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Original Research Article

A cross-sectional comparative study to assess the role of spot urinary calcium to creatinine ratio in prediction of preeclampsia

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ABSTRACT

Background: Preeclampsia is a hypertensive disorder unique to pregnancy, associated with significant maternal and fetal morbidity and mortality. Early prediction and diagnosis are critical for better clinical outcomes. Biochemical markers such as the spot urinary calcium to creatinine ratio (UCa/Cr) have been proposed as a non-invasive, cost-effective tool for identifying preeclampsia. Objective of the study was to evaluate and compare the spot urinary calcium to creatinine ratio in pregnant women with and without preeclampsia and assess its diagnostic utility.

Methods: This cross-sectional comparative study was conducted on pregnant women in the third trimester attending antenatal clinics or admitted to the obstetric unit. Participants were divided into two groups: women diagnosed with preeclampsia (study group) and normotensive pregnant women (control group). A midstream urine sample was collected from each participant for calcium and creatinine estimation, and the UCa/Cr ratio was calculated. The two groups were statistically compared using appropriate tests.

Results: The mean UCa/Cr ratio was significantly lower in the preeclamptic group compared to the normotensive controls ($p < 0.001$). A UCa/Cr cutoff value of < 0.04 demonstrated good sensitivity and specificity in identifying preeclampsia.

Conclusions: The spot urinary calcium to creatinine ratio is a simple, non-invasive, and reliable test that may serve as an adjunct tool for the early detection and diagnosis of preeclampsia. Further large-scale studies are recommended to validate its clinical applicability.

Keywords: Preeclampsia, Hypertensive disorder, Pregnancy, Urinary calcium to creatinine ratio, Biochemical marker, Third trimester, Non-invasive test, Early detection, Maternal and fetal morbidity

INTRODUCTION

Hypertensive disorders are the most common medical complications during pregnancy, affecting approximately 5–10% of all pregnancies.¹ Among these, preeclampsia is the most severe form, occurring in about 3.9% of pregnancies and is the second leading cause of maternal mortality in India, as well as a major contributor to perinatal morbidity and mortality.¹ Preeclampsia is a complex multisystem disorder responsible for over 40% of premature deliveries and causes approximately 40,000 maternal deaths annually, with one maternal death occurring every three minutes worldwide.² Infants of

women with severe hypertension during pregnancy face a fivefold higher risk of death compared to those of normotensive mothers.³

Despite extensive research, the exact cause of preeclampsia remains unclear, making primary prevention unfeasible. As a result, focus has shifted toward early detection and secondary prevention through identification of at-risk women using reliable predictive markers. Calcium metabolism undergoes significant changes during pregnancy, with urinary calcium excretion increasing to 350–620 mg/day compared to 100–250 mg/day in non-pregnant women, likely due to increased intestinal

absorption, renal filtered load, and glomerular filtration rate. In preeclampsia, hypocalciuria is commonly observed, along with decreased levels of 1,25-dihydroxyvitamin D, elevated parathyroid hormone (PTH), reduced urinary cyclic AMP, increased intracellular calcium, and lower ionized calcium levels (7–8 mg/dl).⁴⁻⁸ These abnormalities may result from reduced glomerular filtration rate (GFR), decreased renal plasma flow, increased fetal calcium uptake, or low maternal calcium intake.⁹⁻¹¹

Although 24-hour urine collection is the standard for detecting hypocalciuria, it is often cumbersome and time-consuming. Studies have shown that the spot urinary calcium-to-creatinine (Ca/Cr) ratio is a reliable and simpler alternative, with good correlation to 24-hour urinary calcium measurements.¹⁵

Objectives

This study aims to assess the clinical utility of the spot urinary calcium-to-creatinine ratio in predicting the development of gestational hypertension and preeclampsia in asymptomatic, normotensive pregnant women.

Review of literature

Definition

Hypertension in pregnancy is defined by the National High Blood Pressure Education Program (NHBPEP, 2000) and American College of Obstetrics and Gynecology (ACOG) as a systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg (marked by Korotkoff phase V), recorded on two occasions 4–6 hours apart.¹³

Proteinuria, reflecting glomerular damage and associated with maternal and perinatal morbidity and mortality, is not used to classify preeclampsia as severe or non-severe.¹⁴

Classification (ISSHP & NHBPEP, 2000)

Gestational hypertension: BP $>140/90$ mmHg after 20 weeks gestation without proteinuria, returning to normal within 12 weeks postpartum. Up to 50% may progress to preeclampsia. Preeclampsia/eclampsia syndrome: hypertension with proteinuria developing after 20 weeks in previously normotensive women. A pregnancy-specific syndrome caused by vasospasm and endothelial dysfunction.

