DOI: 10.7860/JCDR/2018/31930.11545

Original Article

Obstetrics and Gynaecology Section

Role of Serum Lactate Dehydrogenase in Pregnancy Induced Hypertension with its Adverse Feto-Maternal Outcome- A Case-control Study

PALLAVI SINGH[1], HARSHA SHAILESH GAIKWAD[2], SHEEBA MARWAH[3], PRATIMA MITTAL[4], CHARANJEET KAUR[5]

ABSTRACT

Introduction: Hypertensive Disorders of Pregnancy (HDP) affect 6-8% of all pregnancies, contributing immensely to maternal morbidity and mortality. Pre-eclampsia is a common, yet a major medical complication of pregnancy and is responsible for 10%-15% of maternal deaths. Lactate Dehydrogenase (LDH) is an intracellular enzyme, which when present in serum signifies tissue damage and haemolysis.

Aim: To study the correlation of serum LDH levels with blood pressure range in patients with gestational hypertension and pre-eclampsia, and the feto-maternal outcome.

Materials and Methods: Based on the eligibility criteria, 150 hypertensive pregnant women were enrolled as cases. Also, 150 normotensive women admitted after 28 weeks of gestation were taken as controls in the study. Serum LDH was measured in both the groups and its correlation with blood pressure was studied, after applying appropriate clinical tests. (Quantitative variables-unpaired t-test/Mann-Whitney test between two groups and ANOVA/Kruskal Wallis test between more than two groups; Qualitative variables: chi-square/Fisher exact test. Pearson/Spearsman correlation coefficient was used to assess correlation between various quantitative parameters) Association of other biochemical parameters with feto-maternal outcome was also studied.

Results: The mean age of women in hypertensive group was 25.06±4.13 years, while that in normotensive group was 24.38±3.8 years. Most women belonged to lower and lower middle class (75.33%). The mean serum LDH levels in hypertensive group was 1011.81±539.31 IU/L, while it was 555.24±237.69 IU/L in normotensive groups (p-value=0.017). Serum LDH levels were significantly associated with the range of blood pressure (p-value<0.0005). High levels of LDH were found in pre-eclamptic women with adverse maternal outcomes (p-value=0.001). Elevated levels of LDH (1058.88±575.19 IU/L) were also associated with Low Birth Weight (LBW) babies and poor foetal outcome, including low Apgar, higher rates of NICU admission and neonatal mortality (p-value<0.0005).

Conclusion: LDH level estimation and its correlation with range of blood pressure is a simple and reliable method for prediction of adverse maternal and foetal outcome in hypertensive disorders of pregnancy. It may also help in assessing the severity of the condition.

Keywords: Adverse maternal effects, Gestational hypertension, Lactate dehydrogenase, Pre-eclampsia, Unfavourable foetal fallouts

INTRODUCTION

Pregnancy is a physiological state associated with varied biochemical and maternal adaptation in response to physical stimuli provided by foetus and placenta [1]. HDP affect 6-8% of all pregnancies [2] and along-with haemorrhage and infection, they form a complex triad, contributing immensely to maternal morbidity and mortality [1,3].

Hypertensive disorders of pregnancy, particularly pre-eclampsia, are responsible for 10% to 15% of maternal deaths [4]. In developing countries, incidence of pre-eclampsia and its morbidity is much higher as compared to high income countries. Over the years, maternal deaths due to pre-eclampsia have been significantly reduced by early diagnosis and management in developed countries; on the contrary it is still responsible for 19% of annual maternal deaths in developing countries including India [5].

Though exact aetio-pathogenesis of pre-eclampsia is unknown, multiple factors have been implicated to have a role in it; the factors being abnormal placental implantation, endothelial dysfunction, maternal immunological tolerance, cardiovascular, genetic, nutritional and environmental factors [6]. Amongst these, endothelial dysfunction is considered to be most important factor resulting in mild to moderate microangiopathy of target organs, leading to excessive leakage of LDH in serum [5-7]. Thus presence of LDH signifies tissue damage and haemolysis [8]. Elevated LDH levels in pre-eclampsia reflect severity as well occurrence of complications in pre-eclampsia [6,9,10].

