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Serum Gamma Glutamyl Transferase (GGT) Level in Preeclampsia

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ABSTRACT

Background: *Preeclampsia is a major cause of maternal and perinatal mortality and morbidity. The intracellular enzyme, Gamma Glutamyl Transferase (GGT) is widely distributed throughout the body in many tissues. As severe preeclampsia may lead to a numerous multisystem complications, estimation of serum GGT level can be used to predict the severity of preeclampsia.The aim of this study wasto assess serum GGT level in preeclamptic women and its comparison with normal pregnant women in third trimester.*

Materials and methods: *The cross sectional analytic study was carried out in the Department of Physiology, Dhaka Medical College (DMC) Dhaka from January 2014 to December 2014. Seventy preeclamptic women and thirty five normotensive pregnant controls were selected.Preeclamptic group was further divided into two subgroups, mild (n=35) and severe (n=35) preeclamptic women. Both the groups were in their third trimester (28-40 weeks) of pregnancy and of same age (18-35 years).*

Results: *The mean serum GGT levels (In mild preeclamptic = 28.4±6.7 U/L & in severe preeclamptic= 63.1±45.9 U/L) were significantly higher in mild and severe preeclamptic women than those of normal pregnant women(in normal pregnant women = 14.8±4.6 U/L).Again, this value was significantly elevated in severe preeclamptic women than mild preeclamptic women.*

Conclusion: *From this study, it can be concluded that serum GGT level is elevatedin patients of preeclampsia. So, measurement of serum GGT level in preeclamptic women may reflects the severity of disease and helps to provide appropriate treatment to the patient.*

Key words: Preeclampsia; Blood pressure; Serum GGT.

Introduction

Preeclampsia is specific to human pregnancy and presents with hypertension, proteinuria and/or edema after 20 weeks of gestation[1]. In the absence of proteinuria, preeclampsia is suspected in pregnant women if the hypertension is associated with headache, visual disturbances, abdominal pain or rapid weight gain[2].

Preeclampsia has been classified by clinical severity in to mild and severe forms. Mild preeclampsia is defined as new onset of hypertension (Systolic blood pressure of > 140 to < 160 mmHg or a diastolic blood pressure > 90 to ≤ 110 mmHg) after 20 weeks of pregnancy with or without proteinuria (> 0.3gm to < 5gm/day). Preeclampsia is considered as severe when blood pressure is markedly elevated (≥160 mmHg/110 mmHg) and associated with significant proteinuria (> 5gm/ day or ≥ 2+ on dipstick) or evidence of other organ dysfunctions[3-4].

Preeclampsia can occur in about 2-8% of all pregnancies and can be devastating and life threatening for both mother and fetus[5]. In developing countries, the rate of maternal deaths from preeclampsia and eclampsia is as high as 15% where as 1.8% in developed countries[6]. The increased incidence of perinatal morbidity and mortality seen in preeclampsia is primarily due to the need for premature delivery and uteroplacental insufficiency[4]. Placental abruption, Intrauterine Growth Restriction (IUGR) and Intrauterine Fetal Death (IUD) are some fetal complications from preeclampsia. Some acute maternal complications of preeclampsia are eclampsia, pulmonary oedema, Disseminated Intravascular Coagulation (DIC) HELLP (Hemolysis, Elevated Liver Enzymes, Low Platelets) syndrome, acute renal failure and death. Long term effects on mother include chronic hypertension, diabetes mellitus, coronary artery disease, neurological deficit and premature death etc[6].

The exact mechanisms which lead to preeclampsia are unknown. The primary pathophysiology in preeclampsia is proposed due to placental hypoxia with consequent endothelial cell injury[7]. Endothelial cells dysfunction can contribute to inappropriate vasoconstriction, platelet aggregation,

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activation of the coagulation system and ultimately decreased blood flow to organs by microthrombi formation[8-9]. Since preeclampsia is a syndrome, it virtually affects all maternal organ system including liver, kidneys, brain, clotting system and primarily the placenta. Gamma Glutamyl Transferase (GGT) is a microsomal enzyme which is widely distributed in many tissues throughout the body, particularly in liver[1,8]. It acts significantly at the cellular level both in endothelium and epithelium. So, an elevated level of serum GGT indicates both endothelial and cellular damage. A biochemical marker of endothelial damage which might predict the severity of the diseases in preeclampsia would be useful clinically to avoid adverse maternal and fetal outcome[10]. Some authors suggested that serum GGT level can be used as potential marker to predict the severity of preeclampsia[1].

In many studies, it was found that serum GGT level was significantly raised among mild and severe preeclamptic subjects as compared to normotensive controls[1,8,11-12]. On the contrary, some authors observed no significant change of serum GGT level in between preeclamptic and normal pregnant women[13].

Therefore the study was done to observe the changes of serum GGT level in preeclamptic women and to compare it with normal pregnant women in third trimester.

