Pattern of Acid-Base Status in Hypertensive Disorders during Pregnancies in Benin City, Nigeria

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ABSTRACT

Background

Maintaining a healthy acid-base balance, ensuring adequate nutrition, and managing conditions like hypertensive disorders in pregnancy are essential aspects of maternal healthcare during pregnancy. Addressing these factors can help mitigate the risks associated with pregnancy complications and promote better outcomes for both the mother and the baby. The aim of this research, therefore, was to investigate and describe the patterns of acid-base status in pregnant women with hypertensive disorders in Benin City, Nigeria.

Materials and Methods

This study employed a prospective case-control design to investigate the acid-base status of pregnant women with preeclampsia, pregnancy-induced hypertension (PIH), and normotensive controls. The study was conducted at the Department of Obstetrics and Gynaecology, Central Hospital, and University of Benin Teaching Hospital, Benin City, Nigeria. A total of 190 pregnant women were recruited for this study. Blood pressure determination and urinalysis were necessary to divide the study participants into proeeclampsia, PIH and normotensive. Blood samples were collected for analysis of plasma lactate, lactate dehydrogenase, and bicarbonate levels to determine acid-base status. The study excluded women with maternal illnesses, multiple pregnancies, and gestational age <28 weeks.

Results

Results indicated that there were no significant differences in lactate as well as plasma bicarbonate levels among the preeclamptic, PIH and normotensive groups. However, the incidence of pregnancy induced hypertension elevated the Lactate dehydrogenase. The acid-base analytes (lactate, plasma bicarbonate, and LDH) did not show significant differences between mild and severe preeclampsia cases.

Conclusion

Notably, the findings revealed a discordant pattern, where a significant increase in lactate dehydrogenase (LDH) enzyme activity was not paralleled by a corresponding elevation in lactate levels, indicating a potential disconnect between LDH enzyme activity and lactate production in the study population.

Keywords: Lactate**,** Plasma bicarbonate, Lactate dehydrogenase, Acid-base status

INTRODUCTION

Hypertensive disorders during pregnancy are a significant cause of maternal and perinatal morbidity and mortality worldwide, particularly in underdeveloped nations like Nigeria. According to the World Health Organization, 15% of expectant mothers are at risk of life-threatening complications. Pregnancy-related Hypertension Disorders (HDP) play a major role in these complications, and managing them is crucial. HDP includes conditions like preeclampsia, eclampsia, gestational hypertension, and chronic hypertension [1,2,3].

Pregnancy induces significant changes in the mother's acid-base balance, characterized by a decrease in partial pressure of carbon dioxide (pCO2) and a rise in oxygen levels. This leads to a compensated respiratory alkalosis, accompanied by increased renal excretion and a drop in bicarbonate levels. The foetus also experiences acid-base changes, with a decrease in pCO2 and an increase in non-carbonic acids like lactate and keto acids. Understanding these changes is essential for managing acid-base status in pregnancies complicated by hypertensive diseases [2,3,4,5,6].

Studies have shown that pregnant women with hypertensive disorders have altered electrolyte levels, including lower sodium levels in preeclampsia and higher calcium levels in pregnancy-induced hypertension [5,7-12]. Additionally, pre-eclamptic women have lower serum calcium levels, which may contribute to the pathophysiology of the condition. Monitoring acid-base status and electrolyte levels is crucial for preventing and treating hypertension problems in pregnancy [8,9,11].

Effective management of hypertensive disorders during pregnancy requires recognition and management of pregnancy-related alterations to the respiratory and metabolic components of the acid-base system. Clinical management strategies should be tailored to individual trimester and hypertension condition patterns. By assessing acid-base status, healthcare providers can enhance foetal and maternal surveillance, improve pregnancy outcomes, reduce complications and morbidity, and optimize neonatal care [9, 10, 12-14]. This study aims to investigate the patterns of acid-base status among pregnant women in Benin City to provide baseline information for enhanced foeto-maternal care.