ACOG (2013b) diagnostic criteria

BP $\geq 140/90$ mmHg on two occasions ≥ 4 hours apart or $\geq 160/110$ mmHg (severe hypertension may be confirmed within minutes).

Proteinuria: ≥ 300 mg/24 hours, protein/creatinine ratio ≥ 0.3 mg/dl, or dipstick 2+ (if quantitative methods unavailable).

Without proteinuria: presence of thrombocytopenia, renal insufficiency, liver dysfunction, pulmonary edema, or new-onset headache or visual symptoms.

Delta hypertension: a sudden rise in mean arterial pressure in late pregnancy, even with BP $<140/90$ mmHg, may indicate preeclampsia.¹⁵

Chronic hypertension: diagnosed before pregnancy or before 20 weeks' gestation, or if hypertension persists >12 weeks postpartum.

Superimposed preeclampsia: new-onset proteinuria (>300 mg/24h) in a chronically hypertensive woman.

Regulation of calcium

Calcium homeostasis is tightly regulated by parathyroid hormone (PTH), vitamin D metabolites, calcitonin, and cytokines such as TGF- β and IL-6. The enzyme 1 α -hydroxylase in the kidney converts 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol (active vitamin D), which promotes calcium absorption in the gut and reabsorption in the kidney.

PTH is released in response to low ionized calcium. It increases calcium reabsorption in the distal tubule, stimulates bone resorption, and enhances intestinal absorption via vitamin D₃ activation, thus normalizing serum calcium.

Calcium in normal pregnancy and preeclampsia

During pregnancy and lactation, calcium needs are met through enhanced intestinal absorption and renal conservation. Hormones like IGF-1, PTH, and 1,25(OH)₂D play significant roles.¹⁶

Normal pregnancy shows mild serum calcium/phosphate changes, but urinary calcium excretion rises—ranging from 350–620 mg/day versus 100–250 mg/day in non-pregnant women.¹⁷ Excretion peaks in the third trimester.¹⁸ In preeclampsia, calcium metabolism is altered: hypocalciuria, decreased 1,25(OH)₂D, increased PTH, reduced urinary cAMP, elevated intracellular calcium, and lowered ionized calcium.¹⁹⁻²² Some studies report reduced 24-hour urinary calcium in preeclampsia, while others find no correlation.^{18,23,24} Causes of hypocalciuria may include dietary deficiency, increased fetal extraction, or renal dysfunction.

Hypocalciuria in preeclampsia

Women with preeclampsia may show low or normal ionized calcium.

Hypothesized sequence

Reduced 1,25(OH)₂D or low dietary calcium $\rightarrow$ $\downarrow$ intestinal absorption $\rightarrow$ $\downarrow$ serum ionized calcium.

Elevated PTH in response.

Increased renal calcium reabsorption → hypocalciuria.

Clinical studies

Sanchez-Ramos L: a single urine sample's calcium-creatinine ratio is as accurate as a 24-hour collection. This low ratio is an early and persistent marker in pregnancy.

According to Amitava et al, 24-hour urine calcium <100 mg supports preeclampsia diagnosis.³¹

Clinical relevance

Low calcium intake may raise BP via PTH/renin stimulation, increasing intracellular calcium in vascular smooth muscle → vasoconstriction, decreased GFR, and further calcium retention. Extracellular ionized calcium is crucial for nitric oxide production and vascular tone regulation.

Benefits of early detection

Preeclampsia can evolve rapidly and fatally. Timely detection through BP monitoring significantly improves outcomes. According to USPSTF, appropriate treatments reduce both maternal and neonatal morbidity and mortality.

METHODS

This cross-sectional comparative study was conducted in the Department of Obstetrics and Gynecology at IQRAA International Hospital and Research Institute, Calicut, Kerala, over a period of two years (December 2020 to November 2022). The study aimed to evaluate the utility of the urinary calcium/creatinine ratio (UCCR) in predicting preeclampsia among pregnant women.