It has been studied in recent times that pre-eclamptic patients with higher levels of LDH are susceptible to have poor maternal and foetal upshots [6,8,9,11,12]. Identification of such high risk patients with elevated LDH levels, along with close monitoring and prompt management may prevent these complications, with subsequent drop in feto-maternal morbidity and mortality. Most of the available data in literature till date, are smaller studies, spanning over smaller duration [6,8,9,11-18].

The present study was conducted to correlate LDH levels with blood pressure ranges and maternal and foetal outcome in women with gestational hypertension, pre-eclampsia.

MATERIALS AND METHODS

A prospective case-control study was conducted, in Department of Obstetrics and Gynaecology in collaboration with Department of Pathology and Biochemistry, Vardhaman Mahavir Medical College and Safdarjang Hospital, New Delhi, India, over 18 months, from September 2014 to March 2016. One hundred fifty women, after 28 weeks of gestation, with HDP as cases, and another 150

normotensive pregnant women as controls, were enrolled in the study after taking their written informed consent. This study was approved by Institutional Ethical Committee. Women were selected consecutively on the basis of definitions given by National High Blood Pressure Education Program (NHBPEP 2000) [10] and ACOG criteria [3]. Following the ACOG criteria, cases were further divided into following subgroups-women with gestational hypertension, mild pre-eclampsia, severe pre-eclampsia and eclampsia [3]. All the women were then categorised according to serum LDH levels into mild (<600 IU/L), moderate (600-800 IU/L) and severe (>800 IU/L) categories.

Exclusion criteria were chronic hypertension, diabetes mellitus, chronic liver renal disease, epilepsy, thyroid disease, smoking, alcohol and drug intake, hyperuricaemia, symptomatic infectious diseases, multiple gestation, and unwillingness to participate.

On the first visit to the hospital, after comprehensive history and physical examination, the included women were subjected to routine investigations for HDP (haemoglobin, bleeding time, clotting time, blood sugar, liver and kidney function test). Then, about 2 mL of venous blood was drawn under aseptic conditions in a plain vial (before initiation of medical treatment) from the women in both the groups, serum was separated by centrifugation and estimation of serum LDH was done.

Concentration of serum LDH, was analysed by using analytical kits from HITACHI analyser 902 (hospital supply) in clinical biochemistry unit. This method is based on the reduction of pyruvate to lactate in the presence of NADH (nicotinamide adenine dinucleotide, reduced form) by the action of LDH enzyme. Pyruvate that remains unchanged reacts with 2,4-dinitrophenylhyrazone, which was then determined calorimetrically in an alkaline medium. The rate of fall of NADH is directly proportional to the activity of LDH. The rate of formed NAD+ is determined by the decrease in its absorbance.

All women were followed through their antenatal period till 72 hours post-delivery (along-with their newborns) and subsequently till 12 weeks postpartum.

Outcomes measured were demographic and maternal characteristics, serum LDH values, its correlation with blood pressure and feto-maternal outcome and correlation of blood pressure with other biochemical markers (liver and kidney function tests).

STATISTICAL ANALYSIS

Sample size calculation and data analysis: To detect a significant difference from 90-95%, correlation among cases and controls, with a power of 80%, and incidence of pre-eclampsia being 5-8%, two tailed α of 5% required a minimum of 150 women in each group, thus achieving a sample size of 300 as deduced through epi info software. All data were entered on predesigned case proforma and deciphered at the end of the study using SPSS 21.0 version. Quantitative variables were compared using unpaired t-test/mann-Whitney test between two groups, and ANOVA/Kruskal Wallis test between more than two groups. Qualitative variables were correlated using chi-square/Fisher exact test. ROC curve was used to assess the predictability of various factors for diagnosing severity of hypertension. Pearson/Spearsman correlation coefficient was used to assess correlation between various quantitative parameters. The level of significance was set at <0.05 for all variables.

RESULTS

Amongst 300 women included in the study, 123 women had LDH levels <600 IU/L, 59 had LDH levels 600-800 IU/L and 118 women had >800 IU/L. Out of the total 150 cases, 47 had gestational hypertension, 65 had mild pre-eclampsia and 38 had severe pre-eclampsia. The mean age of women in hypertensive group was 25.06±4.13 years, while that in normotensive group was 24.38±3.8 years. Thus, both were comparable statistically (p-value=0.153). Most of the patients belonged to lower and lower middle class, hailing from rural (37.33%) and slum (47.67%) areas [Table/Fig-1].

