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Comparative Study of Demographic, Clinical and Haematological Parameters in the Third Trimester of Hypertensive and Normotensive Pregnant Women

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Abstract

The purpose of this study was to assess the demographic, clinical, and haematological characteristics of women with hypertensive disorders of pregnancy (HDP) compared to normotensive pregnant individuals during their third trimester. It was a cross-sectional study involving 270 women with HDP and 270 normotensive individuals admitted to the maternity unit at Nelson Mandela Academic Hospital in South Africa. The definition of HDP followed the International Society for the Study of Hypertension in Pregnancy guidelines. Blood pressure was measured using an automated device and blood samples were collected for measurement of haemoglobin, platelet count, creatinine, alanine transaminase (ALT), aspartate transaminase (AST), and lactate dehydrogenase (LDH). The median age was 27 years while the youngest and oldest were 15 years and 46 years respectively. The unemployment rate was higher among women with HDP compared to normotensive individuals ($p = 0.017$). HIV prevalence showed no significant difference ($p > 0.05$). Hypertensive cases had higher median pulse rates [87(74-98) vs 82 (IQR:67-95) b/m, $p = 0.023$] and lower median platelet counts [(230 (IQR:159-281) vs 240 (IQR:192-293) $x10^9$/L, $p = 0.009$). Additionally, hypertensive cases had significantly ($p < 0.0001$) higher median levels of urea (3 vs 2 mmol/L), creatinine (60 vs 50 umol/L), AST (28 vs 21 u/L), ALT (14 vs 11 u/L), and LDH (383 vs 270 u/L).In conclusion, women with HDP exhibit distinct clinical and haematological differences compared to normotensive controls, highlighting the importance of careful monitoring for patients with HDP to mitigate potential complications.

Keywords: Preeclampsia, hypertensive disorders of pregnancy, Haematological markers, Blood pressure, Employment status, Demographics, Clinical parameters.

INTRODUCTION

Hypertension during pregnancy has become an important public health issue, impacting not only maternal health but also foetal well-being and neonatal outcomes. The primary types of hypertensive disorders in pregnancy include gestational hypertension, preeclampsia (PE), and chronic hypertension (Brown *et al*., 2018;Espinoza *et al*., 2020). Research indicates that these conditions can lead to severe maternal health complications and adverse effects on the foetus, including renal failure, Haemolysis Elevated Liver enzymes and Low Platelets (HELLP) syndrome, abruptio placentae, pulmonary oedema, low birth weight, and preterm birth (Brown *et al*., 2018).

The incidence and demographic characteristics of women affected by hypertensive disorders of pregnancy (HDP) vary significantly across different populations. Globally, hypertensive disorders affect approximately 5-10% of all pregnancies, with variations ranging from 4% to 25% (Hutcheon *et al*., 2011; Jeyabalan, 2013; Wang *et al*., 2021). In sub-Saharan Africa, while data are limited, the prevalence of HDP is estimated to be around 8% (Gemechu *et al*., 2020). In a study of black South African primigravid women in Durban, the prevalence was identified at 12.5%, with PE affecting 5.8% of these women (Moodley *et al*., 2016).

The pathophysiological mechanisms underlying hypertensive disorders in pregnant women, in particular, PE, differ markedly from those in normotensive women. Research suggests that women with PE experience changes in placental function, elevated levels of inflammatory markers, and endothelial

dysfunction (Khan *et al*., 2005). A key factor in this process is the inadequate remodelling of spiral arteries. Ineffective trophoblast invasion results in intermittent blood flow, leading to ischemia-reperfusion cycles that create a hypoxic environment associated with oxidative stress and inflammation (Myatt & Webster, 2009). One proposed mechanism for endothelial dysfunction is the release of soluble fms-like tyrosine kinase (sFlt-1), an anti-angiogenic protein that inhibits vascular endothelial growth factor (VEGF), which exacerbates endothelial dysfunction through oxidative stress and reactive oxygen species (Luttun *et al*., 2002; Maynard *et al*., 2003; Zhou *et al*., 2011). The endothelium, a single layer of cells lining blood vessels, is crucial for regulating vascular tone, inflammatory responses, homeostasis, and coagulation (Galley & Webster, 2004). Impaired endothelial function in cases of PE can lead to increased vascular permeability, platelet consumption, organ ischemia, vasoconstriction, and the clinical manifestations of PE, including elevated blood pressure and complications like renal failure and HELLP syndrome, and others (Curtin L., 1999; Young *et al*., 2010).

