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Study of Serum Lipid Profile and Magnesium in Normal Pregnancy and in Pre-Eclampsia: A Case Control Study

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ABSTRACT

The aim was to evaluate changes in serum magnesium and lipid profile in pre-eclamptics, their correlation with each other and roles of these changes in its Pathophysiology. Serum magnesium levels were determined by Calmagite method and total lipid profile by enzymatic calorimetric method in healthy non pregnant women (Group-1), primigravidas with normal pregnancy (Group-2) and primigravidas with pre-eclampsia (Group-3). Results of Group-2 were compared with Group-1 and results of Group-3 were compared with Group-2. Compared with normal pregnancy, in pre-eclampsia the level of TG (Triglycerides) ($p<0.0001$) and VLDL-C (Very Low Density Lipoprotein Cholesterol) ($p<0.0001$), LDL-C (Low Density Lipoprotein Cholesterol) ($p<0.012$) were significantly increased and HDL-C (High Density Lipoprotein Cholesterol) ($p<0.0001$) and Mg^{++} ($p<0.001$) levels were decreased significantly. In normotensive pregnant women TG ($p<0.0001$) and VLDL-C ($p<0.0001$) and HDL-C ($p<0.0001$) levels were high and LDL-C ($p<0.0001$) level was low compared to healthy non pregnant women. No significant change could be observed in serum Mg^{++} in Group-2 as compared to Group-1 ($p<0.2636$). No significant change could be observed in total cholesterol level in any group. In addition serum TG correlated negatively($r = -0.47$) with serum Magnesium in Pre-eclampsia. The inverse correlation between serum Mg^{++} and serum triglycerides in pre-eclampsia may suggest the role of magnesium in the pathogenesis of pre-eclampsia along with dyslipidemia. During pregnancy, detection of hypomagnesaemia and dyslipidemia early will prevent its metabolic complication and in established pre-eclamptic women it will help in better management of the disease.

Key words: Pre-eclampsia, pregnancy induced hypertension, endothelial dysfunction, hypomagnesemia, dyslipidemia

INTRODUCTION

Hypertension, defined by a blood pressure (BP) of 140/90 mmHg or more, affects up to 8-10% of pregnancies (Iribhogbe *et al*., 2011). It includes a spectrum of conditions namely, Pre-eclampsia, Eclampsia, Pre-eclampsia superimposed on chronic hypertension, chronic hypertension and gestational hypertension. Unlike other hypertensive disorders in pregnancy, pre-eclampsia is a multisystem disease. Its distinctive feature being the sudden onset of proteinuria (≥300 mg/24 h urine) (Roberts and Redman, 1993). Being a hypertensive complication of pregnancy, it is associated with significant morbidity and mortality for mother and baby (Walker, 2000; Brown and

Buddle, 1996). Eclampsia is a convulsive form of Pre-eclampsia that affects 0.1% of all pregnancies. Chronic hypertension is diagnosed with BP readings equal to or greater than 140/90 mmHg prior to pregnancy or before the 20th week of gestation. It represents a major risk factor for pre-eclampsia, which affects 25% of these women, in contrast to 5% of women without preexisting hypertension and otherwise normal pregnancies. Gestational hypertension refers to hypertension occurring for the first time during the second half of pregnancy in the absence of proteinuria. It includes women with pre-eclampsia who have not yet developed proteinuria, those with hypertension only, and a subset of patients in whom BP remains elevated after delivery, leading to the diagnosis of chronic hypertension. Although the first two forms of gestational hypertension typically abate with the termination of pregnancy, the third form may lead to chronic hypertension, which is diagnosed when gestational hypertension persists beyond 12 weeks postpartum.

Although hypertension and proteinuria are the simple clinical criteria for diagnosis of pre-eclampsia the pathophysiological mechanisms that lead to the disorder are by all evidence very diverse (Ness and Roberts, 1996; Mushambi and Halligan, 1996). Pre-eclampsia develops in a particular woman following an unfortunate combination of maternal (trophoblast-independent) risk factors and an excessive maternal response to the trophoblast/trophoblast-derived factors. The maternal risk factors include disorders associated with endothelial dysfunction such as chronic hypertension, diabetes, kidney disease and dyslipidemia. Some studies say that there is no correlation between serum lipids in normal pregnancy and Pre-eclampsia (Ness and Roberts, 1996; Redman *et al*., 1999; Punthumapol and Kittichotpanich, 2008a).