S. NO.	Characteristics	Hypertensive Group (%)	Normotensive group (%)	Total (%)	p-value
1.	**Age (years)**				
	20	18 (12.00%)	22 (14.67%)	40 (13.33%)	0.451
	21-30	119 (79.33%)	120 (80.0%)	239 (79.67%)	
	>30	13 (8.67%)	8 (5.33%)	21 (7.00%)	
2.	**Parity**				
	Multigravida	72 (48.00%)	78 (52.00%)	150 (50.00%)	0.23
	Primigravida	78 (52.00%)	72 (48.00%)	150 (50.00%)	
3.	**Residence**				
	Urban	21 (14.00%)	26 (17.33%)	47 (15.67%)	0.729
	Slum	73 (48.67%)	70 (46.67%)	143 (47.67%)	
	Rural	56 (37.33%)	54 (36.00%)	110 (36.67%)	
4.	**Socioeconomic status (modified Kuppuswami Upper Scale)**				
	Upper	0 (0.00%)	0 (0.00%)	0 (0.00%)	0.192
	Upper middle	0 (0.00%)	0 (0.00%)	0 (0.00%)	
	Middle	37 (24.67%)	29 (19.00%)	66 (22.00%)	
	Lower middle	92 (61.33%)	89 (59.33%)	181 (60.33%)	
	Lower	21 (14.00%)	32 (21.33%)	53 (17.67%)	

[Table/Fig-1]: Comparison of demographic and maternal characteristics of the study population.

The mean serum LDH levels in hypertensive group was 1011.81±539.31 IU/L, while it was 555.24±237.69 IU/L in the normotensive group (p-value<0.0005). Analysing the cases with respect to serum LDH values, the mean LDH value was much lower in women with gestational hypertension and mild pre-eclampsia (n=112; 958.52±501.75 IU/L), when compared to women with severe pre-eclampsia (n=38; 1168.9±618.13 IU/L) This difference was statistically significant (p-value=0.017) [Table/Fig-2].

LDH Levels In Study Population		$p\text{-value}_a$	$p\text{-value}_b$
	Mean LDH value		
Hypertensive group (n=150)		0.017	<0.0005
Gestational hypertension and mild pre-eclampsia	958.52±501.75 IU/L		
Severe pre-eclampsia	1168.9±618.13 IU/L		
Normotensive Group (n=150)	555.24±237.69 IU/L		

[Table/Fig-2]: Correlation of Serum LDH and Blood Pressure.
$p\text{-value}_a$=level of significance for severity of hypertension
$p\text{-value}_b$= level of significance for mean LDH values among cases and controls

It was noted that in women with LDH levels above 800 IU/L (118/300) 28 women had systolic BP>160 and above (23.7%) and 40 women had diastolic BP 110 and above (11.01%). Studying the association of LDH with increase in blood pressure, the rise in LDH was found to be weakly correlated with SBP (correlation coefficient=0.24), but strongly correlated with DBP (correlation coefficient=-0.12) [Table/Fig-3].

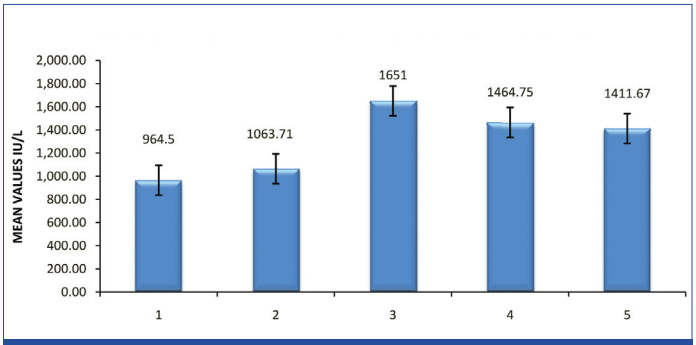
Blood pressure	<600 IU/L	600-799 IU/L	≥ 800 IU/L	Correlation coefficient
Systolic blood pressure				
<140 mmHg	103	27	20	0.24
140-159 mmHg	13	30	70	
>160 mmHg	7	2	28	
Diastolic blood pressure				
< 90 mmHg	106	31	13	-0.12
90-109 mmHg	13	24	65	
>110 mmHg	4	4	40	

[Table/Fig-3]: Distribution of study population according to serum LDH levels and blood pressure.

