

## A STUDY OF COMPARING SERUM LEVELS OF MAGNESIUM AND CALCIUM IN HYPERTENSIVE MOTHERS VERSUS NORMOTENSIVE MOTHERS IN TERTIARY CARE CENTRE

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### ABSTRACT

**Background:** Hypertensive disorders of pregnancy significantly contribute to maternal and perinatal morbidity and mortality. Electrolyte imbalance, particularly involving magnesium and calcium, plays a role in vascular dysfunction. This study aimed to compare serum magnesium and calcium levels between hypertensive and normotensive pregnant women and evaluate their diagnostic significance. **Materials and Methods:** This prospective comparative case-control study included 100 pregnant women attending a tertiary care centre over one year. Participants were divided into hypertensive and normotensive groups. Clinical evaluation, blood pressure measurement, and urine protein assessment were performed. Venous blood samples were collected under aseptic conditions, and serum magnesium and calcium levels were estimated using colorimetric methods with an automated analyser. **Results:** Hypertensive patients showed significantly lower serum magnesium ( $1.43 \pm 0.09$  vs.  $2.25 \pm 0.12$  mg/dL) and calcium levels ( $8.56 \pm 0.31$  vs.  $10.96 \pm 0.45$  mg/dL) ( $p < 0.001$ ). Body mass index was higher ( $28.3 \pm 4.2$  vs.  $23.2 \pm 2.1$  kg/m<sup>2</sup>), and birth weight was lower ( $2.40 \pm 0.51$  vs.  $2.78 \pm 0.42$  kg) in hypertensive patients. Hypomagnesaemia and hypocalcaemia were observed in 100% and 94% of hypertensive patients, respectively. Serum magnesium demonstrated 100% sensitivity and specificity in this sample, while serum calcium showed 94% sensitivity and 100% specificity. Proteinuria, maternal complications, and imminent signs were significantly higher in hypertensive patients, whereas the mode of delivery was comparable. **Conclusion:** Hypertensive disorders of pregnancy are associated with hypomagnesaemia and hypocalcaemia. Serum magnesium may serve as a potential biomarker for identifying hypertensive disorders, and assessment of these electrolytes may aid in clinical evaluation and risk stratification. Further large-scale studies are required to validate these findings.

## INTRODUCTION

Hypertensive disorders of pregnancy (HDP), including gestational hypertension, preeclampsia, and eclampsia, are a major source of maternal and perinatal morbidity and mortality worldwide. It is one of the most common obstetric complications and is associated with placental abruption, preterm labour, and foetal death. This condition affects approximately 5-10% of pregnant women. The incidence of HDP is higher in developing countries because of limited healthcare resources.<sup>[1,2]</sup> The

development of hypertension during pregnancy increases the risk of developing cardiovascular and kidney diseases later in life, which highlights the public importance of HDP. In spite of medical progress, HDP remains challenging because of its complex pathogenesis, which impedes accurate prediction and prevention. Placental ischaemia, caused by altered placental function, impaired spiral artery remodelling, and poor trophoblast invasion, leads to endothelial cell injury. The excess production of soluble fms-like tyrosine kinase-1

results in hypoperfusion of the utero-placental tissues.<sup>[3]</sup>

An imbalance between vasodilators, such as nitric oxide, and vasoconstrictors, such as endothelin, increases vascular resistance and hypertension.<sup>[4]</sup> Oxidative stress and inflammation exacerbate endothelial dysfunction, causing organ damage and developmental abnormalities during pregnancy. There is accumulating evidence for the involvement of immune dysregulation and epigenetics in the development of HDP, highlighting the involvement of the environment and genetics in HDPs.<sup>[5,6]</sup> Endothelial dysfunction connects HDP with acute and chronic disorders. Circulating endothelial cells indicate vascular injury and poor repair processes, providing an explanation for the cardiovascular risks associated with the condition.<sup>[7,8]</sup> HDP can increase the risk of cerebrovascular disorders due to hypertension and unstable vascular structure.<sup>[9]</sup> Micronutrient imbalance plays a major role in controlling vasculature. Proper nutrition provides adequate conditions for maintaining endothelial integrity, controlling oxidative stress and vasoconstriction and reducing vascular tone.<sup>[10]</sup>

