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Assessment of serum Lactate Dehydrogenase in preeclamptic pregnant women

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Abstract

Background: Evidences prove that endothelial cell and altered endothelial cell function play an important role in the pathogenesis of preeclampsia. Therefore serum lactate dehydrogenase (LDH) is useful biochemical markers reflecting the severity of the occurrence of preeclampsia. **Objective:** Determination of serum lactate dehydrogenase (LDH) in severe preeclamptic pregnant women. **Methods:** This is the case-control hospital based study carried in the Al Azhar Medical College and Super speciality Hospital, Thodupuzha. Normal pregnant women (n=55) and women with severe preeclampsia (n=63) were included in the study. Both the groups were in their third trimester and of same age and same gestational age. Serum LDH levels were measured by continuous spectrophotometric pyruvate method for both groups. **Results:** Higher values of serum lactate dehydrogenase (LDH) were found in severe preeclamptic women compared with normal pregnant women in third trimester. **Conclusion:** Elevated levels of serum LDH indicates the tissue damage and might be the cause of the occurrence of preeclampsia.

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Keywords: Preeclampsia, LDH, Third trimester

Introduction

Preeclampsia is a clinical manifestation characterized by hypertension, proteinuria and edema that occurs after 20th week of pregnancy. It is a multisystem disorder of pregnancy with potentially severe consequences for both mother and child [1]. It affects about 5-8% of all pregnancies and is a major cause of maternal, fetal and neonatal mortality and morbidity [2, 3]. The etiology of preeclampsia is unknown but thought to be related to hypoxia in the placenta and endothelial dysfunction [4]. The various causes that lead to these abnormalities are genetic and dietary causes, immunological causes, race, increased oxidative stress etc [5]. Since preeclampsia is a syndrome virtually affecting all maternal organ systems, it is associated with different clinical characteristics, prevention, diagnosis and therapy where a disease requires a close interdisciplinary cooperation [6]. There is increasing evidence that endothelial cell and altered endothelial cell function play an important role in the pathogenesis of preeclampsia. Serum lactate dehydrogenase (LDH) is most often measured to evaluate the

Manuscript received: 5th September 2016 Reviewed: 15th September 2016 Author Corrected: 23rd September 2016 Accepted for Publication: 30th September 2016 presence of tissue damage associated with endothelial damage. Dysfunction of endothelial cells can contribute to inappropriate vasoconstriction and platelet aggregation which are early signs of hypertension [7]. These are multisystem disorders and lead to a lot of cellular death. LDH is an intracellular enzyme and its level is increased in these women due to cellular death. This can be further used as help in making decision, regarding the management strategies to improve the maternal and fetal outcome. So the aims of the present study are to compare serum LDH levels in the normal pregnant women and in women with severe preeclampsia in ante-partum period.

Methods

Design of Study: A prospective comparative case control study. **Setting**: Department of OBG, Al Azhar Medical College and Super speciality Hospital, Thodupuzha, Kerala. Duration of the Study: one year (Janurary 2014 to December 2014). **Study population**: Study was divided into two groups (control pregnant women and severe preeclamptic women). 63 pregnant women clinically diagnosed as preeclampsia during third trimester (28-40 weeks) with the age 18-36 years admitted in obstetrics

Inclusion and exclusion criteria: Inclusion criteria for women included in the study were: should not be using any kind of oral contraceptives, anticoagulant drugs, should be non-smokers and non alcoholics and exclusion criteria was: past history of diabetes, systemic or endocrine disorder, chronic infection, chronic renal disease and hypertension, women in the labour pains, were excluded from the study.

Method of study: Those who fulfill the selection criteria are recruited for the study. After informed consent, data are collected by a pre-structured interview schedule combined with hospital documents and available lab tests.