Analysing the maternal complications in the hypertensive group, 8 (8.08%) women developed eclampsia, 3 (2.00%) had placental abruption and 1 (0.67%) had HELLP syndrome. Noteworthy was the fact, that all these women had LDH values >800 IU/L. However, none of these complications were seen in the normotensive group. Also, as the levels of LDH increased in both the groups, the percentage of women delivering by LSCS increased (p-value=0.001) [Table/Fig-4].

The gestational age at delivery of women in hypertensive group (37.34±2.55 weeks) was significantly lesser than that of women in normotensive group (38.41±1.74 weeks) (p-value<0.0005).

The women with LBW neonates had higher levels of LDH (1058.88±575.19 IU/L) than women with average birth weight (975.82±510.71 IU/L) [Table/Fig-5]. Also, it was seen that as the levels of serum LDH increased, the prognosis of foetus worsened and chances of NICU admission, IUD and neonatal death increased [Table/Fig-6].



[Table/Fig-6]: Association of LDH with foetal outcome.
1. Alive and well; 2. NICU admission; 3. Fresh still birth; 4. Macerated still birth; 5. Neonatal death

S.No		Normotensive group			Hypertensive Group			p-value
1.	Complications	<600 IU/L	600-800 IU/L	>800 IU/L	<600 IU/L	600-800 IU/L	>800 IU/L	
	Eclampsia	0	0	0	0	0	8	0.113
	Placental Abruption	0	0	0	0	0	3	0.455
	HELLP syndrome	0	0	0	0	0	1	0.772
2.	Mode of delivery							
	NVD	103 (100%)	25 (92.6%)	18 (90%)	20 (100%)	29 (90%)	86 (87.8%)	0.001
	LSCS	0 (0%)	2 (7.4%)	2 (10%)	0 (0%)	3 (10%)	12 (12.3%)	

[Table/Fig-4]: Correlation of Serum LDH with maternal complications and mode of delivery.

Foetal Outcome	LDH value (IU/L)						p-value$_a$	p-value$_b$
	Normotensive Group (%)			Hypertensive Group (%)				
	<600	600-800	>800	<600	600-800	>800		
POG								
Preterm	1 (0.97%)	1 (3.8%)	4 (20%)	7 (40.9%)	13 (34.5%)	27 (27.3%)	0.402	<0.0005
Term	103 (99%)	25 (96.2%)	16 (80%)	13 (59.1%)	19 (65.5%)	71 (72.7%)		
Birth weight								
LBW	0 (0%)	0 (0%)	2 (10%)	12 (54.6%)	9 (28.1%)	44 (44.4%)	0.227	<0.0005
Average	103 (100%)	27 (100%)	18 (90%)	8 (45.5%)	23 (71.9%)	54 (55.6%)		
Still birth	0	0	0	0	0	6 (4%)	0.076	<0.0005
Neonatal death	0	0	0	0	0	3 (2%)		
NICU admission	0	0	0	0	0	24 (16%)		
Alive+well	103 (100%)	27 (100%)	20 (100%)	0	0	117 (78%)		

[Table/Fig-5]: Correlation of serum LDH with foetal outcome.
p-value$_a$=level of significance among cases
p-value$_b$=level of significance among cases and controls

Drawing contrast for the other biochemical parameters within cases and controls, it was noted that there was a significant difference in serum bilirubin, SGOT, SGPT, blood urea and serum creatinine between the two groups (p-value<0.0005) [Table/Fig-7].

Biochemical tests	Hypertensive Group	Normotensive Group	p-value
Serum Bilirubin	0.56±0.32	0.37±0.14	<0.0005
Mild pre-eclampsia	0.53/0.33		
Severe pre-eclampsia	0.62/0.29		0.02
SGOT	56.37±40.82	40.25±20.84	<0.0005
Mild pre-eclampsia	56.57/40.27		
Severe pre-eclampsia	55.79/42.96		0.702
SGPT	54.65±35.5	44.91±23.12	<0.0005
Mild pre-eclampsia	59.39/35.91		
Severe pre-eclampsia	40.66/30.61		0.002
Blood urea	30.83±18.71	21.09±7.77	<0.0005
Mild pre-eclampsia	31.21/20.67		
Severe pre-eclampsia	29.71/11.19		0.804
Serum Creatinine	0.6±0.43	0.45±0.13	<0.0005
Mild pre-eclampsia	0.6/0.49		
Severe pre-eclampsia	0.59/0.19		0.207

[Table/Fig-7]: Correlation of blood pressure and other parameters.