Electrolytes, such as magnesium and calcium, are important for ensuring proper vascular smooth muscle function. Magnesium is important for maintaining vascular stability because it functions as a calcium inhibitor and induces vasodilatation. It helps control the levels of intracellular calcium, which in turn prevents unnecessary contraction of the vascular smooth muscle. Magnesium deficiency is associated with high levels of vasoconstriction, inflammation, and endothelial dysfunction, and thus is a cause of HDP.<sup>[11]</sup> Magnesium sulfate is used in the management of preeclampsia and eclampsia, emphasising the significance of this electrolyte in pregnancy. Magnesium imbalance affects calcium metabolism by inhibiting parathyroid hormone secretion.<sup>[12,13]</sup> Calcium, on the other hand, controls the concentration of intracellular calcium to regulate vascular tone. Hypocalcemia in women with hypertensive disorders during pregnancy may cause an increase in parathyroid hormone and renin secretion, which increases intracellular calcium concentration.<sup>[14]</sup>

Although evidence from some studies indicates that calcium supplementation can decrease blood pressure and the incidence of HDP, the results remain inconclusive due to differences in diets, populations, and methodologies.<sup>[14,15]</sup> This underscores the necessity for further research on calcium metabolism. The interrelationship between magnesium and calcium is crucial for maintaining vascular equilibrium. Magnesium functions as a calcium channel blocker, regulating calcium entry into cells and facilitating the vasodilation. Any disruption, particularly with simultaneous magnesium and calcium deficiency, could increase endothelial dysfunction and vascular resistance, hence increasing the likelihood of developing HDP.<sup>[16]</sup> Studies have shown low levels of

magnesium and calcium in patients with preeclampsia, which correlate with negative maternal and foetal complications.<sup>[17,18]</sup>

Despite evidence of the roles of magnesium and calcium in hypertensive disorders during pregnancy, their simultaneous evaluation remains inadequately explored. Most studies have only focused on assessing either of the two electrolytes separately, overlooking the impact of their interactions on the physiological functions of the vessels. Variation in results based on disparities in nutrition, socioeconomic status, and healthcare accessibility restricts the ability to generalise results.<sup>[19]</sup> There is also a lack of data regarding the evaluation of serum magnesium and calcium concentrations from tertiary hospitals located in less developed parts of the world, which suffer from a higher incidence of HDP. Evaluating serum magnesium and calcium levels together may help elucidate their involvement in the development of HDP. Recognising any changes would provide justification for using these markers for earlier diagnosis and risk assessment.

#### **Aim:**

Our study aimed to compare serum magnesium and calcium levels in women with hypertensive disorders of pregnancy and normotensive pregnant women.

## **MATERIALS AND METHODS**

This comparative prospective case-control study was conducted in 100 pregnant women who attended the Department of Obstetrics and Gynaecology, Mahatma Gandhi Memorial Government Hospital, Tiruchirapalli, from January 2024 to January 2025. Ethical approval was obtained from the institutional ethics committee, and written informed consent was obtained before study initiation.

**Sample Size Calculations:** The sample size was determined based on a similar study by Sethi et al., which compared serum calcium and magnesium levels in 100 pregnant women, comprising 50 with preeclampsia and 50 normotensive controls.<sup>[20]</sup> Accordingly, a total of 100 subjects were enrolled in the present study, with 50 hypertensive patients (Group H) and 50 normotensive patients (Group N), maintaining a 1:1 case-to-control ratio.

#### **Inclusion and Exclusion criteria**

Our study included pregnant women aged 18-35 with singleton gestation beyond 20 weeks, diagnosed with gestational hypertension or preeclampsia according to the ACOG criteria, and normotensive pregnant women matched for gestational age.

Women with chronic hypertension before pregnancy, renal disease, diabetes, thyroid or seizure disorders, multiple gestation, history of parathyroid or bone metabolic disorders, those on calcium, magnesium, or vitamin D supplements within a

month before enrolment, and those unwilling to consent were excluded.