MATERIALS AND METHODS

Study Design

This study employed a prospective case-control design to investigate the acid-base status of pregnant women with preeclampsia, pregnancy-induced hypertension (PIH), and normotensive controls.

Study Setting

This research was carried out in Benin City. Two different hospitals which are the most-visited in the City were used for the study; these included Central Hospital, Benin as well as University of Benin Teaching Hospital. Participants were randomly selected based on their readiness to participate in the study; this was after ethical approval was obtained.

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Ethical Considerations

Before the investigations began, it was of utmost importance that informed consent was gotten from the individual partaker. Each participant was briefed on the importance of the study and were assured that their contributions would be treated with strict confidence; they were also guaranteed that they reserved the right to withdraw from the study whenever and however they felt it was necessary. Ethical approval for the study was also obtained from the Hospital's Committee on Research Ethics, via Reference No. ADM/E.22/A/VOL.VII/1469.

Study Participants

In order to avoid bias, selection of participants was random in both hospitals. In total, there were one hundred and ninety (190) participants, who were all pregnant. These women, who attended antenatal care and were willing to be recruited for the study, were subjected to three kinds of diagnostic tests which was used to categorize them into the three groups originally made for the study. The preeclamptic group consisted of the pregnant women with a characteristic high systolic and diastolic blood pressure of not less than 140mmHg and 90 mmHg respectively. Urine analyses carried out also showed positive proteinuria (≥ 300 mg/24 hours).

For the Pregnancy-Induced Hypertension (PIH) Group, the participants, who were also pregnant, had similar high blood pressure, but no evident proteinuria. The final group, which consisted of the control participants presented with normal blood pressure, or BP < 140/90 mmHg; and no proteinuria.

Inclusion and Exclusion Criteria:

Pregnant women within the ages of 20 and 40 with singleton pregnancy, and a gestational age of not less than twenty eight (28) weeks were included in the study. Those who were excluded from the study were pregnancies with historical evidence of chronic illnesses such as diabetes, hypertension, renal and hepatic insufficiencies, cardiovascular disease and thyroid dysfunction. Similarly, pregnant women with multiple gestations were excluded.

Blood Pressure Measurements

Blood pressure was measured with patients in a prone position on at least two separate occasions using a mercury sphygmomanometer. The average of the two readings was recorded as the participant's blood pressure.

Blood Sampling

Venous blood was collected from each participant through a single antecubital venipuncture, yielding a 5ml sample. The blood was then transferred to a heparinized container and promptly separated, with the resulting plasma being carefully pipetted into a 5ml vial. The sample was subsequently stored at a temperature of -4°C until further analysis was conducted.

Laboratory Analysis

Plasma lactate concentration, lactate dehydrogenase enzyme activities, and plasma bicarbonate levels were determined to ascertain the acid-base status of the study participants. Plasma lactate levels and lactate

dehydrogenase enzyme activities were estimated colorimetrically using commercially purchased kits from Randox Diagnostics. Bicarbonate levels were determined using an ion-selective electrolyte analyzer.

Data Analysis:

Descriptive statistics was used to summarize demographic and clinical data. Acid-base status parameters were compared among the three groups using ANOVA or non-parametric tests as appropriate. Statistical significance was set at $P < 0.05$.

RESULTS

Figure 1 shows the distribution of participants into preeclampsia, pregnancy induced hypertension, and normotensive groups respectively, the results showed that the largest proportion of participants, 65.3%, falls into the preeclampsia group.