A total of 104 pregnant women with gestational age between 24–40 weeks were enrolled through purposive non-random sampling. Participants were divided into two groups: group I (control) included 52 normotensive pregnant women, and group II (study group) comprised 52 women diagnosed with preeclampsia based on ACOG 2013 criteria. Inclusion criteria included pregnant women aged 18–40 years, and for group II, the presence of hypertension (BP $\geq 140/90$ mmHg) and proteinuria ($\geq 1+$ on dipstick) or other diagnostic indicators of preeclampsia. Women with comorbidities such as diabetes, renal disorders, thyroid disease, autoimmune conditions, and multiple pregnancies were excluded.

Sample size estimation was based on previously reported mean UCCR values: 0.13 ± 0.06 in normal pregnancies and 0.06 ± 0.05 in preeclamptic patients, with a calculated minimum of 51 participants per group ($Z\alpha=1.96$, $Z\beta=0.84$, $SD=0.054$, and effect size $d=0.03$).

Data collection included clinical examination and relevant maternal and fetal parameters such as age, parity, blood pressure, proteinuria, BMI, and fetal wellbeing. A random urine sample was analyzed using the immunoturbidimetric method to measure calcium and creatinine levels, and UCCR was calculated.

The primary outcome of the study was to assess the sensitivity of UCCR in predicting preeclampsia. Severe preeclampsia was identified by BP $\geq 160/110$ mmHg along with signs like headache, visual disturbances, elevated liver enzymes, oliguria, or fetal growth restriction.

This study provides insights into the diagnostic value of UCCR as a simple, non-invasive test for early prediction and management of preeclampsia.

Statistical analysis

Data was analyzed using the statistical package for the social sciences (SPSS) 26.0 (SPSS Inc., Chicago, IL) and level of significance was set at $p < 0.05$. Descriptive statistics was performed to assess the proportion of each category of the respective groups. Normality of the data was assessed using Shapiro Wilk test. Inferential statistics was done using Chi square test for categorical data to find out the association between the groups and independent t test for the continuous data to find out the difference between the groups.

RESULTS

Age

Preeclamptic women were significantly older (mean 28.46 years) than normotensive controls (mean 25.55 years); $p=0.002$.

Gestational Age

reeclamptic women had slightly higher mean gestational age (34.86 weeks versus 33.48 weeks); $p=0.011$ (Table 1).

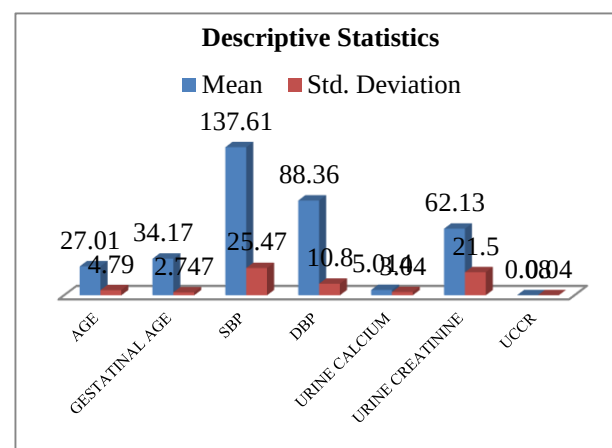


Figure 1: Descriptive statistics.

Table 1: Comparison of mean age and gestational age.

Category	Case (n=52)	Control (n=52)	T value	P value
Comparison of mean age				
Mean	28.46	25.55	3.237	0.002*
SD	5.42	3.52		
Comparison of gestational age				
Mean	34.86	33.48	2.602	0.011*
SD	2.65	2.76		

*P<0.05 is statistically significant (independent t test)

Parity

No statistically significant difference in primigravida versus multigravida between groups; p=0.239.

BMI

Preeclamptic women had a higher BMI (mean=26.10) compared to controls (mean=24.78); p=0.018, indicating obesity as a possible risk factor (Table 2).

Table 2: Comparison of parity and BMI.

Catego-ry	Case (n=52) (%)	Control (n=52) (%)	Chi square value	P value
Comparison of parity				
Multi	29 (55.8)	23 (44.2)	1.385	0.239*
Primi	23 (44.2)	29 (55.8)		
Comparison of BMI			T value	
Mean	26.10	24.78	2.478	0.018**
SD	3.01	2.58		

*P<0.05 is statistically significant (Chi square test), **p<0.05 is statistically significant (independent t test)

SES

No difference between groups; majority (92.7%) in both belonged to middle class; p=1.000.

