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COPARATIVE STUDY OF PRE AND POST PARTUM ELECTROLYTES, UREA AND CREATININE LEVELS OF HYPERTENSIVE MOTHERS IN ENUGU STATE UNIVERSITY TEACHING HOSPITAL, ENUGU, NIGERIA

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ABSTRACT

Hypertensive disorders of pregnancy remain a major cause of maternal and perinatal morbidity and mortality worldwide, particularly in low- and middle-income countries. Pregnancy is associated with profound physiological adaptations involving cardiovascular, renal, and electrolyte regulation, which may become dysregulated in the presence of hypertension. Alterations in serum electrolytes, urea, and creatinine during the pre-partum and post-partum periods provide important insights into renal function and fluid balance in hypertensive mothers. This study comparatively evaluated serum electrolytes, urea, and creatinine levels in pre-partum and post-partum hypertensive women attending Enugu State University Teaching Hospital. A total of fifty (50) participants were recruited and grouped into twenty-five (25) pre-partum hypertensive women and twenty-five (25) post-partum hypertensive women, with normotensive women serving as controls. Serum urea was determined using the ureaseBerthelot method, serum creatinine was measured by the Jaffe reaction, sodium and potassium were analyzed using flame emission photometry, while chloride and bicarbonate were determined by titrimetric methods. Data were expressed as mean ± SEM and analyzed using one-way analysis of variance (ANOVA), with $p < 0.05$ considered statistically significant. The results showed a significant reduction in serum sodium in pre-partum hypertensive women (134.72 ± 0.98 mmol/L) compared to post-partum (138.52 ± 2.16 mmol/L) and control subjects (138.40 ± 1.43 mmol/L) ($p < 0.05$). Potassium and bicarbonate levels were also significantly lower in the pre-partum group (3.46 ± 0.09 mmol/L and 19.32 ± 1.07 mmol/L, respectively) compared to post-partum and controls ($p < 0.05$), while chloride levels showed no significant difference ($p >0.05$). Serum urea and creatinine were significantly elevated in pre-partum hypertensive women (8.11 ± 1.17 mmol/L and 123.01 ± 26.58 µmol/L, respectively) compared to post-partum (4.56 ± 1.99 mmol/L and 82.90 ± 38.03 µmol/L) and control groups (3.94 ± 1.61 mmol/L and 67.95 ± 14.81 µmol/L) ($p < 0.05$). These findings indicate increased renal stress during hypertensive pregnancy with partial biochemical normalization following delivery. Routine monitoring of renal and electrolyte parameters is therefore essential in the management of hypertensive disorders of pregnancy.

Keywords: Hypertension, Pregnancy, Pre-partum, Post-partum, Electrolytes, Urea, Creatinine, Renal function.

INTRODUCTION

Pregnancy is a complex physiological state characterized by profound anatomical, hormonal, metabolic, cardiovascular, and renal adaptations aimed at supporting fetal growth and maintaining maternal homeostasis. These changes begin early in gestation and progress through the pre-partum period, with gradual reversal during the post-partum phase following delivery (1). While these adaptations are normally well regulated, the presence of hypertensive disorders during pregnancy disrupts physiological balance and predisposes both the mother and fetus to adverse outcomes. Hypertensive disorders of pregnancy remain a leading cause of maternal morbidity and mortality globally, accounting for approximately 14% of maternal deaths, with a disproportionate burden in sub-Saharan Africa (2).

Hypertension in pregnancy encompasses a spectrum of conditions including chronic hypertension, gestational hypertension, pre-eclampsia, and eclampsia. These conditions are associated with endothelial dysfunction, altered renal perfusion, impaired sodium handling, and changes in glomerular filtration rate (GFR), all of which significantly influence electrolyte balance and renal function markers such as urea and creatinine (3). The pre-partum period represents the peak of physiological stress, as plasma volume expansion, increased cardiac output, and heightened renal workload coexist with placental vascular demands. Conversely, the post-partum period is marked by gradual normalization of these changes, although recovery may be delayed or incomplete in hypertensive women (4).