### Materials

The materials used in the study included a mercury sphygmomanometer, urine dipstick for proteinuria assessment, sterile syringes, vacutainers, centrifuge, automated biochemical analyser, and reagents for colorimetric estimation of serum calcium and magnesium.

### Methods

Clinical history, including age, parity, gestational age, and obstetric history, was recorded for all patients. General and obstetric examinations emphasised blood pressure measurement and the presence of oedema.

Blood pressure was measured using a mercury sphygmomanometer in the sitting position after 10 minutes of rest, and two readings were recorded at least 4 hours apart, average of the two readings was used for diagnosis. Urine protein levels were assessed using the dipstick method. Under aseptic conditions, 5 ml of venous blood was collected after fasting, when possible, clotted, and centrifuged for serum. Serum calcium and magnesium levels were estimated using colorimetric methods with an automated analyser. Quality control was maintained throughout the biochemical analyses.

### Statistical Analysis

Data were presented as mean, standard deviation, frequency, and percentage. Continuous variables were compared using the independent t-test, while categorical variables were analysed using the chi-square test and Fisher's exact test, where appropriate. Diagnostic performance indices, including sensitivity, specificity, positive predictive value, and negative predictive value, were also calculated. Significance was defined as  $P < 0.05$ , and analysis was performed using IBM-SPSS version,<sup>[21]</sup> (IBM-SPSS Science Inc., Chicago, IL, USA).

## RESULTS

The age distribution showed that most patients were 21-25 years, with more normotensive (38%) than hypertensive (26%) patients, followed by 26-30 years (30% vs. 24%). Patients aged  $\leq 20$  years were similar (16% vs. 14%), while those aged 31-35 years (20% vs. 12%) and  $> 35$  years (16% vs. 4%) were higher among hypertensive patients. Socioeconomic status was mostly Class V (96% normotensive vs. 94% hypertensive), with some in Class IV (4% vs. 6%). Among hypertensive patients, 56% had mild PIH and 44% had severe PIH; no normotensive patients had hypertension. All normotensive patients had normal platelet counts (100%) compared to 76% of hypertensive patients; a low platelet count was only observed in hypertensive patients (24%).

Normotensive patients received no treatment (100%), whereas hypertensive patients received antihypertensives (50%), antihypertensives with magnesium sulfate (30%), and conservative treatment (20%). Vaginal delivery was more common in both groups, and was slightly higher in hypertensive patients (70% vs. 64%), with similar LSCS rates (28% vs. 26%). The rate of instrumental delivery was higher in normotensive patients (10% vs. 2%). Birth weight  $> 2.5$  kg was more frequent in normotensive (92%) than hypertensive patients (76%), whereas low birth weight ( $\leq 2.5$  kg) was more frequent in hypertensive patients (24% vs. 8%). Foetal complications were more frequent in hypertensive patients, including IUGR (10% vs. 2%), preterm birth (10% vs. 4%), and respiratory distress (6% vs. 4%), whereas the absence of complications was higher in normotensive patients (90% vs. 74%). [Table 1]

**Table 1: Demographic, Clinical, and Obstetric Characteristics of Patients**

		N (%)	
		Hypertensive (n=50)	Normotensive (n=50)
Age (in years)	$\leq 20$	7 (14%)	8 (16%)
	21-25	13 (26%)	19 (38%)
	26-30	12 (24%)	15 (30%)
	31-35	10 (20%)	6 (12%)
	$> 35$	8 (16%)	2 (4%)
Socioeconomic Status	Class IV	3 (6%)	2 (4%)
	Class V	47 (94%)	48 (96%)
Hypertension Type	Mild PIH	28 (56%)	0
	Severe PIH	22 (44%)	0
Platelet Status	Normal	38 (76%)	50 (100%)
	Low	12 (24%)	0
Treatment	None	0	50 (100%)
	Conservative	10 (20%)	0
	Antihypertensive	25 (50%)	0
	Antihypertensive + MgSO4	15 (30%)	0
Mode of Delivery	Vaginal	35 (70%)	32 (64%)
	LSCS	14 (28%)	13 (26%)
	Instrumental	1 (2%)	5 (10%)
Birth weight (in Kg)	$\leq 2.0$	4 (8%)	1 (2%)
	2.0-2.5	8 (16%)	3 (6%)
	$> 2.5$	38 (76%)	46 (92%)
Foetal Complications	None	37 (74%)	45 (90%)