Pulse rate

No significant difference in pulse rate between groups; p=0.762 (Table 3).

Systolic BP

Significantly higher in preeclamptic group (155.34 mmHg versus 119.88 mmHg); p<0.0001.

Diastolic BP

Also significantly higher (97.46 mmHg versus 79.33 mmHg); p<0.0001 (Table 4).

These findings confirm the diagnostic criteria of preeclampsia.

Table 3: Comparison of socioeconomic status and pulse rate.

Category	Case (n=52) (%)	Control (n=52) (%)	Chi square value	P value
Comparison of SES				
Low	4 (7.7)	4 (7.7)	0.000	1.000*
Middle	48 (92.7)	48 (92.7)		
Comparison of mean PR				
Mean	75.69	76.11	-0.304	0.762**
SD	4.80	1.22		

*P<0.05 is statistically significant (Chi square test), **p<0.05 is statistically significant (independent t test)

Table 4: Comparison of mean systolic and diastolic blood pressure.

Category	Case (n=52)	Control (n=52)	T value	P value
Comparison of mean SBP				
Mean	155.34	119.88	9.908	0.000*
SD	12.33	22.55		
Comparison of mean DBP				
Mean	97.46	79.33	15.480	0.000*
SD	6.11	5.76		

*P<0.05 is statistically significant (independent t test)

Urine calcium

Significantly lower in preeclampsia group (2.86 mg/100 ml) than controls (7.16 mg/100 ml); p<0.0001.

Urine creatinine

Also lower in preeclamptic group (57.40 mg/100 ml versus 66.85 mg/100 ml); p=0.027 (Table 5).

Indicates renal involvement and altered calcium metabolism in preeclampsia.

Table 5: Comparison of mean urine calcium and creatinine.

Category	Case (n=52)	Control (n=52)	T value	P value
Comparison of mean urine calcium				
Mean	2.86	7.16	-10.160	0.000*
SD	1.86	2.41		
Comparison of mean urine creatinine				
Mean	57.40	66.85	-2.243	0.027*
SD	18.7	23.91		

*P<0.05 is statistically significant (independent t test)

UCCR

Markedly reduced in the preeclamptic group (mean: 0.0488) versus control (mean: 0.1135); p<0.0001.

A significant proportion of preeclamptic women had $\text{UCCR} \leq 0.04$, supporting its diagnostic potential (Table 6).

Table 6: Comparison of mean urinary calcium to creatinine ratio.

Category	Comparison of mean urine calcium			
	Case (n=52)	Control (n=52)	T value	P value
Mean	0.0488	0.1135	-8.772	0.000*
SD	0.033	0.041		

*P<0.05 is statistically significant (independent t test)

DISCUSSION

Preeclampsia is a complex multisystem disorder, primarily rooted in abnormal placentation characterized by inadequate cytotrophoblast invasion of the spiral arteries. This leads to widespread endothelial dysfunction and vasospasm. Recent studies suggest deranged calcium metabolism in preeclampsia, often reflected as hypocalciuria, which may either precede or accompany the condition. Renal tubular dysfunction seen in preeclampsia contributes to reduced calcium excretion and proteinuria. Traditionally, 24-hour urinary protein estimation has been the diagnostic gold standard, but it's time-consuming and impractical, prompting investigation into simpler, reliable markers such as the spot UCCR.

This cross-sectional comparative study was conducted in the Department of Obstetrics and Gynaecology at IQRAA International Hospital and Research Centre, Calicut, over two years. It involved 104 pregnant women, equally divided into two groups: preeclamptic and normotensive controls (52 each).