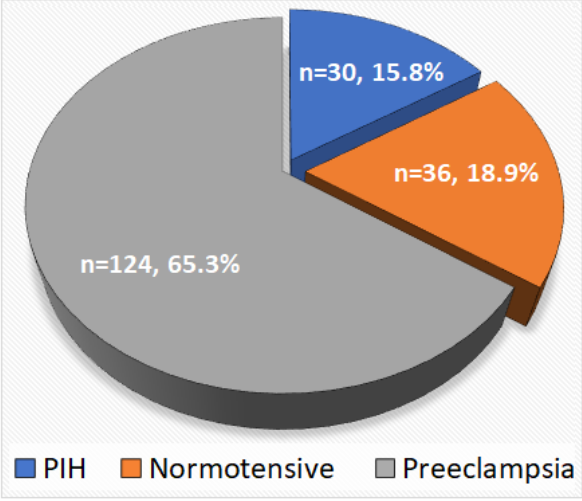

Figure 1: Distribution of participants into preeclampsia, pregnancy induced hypertension, and normotensive groups respectively.

Table 1: Demographic data of respondents

Parameters	Preeclampsia cases (A) n (%) (N=124)	Normotensive (B) n (%) (N=36)	PIH (C) n (%) (N=30)	X^2	p-value
Marital status					
Single	3 (2.4)	2 (5.6)	0	11.480	0.0217*
First Marriage	92 (74.2)	33 (91.7)	10 (100)		
Remarried	29 (23.4)	1 (2.8)	0		
Educational status					
None	4 (3.2)	0	0	4.823	0.567
Primary	17 (13.7)	3 (8.3)	3 (30)		
Secondary	47 (37.9)	16 (44.5)	3 (30)		
Post-secondary	56 (45.2)	17 (47.2)	4 (40)		
Job status					
Employed	102 (82.3)	27 (97.5)	8 (80)	0.942	0.624
Unemployed	22 (17.7)	9 (2.5)	2 (20)		

*significant

Table 1 presents the demographic data of respondents divided into three groups: Pre-eclampsia cases (A), Normotensive (B), and Pregnancy-Induced Hypertension (PIH) (C). The study showed that single women have a higher prevalence of pre-eclampsia and PIH compared to normotensive women. Results showed that single pregnant women 5.6% were normal, compared to 2.4% preeclampsia. There was also 74.2% preeclampsia in those of first marriage while 91.7% were normal. Educational status was another factor that influenced the development of hypertensive disorders. While the majority of participants across all educational levels were normotensive, there was a notable exception among those with only primary education. In this group, 30% developed pregnancy-induced hypertension (PIH), compared to 8.3% who remained normotensive and 13.7% who developed preeclampsia.

Table 2: Grouped data (totals) for acid-base status of respondents presented irrespective of trimester used for assessing

Parameters	Preeclampsia cases (A) (µ±SEM) (n=124)	Normotensive (B) (µ±SEM) (n=36)	PIH (C) (µ±SEM) (n=30)	p-value (A+B)	p-value (A+C)	p-value (B+C)
Lactate (mg/dl)	42.51±0.65	32.72±1.32	38.92±2.14	0.851	0.878	0.876
Plasma bicarbonate (mM/L)	26.62±0.39	24.38±0.98	27.20±1.95	0.051	0.840	0.473
Lactate dehydrogenase (U/l)	86.45±3.41	66.86±5.58	88.60±23.31	0.006	0.875	0.003

Table 2 above provides grouped data on the acid-base status of respondents across three categories: Preeclampsia cases (A), Normotensive (B), and Pregnancy-Induced Hypertension (PIH) (C). The variables measured include Lactate, Plasma bicarbonate, and Lactate dehydrogenase (LDH), along with their respective p-values for comparisons between the groups. The results shows that Lactate levels are highest in the Preeclampsia group (A) and lowest in the Normotensive group (B). The *P*-values showed that there was no statistically significant difference in lactate levels between any of the groups ($p > 0.05$). Plasma bicarbonate levels are slightly higher in the Preeclampsia (A) and PIH (C) groups compared to the Normotensive (B) group. The *P*-values suggest a borderline significant difference between the Preeclampsia (A) and Normotensive (B) groups ($p = 0.051$), but no significant difference between the other groups ($p > 0.05$). This indicates a potential, but not definitive, difference in plasma bicarbonate levels between these groups. LDH levels are highest in the PIH (C) group, followed by Preeclampsia (A), and lowest in the Normotensive (B) group. The *P*-values indicate significant differences between the Preeclampsia (A) and Normotensive (B) groups ($p = 0.006$), as well as between the Normotensive (B) and PIH (C) groups ($p = 0.003$).