DISCUSSION

Several studies have been done for correlating to serum LDH with feto-maternal outcome [7,10,11,13,14,17-26]; all these had different results because of varied sample size, study duration and parameter observed [Table/Fig-8].

In consonance with previous researchers, serum LDH is correlated significantly with severity of blood pressure [6,8]. Detection of serum LDH in pregnant female would help the clinicians, even at Primary Health Centres (PHCs) to diagnose pre-eclampsia early and timely refer them to higher centres for better antepartum surveillance and manage in order to avert the complications associated with it. So it is proposed that efforts should be made to make this simple and cheap testing modality to be available at all PHCs for all antenatal hypertensive women.

Author (year)	Type of Study	Sample Size	Observation	Conclusion
1. Kant RH et al., [7]	Observational	200	LDH, LFT and KFT	Pre-eclampsia- Significant correlation with levels
2. Dey PS et al., [10]	Case-control	101	LDH and GGT	Pre-eclampsia -significant correlation with levels
3. Lavanya YR and Shobharani B [11]	Case-control	60	LDH	Pre-eclampsia -significant correlation with levels
4. Bakhshandeh N et al., [13]	Case-control	100	LDH	Pre-eclampsia-Not significant
5. Sreelatha S et al., [14]	Prospective	80	LDH and UA	Pre-eclampsia and LBW significant correlation with levels
6. Hak J et al., [17]	Case-control	200	LDH	Pre-eclampsia and Foetal outcome- significant correlation with levels
7. Bera S et al., [18]	Case-control	124	LDH, ALT and AST	BP and LBW- significant correlation with levels
8. Gandhi M et al., [20]	Case-control	50	LDH and UA	Pre-eclampsia and SGA-significant correlation with levels
9. Sabiullah M et al., [21]	Case-control	50	LDH	Pre-eclampsia-significant correlation with levels
10. Sharma C et al., [22]	Case-control	60	LDH, AST and Lipid profile	Raised in hypertensive group
11. Umastyari Y et al., [23]	Prospective	150	LDH	Pre-eclampsia and SGA - significant correlation with levels
12. Munde SM et al., [24]	Prospective	80	LDH	Pre-eclampsia foetal weight-significant correlation with levels
13. Andrews L et al., [25]	Prospective	2237/328	LDH,ALT,AST UA, PT and APTT	Pre-eclampsia, Placental abruption, HELLP and DIC-significant correlation with levels
14. Sonagra AD et al., [26]	Cross-sectional	120	LDH, ALP and UA	Pre-eclampsia- significant correlation with levels
15. Present study	Case-control	300	LDH, LFT and KFT	Pre-eclampsia and Feto-maternal outcome- significant correlation with levels

[Table/Fig-8]: Comparative evaluation of LDH in different studies.

Present study showed significantly elevated levels of LDH to be associated with presence of complications in hypertensive group as compared to normotensive group. This was analogous to conclusions from previous investigators [9,13]. This again emphasises the fact that morbidity due to HDP can be decreased only with stringent management of such women, after picking up the clinical entity well within time. For this purpose LDH is quite beneficial. Also higher number of LSCS in women with increased levels of serum LDH proved the fact that clinical suspicion and adequate facilities to diagnose pre-eclampsia in time must be endorsed even at the PHCs; so that such women can be timely transferred to first referral units for operative deliveries and for achieving optimum feto-maternal outcome. However, very few researchers did not find any significant difference in LDH level between normotensive and pre-eclamptic patients, which could be attributed to small sample size of their study [14].

Scrutinising the foetal outcome in the study in females with HDP, high levels of LDH were able to predict worst foetal prognosis in terms of prematurity, birth weight, NICU admission and foetal demise. This was similar to findings of past pollsters who reported that the mean gestational age decreased with increased LDH levels, which could be due to induction of labour at an earlier gestational age [6,12,13,15-19].