Patients with HELLP syndrome may exhibit pathological assessments revealing hepatic intravascular fibrin deposits, which can obstruct hepatic sinusoids, increasing intrahepatic pressure and causing vascular congestion. This may ultimately lead to hepatic necrosis, subcapsular haemorrhage, and a risk of capsular rupture over time (Dusse *et al*., 2015). Such patients typically present with elevated liver enzymes and features of haemolysis.

Recognizing the haematological and clinical differences between normotensive and hypertensive patients is vital for early identification and management of those at risk, along with the development of targeted therapeutic strategies (Belovic *et al*., 2019).

Given the increasing incidence of HDP and their potential long-term impacts, this study aims to outline and compare the demographic, clinical, and haematological characteristics of pregnant women with hypertension against those of normotensive controls. The goal is to promote the groundwork for enhanced screening and management strategies for this vulnerable population.

Aim: The purpose of this study was to assess the demographic, clinical, and haematological characteristics of women with HDP compared to normotensive pregnant individuals during their third trimester.

METHODOLOGY

We conducted a prospective cross-sectional study involving 270 women with HDP and 270 normotensive controls admitted to the maternity unit at Nelson Mandela Academic Hospital in South Africa. HDP was defined according to the International Society for the Study of Hypertension in Pregnancy guidelines (Brown *et al*., 2018). Eclamptic patients were deliberately separated from those with PE without eclampsia due to the seriousness of eclampsia as a complication of PE.

Patients with cardiac diseases, multiple pregnancies, diabetes mellitus, chronic renal disease, antiphospholipid syndrome, or other autoimmune disorders were excluded from the study.

Blood pressure was measured using an automated device, the Microlife WatchBP Office. Measurements were taken three times at five-minute intervals after a 30-minute rest period. Participants were instructed to sit upright with their arm resting on the armrest of the chair and legs uncrossed. The monitor was activated after an appropriately sized cuff was placed on the participant's left mid-arm. The cuff was inflated, allowing the machine to automatically calculate blood pressure, pulse rate, and central pulse pressure values.

Blood samples were collected from each participant using two different types of tubes. For serum haemoglobin and platelet count, 10 mL of venous blood was drawn from the antecubital vein into tubes containing EDTA. The plasma was then separated through centrifugation at 4°C for 15 minutes at 3500 rpm and stored at -80°C until analysis. An additional 10 mL of venous blood was drawn into yellow tubes containing Solution A (trisodium citrate, citric acid, and dextrose) for the measurement of creatinine, alanine transaminase (ALT), aspartate transaminase (AST), and lactate dehydrogenase (LDH). All assays were performed using an Alinity iSTAT analyzer (Abbott, USA).

Statistical analysis

Data were checked for completeness and accuracy before being captured using the IBM SPSS STATISTICS software package version 29 for Windows (IBM Inc., Chicago IL, USA). Blood pressure readings were interpreted using medians and percentiles (q25, q75) due to the skewed nature of the data. Pearson's chi-square test was used to interpret categorical data, while the Kruskal-Wallis H test was employed to assess the association between continuous variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic characteristics of the women with hypertensives disorders of pregnancy and normotensive controls. The study enrolled 540 pregnant women, with 270 having HDP. Among those, 143 had preeclampsia, 102 eclampsia, 14 chronic hypertension, and 11 gestational hypertension.