Demographic analysis revealed the mean age of the preeclamptic group was 28.46 ± 5.42 years, significantly higher than the control group at 25.55 ± 3.52 years ($p=0.002$). In the preeclamptic group, 42.3% were over 30 years old, compared to only 13.5% in the control group. These findings align with Lamminpää et al, who demonstrated an association between advanced maternal age and increased preeclampsia risk.³⁶

Gestational age was also significantly different, with the preeclampsia group averaging 33.48 ± 2.76 weeks, and controls averaging 34.86 ± 2.65 weeks ($p=0.011$). Most preeclamptic patients (55.76%) were under 35 weeks. Gravidity showed no significant difference between groups ($p=0.239$), consistent with previous studies by Qublan et al, Begum et al and Soudan et al.²⁵⁻²⁷

Socioeconomic status was comparable between the groups ($p=1.000$), but body mass index (BMI) showed a significant difference. The preeclampsia group had a higher BMI (26.10 ± 3.01) compared to controls (24.78 ± 2.58), $p=0.018$. Seven women in the preeclampsia group had $\text{BMI} \geq 30$, while only one control did. This

correlates with Mrema et al, who noted increased preeclampsia risk with maternal obesity.³⁷

Pulse rate (PR) showed no significant difference between groups ($p=0.762$), but both systolic (155.34 ± 12.33 mmHg) and diastolic (97.46 ± 6.11 mmHg) blood pressures were significantly elevated in the preeclamptic group compared to the control group (119.88 ± 22.55 and 79.33 ± 5.76 mmHg respectively; $p<0.05$). No participants had a past or family history of preeclampsia.

Urinary biochemical parameters were key in this study. Spot urinary calcium and creatinine concentrations were significantly reduced in preeclamptic patients. Mean calcium concentration in preeclampsia was 2.86 ± 1.86 mg/100ml versus 7.16 ± 2.41 mg/100 ml in controls ($p<0.05$). Mean creatinine concentration was also lower in preeclampsia (57.40 ± 18.7 mg/100 ml) compared to controls (66.85 ± 23.91 mg/100 ml, $p=0.027$).

These findings support theories that increased tubular reabsorption of calcium due to altered renal perfusion and glomerular filtration contributes to hypocalciuria in preeclampsia. Marya et al noted increased urinary calcium excretion in normal pregnancies due to higher glomerular filtration rates, a phenomenon altered in preeclampsia.³⁰

Ingec et al confirmed reduced urinary calcium in preeclamptic and eclamptic patients despite unchanged serum calcium levels.³⁸ Similarly, McMaster et al conducted a meta-analysis of 21 studies and concluded that urinary calcium excretion was consistently lower in preeclamptic women.³⁹

Further studies, including Huikeshoven and Zuiderhoudt, Vural et al, and Donovan McGrowder et al, reinforced that hypocalciuria is a consistent and measurable feature of preeclampsia, likely due to increased calcium reabsorption.^{12,28,29} Taufield et al reported significantly lower urinary calcium in preeclampsia compared to other hypertensive and normotensive pregnant patients.²⁰

In this study, the UCCR was significantly reduced in the preeclampsia group (0.0488 ± 0.033) compared to the control group (0.1135 ± 0.041). This supports findings by Kazerooni et al, Ozcan et al, and Gokee et al, who found strong correlation between UCCR and total urinary calcium excretion.³²⁻³⁴ Kar et al showed that a $\text{UCCR} \leq 0.04$ during mid-trimester predicted preeclampsia with high accuracy. In the current study, 57.69% of preeclamptic women had a $\text{UCCR} \leq 0.04$, while none of the controls did.³⁵

Ye et al demonstrated that UCCR has strong predictive value for pregnancy-induced hypertension, with high sensitivity (76.2%) and specificity (97.5%) at a cut-off of 0.04. Begum et al in Bangladesh also found significantly reduced UCCR in preeclamptic patients.²⁶

From these results it is evident that spot urinary calcium creatinine ratio is a very useful indicator of hypocalciuria and is very important that hypocalciuria is seen in most studies with preeclamptic patients. In this study on comparing the spot urinary calcium creatinine ratio of both groups showed highly significant variation, hence spot urinary calcium and creatinine ratio can be regarded as a predictor for preeclampsia.

CONCLUSION

Preeclampsia is a serious multisystem disorder in pregnancy, and early prediction is essential to reduce its adverse outcomes. This study found that the spot UCCR is significantly lower in preeclamptic women compared to normotensive pregnant women, and it correlates well with calcium excretion. Unlike 24-hour urine calcium estimation, which is cumbersome and costly, spot UCCR is simple, inexpensive, quick, non-invasive, and patient-friendly, making it a promising screening tool.