Detailed information regarding socio-demographic factors, previous pregnancy losses-their gestational age, documentation of fetal heart, any congenital anomalies, complications during antenatal period like severe Preeclampsia, IUGR and abruption remote from term, weight of foetuses and investigation results were collected. Also detailed history regarding past history of Hypertension, Gestational diabetes, hypo-thyroidism, SLE, nephritic

Original Research Article

syndrome, cardiac disease, personal history of thrombosis like DVT, pulmonary embolism, family history of thrombosis, history of drugs, contraception, addiction, smoking was also taken.

Preeclamspia was diagnosed according to American College of Obstetrics and Gynecology (ACOG) criteria as severe if diastolic blood pressure increased to at least 110mmHg, proteinuria >5000mg per day and the presence of headache, visual disturbances, epigastric pain, oliguria, elevated LFT, elevated RFT, thrombocytopenia. Serum LDH levels were measured.

Sample collection: Blood samples were collected in the morning in a plain bulb with aseptic conditions. In the preeclampsia group, blood samples were collected when the patients presented for evaluation and before initiation of medical therapy. Serum LDH levels were measured by continuous spectrophotometric pyruvate method where the reduction of pyruvate to lactate takes place in the presence of NADH by the action of lactate dehydrogenase. The results were expressed as mean \pm SD and groups were compared using Students t test.

Statistical analysis: Data analysis was carried out by using SPSS software, version 16. The level of significance was set at < 0.05.

Results

The Anthropometric details of both study groups are mentioned in table1. Maternal age and body mass index (BMI) were not significantly different between the groups. Gestational age, systolic and diastolic blood pressures were significantly higher in preeclamptic groups as compared to normal pregnant women (p<0.0001). The same when compared between mild and severe preeclamptic groups, it was found to be significantly higher in severe preeclamptic group (p<0.0001). Serum LDH was found to be highest in preeclamptic women as compared to normal pregnant women (p<0.0001, Table 2).

Anthropometric factors	Control group	Preeclampsia group	
	Mean	Mean	p value
	±SD	±SD	
Age (yrs)	22.27±	23.13±	
	2.77	3.46	0.624
BMI (Kg/m ²)	22.17±	23.69±	
	1.75	1.41	0.329
Gestational age (wks)	37.58±	36.11±	
	3.64	1.86	< 0.0001
Systolic blood pressure (mm of Hg)	113.62±	155.44±	
	5.42	4.82	< 0.0001
Diastolic blood pressure (mm of Hg)	$78.41\pm$	110.05±	
	5.81	3.55	< 0.0001

Table-1:	Comparison o	f Anthropomet	ric factors in	both groups.

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Parameters	Controls	Preeclampsia group	
	Mean	Mean	p value
	±SD	±SD	
	201.14±	513.37±	
LDH	78.47	203.44	<0.0001

Table-2: Comparison of LDH in both groups.

Discussion

Preeclampsia is considered an idiopathic multisystem disorder that is specific to human pregnancy [8]. Several potential candidate biochemical markers have been proposed to predict the severity of preeclampsia [5, 9]. The multi organ dysfunction in preeclampsia caused by vascular endothelial damage, including maternal liver, kidney, lungs, nervous system, coagulation system will leads to excessive LDH leakage and elevated levels in serum due to cellular dysfunction, which may cause the occurrence of preeclampsia. These results are also supported by HS Qublan [10].

Qublan HS, et al. [10] found in their study that the mean LDH levels in normal controls was 299 ± 79 IU/l, in patients with mild preeclampsia was 348 ± 76 IU/l and in patients with severe preeclampsia was 774 ± 69.61 IU/l. Thus they demonstrated a significant association of serum LDH levels with severe preeclampsia (P < 0.001).

Lactate dehydrogenase (LDH) is an intracellular enzyme that converts lactic acid to pyruvic acid and elevated levels indicate cellular death and leakage of enzyme from the cell [11]. The results from our study showed that the levels of serum LDH was significantly higher in preeclamptic women as compared to normal pregnant women as shown in table 2 and was also supported by several other studies [10, 12].