Table 3: Acid base analytes of respondents presented on the basis of trimesters

Parameters	Trimester	Preeclampsia cases (A) (n=62)	Normotensive (B) (n=16)	PIH (C) (n=15)	(A+B)	(A+C)	(B+C)
					p-values		
Lactate (mg/dl)	Second	32.612	25.790	34.384	0.596	0.638	0.624
	Third	32.407	27.865	38.746	0.735	0.034	0.028
Plasma bicarbonate (mM/L)	Second	20.483	24.687	21.800	0.061	0.864	0.183
	Third	19.766	25.350	20.600	0.232	0.037	0.052
Lactate dehydrogenase (U/l)	Second	84.466	67.933	75.946	0.096	0.651	
	Third	88.682	66.868	101.266	0.533	0.531	0.042

Table 3 above presents the means of acid-base analytes (lactate, plasma bicarbonate, and lactate dehydrogenase) of respondents categorized into three groups: Preeclampsia cases (A), Normotensive (B), and Pregnancy-Induced Hypertension (PIH) (C) across the second and third trimesters. The p-values indicate the statistical significance of the differences between the groups (A+B, A+C, B+C). The results show that in the second trimester, lactate levels were slightly higher in the PIH group compared to both the preeclampsia and normotensive groups. However, the p-values for the comparisons (A+B, A+C, B+C) are not statistically significant (0.596, 0.638, and 0.624, respectively), indicating that the differences observed may be due to chance rather than a true difference between the groups. In the third trimester, lactate levels increased across all groups, with the PIH group showing the highest levels. The differences between the groups become statistically significant when

comparing PIH (C) to both Preeclampsia cases (A) and Normotensive (B), with p-values of 0.034 and 0.028, respectively. Plasma bicarbonate levels were slightly lower in the preeclampsia group compared to the normotensive group in the second trimester, with the PIH group showing intermediate levels. However, none of the differences reached statistical significance, as indicated by p-values of 0.061, 0.864, and 0.183 for the comparisons A+B, A+C, and B+C, respectively. In the third trimester, plasma bicarbonate levels decreased in the preeclampsia group and remained relatively stable in the PIH group, while the normotensive group maintained higher levels. The difference between preeclampsia (A) and PIH (C) were not statistically significant (p-value = 0.037). Lactate dehydrogenase (LDH) levels were highest in the preeclampsia group and lowest in the normotensive group during the second trimester. The differences between groups was not statistically significant, with p-values of 0.096, 0.651, and 0.304 for the comparisons A+B, A+C, and B+C, respectively. LDH levels increased in all groups in the third trimester, with the PIH group showing the highest levels, followed by the preeclampsia group. The comparison between normotensive (B) and PIH (C) groups was not statistically significant (p-value = 0.042).

Table 4: Analyte composition of preeclamptic subjects separated on the basis of severity of disease

Analytes	Groups	N	Mean	Std. Error Mean	p-value
Lactate (mg/dl)	Mild	39	32.68	1.2407	0.834
	Severe	84	32.381	0.7828	
Plasma bicarbonate (mM/L)	Mild	39	21.635	0.7037	0.927
	Severe	84	24.557	0.4738	
Lactate dehydrogenase (U/l)	Mild	39	87.464	5.2115	0.866
	Severe	84	86.21	4.4475	

