Original Article

Role of Magnesium in Preeclampsia

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Abstract

Background: The present study was planned to study the role of Mg in preeclampsia and its correlation with dietary intake of proteins and calories by analyzing serum Mg at 20 weeks, delivery, and cord blood Mg levels. **Methods:** The study was conducted in 102 pregnant women aged 17–36 years attending the outpatient department (OPD) before 20 weeks of gestation. The study samples were drawn twice: once before 20 weeks during visit in the OPD and second within 1 h of childbirth. Serum was analyzed for Mg levels using spectrophotometric method. Furthermore, thyroid-stimulating hormone and glucose challenge test were done as per the standard methods. **Results:** In normal pregnant women, the mean serum Mg level at <20 weeks of gestation was 2.03 ± 0.34 mg/dl, at term was 1.93 ± 0.41 mg/dl, and in cord blood was 1.84 ± 0.35 mg/dl. **Conclusion:** The study data support the hypothesis that Mg deficiency might be the causative factor in the development of preeclampsia.

Keywords: Apgar, birthweight, cord blood, magnesium, outcome, serum

Introduction

Preeclampsia is one of the most common causes of maternal and fetal morbidity and mortality affecting about 5%–7% of all pregnancies.[1] Early detection and prompt management helps in reducing the complications of this condition. The etiopathogenesis of preeclampsia is not exactly known. However, immunological, oxidative stress, and endothelium damage have been reported to be involved.

A number of dietary deficiencies or excesses have been blamed as the cause for preeclampsia over centuries. Studies have shown relationship between dietary deficiencies and incidence of preeclampsia. Some studies conclude malnutrition as a risk factor in the etiology of preeclampsia because of its higher incidence in developing countries. Dietary deficiency of magnesium (Mg) has been established to play a role in blood pressure regulation and hence the development of preeclampsia.[2]

Mg deficiency during pregnancy can induce maternal and fetal nutritional problems and consequences that might last in offspring throughout life. Maternal Mg intake is not only associated with pregnancy outcome but also associated with infant outcome.[3,4] Mg deficiency is associated with uterine hyperexcitability,[5] premature labor, and preeclampsia. Mg deficiency can also lead to problems with regulating body temperature in babies and can result in sudden infant death syndrome[6] and intrauterine growth retardation. Mg is an essential element for fetal well-being, and supplementation of Mg may be benefited to fetal outcome. Use of antenatal Mg sulfate for fetal neuroprotection has been recommended.[7]

Mg is important in energy-requiring metabolic processes, protein synthesis, membrane integrity, nervous tissue conduction, neuromuscular excitability, muscle contraction, hormone secretion, and intermediate metabolism.[3] Mg levels may have significant effects on cardiac excitability and vascular tone, contractility, and reactivity. Mg causes vascular muscle relaxation.[2,4] Hypomagnesemia in most of pregnant women is associated with hemodilution, renal clearance, and consumption of minerals by the growing fetus. There is a decrease in ionized and total Mg levels with increasing gestational age during normal pregnancy, and there is evidence of Mg disturbance in women who later

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developed preeclampsia. Pregnancy-induced hypertension is characterized by vasospasm, elevated blood pressure, and increased neuromuscular irritability, and these features are common to syndromes of Mg deficiency.[5]

A study on 42 normal pregnant women and 66 preeclamptic (34 with mild and 32 with severe preeclampsia) reported low Mg levels in both mild and severe preeclampsia as compared to normal pregnant women. They attributed that reduction in serum levels of Mg during pregnancy might be a possible contributor in the etiology of preeclampsia.[6]

However, status of Mg at delivery and in cord blood is not known. Hence, the present study was planned to study the role of Mg in preeclampsia and its correlation with dietary intake of proteins and calories by analyzing serum Mg at 20 weeks, delivery, and cord blood Mg levels.

Methods

The present prospective study was conducted at the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynecology, Pt. B. D. Sharma, PGIMS, Rohtak.