In another study by Jaiswar S.P, et al. [13], the control arm had mean LDH levels of 278.3 \pm 119.2 IU/l (normotensives). In severe preeclampsia group it was 646.95 \pm 401.64 IU/l. Jaiswer SP, et al. [13] also demonstrated a significant rise in the LDH levels with increasing severity of the disease (P < 0.001). In the present study, the LDH levels were significantly raised with the severity of the disease (P < 0.001) and this was in accordance with our study.

This study had limitations, namely the only severe preeclamptic women and the relatively less number of pregnant women. Therefore a large sample size prospective study with mild and eclampsia cases are required to establish the etiology, risks factors, and prognosis of the patients.

Conclusion

Elevated levels of serum LDH in severe preeclampia suggest that there is cellular damage and dysfunction and this could be used as a biochemical marker to know the severity of the disease.

Funding: Nil, Conflict of interest: Nil Permission from IRB: Yes

References

1. F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, John C. Hauth, Dwight J. Rouse, Catherine Y. pong. Williams OBSTETRICS- 23rd ed. New York, NY: McGraw-Hill; 2010:706-756.

2. WHO, 2004. Bethesda, MD. Global Burden of Disease for the Year 2001 by World Bank Region, for Use in Disease Control Priorities in Developing Countries, National Institutes of Health: WHO. Make every mother and child count. World Health Report, 2005, Geneva: World Health Organization, 2005. 2nd ed.

3. Stekkinger E, Zandstra M, Peeters LL, Spaandernen ME. Early-onset preeclampsia and the prevalence of postpartum metabolic syndrome. Obstet Gynecol. 2009 Nov;114 (5):1076-84. doi: 10.1097/AOG. 0b013e3181b 7b242.

4. Kay HH, Zhu S, Tsoi S. Hypoxia and lactate production in trophoblast cells. Placenta. 2007 Aug-Sep;28(8-9):854-60. Epub 2007 Feb 2.

5. Lopez-Jaramillo P, Casas JP, Serrano N.Preeclampsia: from epidemiology observation to molecular mechanisms. Braz J Med Biol Res. 2001 Oct;34(10):1227-35.

6. Jan AK, Jamil M. Management of preeclampsia and Eclampsia. JPMI 2000;14(1):67-71.

7. Pasaoglu H, Bulduk G, Ogus E, Pasaoglu A et al. Nitric oxide, lipid peroxide and uric acid levels in preeclampsia and eclampsia. Tohoku J Exp Med. 2004 Feb;202(2): 87-92.

8. Norwitz ER, Hsu CD, Repke JT. Acute complications of preeclampsia.Clin Obstet Gynecol. 2002 Jun; 45 (2): 308-29.

9. Page NM. The endocrinology of preeclamspia. Clin Endocrinol (Oxf). 2002 Oct;57(4):413-23.

10. Qublan HS, Ammarin V, Bataineh O, Al Shraideh Z,Tahat Y, Awamleh I, et al. Lactic dehydogenase as a biochemical marker of adverse pregnancy outcome in severe preeclampsia. Med Sci Monit. 2005 Aug;11(8): CR393-7. Epub 2005 Jul 25.

Original Research Article

11. Krefetz RG: Enzymes. Clinical Chemistry, 4th ed. Lippincott Williams and Wikins; Philadelphia 2000; 196-98.

12. Malarewicz A, Gruszka O, Szymkiewicz J, Rogala J. The usefulness of routine laboratory tests in the evaluation of sudden threat of pregnant woman and fetus in preeclampsia. Ginekol Pol 2006; 77(4):276-84.

13. Jaiswar SP, Gupta Amrit, Sachan Rekha, Natu SN, Shaili Mohan. Lactic Dehydrogenase: A biochemical marker for preeclampsia-eclampsia. JOGI 2011; 61(6): 645-648.

How to cite this article?

Nair R, Narayanan V. Assessment of serum Lactate Dehydrogenase in preeclamptic pregnant women. *Obg Rev:J obstet Gynecol* 2016;2(3):26-29. doi: 10.17511/joog.2016.i03.01.