Higher number of preterm, LBW babies, intrauterine deaths, neonatal deaths and high NICU admissions, in hypertensive group could be due to early settings of placental insufficiency, followed by foetal growth restriction and hence recourse to early induction in those women after weighing the clinical scenario.

In order to establish predictive value of LDH as a marker for HDP, the present author's correlated blood pressure with other available biochemical tests viz., serum bilirubin, SGOT, SGPT, blood urea and serum creatinine. Analogous to previous investigators, a positive correlation with the same in the present study highlighted the long-term significance of LDH in predicting development of HDP in antenatal women (at risk) [15,18,26]. Altered levels of LFT and KFT in severe pre-eclampsia provides us with a gamut of minimum laboratory parameters that must be performed in each women of HDP; adding to predictive value of LDH.

Ample knowledge of the above in all young gynaecologists and the grass-root multipurpose health workers and mid wives, along-with timely intervention would strengthen maternal surveillance and curtail the rate limiting step in pathogenesis of severe pre-eclampsia, averting its dreaded complications.

LIMITATION

Having drawn from a smaller populace over a period of 18 months, the results cannot be applied to the national population. For the latter, larger and more powered studies are advocated.

CONCLUSION

Elevated levels of LDH in HDP are indicative of cellular damage and dysfunction; thus LDH can be used as a biochemical marker, reflecting the severity of the disease. Detection of high-risk pregnancies with increased levels of LDH, besides careful monitoring in antenatal period and proper management would be the key to decrease both maternal and foetal morbidity and mortality. Thus all PHCs and sub-centers should be equipped with detection of LDH testing kits.

ACKNOWLEDGEMENTS

We are thankful to our patients for their cooperation and support in helping this study reach a successful culmination.

REFERENCES

[1] Cunningham FG, Leveno KJ, Bloom SL, et al. (2014) Chapter 40. Hypertensive disorders. In: Williams Obstetrics (24th edn). New York, Mc Graw-Hill. 2014;728-79.
[2] Kamath S. Hypertension in pregnancy. JAPL. 2006;54:269-70.
[3] American College of Obstetrician and Gynecologists. Women's Health Care Physicians. Hypertension in Pregnancy. 2013. Washington, DC. Chapter -2, Establishing the Diagnosis of Preeclampsia and Eclampsia. 2013; pp.17-21
[4] Jim B, Sharma S, Kebede T, Acharya A. Hypertension in pregnancy: a Comprehensive update. Cardiology in Review. 2010;18:178-89.
[5] Trends in maternal mortality: 1990 to 2010. WHO, UNICEF, UNFPA and the World Bank estimates. Available at: http://whqlibdoc.who.int/publications/2012/9789241503631_eng.pdf. Accessed on July 12, 2012.
[6] Qublan H, 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 pre-eclampsia. International Medical Journal of Experimental and Clinical Research. 2005;11(8):CR393-397.