The median age was 27 years while the youngest and oldest were 15 years and 46 years respectively. The non-hypertensive controls (median =

28 years) were significantly older than the hypertensive cases (median = 25 years) (p<0.0001). In terms of age groups, the majority (63.1% n=341) were in the 20 to 34 years age group and the minority (17.2% n=93) were younger than 20 years.

The other demographic characteristics of the women with HDP and normotensive controls are shown in **Table 1**. The unemployment rate was significantly higher among women with HDP than among the normotensive controls (p = 0.017). However, no significant differences (p>0.05) were found between women with HDP and the normotensive controls for all other characteristics.

Nearly a third of women in this study (n = 175; 95% CI=28.6% - 36.4%) were classified as obese, while 67 (12.4%; 95% CI= 9.8% - 15.4%) were severely obese, and 3 (0.6%; 95% CI= 0.2% - 1.5%) were underweight.

Of all the women who participated in the study, 153 (28.3%; 95% CI= 24.7% - 32.2%) were HIV-positive, while the rest were HIV negative. There was no significant difference in the HIV prevalence among the women with HDP and the normotensive controls (p>0.05). Most of the HIV-positive women had viral loads below the detectable limits, and only 19 (12.4%; 95% CI= 7.9% - 12.4%) had not yet achieved viral suppression. There were no significant differences between HIV-positive women with HDP and HIV-positive normotensive controls with regard to viral suppression (p>0.05).

Table 1: General characteristics of hypertensives cases and normotensive controls

Characteristics	Hypertensive cases; n= 270	Normotensive controls; n=270	Total	Pearson X^2 Sig.
	n (%)	n (%)	n (%)	
Ethnicity				
Black African	269 (99.6)	268 (99.2)	537 (99.4)	0.624*
Mixed race	1 (0.4)	2 (0.7)	3 (0.6)	
Marital status				
Single	210 (77.8)	207 (76.7)	417 (77.2)	0.837*
Married	60 (22.2)	62 (23.0)	122 (22.6)	
Divorced	0 (0.0)	1 (0.4)	1 (0.2)	
Level of education				
Tertiary	35 (13.0)	46 (17.0)	81 (15.0)	0.194*
Secondary	207 (76.7)	189 (70.0)	396 (73.3)	
Primary	27 (10.0)	35 (13.0)	62 (11.5)	
No formal education	1 (0.4)	0 (0.0)	1 (0.2)	
Occupation				
Unemployed	234 (86.7)	232 (85.9)	466 (86.3)	0.017
Student	14 (5.2)	4 (1.5)	18 (3.3)	
Employed	22 (8.1)	34 (12.6)	56 (10.4)	
Nutritional status				
Underweight (BMI <18.5)	2 (0.7)	1 (0.4)	3 (0.6)	0.196*
Normal weight (BMI 18.5-24.9)	81 (30.0)	59 (21.9)	140 (25.9)	
Overweight (BMI ≥25.0-29.9)	74 (27.4)	81 (30.0)	155 (28.7)	
Obese (BMI ≥30.0-39.9)	79 (29.3)	96 (35.6)	175 (32.4)	
Severely obese (BMI ≥40.0)	34 (12.6)	33 (12.2)	67 (12.4)	
HIV status				
Positive	66 (24.4)	87 (32.2)	153 (28.3)	0.056
Negative	204 (75.6)	183 (67.8)	387 (71.7)	
Viral suppression				
Unsuppressed (>1000 cp/mL)	10 (15.2)	9 (10.3)	19 (12.4)	0.609*
Suppressed (<1000 cp/mL)	0 (0.0)	1 (1.1)	1 (0.7)	
Less than detectable limit (<50 cp/mL)	56 (84.8)	77 (88.5)	133 (86.9)	

*BMI = body mass index (kg/m^2); cp/mL = copies/mL; *Fisher's Exact p-value*

Clinical and haematological characteristics in hypertensive cases and normotensive controls

The clinical and haematological characteristics of the sample population are presented in **Table 2** and show that central pulse pressure and haemoglobin were not different across the study groups. All other measurements: pulse rates, platelet, urea, creatinine, aspartate transferase, alanine transaminase, and lactate dehydrogenase differ significantly between the two groups.