Magnesium is the 4th most abundant cation in the body and is present in more than 300 enzymatic systems where it is crucial for ATP metabolism (Byrd and Roy, 2003). Magnesium may influence blood pressure by modulating vascular tone and structure through its effects on myriad biochemical reactions that control vascular contraction/dilation, growth/apoptosis, differentiation and inflammation. Magnesium acts as a calcium channel antagonist. It stimulates production of vasodilator prostacyclins and nitric oxide and alters vascular responses to vasoconstrictor agents. Its deficiency can also play a role in hypertension of pregnancy (Yogi *et al*., 2011). Regarding Mg there are lots of contrast studies. Some studies do not support the hypothesis that low serum magnesium is a risk factor for developing hypertension and vascular dysfunction, whereas very few findings support the hypothesis that hypomagnesaemia is one of possible etiologies of pre-eclampsia (Khan *et al*., 2010; Sukonpan and Phupong, 2005; Punthumapol and Kittichotpanich, 2008b).

While maternal obesity, diabetes mellitus and chronic hypertension are each probable risk factors for Pre-eclampsia, less is known about the relationship between other conditions such as dyslipidemia, hypomagnesaemia and the risk of pre-eclampsia (O'Brien *et al*., 2003; Bryson *et al*., 2003; Ros, *et al*., 1998; Rey and Couturier, 1994).

So the aim of present study was to evaluate the risk of pre-eclampsia in association with maternal lipids and to find out whether hypomagnesaemia is associated with hypertension of pregnancy. We also found out whether there was any correlation between hypomagnesaemia and dyslipidemia.

MATERIALS AND METHODS

Three groups of women were recruited for the study. First group (G1) comprised 25 healthy non-pregnant women taken as controls. Second group (G2) comprised of 20 age and gestational age matched normotensive pregnant women and third group (G3) consisted of 20 pre-eclamptic women who were selected after admission to Obstetrics and Gynecology department, J.L.N. Government Medical College, Ajmer. This research was conducted from 21 June 2010 to 10 November 2010. Pre-eclampsia was defined as development of blood pressure >140/90 mm Hg after 20 weeks gestation and proteinuria of ≥300 mg as confirmed by 24 h urine collection in women with no known history of hypertension, renal disease, endocrine abnormalities and had single pregnancy and had no family history of lipid or carbohydrate disorders (Lampinen *et al*., 2008).

The Pre-eclamptic women had an average blood pressure at admission of 157±4/103±2 mm Hg as measured by standard mercury sphygmomanometer and urinary protein averaged 1.8±0.3 g day^{-1}.

Normotensive non-pregnant and normotensive pregnant women were carefully matched for age with the pre-eclamptic group and had average blood pressure of 116±2/71±2 mm Hg. The women included in the study were 25 to 35 years old. Women with concomitant disease such as diabetes or a history of gestational diabetes, chronic hypertension, and kidney disease or coagulation disorders were excluded. All pregnant women were in the third trimester of pregnancy.

All women gave informed consent to participate in the study, which was approved by the locally appointed ethics committee.

Blood samples were collected from anticubital vein by aseptic techniques following a fast of 12 h for the analysis of lipids (total cholesterol, low density lipoproteins, high-density lipoproteins, and triglycerides) and magnesium. Serum Total Cholesterol was measured by using CHOD-PAP method (Allain *et al*., 1974). Serum triglyceride was measured by using GPO-POD enzymatic colorimetric method (Bucolo and David, 1973). Serum HDL-Cholesterol was measured by using Phosphotungestic acid method (Assmann *et al*., 1983). VLDL-C and LDL-C were calculated according to Friedwald W.T.'s equation. Serum magnesium level was measured by using Calmagite method (Elin, 1991).

STATISTICAL ANALYSIS

The values of laboratory parameters are presented as the Mean±SD. A Student's unpaired t-test was used for cross sectional comparisons of continuous variables between the 2 groups. The association between lipid parameters and magnesium level was analyzed by means correlation test. The correlation coefficient was obtained by the method of least squares. To determine correlation between quantitative variables, Pearson's coefficient of determination was used. For this purpose, Pearson Correlation (v1.0.3) in Free Statistics Software (v1.1.23-r6) was used (Wessa, 2008). The results were considered statistically significant when the probability of the null hypothesis was less than at least 5% ($p<0.05$). The main statistical comparisons were performed between healthy non-pregnant and healthy pregnant women, between healthy pregnant women and pre-eclamptic women.