	IUGR	5 (10%)	1 (2%)
	Preterm	5 (10%)	2 (4%)
	Respiratory distress	3 (6%)	2 (4%)

Parity distribution showed more primigravida patients among hypertensive (74%) than normotensive (58%) patients, followed by gravida 2 (20% vs. 30%) and gravida  $\geq 3$  (6% vs. 12%); this was not statistically significant ( $p=0.125$ ). In contrast, BMI categories were significantly associated ( $p<0.001$ ), with obesity much higher in hypertensive patients (36% vs. 0%) and overweight more frequent (34% vs. 4%), while normal BMI was predominant in normotensive patients (94% vs. 30%), and underweight was only in normotensive patients (2%).

Proteinuria was strongly associated with hypertensive disorders ( $p<0.001$ ), with its absence more common in normotensive patients (90% vs. 20%), while varying degrees of proteinuria (1+, 2+, and 3+) were mainly observed in hypertensive

patients (40%, 24%, and 16%) compared to normotensive patients (10%, 0%, and 0%). Maternal complications were higher among hypertensive patients ( $p<0.05$ ), with complications such as abruption (12%), eclampsia (6%), and HELLP syndrome (2%) only in hypertensive patients, while there were none in normotensive patients.

Imminent signs were also significantly associated ( $p<0.001$ ), with their absence being more common in normotensive patients (100% vs. 60%). Among hypertensive patients, headache (16%), blurring of vision (8%), epigastric pain (6%), and multiple symptoms (10%) were observed exclusively, and the overall presence of any symptom was noted in 40% of hypertensive patients, while none of the normotensive patients exhibited these signs. [Table 2]

**Table 2: Association of Parity, BMI, Proteinuria, Maternal Complications, and Imminent Signs with Hypertensive Disorders of Pregnancy**

		N (%)		P value
		Hypertensive (n=50)	Normotensive (n=50)	
Parity	Primigravida	37 (74%)	29 (58%)	0.125
	Gravida 2	10 (20%)	15 (30%)	
	Gravida $\geq 3$	3 (6%)	6 (12%)	
BMI Categories	Underweight	0	1 (2%)	<0.001
	Normal	15 (30%)	47 (94%)	
	Overweight	17 (34%)	2 (4%)	
	Obese	18 (36%)	0	
Proteinuria	Nil	10 (20%)	45 (90%)	<0.001
	1+	20 (40%)	5 (10%)	
	2+	12 (24%)	0	
	3+	8 (16%)	0	
Maternal Complications	None	40 (80%)	50 (100%)	<0.05
	Abruption	6 (12%)	0	
	Eclampsia	3 (6%)	0	
	HELLP	1 (2%)	0	
Imminent Signs	None	30 (60%)	50 (100%)	<0.001
	Headache	8 (16%)	0	
	Blurring of vision	4 (8%)	0	
	Epigastric pain	3 (6%)	0	
	Multiple symptoms	5 (10%)	0	
	Any symptom present	20 (40%)	0	

The mean age was higher in hypertensive patients ( $29.4 \pm 7.8$  years) than in normotensive patients ( $26.1 \pm 5.9$  years) ( $p<0.05$ ,  $d=0.47$ ). Gestational age was similar ( $30.2 \pm 5.8$  vs.  $29.8 \pm 5.7$  weeks;  $p=0.732$ ). Height showed no significant difference ( $155.1 \pm 7.9$  cm vs.  $154.8 \pm 8.2$  cm;  $p=0.855$ ). Weight was significantly higher in hypertensive patients ( $68.2 \pm 12.4$  kg vs.  $55.8 \pm 7.9$  kg) ( $p<0.001$ ,

