to collect some information about you by reviewing the medical records and by interviewing you. All information will be kept confidential, and neither your name nor other identifying information will be recorded. If you do not want to answer all of or some of the questions, you do have the right to do so. However, your willingness to answer all of the questions would be appreciated.

Would you participate in responding to the questions in this questionnaire?

Yes □ No □

Name of interviewer: _____ signature_____

Name of the supervisor _____ signature _____

Instruction: The respondents must be mother herself. Please encircle the letter corresponding to the correct response or write the response of the respondent on the space provided.

Identification

1. Questionnaire I.D:_____

2. Date of checking_____

Remark: 1. Complete 2. Incomplete

Part I: Socio-demographic Information			
No.	**Questions**	**Response**	**code**
Q101	age in years	1.18-24 2.25-34 3. 35-48	
Q102	Religion	1.Orthodox 2.Protestant 3.Muslim 4.other	
Q103	Residence	1.Urban 2.Rural	
Q104	Marital status	1.Married 2.Unmarried 3.Widowed/Divorced 4.Not stated	
Q105	Occupation	1.House wife 2.Employee 3.Student 4.Other	
Q106	Educational status	1.Illiterate 2.Write and read 3. Completed primary school (1-8) 4. High school (9-12) 5. Higher level education	

Part II: Obstetric characteristics			
Q201	Gravidity	1.Once 2.Two or .more 3.more than five	
Q202	Parity	1.Once 2.Two or more 3.more than five	
Q204	GA in weeks	1. 20-34week 2. >34 week	
Q205	Where did you have ANC follow-up During the pregnancy	1.Health post 2.Health centre 3.Hospital 4.Private Clinic 5.Mixed 6.No ANC	
Q206	If yes for Q205, how many times?	1.<4x 2.>4x	
Q207	Onset of preeclampsia	1. 20-34week 2. >34 week	
Q208	Family history of preeclampsia	1, Yes 2.. No	
Q209	Previous history of preeclampsia	1, Yes 2.. No	
Part III Assessment criteria for diagnosing preeclampsia			
Q301	Blood pressure at admission	……..…… mmHg	
Q302	Has Proteinuria.	1. Yes 2. No	
Q303	What was the Diagnosis?	1. Preeclampsia without Severe future	

		2. Preeclampsia with Severe future 3. Eclampsia	
Part IV lifestyle			
Q401	Height	----------Cm	
Q402	Weight	----------Kg	
Q403	BMI	----------$\frac{Kg}{Cm^2}$	
Part V ABO Blood Group and Rh Distribution			
Q501	Blood group	1. O 2. A 3. B 4. AB	
Q502	If O for Q501	What neonate Blood Group-------	
Q503	RH Factor	1. Rh $^{-ve}$ 2. Rh $^{+ve}$	
Q504	If Rh $^{-ve}$ for Q503	A. What paternal Rh---------- B. What neonate Rh----------	
Part VI Maternal out come			
Q601	Was induced labour /pregnancy terminated?	a.Yes **1.** No	
Q602	What was the mode of delivery?	a. Spontaneous vaginal delivery (SVD) b. Breech delivery c. Instrumental Delivery (Vacuum extraction or Forceps delivery) d. Caesarean section e. other(mention)	

		…….	
Q603	Was there any maternal complication?	a. No complication b. Placental abruption c. Thrombocytopenia (<100 000/µl) d. Eclampsia/convulsion e. Pulmonary edema f. DIC/HELLP syndrome g. Post-partum hemorrhage h. Renal failure/Oliguria i. Maternal Death	
Q604	What was the outcome of the mother?	a. Improved & discharged b. Referred c. Died d. Left against medical advice	
Part VII Neonatal outcome			
Q701	What was sex of the neonate	a. Male b. female	
Q702	What was the Weight(s) of the neonate	..... grams	
Q703	What was the APGAR Score for neonate in 1st and 5th minute of life	……	
Q704	What was the neonatal outcome	a. Preterm/ Prematurity b. Stillbirth (IUFD) c. Early Neonatal Death d. Other (mention) **.....**	
PART VIII Complete Blood Count (CBC)			

Q801	**Red Blood Cells (RBC):**	1. Count…….. million cells/µL 2. Hb …… g/dL. 3. HCT……..%. 4. MCV…… fL. 5. MCH…… pg. 6. MCHC……. g/dL. 7. RDW……%	
Q802	**White Blood Cells (WBC)**	1. Neutrophils…….. cells/µL 2. Lymphocytes……. cells/µL. 3. Monocytes…… cells/µL. 4. Eosinophils……cells/µL. 5. Basophils……cells/µL.	
Q803	**Platelet**	1. Platelet count……./microliter 2. MPV…..…… fL	