Renal adaptation is central to normal pregnancy. Physiologically, pregnancy is associated with a 40–50% increase in renal blood flow and GFR, leading to lower serum urea and creatinine concentrations compared to non-pregnant women (5). Sodium and water retention occur to support plasma volume expansion, mediated by hormonal changes involving aldosterone, estrogen, progesterone, and the renin–angiotensin–aldosterone system (RAAS). Potassium balance is typically maintained through enhanced renal excretion despite increased intake, while bicarbonate levels may decrease slightly due to pregnancy-induced respiratory alkalosis (6). These tightly regulated processes ensure adequate uteroplacental perfusion and fetal development.

In hypertensive pregnancy, however, these renal and electrolyte adaptations become dysregulated. Reduced renal perfusion pressure, glomerularendotheliosis, and altered tubular function impair the kidney's ability to excrete nitrogenous waste and regulate electrolytes efficiently (7). Elevated serum urea and creatinine in hypertensive pregnant women reflect reduced GFR and increased renal stress, while disturbances in sodium, potassium, and bicarbonate levels may indicate fluid imbalance, acid–base disturbances, and altered tubular handling (8). Such biochemical abnormalities are clinically significant, as they may precede overt renal failure and contribute to complications such as pulmonary edema, metabolic acidosis, and cardiovascular instability. The pre-partum period is particularly critical,

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as hypertensive pathology often worsens with advancing gestation. Studies have shown that serum creatinine and urea levels tend to rise progressively in hypertensive pregnancies compared to normotensive pregnancies, while hyponatremia and reduced bicarbonate levels may reflect dilutional effects and metabolic alterations (9). Potassium imbalance, though less consistent, has been linked to altered aldosterone sensitivity and renal tubular dysfunction in severe hypertension (10). In contrast, the post-partum period is associated with gradual restoration of renal function and electrolyte balance as placental influence is removed and systemic vascular resistance normalizes. However, persistent abnormalities may indicate underlying renal disease or incomplete recovery (11).

Comparative evaluation of biochemical parameters between pre-partum and post-partum hypertensive women provides valuable insight into the dynamic impact of pregnancy and delivery on renal physiology. Such comparisons help distinguish transient pregnancy-related changes from persistent pathological alterations requiring long-term follow-up. In resource-limited settings like Nigeria, where access to advanced diagnostic tools may be limited, routine biochemical markers such as electrolytes, urea, and creatinine serve as cost-effective indicators for monitoring maternal renal health (12).
Despite the clinical importance of these parameters, data on comparative pre-partum and post-partum renal biochemical changes in hypertensive Nigerian women remain limited. Most available studies focus on pre-eclampsia or single-time-point measurements, with few evaluating post-delivery recovery patterns. Enugu State University Teaching Hospital serves a large and diverse population, making it an appropriate setting for investigating these biochemical changes in hypertensive mothers. Understanding local patterns is essential for improving antenatal and postnatal care protocols and reducing maternal complications associated with hypertensive disorders. This study therefore aimed to comparatively assess serum electrolytes (sodium, potassium, chloride, and bicarbonate), urea, and creatinine levels in pre-partum and post-partum hypertensive women attending Enugu State University Teaching Hospital, using standardized biochemical methods. By comparing these groups with normotensive controls, the study seeks to elucidate the extent of renal and electrolyte disturbances associated with hypertensive pregnancy and the degree of biochemical recovery following delivery. The findings are expected to contribute to improved clinical monitoring, early intervention, and better maternal health outcomes in hypertensive pregnancies.

MATERIALS AND METHODS

This comparative cross-sectional study was conducted at Enugu State University Teaching Hospital, Enugu, Nigeria. A total of fifty (50) adult women diagnosed with hypertension were recruited for the study after obtaining informed consent. The participants were divided into two groups consisting of twenty-five (25) pre-partum hypertensive women and twenty-five (25) post-partum hypertensive women, while normotensive women served as control subjects. Venous blood samples were collected under aseptic conditions from all participants and allowed to clot, after which serum was separated by centrifugation at 3000 rpm for 10 minutes and used for biochemical analysis. Serum urea concentration was determined using the urease–Berthelot method (13), while serum creatinine was measured using the Jaffe reaction according to standard laboratory guidelines (14). Serum sodium and potassium levels were analyzed using a flame emission photometer (15). Serum chloride and bicarbonate concentrations were determined by titrimetric methods (16,17). Statistical analysis was performed using one-way analysis of variance (ANOVA) with significant set at p<0.05