RESULTS

Women with an uncomplicated course of pregnancy were investigated during routine check-ups in the third trimester of pregnancy. The gestational age at blood sampling in Pre-eclamptic women

Table 1: Comparison of biochemical parameters in Healthy non pregnant women and Normotensive pregnant women

Parameter	Normal range (Mg dL^{-1})	Healthy non pregnant women (Group 1) (n = 25)	Normotensive pregnant women (Group 2) (n = 20)	t-value	p-value
Total cholesterol	140-240	211.88±11.00	218.10±16.45[c]	1.5158	0.13
Triglycerides	25-160	117.28±12.13	215.30±27.32[a]	16.09	0.0001***
HDL-C	35-80	47.96±9.25	59.47±8.54[a]	4.27	0.0001***
VLDL-C	5-32	23.45±2.42	43.06±5.46[a]	16.10	0.0001***
LDL-C	105-135	140.46±14.79	115.56±12.02[a]	6.08	0.0001***
Magnesium	1.59-3.05	2.62±0.33	2.42±0.80[c]	1.1327	0.2636

Values are as Mean±SD. ***Significant at p<0.001, **Significant at p<0.01, *Significant at p<0.0, (a: Significant, p<0.001), (c: Not significant, p>0.05)

Table 2: Comparison of biochemical parameters in Normotensive pregnant women and Pre-eclamptic women

Parameter	Normal range (Mg dL^{-1})	Normotensive pregnant women (Group 2) (n = 20)	Pre-eclamptic women (Group 3) (n = 20)	t-value	p-value
Total cholesterol	140-240	218.10±16.45	236.80±35.32[b]	2.14	0.038*
Triglycerides	25-160	215.30±27.32	275.70±38.35[a]	5.73	0.0001***
HDL-C	35-80	59.47±8.54	45.95±8.03[a]	5.14	0.0001***
VLDL-C	5-32	43.06±5.46	55.14±7.67[a]	5.73	0.0001***
LDL-C	105-135	115.56±12.02	135.71±32.20[b]	2.62	0.012*
Magnesium	1.59-3.05	2.42±0.80	1.55±0.80[a]	3.41	0.001**

Values are as Mean±SD. ***Significant at p<0.001, **Significant at p<0.01, *Significant at p<0.05, (a: Significant, p<0.001) (b: Significant, p< 0.05)

Table 3: Pearson's coefficient of determination between serum Mg^{++} and lipid profile parameters in Pre-eclamptic women

Lipid parameters	Pearson's coefficient of correlation (r)	t-value	p-value
Total cholesterol	-0.14	-0.63	0.53
Triglycerides	-0.47*	-2.26	0.036*
HDL-C	0.182	0.787	0.44
VLDL-C	-0.47*	-2.26	0.036*
LDL-C	-0.09	-0.40	0.68

*Significant at p<0.05

(Group 3) was (33±0.8 week). This matches the gestational age (32±1 week) of normotensive pregnant women (Group 2). In this study we compared the level of various serum lipid parameters and serum magnesium of normotensive pregnant women with the lipid parameters and serum magnesium of healthy non-pregnant women. We also compared the Pre-eclamptic women with normotensive pregnant women for the same parameters.

Mean fasting total cholesterol level in Group 1 (Healthy non pregnant women) was 211.88±11.00 and in Group 2 (Normotensive pregnant women) it was 218.1±16.45. No significant alteration in total cholesterol level could be observed in Group 2 compared to Group 1 (Table 1).

Mean serum Triglyceride (TG) concentration was significantly increased (p<0.0001) in Group 2 (Normotensive pregnant women, 215.3±27.32) compared with Group 1 (Healthy non pregnant women, 117.28±12.13), the mean value being raised almost two folds. Mean serum VLDL-C level also rose significantly (p<0.0001) in normal pregnancy (43.06±5.46) in comparison to healthy non-pregnant women (23.45±2.42). We observed significant decrease in LDL-C