The study was conducted in 102 pregnant women aged 17–36 years attending the outpatient department (OPD) before 20 weeks of gestation. These women were followed till delivery. Informed and written consent was obtained from all the women, and the study was approved by the ethical board of the institute.

Women with medical disorders such as hypertension, diabetes mellitus, epilepsy, hyperthyroidism, hypothyroidism, severe anemia, malabsorption syndromes, osteomalacia, and fluorosis were excluded from the study. Calcium and iron supplementation was given to all the pregnant women with a period of gestation >20 weeks. They were asked to take 1000 mg calcium and 100 mg of elemental iron.

The study samples were drawn twice: once before 20 weeks during visit in the OPD and second within 1 h of childbirth. Three milliliters of maternal venous blood was drawn into red vacutainer tubes. Serum was analyzed for Mg levels using spectrophotometric method.[7] SPSS version 23 was used for statistical analysis (It was Randox kit run on autoanalyzer). Results were expressed as mean ± standard deviation and unpaired *t*-test and Pearson's correlation test was applied.

Results

Of 102 pregnant women, only 11 developed preeclampsia on follow-up after 20 weeks. The mean age of normal pregnant women was 25.05 ± 3.04 years and of preeclamptic women was 25.63 ± 1.91 years. The mean calorie intake of normal pregnant women was 2042.85 ± 150.65 kilocalories/day and of preeclamptic women was 1990.90 ± 137.51 kilocalories/day. The mean protein intake for normal pregnant women was 48.89 ± 4.73 g/day and preeclamptic women was 45.81 ± 4.77 g/day.

The mean hemoglobin level was 9.13 ± 0.70 g/dl, the mean thyroid-stimulating hormone was 2.12 ± 0.58 mIU/L, and the mean glucose challenge test was 93.07 ± 14.93 mg/dl [Table 1]. The mean birthweight of baby of normal pregnant women was 2.62 ± 0.45 kg and baby of preeclamptic women was 2.60 ± 0.51 kg. The mean Apgar score for normal group at 1 min was 6.6 ± 0.84 and at 5 min the score was 8.7 ± 0.45; whereas, the mean Apgar score for preeclamptic group at 1 min was 6.18 ± 0.6 and at 5 min the score was 8.27 ± 0.64.

In normal pregnant women, the mean serum calcium level at <20 weeks of gestation was 9.22 ± 0.74 mg/dl, at term was 8.85 ± 0.95 mg/dl, and in cord blood was 8.59 ± 0.89 mg/dl. In preeclamptic women, the mean serum calcium levels at <20 weeks of gestation was 8.16 ± 1.19 mg/dl, at term was 6.40 ± 2.12 mg/dl, and in cord blood was 6.03 ± 1.85 mg/dl.

In normal pregnant women, the mean serum Mg level at <20 weeks of gestation was 2.03 ± 0.34 mg/dl, at term was 1.93 ± 0.41 mg/dl, and in cord blood was 1.84 ± 0.35 mg/dl. In preeclamptic women, the mean serum calcium levels at <20 weeks of gestation was 1.80 ± 0.30 mg/dl, at term was 1.61 ± 0.38 mg/dl, and in cord blood was 1.61 ± 0.41 mg/dl.

The correlation between serum Mg at <20 weeks with preeclamptic women was negative and statistically significant ($r = -0.205$ and $P = 0.039$) [Table 1]. The correlation between serum Mg at term with preeclamptic women was negative and statistically significant ($r = -0.219$, $P = 0.027$) [Table 2].