Materials and methods

The cross sectional analytic study was carried out in the Department of Physiology, Dhaka Medical College (DMC) Dhaka from January 2014 to December 2014. Ethical permission was taken from Ethical Review Committee of Dhaka Medical College.A total number of 70 pregnant women with preeclampsia and 35 healthy normotensive pregnant controls were selected from Department of Obstetrics & Gynecology of DMC Hospital. Preeclamptic group was further divided into two subgroups mild (n=35) and severe (n=35) preeclamptic women[3-4]. Both the groups were in their third trimester (28-40 weeks) and of same age (18-35 years). Cases with any medical history of preexisting hypertension, diabetes, renal disease, thyroid disease or liver disease were excluded from the study. Detailed clinical and anthropometric data was recordedby using a prefixed questionnaire.

The results were expressed as means ± SD and compared by applying one way ANOVA test, unpaired Student's 't' test. Procedure of estimation of serum GGT level: With all aseptic precaution, 5ml of venous blood was collectedfrom all cases from antecubital vein. The blood was allowed to clot and serum was separated for the estimation of serum GGT levelby continuous spectrophotometric method[14]

Results

There were no significant differences among the three groups in respect of age and Body Mass Index (BMI). The mean gestational weeks of severe preeclamptic women was significantly lower than mild preeclamptic ($p < 0.05$) and normal pregnant ($p < 0.001$) groups. But there was no significant difference between mild preeclamptic and normal pregnant in respect of this value (Table I).

Table I : Demographic data of the three groups (n=105).

Groups	Age (Years)	BMI (kg/m^2)	Gestational weeks
Normal pregnant (n=35)	25.08±5.39	29.13±3.29	35.6±2.80
Mild preeclamptic (n=35)	26.09±4.58	30.05±3.92	34.3±3.38
Severe preeclamptic (n=35)	25.17±5.03	30.34±2.73	32.6±3.46$^{***, \#}$

Results are expressed as Mean ± SD.

(***$p < 0.001$, when compared to normal pregnant and #$p < 0.05$, when comparison was done between mild preeclamptic and severe preeclamptic women). n = Number of subjects.

In this study, the mean systolic blood pressure and diastolic blood pressure were significantly higher ($p<0.001$) in mild preeclamptic and severe preeclamptic women in comparison to those of their control group. Again, these value were also significantly higher ($p<0.001$) in severepreeclamptic women than that of mild preeclamptic women (Figure 1).

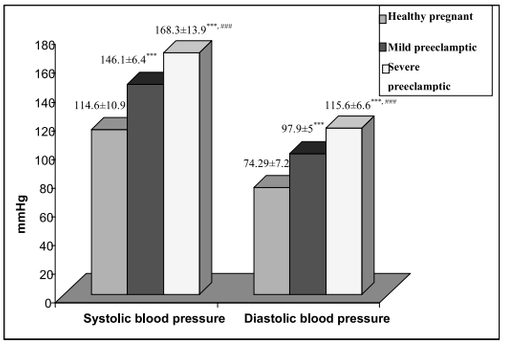

Figure 1: Mean blood pressure of the three groups (n=105).

Results are expressed as Mean ± SD. (***$p < 0.001$, when compared to normal pregnant and ###$p < 0.001$, when comparison was done between mild preeclamptic and severe preeclamptic women). n = Number of subjects.

Figure 2, showing that the mean serum GGT levelwas significantly higher ($p<0.001$) in mild preeclamptic and severe preeclamptic women than that of normal pregnant women. Again, the value was also significantly higher ($p<0.001$) in severe preeclamptic women than that of mild preeclamptic women.

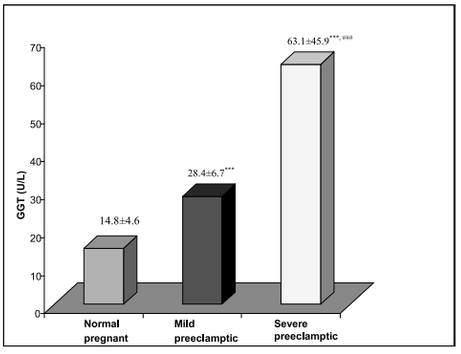

Figure 2: Mean serum GGT level of the three groups (n=105).

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Results are expressed as Mean ±SD. (***p < 0.001, when compared to normal pregnant and ###p < 0.001, when comparison was done between mild preeclamptic and severe preeclamptic women). n = Number of subjects.

Discussion

In this study, the mean serum GGT level was significantly higher (p< 0.001) in preeclamptic women than that of healthy pregnant women. Serum GGT level was also significantly higher (p<0.001) in severe preeclamptic women than that of mild preeclamptic women. This finding was in agreement with the study of many researchers of different countries[1,8,10-11,14,15].

On the other hand, no significant changes of serum GGT level in between preeclamptic and normal pregnant women were found by Delic & Stefanovic[13].