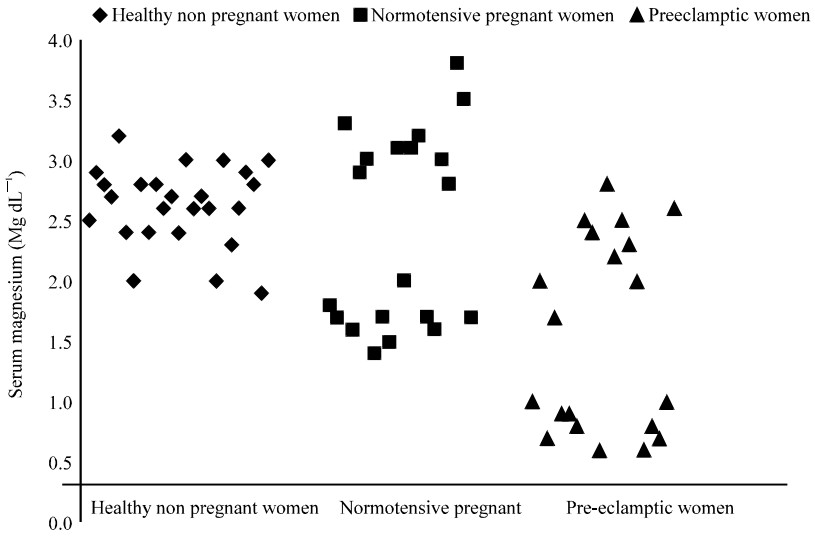

Fig. 1: Scatter plot showing single values of serum Mg^{++} in healthy non pregnant women (n = 25) normotensive pregnant women (n = 20) and Pre-eclamptic women (n = 20)

($p<0.0001$) in normotensive pregnant women (115.56±12.02) compared to healthy non pregnant women (140.46±14.79). In this study we found that the mean value of HDL-C was about 25% higher in the third trimester of normal pregnancy (59.47±8.54) over the non pregnant healthy women (47.96±9.25) and the alteration was statistically significant ($p<0.0001$). No significant fall ($p<0.2636$) in serum Mg^{++} level was found in normal pregnancy in comparison to healthy non pregnant women (Table 1).

Mean fasting total cholesterol level in Group 2 (Normotensive pregnant women) was 218.1±16.45 and in Group 3 (Pre-eclamptic women) it was 236.8±35.32. No significant alteration in total cholesterol level could be observed in Group 3 compared to Group 2. Significant increase in mean serum TG concentration ($p<0.0001$) was found in Group 3 (Pre-eclamptic women, 275.7±38.35) in comparison to group 2 (normotensive pregnant women, 215.3±27.32). Significant increase in LDL-C ($p<0.012$) was observed in Pre-eclamptic women (135.71±32.20) compared to normotensive pregnant women (115.56±12.02). Significant decrease in HDL-C level ($p<0.0001$) was observed in Pre-eclamptic women (45.95±8.03) compared to the normotensive pregnant women (59.47±8.54).There was a significant fall ($p<0.001$) in serum Mg^{++}level in Pre-eclamptic women (1.55±0.80) compared to the normotensive pregnant women (2.42±0.80) (Table 2).

Figure 1 shows the single values of serum Mg^{++} in healthy non pregnant women (n = 25) normotensive pregnant women (n = 20) and Pre-eclamptic women (n = 20).

The evaluation of correlation of serum Mg level with various lipid parameters in Pre-eclamptic women using linear correlation evaluation methods, showed converse linear statistically significant correlation between serum Mg level and TG level and VLDL levels (r = -0.47) (Fig. 2). No significant correlation could be observed between serum Mg level and other lipid parameters (Table 3).

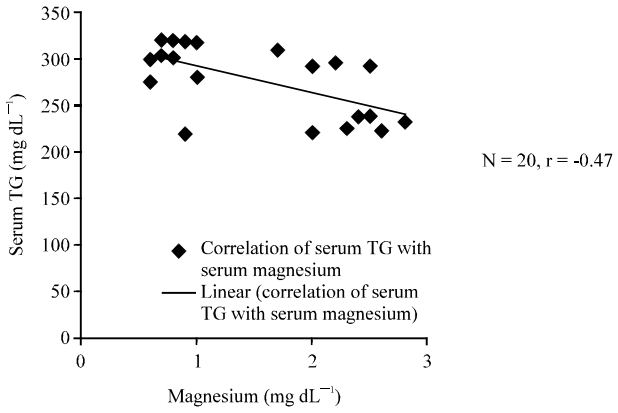

Fig. 2: Graphical presentation of negative correlation between serum magnesium and serum TG in Pre-eclamptic women

DISCUSSION

In present study we could not find any significant increase in total cholesterol level in any of the groups. These findings are similar to some previous studies (Sattar *et al*., 1997; Jayanta *et al*., 2006). Sahu *et al*. (2009) observed significant rise in the fasting triglycerides, total cholesterol and LDL-C levels in PIH ($p<0.0001$) compared to pregnant women in their third trimester of pregnancy. Osadolor *et al*. (2005) also observed that there were significant increases in serum T-CHOL, ($p<0.05$), TG ($p<0.01$) and LDL-C ($p<0.05$) in hypertensives relative to normotensive controls.