Discussion

Jafrin *et al*.[5] have reported that the mean serum Mg of pregnant women with pregnancy-induced hypertension was significantly decreased in comparison to that of normal pregnant women. The mean serum level of Mg in normal pregnant group was

Table 1: Hemoglobin, thyroid-stimulating hormone, and glucose challenge test

Parameters	Mean±SD	
	Normal pregnant women	Preeclamptic women
Hemoglobin (g/dl)	9.15±0.71	8.90±2.72
TSH (mIU/L)	2.12±0.60	2.05±0.75
GCT (mg/dl)	94.35±9.19	95.54±7.43

TSH: Thyroid-stimulating hormone, GCT: Glucose challenge test, SD: Standard deviation

Table 2: Correlations between maternal serum magnesium levels and preeclampsia

	Serum magnesium at <20 weeks		Serum magnesium at term	
	Correlation coefficient	*P*	Correlation coefficient	*P*
Preeclamptic women	−0.205	0.039	−0.219	0.027

1.91 ± 0.08 mg/dl and in the group with pregnancy-induced hypertension was 1.75 ± 0.10 mg/dl, indicating that reduction in serum levels of Mg during pregnancy might be a possible contributor in the etiology of pregnancy-induced hypertension and Mg supplementation as diet or drugs may be of value to prevent pregnancy-induced hypertension.[5]

In the present study, Mg supplementation was not given to pregnant women. However, the observed changes in Mg values are in agreement with the previous studies. The correlation between maternal serum Mg and preeclamptic women was negative and statistically significant at term ($r = -0.219$, $P = 0.027$); [Table 2].

Mg sulfate is indicated for the prevention and control of seizures in preeclampsia and eclampsia. Therapeutic levels of Mg have also been found to produce specific placental effects such as vasodilation.[7,8] From an *in vitro* study of human umbilical artery resistance, Mg sulfate was found to exert a relaxant effect on umbilical arterial tone attenuating the vasoconstrictor effect of angiotensin II and endothelin-1 in the fetal–placental vasculature. Mg sulfate used for the treatment of preeclampsia or hypertensive disease in pregnancy may have beneficial effects on the fetoplacental circulation. Prenatal treatment with Mg sulfate may influence calcium homeostasis and nonenzymatic antioxidant reserve in erythrocytes of preterm newborns.[7,8]

Conclusion

The study data supported the hypothesis that Mg deficiency might be the causative factor in the development of preeclampsia. The consequence of the deficiencies of the Mg may be responsible for the clinical manifestations that are observed in preeclampsia and eclampsia patients since both ions are important in cellular and neuronal metabolism as well as cell membrane stability.

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Conflicts of interest

There are no conflicts of interest.

References

1. Ziael S, Ranjkesh F, Faghihzadeh S. Evaluation of 24-hour copper in pre-eclamptic vs. normotensive pregnant and non-pregnant women. Int J Fertil Steril 2008;2:9-12.
2. Tong GM, Rude RK. Magnesium deficiency in critical illness. J Intensive Care Med 2005;20:3-17.
3. Khoushabi F, Shadan MR, Miri A, Sharifi-Rad J. Determination of maternal serum zinc, iron, calcium and magnesium during pregnancy in pregnant women and umbilical cord blood and their association with outcome of pregnancy. Mater Sociomed 2016;28:104-7.
4. Sanders GT, Huijgen HJ, Sanders R. Magnesium in disease: A review with special emphasis on the serum ionized magnesium. Clin Chem Lab Med 1999;37:1011-33.
5. Jafrin W, Mia AR, Chakraborty PK, Hoque MR, Paul UK, Shaha KR, *et al.* An evaluation of serum magnesium status in pre-eclampsia compared to the normal pregnancy. Mymensingh Med J 2014;23:649-53.
6. Leary NO, Pembroke A, Duggan PF. Single stable reagent (Arsenazo III) for optically robust measurement of calcium in serum and plasma. Clin Chem 1992;38:904-8.
7. Idogun ES, Imarengiaye CO, Momoh SM. Extracellular calcium and magnesium in preeclampsia and eclampsia. Afr J Reprod Health 2007;11:89-94.
8. David R, Leitch IM, Read MA, Boura ALA, Walters WAW. Actions of magnesium, nifedipine and clonidine on the fetal vasculature of the human placenta. Aus NZ J Obstet Gynaecol 1996;36:267-71.