The median pulse rate for the sample population was 84 (IQR:70-97) b/m). Hypertensive cases had significantly higher median pulse rates [87(IQR:74-98) b/m; p=0.023] than non-hypertensive controls [82 (IQR:67-95) b/m]. The median pulse rate for the sample population was 84 b/m, with hypertensive cases exhibiting a significantly higher median pulse rate of 87 (IQR:74-98) b/m compared to 82 (IQR:67-95) b/m in non-hypertensive controls (p=0.023). Additionally, the median platelet count was significantly lower in hypertensive cases, at 230 (IQR:159-281) $x10^9$/L, compared to non-hypertensive controls, which had a count of 240 (IQR:192-293) 10^9/L (p=0.009).

Moreover, hypertensive cases showed significantly higher median levels of urea (3 vs. 2 mmol/L), creatinine (60 vs. 50 µmol/L), aspartate transferase (28 vs. 21 u/L), alanine transaminase (14 vs. 11 u/L), and lactate dehydrogenase (383 vs. 270 u/L) when compared to non-hypertensive controls, with p-values indicating strong statistical significance (p<0.0001).

Table 2: Clinical and haematological characteristics in hypertensive cases and normotensive controls

	Cases; n=270	**Controls; n=270**	**Total; n=270**	**Pearson X^2 Sig.**
Clinical characteristics	**Median (IQR)**	**Median (IQR)**	**Median (IQR)**	
Pulse rate (beats/minutes)	87(74-98)	82 (67-95)	84 (70-97)	0.023
Central pulse pressure	65 (52-80)	67 (56-86)	66 (55-82)	0.148
Haemoglobin (g/dL)	12(11-13)	11(11-12)	12 (11-13)	0.271
Platelet ($x10^9$ /L)	230 (159-281)	240 (192-293)	234 (178-289)	0.008
Urea (mmol/L)	3 (2-4)	2 (2-3)	2(2-3)	<0.0001
Creatine (umol/L)	60(52-76)	50.0 (44-59)	54 (47-67)	<0.0001
Aspartate transaminase (u/L)	28 (19-47)	21 (17-26)	23 (18-36)	<0.0001
Alanine transaminase (u/L)	14 (10-24)	11 (9-15)	12 (9-18)	<0.0001
Lactate dehydrogenase (u/L)	383 (278-557)	270 (225-325)	305 (235-444)	<0.0001

Further analysis revealed that the pulse rate, central pulse pressure, haemoglobin, and platelet counts were statistically the same (p>0.05) among the cases irrespective of the type of HDP (**Table 3**). It was also observed that the cases with eclampsia had significantly (p<0.0001) higher creatinine [64(IQR:54-81) umol/L], aspartate [34(IQR:23-57)) u/L], and lactate dehydrogenase [452(IQR:330-648)) u/L].

Table 3: Clinical and haematological characteristics according to the studied hypertensive disorders of pregnancy

	A; n= 11	**B; n= 14**	**C; n= 68**	**D; n= 75**	**E; n= 102**	**Kruskal-Wallis Sig.**
Clinical characteristics	**Median (IQR)**	**Median (IQR)**	**Median (IQR))**	**Median (IQR)**	**Median (IQR)**	
Pulse rate (beats/minutes)	79 (69-87)	87(74-99)	82 (73-96)	85.(73-95)	90 (77-102)	0.106
Central pulse pressure	69.0 (64-75)	62 (49-73)	65 (55-80)	59 (52-80)	71(57-83)	0.117
Haemoglobin (g/dL)	12(11-13)	12 (12-13)	12 (11-13)	12 (11-13)	11(11-13)	0.820
Platelet ($x10^9$ /L)	234 (212-335)	236 (172-326)	232(168-285)	200 (148-270)	244 (154-282)	0.511
Urea (mmol/L)	2 (2-3)	2(2-3)	3 (2-4)	4 (2-5)	3 (2-3)	0.002
Creatine (umol/L)	57 (53-67)	55(41-65)	54 (51-65)	63 (54-86)	64(54-81)	0.001
Aspartate transaminase (u/L)	19(15-22)	19(15-23)	23(18-30)	31(21-52)	34(23-57)	<0.0001
Alanine transaminase (u/L)	10 (8-14)	11 (10-14)	13(9-19)	17(11-30)	15(11-29)	0.006
Lactate dehydrogenase (u/L)	234 (190-305)	252(195-320)	297(245-389)	434 (316-613)	452(330-648)	<0.0001