As described above, in present study significant increase in TG was observed in both normotensive pregnant women and Pre-eclamptic women. Many other studies showed the same results (Jayanta *et al*., 2006; Chiang *et al*., 1995; Howlader *et al*., 2007). But Turpin *et al*. (2008) in their study found that serum Triglycerides level decreases in preeclampsia compared to normotensive pregnant women. Significant modulators of lipoproteins during pregnancy include insulin, oestrogen, lipoprotein lipase (LPL) (Silliman *et al*., 1994). In some studies lowering of lipoprotein lipase (LPL) was shown to increase serum TG level in normal pregnancy (Herrera *et al*., 1988). Other studies have shown that the principle modulator of this hypertriglyceridemia is oestrogen as pregnancy is associated with hyperoestrogenaemia. Oestrogen inhibits the hepatic lipid oxidation so the net effect is increased delivery of free fatty acids into hepatic biosynthesis of endogenous triglycerides which is carried by VLDL (Jayanta *et al*., 2006; Silliman *et al*., 1994; Alvarez *et al*., 1996). However, hypertriglyceridemia in pre-eclampsia is probably not due to hyperoestrogenaemia as the levels of oestrogen decrease in pre-eclampsia.

Some studies have concluded that hypertriglyceridemia may be modulated by hyperinsulinism found in pregnancy (Adegoke *et al*., 2003). Insulin resistance and resultant hyperinsulinemia are characteristic of normal pregnancy and are maximal in the third trimester. This is probably mediated by several hormonal changes, including elevations in levels of human placental lactogen; progesterone, cortisol, and estradiol (Barbieri, 1999). Many of markers, which correlate with insulin resistance likewise, vary over the course of pregnancy. For example, levels of triglycerides, small dense LDL particles, and free fatty acids increase as normal pregnancy and associated insulin resistance progress (Belo *et al*., 2002).

In pregnancies complicated by hypertension, there appears to be an exaggeration of insulin resistance and associated metabolic changes. In a report, Pre-eclamptic women were more insulin resistant than normotensive controls, so in pre-eclampsia TG level further increases due to the exaggeration of insulin resistance (Kaaja *et al*., 1999). Increased TG, found in Pre-eclampsia, is likely to be deposited in predisposed vessels, such as the uterine spiral arteries and contributes to the endothelial dysfunction, both directly and indirectly through increased generation of small, dense LDL (Sattar *et al*., 1997). It was indicated that small dense LDL had a greater capacity to stimulate thromboxane synthesis by endothelial cells thereby causing vasoconstriction (Weisser *et al*., 1993). Small dense LDL are also more susceptible to oxidative modifications forming peroxides which inhibit EDRF (Endothelium Derived Relaxation Factor) and also leads to foam cell formation of deciduas (Krauss, 1997; Pierucci *et al*., 1996). Study by Uboh *et al*. (2008) supports this hypothesis as they observed increased levels of free radical products of lipid peroxidation (Malondialdehyde) in pre-eclamptic condition compared to normotensive pregnant women and healthy non pregnant women. Oxidized LDL also impairs endothelial function by expression of adhesion molecules, inhibition of endothelial prostacyclin synthesis, increased endothelin production and release and increased platelet aggregability (Vogel, 1999).

In present study we found that LDL-C level decreases in normal pregnancy which can be explained by increased oestrogen levels in pregnancy. In pre-eclampsia LDL-C level increased compared to normal pregnancy which can be due to decreased level of oestrogen in pre-eclampsia (Bradley and Crook, 1995). A significant increase in LDL-C in third trimester of gestational hypertensive disorders was also reported in many studies (Gratacos *et al*., 2003; Wakatsuki *et al*., 2000).

VLDL-C level increased in present study in both normal pregnancy and pre-eclampsia. Same was reported in other studies. Another study concluded that VLDL-C level might rise up to 2.5 folds at term over the pre-pregnancy level. It is due to hypertriglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into the blood circulation (Herrera *et al*., 1988; Teichmann *et al*., 1988). We also found that HDL-C level increases in normal pregnancy compared to non-pregnant women and in pre-eclamptic women HDL-C levels decreases compared to normotensive pregnant women. Other studies also reported lesser quantities of serum alpha lipoprotein fraction in women with pre-eclampsia in the third trimester of pregnancy (Sattar *et al*., 1997; Enquobahrie *et al*., 2004). Whereas Turpin *et al*. (2008) in their study found that serum HDL-C level increases in preeclampsia compared to normotensive pregnant women.