The study suggests that spot UCCR could be used for early detection of preeclampsia, particularly in developing countries where the disease burden is high. However, its predictive value should be confirmed through larger prospective studies.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

- Balakrishnan S. Textbook of Obstetrics. 3rd edition. New Delhi, India; Paras Medical Publisher. 2020.
- Dutt V, Dev NM. Evaluation of urinary calcium creatinine ratio as a diagnostic tool in the management of preeclampsia. *Int J Med Sci Public Health*. 2016;5:1635-8.
- Mozdzien G, Schininger M, Zazgornik J. Kidney function and electrolyte metabolism in healthy pregnant women [German]. *Wien Med Wochenschr*. 1995;145:12-7.
- Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF. WHO analysis of causes of maternal death: a systematic review. *Lancet*. 2006;367(9516):1066-74.
- Maikranz P, Holley JL, Parks JH, Lindheimer MD, Nakagawa Y, Coe FL. Gestational hypercalciuria causes pathological urine calcium oxalate supersaturations. *Kidney Int*. 1989;36:108-13.
- Belizán JM, Villar J. The relationship between calcium intake and edema-, proteinuria-, and hypertension-gestosis: An hypothesis. *Am J Clin Nutr*. 1980;33:2202-10.
- Kovacs CS. Calcium and Bone Metabolism in Pregnancy and Lactation. *J Clin Endocrinol Metabolism*. 2001;86(6):2344-8.
- Ingec M, Nazik H, Kadanali S. Urinary calcium excretion in severe preeclampsia and eclampsia. *Clin Chem Lab Med*. 2006;44(1):511-3.
- Szmidi-Adjide V, Vendittelli F, David S, Bredent-Bangou J, Janky E. Calciuria and preeclampsia: A Case-control study. *Eur J Obstet Gynecol Reprod Biol*. 2006;125(2):193-8.
- Ramos JGL, Martins-Costa SH, Kessler JB, Costa CA, Barros E. Calciuria and preeclampsia. *Braz J Med Biol Res*. 1998;31(4):519-22.
- Suarez VR, Trelles JG, Miyahira JM. Urinary Calcium in asymptomatic primigravidas who later developed preeclampsia. *Obstet Gynecol*. 1996;87:79-82.
- Huikeshoven FJM, Zuijderhoudt FMJ. Hypocalciuria in hypertensive disorders of pregnancy and how to measure it? *Eu J Obs Gynecol*. 1990;36(1-2):81-5.
- American College of Obstetricians and Gynaecologists: Hypertension in pregnancy. Report of American College of Obstetricians and Gynaecologist's Task force on hypertension in pregnancy. *Obstet Gynecol*. 2013;122:1122.
- Chesley LC. Diagnosis of preeclampsia. *Obstet Gynecol*. 1985;65:423.
- Macdonald-Wallis C, Lawlor DA, Fraser A, May M, Nelson SM, Tilling K. Blood pressure change in normotensive, gestational hypertensive, preeclamptic, and essential hypertensive pregnancies. *Hypertension*. 2012;59(6):1241-8.
- Vargas Zapata CL, Donangelo CM, Woodhouse LR, Abrams SA, Spencer EM, King JC. Calcium homeostasis during pregnancy and lactation in Brazilian women with low calcium intakes: a longitudinal study. *Am J Clin Nutr*. 2004;80(2):417-22.
- Maikranz P, Holley JL, Parks JH, Lindheimer MD, Nakagawa Y, Coe FL. Gestational hypercalciuria causes pathological urine calcium oxalate supersaturations. *Kidney Int*. 1989;36:108-13.
- Pedersen EB, Johannesen P, Kristensen S, Rasmussen AB, Emmertsen K, Møller J, et al. Calcium, parathyroid hormone and calcitonin in normal pregnancy and preeclampsia. *Gynecol Obstet Invest*. 1984;18(3):156-64.
- Belizán JM, Villar J. The relationship between calcium intake and edema-, proteinuria-, and hypertension-gestosis: An hypothesis. *Am J Clin Nutr*. 1980;33:2202-10.
- Taufield PA, Ales KL, Resnick LM, Druzin ML, Gertner JM, Laragh JH. Hypocalcemia in preeclampsia. *N Eng J Med*. 1987;316:715-8.
- August P, Marcaccio B, Gertner JM, Druzin ML, Resnick LM, Laragh JH. Abnormal 1,25 dihydroxyvitamin D (1,25 OH) 2D3 metabolism in preeclampsia. *Am J Obstet Gynecol*. 1992;166:1295-9.
- Seely EW, Wood RJ, Brown EM, Graves SW. Lower serum ionized calcium and abnormal calciotropic hormone levels in preeclampsia. *J Clin Endocrinol Metab*. 1992;74:1436-40.

