

Original Article

Serum level of Lactate dehydrogenase, Homocystein, Hemoglobin and platelet in preeclampsia

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ABSTRACT

Objectives: Pre-eclampsia affects approximately 5-8% of pregnant women. The aim of this study was to compare the serum level of Lactate dehydrogenase (LDH), Homocystein, Hemoglobin and platelet in pregnant women diagnosed as pre-eclampsia and a normal group in Gorgan city, Northeastern Iran from 2007-2008.

Methodology: In this case control study, 50 cases of pre-eclampsia were compared with the control group women hospitalized in Dezyani hospital. Pre-eclampsia criteria were: Blood pressure more than or equal to 140/ 90 mm hg and Proteinuria greater or equal to 300 mg/ 24 hours urine sample in the third trimester. Hemoglobin, platelet, LDH and hemocystein were measured. Data were analyzed by the mean of SPSS-14 program & Chi-2 or t-student were used.

Results: The difference of BMI and family incomes was significant between two groups (P-value<0.01). LDH level was not statistically different between healthy and pre-eclamptic individuals. Six cases (12%) in controls and 9 cases (18%) in pre-eclamptic group had thrombocytopenia (P-value>0.01). Hemocystein level was more than normal range in five patients with pre-eclampsia (P-value<0.001).

Conclusions: In this study, hemocystein level was significantly higher in pre-eclampsia patients but LDH, hemoglobin and platelet level had no significant difference.

KEY WORDS: Hemocystein, Lactate Dehydrogenase (LDH), Hemoglobin, Platelet, Preeclampsia, Eclampsia, Proteinuria.

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INTRODUCTION

Mother's body faces a great cardiovascular and metabolic challenge during pregnancy. One of the most prevalent events is increasing blood pressure. Hypertensive disorders which affect 10% of all pregnancies and contribute greatly to maternal and perinatal mortality throughout the world, includes a wide spectrum of conditions, including pre-eclampsia and eclampsia, pre-eclampsia superimposed on chronic hypertension, chronic hypertension, and gestational hypertension.¹

Preeclampsia can be devastating and life-threatening for both mother and fetus. Overall, 10% to 15% of maternal deaths are associated with preeclampsia and eclampsia. In developing countries, it is much higher in some parts of Africa and Asia compared to Western nations.^{2,3}

Pre-eclampsia affects approximately 5–8% of pregnant women. Maternal death rates from pre-eclampsia have been significantly reduced by careful patient management in the developed world, but not in developing countries, which account for 99% of total annual global maternal deaths.⁴

This disease is a especial situation that occurs only in human pregnancy and presents with hypertension and proteinuria after 20 weeks of pregnancy. The main cause of preeclampsia is unknown, however, abnormal placentation is thought to be responsible to an inflammatory-type response with endothelial dysfunction. Different etiologies have been known in preeclampsia include immunologic factors, genetic, nutrition, race, increased insulin resistance, oxidative stress and imbalance of prostaglandins oxidative stress by free radicals. Different markers are determined for the early diagnosis of pre-eclampsia like kidney and liver markers, vascular function markers (like homocystein clotting system, fibrinolytic platelet,...), etc.⁵

Early onset disease would be result of a poor early placentation and late onset pre-eclampsia is originated from exaggerated systemic inflammatory response such as predisposing cardiovascular or metabolic risks for endothelial dysfunction. Prevention and prediction are still not possible due to the unknown origin, and preventing maternal morbidity (eg, eclampsia) and mortality could be done via clinical management.³

The most common hematologic disorder in pre-eclampsia is thrombocytopenia (platelet count < 100,000/mm³) with unknown mechanism. Some authors have concluded that lactic dehydrogenase (LDH) is a useful biochemical marker that reflects the severity and occurrence of complications in pre-eclampsia.^{5,6} LDH and platelet count is found to be useful in predicting the progression of severe preeclampsia (HELLP syndrome).⁷ Amburgey et al reported that in preeclamptic women, maternal hemoglobin concentration is significantly elevated prior to delivery and found a statistically significant inverse correlation to birth weight percentile of the newborns.⁸

The aim of this study was to compare the serum level of Lactate dehydrogenase (LDH), Homocystein, Hemoglobin and platelet in pregnant women diagnosed as pre-eclampsia and a normal group.