According to some studies oestrogen is responsible for induction of TG and HDL and suppression of serum LDL (Krauss, 1997). So in normotensive pregnant women the increase in HDL-C and decrease in LDL-C can be explained by hyperoestrogenemia. In Pre-eclampsia the oestrogen level decreases so reduced HDL-C and raised LDL-C levels may be due to hypoestrogenemia. This reduced HDL-C level may also contribute to the reduced prostacyclin level seen in pre-eclampsia (Kaaja *et al*., 1995). Lower serum HDL-C level may also reduce antioxidative protection for other lipoproteins (Mackness *et al*., 1995).

In the present study, we were unable to find any differences in serum Mg_{++} in normotensive pregnant women compared with healthy non-pregnant women but we found decreased serum Mg^{++} level in pre-eclamptic women compared to both normotensive pregnant women and healthy non-pregnant women. In some studies the serum magnesium in PIH decreased significantly

($p<0.01$) and the decrease of magnesium concentration may be one of the important factors responsible for the pathophysiological changes of PIH (Qi *et al*., 1997; Borekci *et al*., 2009). But in some studies there was no difference in serum magnesium among normal pregnancy and Pre-eclampsia (Punthumapol and Kittichotpanich, 2008a; Kumru *et al*., 2003).

According to one study, in normal pregnancy hemodilution effect of oestrogen and increased demand of fetus decreases the serum magnesium level and in pre-eclampsia urinary excretion of magnesium also increases so the level decreases further (Kesteloot, 1984). Magnesium deficiency causes hemodynamic abnormalities such as arterial wall thickening, abnormal vascular tone and endothelial dysfunction which are due to alteration in the biology of cellular and non cellular components of arterial wall. There may be a causal relationship between hypomagnesaemia and pre-eclampsia since magnesium is involved in blood pressure regulation through an intracellular inhibition of NO synthase in endothelial cells (Sanders *et al*., 1999). In magnesium deficiency, the production of ATP and ATP dependent sodium / potassium and calcium pump are also impaired, providing another hypothesis to unify the clinical thinking about pre-eclampsia (Newman and Amarasingham, 1993).

Previous studies mainly focused on separate study of lipid profile and magnesium in pre-eclampsia. There are no studies on correlation of lipid profile and magnesium in pregnancy induced hypertension. In a study, statistically significant inverse correlation was observed between intralymphocyte free Mg_i and plasma triglycerides in essential hypertensive subjects ($r = -0.521$, $P = 0.002$) (Pietro *et al*., 1996). This study focused on ionized Mg_i in hypertensive subjects rather than total Mg in hypertensive pregnant women, thus preventing direct comparison with our data.

In present study we found statistically significant inverse correlation between serum Mg and triglycerides in pre-eclamptic women ($r = -0.47$) who also had elevated levels of LDL-C, TG, VLDL-C and low HDL-C. So we suggest that a low serum Mg level might in some way be linked to dyslipidaemia thus explaining the pathogenesis of pre-eclampsia. The relationship between total magnesium and plasma lipids, although imperfectly understood, has been known for some time (Pietro *et al*., 1996; Rayssiguier, 1986).

Due to insufficient data, we cannot account for the association between serum triglycerides and serum Mg^{++}.

In a study, ischemic heart disease patients were treated with oral magnesium for 3 months. Interestingly plasma triglycerides decreased in them (Rasmussen *et al*., 1989).

In another study, hyperlipidemic patients of Frederickson types IV and IIb were supplemented with oral physiological magnesium in addition to the usual dietary measures.

It was found to reduce serum triglycerides (values, Mean±SD, decreased from 198.17±47.01 to 163.20±40.55 mg dL^{-1}, $p<0.05$) (Kisters *et al*., 1993). Furthermore, an important feature of hyperlipidemia associated with magnesium deficiency in experimental animal models is the accumulation of triglyceride-rich lipoproteins (Altura *et al*., 1990).

CONCLUSION

So we can hypothesize that intake of magnesium supplementation in pregnancy can improve the lipid status and decrease the risk of developing pre-eclampsia. In pre-eclampsia oral intake of magnesium supplementation can decrease the level of pathological lipids (mainly TG) and their role in the pathogenesis of pre-eclampsia. But further studies are needed to prove this hypothesis. If

proven it will really be helpful to lower the incidents of this disease and early detection of these parameters is going to aid in better management of pre-eclampsia.

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