RESULTS

Table 4.1 comparison of the mean level of serum electrolytes between pre-partum, post-partum hypertensive women and the control subjects

	Pre-partum	Post-partum	control	ANOVA	P
Sodium	134.72±0.98	138.52±2.16	138.40±1.43	42.21	<0.05
Potassium	3.456±0.092	3.88±0.19	3.790±0.09	113.21	<0.05
Chlorides	98.80±0.87	99.56±1.26	99.60±1.08	1.43	>0.05
bicarbonate	19.32±1.07	23.48±2.22	24.00±1.25	99.99	<0.05

Results are expressed as Mean ±SEM. P<0.05 is considered significant and P>0.05 non significant.

Table 4.2 comparison of the mean level of urea and creatinine between pre-partum, post-partum hypertensive women and the control subjects

Variables (mmol/l)	Pre-partum	Post-partum	control	ANOVA	P
Urea	8.11±1.17	4.564±1,99	3.94±1.61	64.11	<0.05
Creatinine	123.010±26.58	82.896±38.03	67.950±14.81	26.98	<0.05

Results are expressed as Mean ±SEM. P<0.05 is considered significant and P>0.05 non significant.

Results summary

Table 4.1 shows the comparison of mean serum electrolyte levels among the study groups. Serum sodium concentration was significantly lower in pre-partum hypertensive women (134.72 ± 0.98 mmol/L) compared to post-partum hypertensive women (138.52 ± 2.16 mmol/L) and control subjects (138.40 ± 1.43 mmol/L). This difference was statistically significant as indicated by ANOVA ($F = 42.21$, $p < 0.05$). Serum potassium levels also showed a significant variation among the groups, with pre-partum hypertensive women recording lower values (3.46 ± 0.09 mmol/L) compared to post-partum women (3.88 ± 0.19 mmol/L) and controls (3.79 ± 0.09 mmol/L) ($F = 113.21$, $p < 0.05$). In contrast, serum chloride levels did not differ significantly among pre-partum, post-partum, and control groups, with mean values of 98.80 ± 0.87 mmol/L, 99.56 ± 1.26 mmol/L, and 99.60 ± 1.08 mmol/L respectively ($F = 1.43$, $p = 0.244$).

Serum bicarbonate concentration showed a marked and statistically significant reduction in pre-partum hypertensive women (19.32 ± 1.07 mmol/L) when compared with post-partum hypertensive women (23.48 ± 2.22 mmol/L) and control subjects (24.00 ± 1.25 mmol/L). The difference observed was highly significant ($F = 99.99$, $p < 0.001$), indicating altered acid–base balance during hypertensive pregnancy.

Table 4.2 presents the comparison of renal function parameters among the groups. Serum urea levels were significantly higher in pre-partum hypertensive women (8.11 ± 1.17 mmol/L) compared to post-partum hypertensive women (4.56 ± 1.99 mmol/L) and controls (3.94 ± 1.61 mmol/L). This difference was statistically significant ($F = 64.11$, $p < 0.05$). Similarly, serum creatinine concentration was significantly elevated in pre-partum hypertensive women (123.01 ± 26.58 µmol/L) compared with post-partum hypertensive women (82.90 ± 38.03 µmol/L) and control subjects (67.95 ± 14.81 µmol/L), with ANOVA indicating a significant difference among groups ($p < 0.05$).

DISCUSSION

Hypertensive disorders of pregnancy are associated with significant physiological and biochemical disturbances that affect maternal renal function and electrolyte homeostasis. In this study, pre-partum hypertensive women exhibited more pronounced alterations in serum electrolytes, urea, and creatinine levels compared to post-partum hypertensive women and normotensive controls, highlighting the impact of pregnancy-related hypertension on renal and metabolic regulation. The partial normalization of these parameters observed in the post-partum period further supports the role of pregnancy-specific factors in driving these biochemical changes.