METHODOLOGY

In this case control study, 50 cases of pre-eclampsia were selected from women hospitalized

in Dezyani hospital, Gorgan city, Northeastern Iran from 2007-2008. For the control group, fifty healthy pregnant women were matched about age and parity. Pre-eclampsia criteria were as followings: 1-Blood pressure more than or equal to 140/ 90 mm hg and 2- Proteinuria greater or equal to 300 mg/ 24 hours urine sample in third trimester.

Demographic data was gathered in a checklist. Verbal informed consent was taken from all participants. Blood samples were tested for hemoglobin, platelet, LDH, homocystein and cratinine. Creatinine was measured with photometry. Measurement of LDH was done with PARS AZMOON kit with DJKC method and based on the conversion of pyroate to lactate and photomett clinic II that the enzyme was evaluated with kinetic method. Homocystein was measured by Axis-shield-kit ELISA method. Data were analyzed by the mean of SPSS-14 program and Chi-2 or t-student were used for the relation between variables.

RESULTS

The mean age was 26.8± 4.67 years and 26.4± 4.62 years in pre-eclampsia group and controls, respectively. Number of abortion and history of it was not significant between the two groups. In both groups 50% were anemic (Hemoglobin<9ng/dl).

LDH level was not statistically different between healthy and pre-eclamptic individuals. Six cases (12%) in controls and 9 cases (18%) in pre-eclamptic group had thrombocytopenia but it was not statistically significant. Homocystein level was more than normal range in five patients with pre-eclampsia (P-value <0.001). Table-I

Table-I: Mean (± SD) of demographic data and measured laboratory tests in pregnant healthy women compared to preeclamptics.

Variables	Pre-eclamptics	Healthy
Age (years)	26.8± 4.67	26.4± 4.62
Parity	0.96± 1.21	0.8± 1.1
Gravid	2.1± 1.26	2.06± 1.23
BMI (kg/m ²)	30.03± 6.2	27.21± 3.76
Hb (ng/dl)	11.01± 1.49	10.92± 0.9
Plt (/mm ³)	203820± 53151.18	196940± 47857.53
LDH (U/L)	230.92± 112.87	187.06± 79.08
Homocystein (µmol/L)	11.23± 4.46	5.47± 2.65

Table-II: Comparing the income level in pregnant healthy women and preeclampsics.

<i>Income level</i>	<i>Less than 150 \$</i>	<i>150-300 \$</i>	<i>More than 300 \$</i>	<i>Total</i>
<i>Group</i>				
Pre-eclampsics	18 (36%)	31 (62%)	1 (2%)	50 (100%)
Healthy	35 (70%)	14 (28%)	1 (2%)	50 (100%)

Differences between the family incomes were statistically significant (P -value <0.01), in pre-eclampsia group most of the patients were median income. Table-II

Mean of BMI was 30.03 ± 6.2 kg/m² and 27.21 ± 3.76 kg/m² in pre-eclampsia and control group, respectively. Number of patients in pre-eclampsia group was increased with increasing BMI and the difference was significant between two groups (P -value <0.01). Table-III.

DISCUSSION

In the present study, homocystein level was significantly higher in pre-eclampsia patients and all high homocystein levels were seen in severe preeclampsia but LDH level had no significant difference. Mean LDH levels were higher in our preeclamptic women, may be because of the cellular damage due to preeclampsia that release LDH enzyme.

Braekke et al in 2007 and Makedos et al in 2007 had the same results but Fernandes et al in 2005 did not find significant relation between homocystein concentration and preeclampsia.¹⁰⁻¹² Ingee et al in 2005, showed that homocystein concentration in plasma increased in severe preeclampsia and eclampsia.⁹⁻¹²

Our results showed that BMI > 30 could be one of the factors that had significant relation with increasing risk of preeclampsia. Other studies showed that patients with BMI >30 had increased risk of pre-eclampsia.¹³ So, it should be considered as a modifiable risk factor in the progression of preeclampsia and preventing programs should be scheduled for all at risk women.

As shown in Table-II, a significant relationship was reported between family income and pre-eclampsia incidence. Some researchers concluded that pre-eclampsia incidence is lower in better so-

cioeconomic condition¹⁴ but in this study we didn't have such finding. We recorded income by asking patients and may be it is not a proper way for evaluating the income. Further studies are needed.

In our study, half of the women were anemic in both groups thus no difference was seen. Phaloprakarn et al concluded that women with higher hemoglobin level had more pre-eclampsia risk¹⁵ and maternal hemoglobin concentrations are significantly elevated prior to delivery in pre-eclampsia⁸, but our reports did not show this result.