Table 4 above presents the means and standard errors of three acid-base analytes—lactate, plasma bicarbonate, and lactate dehydrogenase—in preeclamptic subjects, categorized into mild and severe cases. The results show that the mean lactate levels in both mild and severe preeclampsia cases are very similar (32.68 mg/dl vs. 32.381 mg/dl), and the difference between the two groups is not statistically significant (p-value = 0.834). Plasma bicarbonate levels are slightly lower in mild preeclampsia compared to severe preeclampsia (21.635 mM/L vs. 24.557 mM/L), but the difference is not statistically significant (*p* value = 0.927). The mean levels of lactate dehydrogenase (LDH) are almost identical between mild and severe preeclampsia cases (87.464 U/l vs. 86.21 U/l), with a non-significant p-value (0.866).

Table 5: Comparing Analyte composition of preeclamptic subjects separated on the basis of BMI

Parameters	Normal (n=23)	Overweight (n=71)	p-value	Normal (n=23)	Obese (n=30)	p-value
Lactate (mg/dl)	38.7748	41.8538	0.527	31.775	32.259	0.822
Plasma bicarbonate (mM/L)	21.2065	1.3627	0.034	23.207	23.967	0.577
Lactate dehydrogenase (U/l)	78.099	91.2705	0.141	78.099	81.958	0.716

Table 5 above presents a comparison of three analytes—lactate, plasma bicarbonate, and lactate dehydrogenase—among preeclamptic subjects, divided based on BMI into "Normal," "Overweight," and "Obese" categories. The results show that despite a slightly higher lactate level in overweight and obese subjects compared to normal-weight subjects, the p-values indicate no statistically significant difference ($p > 0.05$). A significant decrease in plasma bicarbonate levels is observed in overweight subjects compared to normal-weight subjects ($p = 0.034$). However, this difference is not observed between normal and obese subjects ($p = 0.577$).

DISCUSSION

The high percentage (65.3%) of participants with preeclampsia suggests that it is a significant health concern in the studied population. This could be due to various factors such as genetic predisposition, environmental influences, or underlying medical conditions. The relatively small percentage (18.9%) of normotensive participants suggests that hypertension-related conditions (preeclampsia and PIH) are common during pregnancy in this population. This highlights the importance of regular blood pressure monitoring and prenatal care to identify and manage these conditions.

Overall, acid-base status, as measured by lactate, plasma bicarbonate, and LDH, shows some differences between preeclampsia, PIH, and normotensive pregnancies. LDH levels appear to be the most promising biomarker for distinguishing between these conditions. Further research is needed to confirm these findings and explore the underlying mechanisms. The acid-base status of pregnant individuals, as indicated by lactate, plasma bicarbonate, and lactate dehydrogenase (LDH) levels, exhibits distinct variations between preeclampsia, pregnancy-induced hypertension (PIH), and normotensive pregnancies, suggesting potential diagnostic utility. Notably, LDH levels emerge as a particularly promising biomarker for differentiating these conditions, potentially offering a valuable tool for clinicians. However, additional research is essential to validate these findings, elucidate the underlying pathophysiological mechanisms, and determine the clinical applicability of LDH as a diagnostic biomarker, ultimately enhancing our understanding of these complex pregnancy-related disorders and informing improved patient management strategies.

Generally, Lactate levels do not differ significantly with the severity of preeclampsia, suggesting that lactate may not be a reliable biomarker for distinguishing between mild and severe preeclampsia. Plasma bicarbonate levels were relatively consistent across different severities of preeclampsia, indicating that this analyte may not be useful for determining the severity of preeclampsia.

Although there were significant elevations of LDH, their concentrations were not significantly influenced by the severity of preeclampsia, suggesting that LDH may not be a reliable biomarker for distinguishing between mild and severe preeclampsia.