A= Gestational Hypertension; B= Chronic Hypertension; C= Preeclampsia Without Severe Features; D= Preeclampsia with Severe Features; E=-Eclampsia

DISCUSSION

This study assessed the demographic, clinical, and haematological characteristics of 540 pregnant women, contrasting those with HDP against normotensive controls. One significant finding was the increased unemployment rate among women experiencing HDP when contrasted with those who were normotensive. This observation is consistent with existing research that links lower employment status and income levels to an increased occurrence of HDP. For example, studies by Tebeu *et al*., Guerrier *et al*., and Ajah *et al*. support this correlation, highlighting the unemployment status and other socioeconomic factors that impact health(Ajah *et al*., 2016; Guerrier *et al*., 2013; Tebeu *et al*., 2011). Additionally, a thorough review by Choe *et al*. analysed a Korean National Health Insurance database with 65,479 obstetric deliveries, revealing that women from lower household income categories face a higher independent risk of developing PE(Choe *et al*., 2016). This illustrates the multifaceted nature of HDP, demonstrating that both biological and social-economic factors shape maternal health outcomes.

Notably, a larger proportion of women with eclampsia was intentionally included in this study, as previous research often overlooks this serious

complication of PE (Makhanya *et al*., 2016; Moodley, 2008; Moodley *et al*., 2018; Namugowa *et al*., 2017). Eclampsia generally occurs more frequently in younger women, likely influencing the maternal age among those with HDP in this study(Vousden *et al*., 2019). As a result, the control group without hypertension was significantly older than the group with hypertension. The median age of participants was 28 years, with most in the 25 to 35 age range. This age distribution is particularly interesting, given that prior studies have delivered mixed findings on the association between maternal age and the risk of developing HDP. Some suggest that women aged 25 to 35 may have a higher risk, while others indicate increased risks for those older than 35 or younger than 25 (Bromfield *et al*., 2023; Hinkosa *et al*., 2020; Poudel *et al*., 2021). These inconsistencies highlight the complexity of the relationship between age and HDP, possibly reflecting varying demographic, environmental, and healthcare-related influences on these outcomes.

The relationship between HIV and HDP is a complex and evolving area of research, as both conditions share a common pathway involving endothelial cell dysfunction (Naicker *et al*., 2021). Despite this shared mechanism, studies have produced varying results regarding the association between HIV and HDP. In the current study, we found that the prevalence of HIV among participants with hypertension was comparable to, or even lower than, that among individuals without hypertension. While some studies and meta-analyses, such as those conducted by Browne *et al*. (2015) and Shiadeh *et al*. (2018), found no significant correlation between HIV positivity and HDP, Calvert & Ronsmans (2015) in their study, indicated a potential association(Calvert & Ronsmans, 2015;Shiadeh *et al*., 2019). Notably, the meta-analysis by Calvert & Ronsmans included fewer studies compared to the others, which may explain the differing conclusions. Adding to this discussion, a recent case-control study by Sikhosana *et al*. (2022) offers a unique perspective that partly aligns with the findings of the present study(Sikhosana *et al*., 2022). It suggests that untreated HIV could actually have a protective effect against conditions such as PE. This intriguing assertion presents a paradox: although HIV is generally viewed as a detrimental condition, its untreated state may offer unexpected benefits in the context of HDP. However, this protective effect seems to disappear once antiretroviral therapy is initiated, necessitating further investigation into the underlying mechanisms involved(Sikhosana *et al*., 2022).