$d=1.18$ ). Body mass index was also significantly elevated in hypertensive patients ( $28.3 \pm 4.2$  kg/m<sup>2</sup> vs.  $23.2 \pm 2.1$  kg/m<sup>2</sup>) ( $p<0.001$ ,  $d=1.48$ ). Systolic blood pressure was markedly higher in hypertensive patients ( $158.4 \pm 11.2$  mmHg vs.  $118.2 \pm 9.8$  mmHg) ( $p<0.001$ ,  $d=3.83$ ). Diastolic blood pressure was also significantly higher ( $102.1 \pm 8.7$  mmHg vs.  $74.3 \pm 7.1$  mmHg) ( $p<0.001$ ,  $d=3.48$ ). [Table 3]

**Table 3: Comparison of Continuous Demographic, Anthropometric, and Hemodynamic Parameters Between Patients**

	Mean $\pm$ SD		Mean Difference (95% CI)	p-value	Cohen's d
	Hypertensive	Normotensive			
Age (years)	$29.4 \pm 7.8$	$26.1 \pm 5.9$	3.3 (0.54-6.06)	<0.05	0.47
Gestational age (weeks)	$30.2 \pm 5.8$	$29.8 \pm 5.7$	0.4 (-1.89-2.69)	0.732	0.07
Height (cm)	$155.1 \pm 7.9$	$154.8 \pm 8.2$	0.3 (-3.00-3.60)	0.855	0.04
Weight (kg)	$68.2 \pm 12.4$	$55.8 \pm 7.9$	12.4 (8.24-16.56)	<0.001	1.18
BMI (kg/m <sup>2</sup> )	$28.3 \pm 4.2$	$23.2 \pm 2.1$	5.1 (3.73-6.47)	<0.001	1.48
Systolic BP (mmHg)	$158.4 \pm 11.2$	$118.2 \pm 9.8$	40.2 (36.05-44.35)	<0.001	3.83
Diastolic BP (mmHg)	$102.1 \pm 8.7$	$74.3 \pm 7.1$	27.8 (24.63-30.97)	<0.001	3.48

Serum magnesium was significantly lower in hypertensive patients ( $1.43 \pm 0.09$  mg/dl) than in normotensive patients ( $2.25 \pm 0.12$  mg/dl), with a highly significant difference ( $p < 0.001$ ) and large effect size ( $d = 7.45$ ). Serum calcium was also significantly reduced in hypertensive patients ( $8.56 \pm 0.31$  mg/dl) compared to normotensive patients ( $10.96 \pm 0.45$  mg/dl) ( $p < 0.001$ ), with a large effect size ( $d = 6.15$ ). Platelet count was lower in hypertensive patients ( $2.6 \pm 0.8$  lakhs) than in normotensive patients ( $3.0 \pm 0.7$  lakhs), with a significant difference ( $p < 0.01$ ) and moderate effect size ( $d = 0.53$ ).

Haemoglobin levels were similar ( $10.4 \pm 1.2$  g/dl vs.  $10.6 \pm 1.2$  g/dl), with no significant difference ( $p = 0.407$ ). Blood urea ( $25.1 \pm 8.2$  mg/dl vs.  $24.9 \pm 7.8$  mg/dl;  $p = 0.901$ ) and serum creatinine ( $0.80 \pm 0.20$  mg/dl vs.  $0.79 \pm 0.19$  mg/dl;  $p = 0.8$ ) levels also showed no significant differences. Birth weight was significantly lower in hypertensive patients ( $2.40 \pm 0.51$  kg) than in normotensive patients ( $2.78 \pm 0.42$  kg) ( $p < 0.001$ ) with a large effect size ( $d = 0.81$ ). These findings indicate that hypertensive disorders are strongly associated with reduced serum magnesium and calcium levels and lower birth weight, while other biochemical parameters remain comparable. [Table 4]