The significant reduction in bicarbonate levels in the overweight category suggests a potential disruption in acid-base balance in this group, which might be related to metabolic alterations associated with increased BMI. The lack of a similar significant difference in obese subjects warrants further investigation to understand the underlying mechanisms. No significant differences were observed in LDH levels between normal and overweight or obese subjects ($p > 0.05$). This suggests that LDH levels are not significantly influenced by BMI in preeclamptic subjects. The absence of significant variation across different BMI categories indicates that LDH might not be a sensitive marker for BMI-related differences in preeclamptic patients.

Pregnancy-related hypertension disorders (HDP) continue to be a major cause of maternal and perinatal morbidity and mortality across the globe, especially in developing nations such as Nigeria. This study emphasizes the intricate interactions that occur during pregnancy between the mother's acid-base balance and hypertensive diseases, underscoring the necessity of individualized therapeutic care to enhance both the mother's and the fetus's outcomes[1].

During pregnancy, there are significant changes to the respiratory component of the mother's acid-base system, including a progressive rise in oxygen tension and reduction in arterial CO2 tension[5]. As a result of these modifications, the mother's arterial pH remains largely constant due to compensatory respiratory alkalosis and increased renal excretion of bicarbonate. The proper oxygenation of the foetus is ensured by the diffusion of blood gasses between the mother's and the foetal circulations, which is greatly aided by the maternal acid-base condition. Research has shown that maternal partial pressure of carbon dioxide (pCO2) decreases significantly during pregnancy, with a low of 31.3 mmHg observed in the final trimester.

This decrease facilitates the transfer of CO_2 from the fetus to the mother by improving the transplacental gradient for CO_2. Foetuses are naturally low in PaO_2 and high in $PaCO_2$, therefore hypoxia poses a serious concern even with the mother's buffering capabilities. Maintaining appropriate maternal-foetal oxygen exchange is crucial because the foetal response to hypoxia involves preferential blood flow redistribution to essential organs[5,6].

Significant variations were also found between the groups based on the respondents' acid-base state. In comparison to the normotensive group, the preeclampsia and PIH groups had somewhat higher plasma bicarbonate levels, but the lactate levels were highest in the preeclampsia group. Nevertheless, there was no discernible difference in lactate levels under the various circumstances. However, when compared to the normotensive group, the PIH group's LDH levels were considerably higher, indicating that LDH may serve as a marker for hypertension disorders during pregnancy. Further highlighting the intricate interactions between these variables and hypertensive

diseases is the examination of acid-base analytes in various trimesters and BMI ranges. PIH was linked to elevated lactate levels in the third trimester, whereas preeclampsia cases had considerably lower plasma bicarbonate levels. However, there was no significant change in lactate, plasma bicarbonate, or LDH levels according to the degree of preeclampsia, suggesting that these markers are essentially stable regardless of the condition's severity.

The study revealed that significant elevation of the LDH enzyme was not accompanied by elevated lactate levels. LDH uses NAD+ as a coenzyme, which is regenerated to NADH during the reaction. In hypoxic conditions, NADH accumulates, inhibiting LDH activity.

An increase in enzyme concentration doesn't necessarily mean an increase in enzyme activity. Here are some scenarios where this might not hold true. Enzyme inhibition, limiting substrate saturation, co-factor or prosthetic group limitations, as well as protein folding or aggregation which might have been associated with preeclamptic conditions may be the reason for this results. Further investigations are therefore required to elucidate the impact of anoxic conditions as well as the afore-mentioned factors on the limiting LDH activity as against elevated concentrations.

CONCLUSION

This study sheds new light on the acid-base dynamics in pregnancies with hypertensive disorders. The study found a surprising mismatch, where elevated lactate dehydrogenase (LDH) enzyme activity was not accompanied by a corresponding surge in lactate levels, implying a potential separation of LDH enzyme activity from lactate generation in the population under study. Further research will elucidate the underlying mechanisms, paving the way for improved diagnosis, treatment, and prevention of adverse outcomes.

Acknowledgements

The authors are grateful to Mr Owalum Linus Onawo for the assistance during the study.

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