In preeclampsia, there occurs abnormal trophoblastic implantation of placenta that ultimately causes reduced placentalperfusion. Decreased blood supply to the placenta results in production and release of different placental factors into the maternal circulation.These factors act on endothelial cells and leading to endothelial dysfunction[7]. This altered endothelial function results in imbalance of vasoconstrictor and dilator agents and also the endothelial-platelet dysfunction leads to alteration of haemostasis process. These ultimately results in intense maternal vasospasm, adhesion and aggregation of platelets and activation of the coagulation process. Subsequently, there is hypoxic damage to the maternal all organ system[16-17]. Some authors suggested that high serum GGT level in preeclampsia is found due to this widespread endothelial injury including within the uteroplacental circulation and hepatobiliary system[1,10,15,18].

In the present study, increased serum GGT level in preeclamptic women was found than normal pregnant women may be due to these facts.

Conclusion

From the results of this study, it can be concluded that serum GGT level is elevated in women withpreeclampsia than that of women normal pregnancy. Therefore, measurement of serum GGT level may reflect the severity of preeclampsia and helps to provide appropriate treatment to ensure a satisfactory outcomefor mother and fetus.

Disclosure

All the authors declared no competing interests.

Reference

1. Munde SM, Hazari NR, Thorat AP, Gaikwad SB, Hatolkar VS. Gamma glutamyltransferase and Lactate dehydrogenase as biochemical markers of severity of preeclampsia. Int J Med Health Pharm Biomed Eng. 2014; 8(1): 50-53.

2. Page NM. The endocrinology of pre-eclampsia. ClinEndocrinol. 2002; 57: 413–423.

3. Pennington KA, Schlitt JM, Jackson DL, Schulz LC, Schust DJ. Preeclampsia: multiple approaches for a multifactorial disease. Dis models & mech. 2012; 5: 9-18.

4. Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ. Maternal preeclampsia and neonatal outcomes. J Pregnancy. 2011; 2011: 1- 7.

5. Nosrat BS, Azarhoosh R, Borghei A, Sedaghati M, Besharat S, Ghaemi E. Serum level of lactate dehydrogenase, homocystein, hemoglobin and platelet in preeclampsia. Pak J Med Sci. 2011; 27(5): 1014-1017.

6. Staff AC, Benton SJ, Dadelszen PV, Roberts JM, Taylor RN, Powers RW, D. Stephen Charnock-Jones and Christopher W.G. Redman. Redefinining preeclampsia using placenta-derived biomarkers. Hypertens. 2013; 61: 932-942.

7. Duley L. Pre-eclampsia and the hypertensive disorders of pregnancy. Br Med Bull. 2003; 67: 161–176.

8. Sarkar PD, Sogani S. Evaluation of serum lactate dehydrogenase and gamma glutamyltransferase in preeclamptic pregnancy and its comparison with normal pregnancy in third trimester. Int J Res Med Sci. 2013; 1(4): 365-368.

9. Wagner LK. Diagnosis and management of preeclampsia. Am Fam Physician. 2004; 70(12): 2317-2324.

10. Churchill D, Kilby MD, Bignell A et al. Gamma-glutamyltransferase activity in gestational hypertension. Br J ObstetGynaecol. 1994;101:251-253.

11. Babu R, Venugopal B, Sabitha K, Ravikiran BS, Reddy EP. Comparative study of liver and kidney biochemical parameters in normal and pre-eclamptic gestation. J curr trends clin med lab biochemistry. 2013; 1(3): 26-30.

12. Girling JC, Dow E, Smith JH. Liver function tests in preeclampsia: Importance of comparison with a reference range derived for normal pregnancy. Br J ObstetGynaecol. 1997;104:246-250.

13. Delic R, Stefanovic M. Optimal laboratory panel for predicting preeclampsia. J Matern Fetal Neonatal Med. 2010; 23(1): 96–102.

14. Schumann G, Bonora R, Ceriotti F, Ferard G, Ferrero CA, Franck PFH, Gella FJ, Hoelze W, Jorgensen PJ, Kanno T, Kessner A, Klauke R, Kristiansen N, Lessinger JM, Linsinger TPJ, Misaki H, Panteghini M, Pauwels J, Schiele F, Schimmel HG, Weidemann G, Siekmann L. IFCC primary reference procedures for the measurement of catalytic activity concentrations of enzymes at 37°C. ClinChem Lab Med. 2002; 40(6): 643–648.

15. Udenze IC, Arikawel AP, Azinge EC, et al. Liver function tests in Nigerian women with severe preeclampsia. J Clin Sci. 2014;11(1): 7-11.

16. Var A, Yildirim Y, Onur E, Kuscu NK, Uyanik BS, Goktalay K, Guvenc Y. Endothelial dysfunction in preeclampsia. GynecolObstet Invest. 2003; 56: 221-224.

17. Andrews L, Haidas N, Vaishnav S, et al. Biochemical & hematological investigations in pregnancy induced hypertension. J Cell Tissue Res. 2012;12(1):3009-3013.

18. Gruccio S, Di Carlo MB, Pandolfo M, Cruza GS, Touzona MS, Negria G, Giulianob R, Vegab HR, Blancoc MV, Perazzi BE. Biochemical profiling study in umbilical cord blood as predictors of neonatal damage. Int J ClinPediatr. 2014; 3(1): 5-11.