**Table 4: Comparison of Biochemical and Haematological Parameters Between Patients**

	Mean $\pm$ SD		Mean Difference	p-value	Cohen's d
	Hypertensive	Normotensive			
Serum Magnesium (mg/dl)	$1.43 \pm 0.09$	$2.25 \pm 0.12$	-0.82	<0.001	7.45
Serum Calcium (mg/dl)	$8.56 \pm 0.31$	$10.96 \pm 0.45$	-2.4	<0.001	6.15
Platelet Count (lakhs)	$2.6 \pm 0.8$	$3.0 \pm 0.7$	-0.4	<0.01	0.53
Hemoglobin (g/dl)	$10.4 \pm 1.2$	$10.6 \pm 1.2$	-0.2	0.407	0.17
Blood Urea (mg/dl)	$25.1 \pm 8.2$	$24.9 \pm 7.8$	0.2	0.901	0.02
Serum Creatinine (mg/dl)	$0.80 \pm 0.20$	$0.79 \pm 0.19$	0.01	0.8	0.05
Birth Weight (kg)	$2.40 \pm 0.51$	$2.78 \pm 0.42$	-0.38	<0.001	0.81

Serum magnesium demonstrated 100% sensitivity and specificity in this sample, with 50 true positives in hypertensive patients and 50 true negatives in normotensive patients, and no false results. The sensitivity, specificity, predictive values, and diagnostic accuracy were 100%. The 95% confidence interval for sensitivity and specificity was 92.9%-100%, with a large effect size (Cohen's  $d = 7.45$ ). Serum calcium also performed well, with

47 true positives, 50 true negatives, no false positives, and three false negatives. The sensitivity, specificity 100 %, positive predictive value 100 %, and negative predictive value were 94 %, 100 %, 100 %, and 94.3%, respectively, with an accuracy of 97 %. The 95% confidence interval for sensitivity was 83.5% to 98.7%, and for specificity, 92.9% to 100%, with a large effect size (Cohen's  $d = 6.15$ ). [Table 5]

**Table 5: Diagnostic Performance of Serum Magnesium and Calcium in Detecting Hypertensive Disorders of Pregnancy**

	Serum Magnesium (<1.8 mg/dl)	Serum Calcium (<9.0 mg/dl)
True Positive (TP)	50	47
False Positive (FP)	0	0
True Negative (TN)	50	50
False Negative (FN)	0	3
Sensitivity (%)	100%	94%
Specificity (%)	100%	100%
Positive Predictive Value (PPV)	100%	100%
Negative Predictive Value (NPV)	100%	94.30%
Accuracy (%)	100%	97%
95% CI (Sensitivity)	92.9-100%	83.5-98.7%
95% CI (Specificity)	92.9-100%	92.9-100%
Effect Size (Cohen's d)	7.45 (Very Large)	6.15 (Very Large)

## DISCUSSION

Our study revealed significant differences in serum magnesium and calcium levels between hypertensive and normotensive patients, highlighting the clinical and obstetric disparities. These findings underscore the role of micronutrient imbalance in hypertensive disorders of pregnancy (HDP). Hypertensive patients were older ( $29.4 \pm 7.8$  vs.  $26.1 \pm 5.9$  years;  $p < 0.05$ ) and had higher BMI ( $28.3 \pm 4.2$  vs.  $23.2 \pm 2.1$  kg/m<sup>2</sup>;  $p < 0.001$ ), with obesity more common. Sethi et al. reported similar

BMI results in preeclamptic women ( $26.00 \pm 2.18$  vs.  $24.34 \pm 2.12$  kg/m<sup>2</sup>;  $p = 0.0002$ ). Parity showed no significant association ( $p = 0.125$ ), aligning with Sethi et al., but differing from earlier primigravida-focused findings.<sup>[20]</sup>

An important finding of our study was that serum magnesium was significantly lower in hypertensive patients ( $1.43 \pm 0.09$  mg/dl) than in normotensive patients ( $2.25 \pm 0.12$  mg/dl;  $p < 0.001$ ), consistent with Gupta et al.'s report of lower serum magnesium levels in hypertensive women ( $1.723 \pm 0.414$  vs.  $1.979 \pm 0.405$  mg/dl;  $p = 0.001$ ).<sup>[21]</sup> Sethi et al. and

Yousuf et al. also noted reduced magnesium levels in women with preeclampsia, suggesting the role of hypomagnesaemia in endothelial dysfunction and vascular resistance in HDP.[20-22] However, a study by Sah and Goel found no significant difference ( $1.90 \pm 0.31$  vs.  $1.92 \pm 0.23$  mg/dl;  $p=1.00$ ), possibly due to sample size and population disparities.<sup>[23]</sup> Our study indicated a larger effect size ( $d=7.45$ ), suggesting a stronger association, possibly due to the more severe cases.