Regarding other clinical characteristics, both study groups in the current study demonstrated similar central pulse pressure but the heart rate was significantly elevated in hypertensive cases compared to normotensives. Previous research has established that central pulse pressure has a stronger correlation with carotid hypertrophy and the extent of atherosclerosis and is a better predictor of cardiovascular disease incidence compared to brachial pulse pressure (Roman *et al*., 2010; Roman *et al*.,2007; Safar *et al*.,2002). However, the present study, which did not specifically compare participants with cardiovascular diseases to those without, revealed no significant difference in central pulse pressure between preeclamptic and normotensive individuals. Our findings are consistent with those of Hernández-Mora *et al*., who studied 78 women in the immediate puerperium—39 with PE and 39 with normotension—finding no difference in pulse pressure between the two groups(Hernández-Mora *et al*., 2023). Additionally, Savvidou *et al*. (2011) showed that women at risk of developing PE, based on uterine artery doppler findings, did not have significantly different levels of central pulse pressure compared to those who were not at risk(Savvidou *et al*., 2011). In contrast, our findings differ from those reported by Arioz *et al*. (2008), who conducted a case-control study with 30 preeclamptic and 30 normotensive pregnant women, observing significantly higher peripheral pulse pressure in the preeclamptic group (Arioz *et al*., 2008). Furthermore, Namugowa *et al*. found that among 197 participants, both central and peripheral pulse pressures were significantly elevated in women with PE (Namugowa *et al*., 2017). Similarly, Torrado *et al*. (2015) reported higher central pulse pressure in 8 preeclamptic individuals compared to 16 normotensive individuals(Torrado *et al*., 2015). The current study involved more participants than previous studies, which may explain the discrepancies in findings.

In this study, individuals with hypertension displayed a significantly higher heart rate compared to those with normal blood pressure. This observation is consistent with the findings of Namugowa *et al*. (2017), which indicated that individuals with PE had higher peripheral heart rates than those with normal blood pressure. Overall, this aligns with existing literature on hypertension in the general population (Dalal *et al*., 2019).

In addition to blood pressure findings, the present study found significantly elevated mean serum levels of ALT, AST, and LDH and significantly low platelet count in patients with HDP compared to normotensive patients. The increase in these liver enzymes, and low platelet count can be attributed to placental ischemia, which triggers a systemic inflammatory response. In PE, the stressed syncytiotrophoblast releases anti-angiogenic factors, resulting in endothelial damage, vasoconstriction, oxidative stress, and the formation of micro-emboli (Burton *et al*., 2019; Ives *et al*., 2020). This endothelial damage can also lead to haemolysis as red blood cells travel through constricted vessels, further contributing to elevated liver enzyme levels (Abildgaard & Heimdal, 2013). These findings are consistent with previous research that has reported increases in these biomarkers among preeclamptic patients (Ekun *et al*., 2018; Hassanpour & Karami, 2018; Verma *et al*., 2022)

Moreover, our study found significantly higher mean plasma levels of urea and creatinine in the preeclamptic group compared to normotensive pregnant women, consistent with prior research (Ekun *et al*., 2018; Vyakaranam *et al*., 2015). In cases of PE, vasoconstriction and decreased blood flow lead to reduced renal plasma flow and glomerular filtration rate, ultimately causing elevated levels of urea and creatinine(Jeyabalan & Conrad, 2007; Lam & Dierking, 2017). The significantly elevated renal and liver parameters in eclamptic cases suggest that these women experience more severe organ dysfunction compared to those with other forms of HDP and may require targeted monitoring.

CONCLUSION

This study highlights significant differences in employment status, clinical characteristics, haematological and haematological markers between women with hypertensive disorders of pregnancy and normotensive controls. While central pulse pressure did not show the expected variations, heart rates were notably elevated in the HDP group, aligning with existing literature. Elevated liver enzymes and renal markers, along with altered hemodynamic, emphasize the necessity for careful monitoring to mitigate potential complications of HDP.

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