Hence it could be concluded that some modifiable risk factors such as BMI and family income are among the important factors which could significantly affect the pregnancy via occurrence of pre-eclampsia and there are serum factors like homocystein which is higher in those suffered from pre-eclampsia. So it could be suggested to pay more attention to BMI of pregnant women, before and during the pregnancy and also evaluate homocystein as a probable marker of pre-eclampsia in further large studies.

Limitations: It was not planned to follow up all patients and we were not aware of their medications.

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REFERENCES

1. Fabry IG, Richart T, Chengz X, Van Bortel LM, Staessen JA. Diagnosis and treatment of hypertensive disorders during pregnancy. *Acta Clin Belg* 2010;65(4):229-36.
2. Jim B, Sharma S, Kebede T, Acharya A. Hypertension in Pregnancy: A Comprehensive Update. *Cardiology in Review* 2010;18:178-189.

Table-III: Comparing BMI in pregnant healthy women and pre-eclampsics.

<i>BMI</i>	<i>Less than 19.6 kg/m2</i>	<i>19.6-24.9 kg/m2</i>	<i>25-30 kg/m2</i>	<i>30-35 kg/m2</i>	<i>> 35 kg/m2</i>	<i>Total</i>
<i>Group</i>						
Pre-eclampsics	1 (100%)	7 (31.8%)	16 (38.1%)	21 (75%)	5 (71.4%)	50 (100%)
Healthy	0	15 (68.2%)	26 (61.9%)	7 (25%)	2 (28.6%)	50 (100%)
Total	1 (100%)	22 (100%)	42 (100%)	28 (100%)	7 (100%)	100 (100%)

3. Steegers EAP, Daddelen PV, Duvekot JJ, Pijnenborg R. Preeclampsia. *Lancet* 2010;376:631-44.
4. James JL, Whitley GS, Cartwright JE. Pre-eclampsia: Fitting together the placental, immune and cardiovascular pieces. *J Pathol* 2010;221:363-378.
5. 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 preeclampsia. *Med Sci Monit* 2005;11(8):CR393-7.
6. Kay HH, Zhu S, Tsoi S. Hypoxia and lactate production in trophoblast cells. *Placenta* 2007;28(8-9):854-60.
7. Hupuczi P, Nagy B, Sziller I, Rigó B, Hrubby E, Papp Z. Characteristic laboratory changes in pregnancies complicated by HELLP syndrome. *Hypertens Pregnancy* 2007;26(4):389-401.
8. Amburgey OA, Ing E, Badger GJ, Bernstein IM. Maternal hemoglobin concentration and its association with birth weight in newborns of mothers with preeclampsia. *J Matern Fetal Neonatal Med* 2009;22(9):740-4.
9. Ingec M, Borekci B, Kadanali S. Elevated plasma homocysteine concentrations in severe preeclampsia and eclampsia. *Tohoku J Exp Med* 2005;206(3):225-31.
10. Braekke K, Ueland PM, Harsem NK, Karlsen A, Blomhoff R, Staff AC. Homocysteine, cysteine, and related metabolites in maternal and fetal plasma in preeclampsia. *Pediatr Res* 2007;62(3):319-24.
11. Makedos G, Papanicolaou A, Hitoglou A, Kalogiannidis I, Makedos A, Vrazioti V, et al. Homocysteine, folic acid and B12 serum levels in pregnancy complicated with preeclampsia. *Arch Gynecol Obstet* 2007;275(2):121-4.
12. Fernández M, Fernández G, Diez-Ewald M, Torres E, Vizcaíno G, Fernández N, et al. Plasma homocysteine concentration and its relationship with the development of preeclampsia. Effect of prenatal administration of folic acid [Article in Spanish]. *Invest Clin* 2005;46(2):187-95.
13. Hauger MS, Gibbons L, Vik T, Belizán JM. Prepregnancy weight status and the risk of adverse pregnancy outcome. *Acta Obstet Gynecol Scand* 2008;87(9):953-9.
14. Silva LM, Coolman M, Steegers EA, Jaddoe VW, Moll HA, Hofman A, et al. Low socioeconomic status is a risk factor for preeclampsia: the Generation R Study. *J Hypertens* 2008;26(6):1200-8.
15. Phaloprakarn C, Tangjitgamol S. Impact of high maternal hemoglobin at first antenatal visit on pregnancy outcomes: a cohort study. *J Perinat Med* 2008;36(2):115-9.