The significantly reduced serum sodium levels observed in pre-partum hypertensive women suggest altered sodium handling and fluid balance during hypertensive pregnancy. Physiologically, pregnancy is characterized by sodium and water retention to support plasma volume expansion; however, in hypertensive states, endothelial dysfunction and impaired renal perfusion may disrupt tubular sodium reabsorption, resulting in relative hyponatremia (5). Similar reductions in serum sodium have been reported in hypertensive pregnancies and pre-eclampsia, where abnormal activation of the rennin angiotensin aldosterone system and increased vascular permeability contribute to dilutional and renal sodium loss (3,10).

Serum potassium levels were also significantly lower in pre-partum hypertensive women compared to post-partum women and controls. This finding may be attributed to increased aldosterone activity and enhanced renal potassium excretion commonly seen in hypertensive states. Altered tubular handling of potassium in pregnancy-induced hypertension has been documented and may predispose affected women to electrolyte imbalance and cardiovascular instability (8). The normalization of potassium levels in the post-partum group suggests recovery of renal tubular function following delivery.

The marked reduction in serum bicarbonate concentration in pre-partum hypertensive women indicates the presence of acid–base imbalance. Pregnancy is normally associated with mild respiratory alkalosis and compensatory renal bicarbonate excretion; however, hypertensive pregnancy may exaggerate this process, leading to metabolic disturbances (6,4). Reduced bicarbonate levels have been linked to increased metabolic demand, impaired renal compensation, and reduced buffering capacity, particularly in severe hypertensive states (Magee et al., 2022). The restoration of bicarbonate levels in the post-partum group further emphasizes the transient nature of these changes when pregnancy-related stressors are removed. In contrast, serum chloride levels did not differ significantly among the study groups. This finding suggests that chloride homeostasis may be less affected by hypertensive pregnancy compared to other electrolytes, possibly due to its close coupling with sodium and water balance and tighter renal regulation. Similar observations have been reported in previous studies where chloride levels remained relatively stable despite significant changes in other electrolytes in hypertensive pregnant subjects. (9).

Renal function parameters showed significant impairment in pre-partum hypertensive women, as evidenced by elevated serum urea and creatinine levels. Under normal pregnancy conditions, increased GFR leads to reduced serum urea and creatinine concentrations. The elevated levels observed in this study therefore indicate reduced renal filtration and increased renal stress associated with hypertensive pregnancy (7). Glomerularendotheliosis, a hallmark of hypertensive pregnancy disorders, reduces effective filtration surface area and contributes to the accumulation of nitrogenous waste products in the circulation (8).The significant reduction in urea and creatinine levels in post-partum hypertensive women suggests partial recovery of renal function following delivery. This finding is consistent with previous reports demonstrating improved renal hemodynamics and normalization of biochemical markers after removal of the placenta, which plays a central role in the pathogenesis of hypertensive disorders of pregnancy (11). However, the persistence of higher values compared to controls may indicate residual renal dysfunction or increased long-term risk of chronic kidney disease, emphasizing the need for continued post-partum monitoring. The findings of this study align with existing literature demonstrating that hypertensive pregnancy significantly alters electrolyte balance and renal function, with the most severe changes occurring during the pre-partum period. The comparative design employed in this study provides valuable insight into the dynamic biochemical changes associated with pregnancy and delivery. Routine assessment of serum electrolytes, urea, and creatinine during antenatal and post-natal care may therefore aid in early detection of renal impairment, guide clinical management, and reduce maternal complications associated with hypertensive disorders.

CONCLUSION

The findings emphasize the clinical importance of routine monitoring of serum electrolytes, urea, and creatinine in hypertensive pregnancies, both before and after delivery. Early detection of biochemical abnormalities may facilitate timely intervention, reduce maternal complications, and improve pregnancy outcomes. This study contributes valuable local data from Enugu State University Teaching Hospital and supports the incorporation of renal and electrolyte assessment into standard antenatal and post-natal care protocols for women with hypertensive disorders in pregnancy.

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