In our study, serum calcium levels were significantly lower in hypertensive patients ( $8.56 \pm 0.31$  mg/dl) than in normotensive patients ( $10.96 \pm 0.45$  mg/dl;  $p<0.001$ ). Gupta et al. ( $9.461 \pm 1.164$  vs  $10.119 \pm 1.27$  mg/dl) and Yousuf et al. ( $9.7 \pm 1.7$  vs  $10.2 \pm 1.4$  mg/dl;  $p=0.031$ ) reported similar findings.<sup>[21,22]</sup> Sethi et al. found lower calcium levels in preeclamptic patients ( $3.34 \pm 1.35$  vs  $4.37 \pm 0.74$ ;  $p<0.0001$ ), suggesting that hypocalcaemia promotes vasoconstriction via increased intracellular calcium and renin-angiotensin activation.<sup>[20]</sup> Conversely, Sah and Goel found no significant difference ( $9.00 \pm 0.47$  vs.  $9.12 \pm 0.37$  mg/dl;  $p=0.34$ ), indicating that methodological and population factors may influence the calcium-HDP relationship.<sup>[23]</sup> A meta-analysis by He et al. supported this trend, showing significantly lower calcium (MD =  $-0.26$ ; 95% CI  $-0.36$  to  $-0.15$ ) and magnesium levels in hypertensive pregnancies.<sup>[24]</sup>

Our study showed that electrolyte imbalance correlates with disease severity. Proteinuria and maternal complications (abruption, 12%; eclampsia, 6%; and HELLP syndrome, 2%) were exclusive to hypertensive patients. Sethi et al. reported a decrease in calcium and magnesium levels with increasing preeclampsia severity, suggesting that these electrolytes indicate disease progression.<sup>[20]</sup> For foetal outcomes, birth weight was lower in hypertensive patients ( $2.40 \pm 0.51$  vs.  $2.78 \pm 0.42$  kg;  $p<0.001$ ), with higher IUGR, preterm birth, and respiratory distress rates. These findings align with those of Yousuf et al., who reported lower birth weight ( $2.35 \pm 0.48$  vs.  $2.85 \pm 0.45$  kg;  $p=0.022$ ) and higher incidence of low birth weight (47.5% vs. 18.3%;  $p=0.002$ ) in hypertensive pregnancies.<sup>[22]</sup>

Unlike Yousuf et al., who observed higher caesarean rates (75% vs. 53.3%;  $p<0.001$ ), our study found comparable delivery modes, likely reflecting differences in clinical management and severity.<sup>[22]</sup>

An important strength of our study was the diagnostic evaluation of electrolytes. Serum magnesium demonstrated 100% sensitivity and specificity in this study sample, whereas serum calcium levels showed 94% sensitivity and 100% specificity. Consistent findings across studies support their potential as adjunct biomarkers, although variability in cut-off values suggests the need for population-specific validation.

## CONCLUSION

Our study shows that hypertensive pregnancy disorders are linked to altered mineral metabolism, especially hypomagnesaemia and hypocalcaemia. Hypertensive mothers had significantly lower serum magnesium and calcium levels than normotensive mothers, with hypomagnesaemia in all cases and hypocalcaemia in most cases. Serum magnesium levels demonstrated high diagnostic accuracy, suggesting their potential as adjunct biomarkers for screening hypertensive disorders. Hypertensive mothers also had higher body mass indices and adverse foetal outcomes, including low birth weight, but no significant difference in delivery mode was noted, suggesting effective management. These findings emphasise the may be useful in high-risk populations in antenatal care for high-risk pregnancies to enable early detection and intervention. Further large-scale studies are needed to validate these results and explore micronutrient supplementation and preventive strategies.

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