23. Nugent CE, Punch MR, Barr M Jr, LeBlanc L, Johnson MP, Evans MI. Persistence of partial molar placenta and severe preeclampsia after selective termination in a twin pregnancy. *Obstet Gynecol.* 1996;87(5 Pt 2):829-31.
24. Roelofsen JM, Berkel GM, Uttendorfsky OT, Slegers JF. Urinary excretion rates of calcium and magnesium in normal and complicated pregnancies. *Eur J Obstet Gynecol Reprod Biol.* 1988;27:227-36.
25. Qublan HS, Ammarin V, Bataineh O, Al-Shraideh Z, Tahat Y, Awamleh I, et al. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. *Med Sci Monit.* 2005;11(8):CR393-7.
26. Begum H, Rahman MN, Ferdous M, Basak SK, Yasmin N, Parveen A. Evaluation of Spot Urinary Calcium to Creatinine Ratio in Preeclampsia: A Cross Sectional Comparative Study. *J Natl Inst Neurosci Bangl.* 2017;3(1):14-20.
27. Saudan PJ, Shaw L, Brown MA. Urinary calcium/creatinine ratio as a predictor of preeclampsia. *Am J Hypertens.* 1998;11(7):839-43.
28. Vural P, Akgul C, Canbaz M. Calcium and Phosphate Excretion in Preeclampsia. *Tour J Med Sci.* 2000;30(1):39-42.
29. Growder D, Williams A, Gordon L, Crawford T, Alexander Lindo R, Irving R, et al. Hypocalciuria in preeclampsia and gestational hypertension due to decreased fractional excretion of calcium. *Arch Med Sci.* 2009;5:80-5.
30. Marya RK, Rathee S, Manrow M. Urinary calcium excretion in pregnancy. *Gynecol Obstet Investig.* 1987;23:141-4.
31. Pal A, Roy D, Adhikary S, Roy A, Dasgupta M, Mandal AK. A prospective study for the prediction of preeclampsia with urinary calcium level. *J Obstet Gynaecol India.* 2012;62(3):312-6.
32. David A, Padmaja P. Calcium-to-Creatinine Ratio in a Spot Sample of Urine, for Early Prediction of Hypertensive Disorders of Pregnancy: A Prospective Study. *J Obstet Gynaecol India.* 2016;66(Suppl 1):94-7.
33. Ozcan T, Kaleli B, Ozeren M, Turan C, Zorlu G. Urinary calcium to creatinine ratio for predicting preeclampsia. *Am J Perinatol.* 1995;12(5):349-51.
34. Gokce C, Gokce O, Baydinc C, Ijhan N, Alasehrli E, Ozkucuk F, et al. Use of random urine sample to estimate total urinary calcium and phosphate excretion. *Arch Intern Med.* 1991;151(8):1587-8.
35. Kar J, Srivastava K, Mishra RK. Role of urinary calcium creatinine ratio in prediction of pregnancy induced hypertension. *J Obstet Gynaecol India.* 2002;52:39-42.
36. Lamminpää R, Vehviläinen-Julkunen K, Gissler M, Heinonen S. Preeclampsia complicated by advanced maternal age: a registry-based study on primiparous women in Finland 1997-2008. *BMC Pregnancy Childbirth.* 2012;12:47.
37. Mrema D, Lie RT, Ostbye T, Mahande MJ, Dalveit AK. The association between prepregnancy body mass index and risk of preeclampsia: a registry based study from Tanzania. *BMC Pregnancy Childbirth.* 2016;18:56.
38. Ingec M, Nazik H, Kadanali S. Urinary calcium excretion in severe preeclampsia and eclampsia. *Clin Chem Lab Med.* 2006;44(1):51-3.
39. McMaster KM, Kaunitz AM, Burbano de Lara P, Sanchez-Ramos L. A systematic review and metaanalysis of hypocalciuria in preeclampsia. *Int J Gynaecol Obstet.* 2017;1389(1):3-11.

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