[7] Kant RH, Mir N, Sarwar S, Gupta S, Najeeb R. Role of maternal serum lactate dehydrogenase as a biochemical marker in pre-eclampsia. IOSR Journal of Dental and Medical Sciences. 2015;14(7):12-19.
[8] Butt AA, Michaels S, Greer D, Clark R, Kissinger P, Martin DH. Serum lactate dehydrogenase level as a clue to the diagnosis of histoplasmosis. AIDS Read 2002;12(7):317-21.
[9] Malik KK, Akmal N, Akhter N. Correlation of lactate dehydrogenase levels with the severity of pre-eclampsia. Tohoku J Exp Med. 2004;202:87-92.
[10] Dey PS, Jonal S. Evaluation of serum LDH and gamma glutamyltransferase in pre-eclamptic pregnancy in third trimester. Int J Res Med Sci. 2013:1(4) 365-68.
[11] Lavanya YR, Shobharani B. Study of liver enzymes and proteinuria in pregnancy induced hypertension. IOSR Journal of Dental and Medical Sciences. 2015;14(2):34-36.
[12] Malarewicz A, Gruszka O, Szymkiewicz J, Rogale J. The usefulness of routine laboratory tests in the evalueation of sudden threat of pregnant woman and fetus in pre-eclampsia. Ginecol Pol. 2006;77(4):276-84.
[13] Bakshandeh NS, Azarhoosh R, Borghei A, Sedaghati M, Besharat S, Ghaemi E. Serum level of lactate dehydrogenase, homocystein, haemoglobin and platelet in preelampsia. Pak J Med Sci. 2011;27:1014-17.
[14] Sreelatha S, Bharathi, Ramya, Shwetha. Estimation of serum LDH and uric acid in pre-eclampsia and its correlation with maternal and perinatal outcome. International Journal of Advances in Case Reports. 2015;2(7):447-49.
[15] Jaiswar SP, Gupta A, Sachan R, Natu SN, Shaili M. Lactic dehydrogenase: a biochemical marker for preeclampsia–eclampsia. J Obst Gynaecol India. 2011;61(6):645-48.
[16] Mary VP, Chellatamizh M, Padmanaban S. Role of serum LDH in preeclampsia as a prognostic factor-a cross sectional case control study in tertiary care hospital. Int J Reprod Contracept Obstet Gynecol. 2017;6(2):595-98.
[17] Hak J, Nisa UN, Gupta S. LDH levels in pregnancy and its association with severity of the disease and feto-maternal outcome in pre-eclampsia and eclampsia. JK Science. 2015;17(3):110-13.
[18] Bera S, Gupta S, Saha SR, Kunti S, Biswas S, Ghosh D. Study of liver enzyme especially lactate dehydrogenase to predict fetal outcome in pregnancy induced hypertension. Sch J App Med Sci. 2014;2:5A1569-72.
[19] Agrawal P, Rajoria L, Vyas J, Savli A, Balsane R, Gupta S. Serum LDH in preeclampsia and eclampsia and maternal outcomes. Sch J App Med Sci. 2016;4:6C:2052-55.
[20] Gandhi M, Chavda R, Saini HB. Comparative study of serum LDH and uric acid in hypertensive versus normotensive pregnant woman. International Journal of Biomedical Science. 2015;6(01):25-28.
[21] Sabiullah M, Rekha SP, Venkateswarlu U. Study of serum lactate dehydrogenase and uric acid in preeclampsia. IJPRBS. 2015;4(3):160-66.
[22] Sharma C, Soni N, Kaushik GG, Sharma RP. Study of serum lipid profile ast and ldh in pre-elampsia and eclampsia. International Journal of Advanced Research. 2015;3(2):326-29.
[23] Umasatyasri Y, Vani I, Shamita P. Role of LDH (Lactate Dehydrogenase) in preeclampsia-eclampsia as a prognostic marker: an observational study. IAIM. 2015;2(9):88-93.
[24] Munde SM, Hazari NR, Thorat AP, Gaikwad SB, Hatolkar VS. Gamma glutamyl transferase and lactate dehydrogenase as biochemical markers of severity of preeclampsia. World Academy of Science, Engineering and Technology International Journal of Medical and Health Sciences. 2014;8(1):201.
[25] Andrews L, Mehta L, Sharma L, Haridas N, Vaishnav S, Jadav P. Maternal outcome in relation to biochemical parameters in hypertensive disorders in pregnancy. IOSR Journal of Dental and Medical Sciences. 2014;13:18-22.
[26] Sonagra AD, Dattatreya K, Murthy J, DS. Serum LDH, ALP and Uric acid in Hypertensive Disorders of Pregnancy. IJBPS. 2012;2:201-09.

PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of Obstetrics and Gynaecology, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, Delhi, India.
2. Professor, Department of Obstetrics and Gynaecology, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, Delhi, India.
3. Assistant Professor, Department of Obstetrics and Gynaecology, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, Delhi, India.
4. Professor and Consultant, Department of Obstetrics and Gynaecology, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, Delhi, India.
5. Professor and Consultant, Department of Biochemistry, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, Delhi, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sheeba Marwah,
Assistant Professor, Department of Obstetrics and Gynaecology, Vardhaman Mahavir Medical College and Safdarjung Hospital,
New Delhi-110029, Delhi, India.
E-mail: sheebamarwah@yahoo.co.in

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jul 26, 2017**
Date of Peer Review: **Nov 02, 2017**
Date of Acceptance: **Feb 12, 2018**
Date of Publishing: **May 01, 2018**