Section: Obstetrics and Gynecology

Original Research Article

EXPLORING THE SIGNIFICANCE OF LACTATE DEHYDROGENASE LEVELS IN PREECLAMPSIA

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Abstract

Background: Hypertensive disorders pose significant challenges during pregnancy and are part of the obstetric deadly triad, alongside hemorrhage and infection. Eclampsia and preeclampsia have become increasingly prevalent causes of maternal mortality, particularly in developing regions. Monitoring the spectrum of hypertensive disorders, including these conditions, can be facilitated by assessing lactate dehydrogenase (LDH), a crucial biochemical marker. This study aims to compare serum LDH levels in normal pregnant women with those in women experiencing preeclampsia and eclampsia during the antepartum period. **Materials and Methods:** Conducted as a prospective comparative hospital-based study, this research took place in the Department of Obstetrics and Gynecology at SDM College of Medical Sciences and Hospital. A total of 130 antenatal cases were included based on predetermined criteria, comprising 30 normal pregnant women, 50 with non-severe preeclampsia, and 50 with severe preeclampsia. Symptoms and complications of preeclampsia, along with fetal outcomes, were assessed with consideration for LDH levels (<600, 600-800, and >800 IU/L). **Result:** The study revealed elevated serum LDH levels in both non-severe and severe preeclamptic women compared to normal pregnant women, and this difference was statistically significant. Severe preeclamptic cases demonstrated maternal complications such as abruptio placentae, post-partum hemorrhage, intrapartum hemorrhage, acute kidney injury, post-partum eclampsia, pulmonary edema, and HELLP syndrome. All these complications were observed in women with LDH levels exceeding 600 IU/L, contributing to increased ICU admissions and prolonged hospital stays. **Conclusion:** The findings highlight a substantial correlation between LDH levels and the severity of preeclampsia, as well as maternal and fetal outcomes. Instances of maternal and fetal complications were more prevalent in individuals with LDH levels exceeding 600 IU/L. Consequently, serum LDH emerges as a valuable biochemical marker for predicting the severity of preeclampsia, offering insight into maternal complications and fetal outcomes.

INTRODUCTION

Hypertensive disorders present unresolved challenges in the context of pregnancy, constituting a significant aspect of the obstetric deadly triad alongside hemorrhage and infection. In the developing world, eclampsia and preeclampsia have witnessed increasing rates as contributors to maternal mortality.[1]

Despite numerous hypotheses attempting to elucidate the pathogenesis and manifestations of this spectrum, the condition remains an enigma, with several components yet to be fully understood. This disorder manifests as a multisystem condition with a progressive course, posing risks for adverse maternal and perinatal outcomes. Therefore, swift diagnosis and appropriate management are imperative to address the complexities associated with this condition.[2,3]

Early detection of the disease process is facilitated through the evaluation of patients using biochemical markers. An essential biochemical marker for monitoring the spectrum of hypertensive disorders of pregnancy is lactate dehydrogenase (LDH).[4] Lactate dehydrogenase (LDH) is an intracellular enzyme primarily responsible for converting pyruvate to lactate. When elevated levels of LDH are detected in the extracellular space, it serves as evidence of cell death and the loss of cellular integrity. This principle is applied and utilized in the monitoring of cases of preeclampsia and eclampsia, providing insights into the severity of these conditions.[5]

International Society for the study of hypertension in pregnancy (ISSHP) defines Hypertension

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as a systolic blood pressure of ≥140mm Hg and diastolic blood pressure of ≥ 90 mm Hg measured on 2 different occasions at least 6 hours apart within the last 7 days.

The National High Blood Pressure Education Programme (NHBPEP) standards, created in 2000, classify hypertensive disorders during pregnancy. Gestational hypertension is defined as systolic blood pressure (SBP) > 140 mm Hg and diastolic blood pressure (DBP) ≥ 90 mm Hg after 20 weeks of pregnancy in women who were previously normotensive. Preeclampsia and eclampsia are defined as SBP ≥ 140 mm Hg and DBP ≥ 90 mm Hg, measured on two occasions at least 6 hours apart within 7 days after 20 weeks of gestation. Proteinuria more than 1g/L, a dipstick reading of 2+ or higher, or a 24-hour urine protein value greater than 0.3 g all corroborate the diagnosis of preeclampsia. Eclampsia is defined as the occurrence of generalised tonic-clonic seizures in the setting of preeclampsia that are not caused by other factors. New start proteinuria in hypertensive women after 20 weeks of gestation, or a rapid rise in blood pressure, proteinuria, or thrombocytopenia in women with pre-existing hypertension prior to 20 weeks of gestation, are indicators of pre-eclampsia superimposed on chronic hypertension. Chronic hypertension is not related to multiple pregnancy or gestational trophoblastic disease; it is detected either before 20 weeks of gestation or prior to pregnancy. It can also be detected beyond 20 weeks of pregnancy and continue over 12 weeks after giving birth. A recent epidemiological report released by the World Health Organization (WHO) has estimated that preeclampsia is directly responsible for 70,000 maternal deaths annually worldwide. Beyond the significant impact on maternal mortality and morbidity, it is also associated with 500,000 infant deaths per year. Given its heterogeneous nature, preeclampsia affects multiple organ systems. In the context of India, the reported incidence of preeclampsia is approximately 8-10% of overall pregnancies. A comprehensive population study indicated a prevalence of hypertensive disorders of pregnancy at 7.8%, with preeclampsia accounting for 5.4% of cases.[6]

Regardless of the region or ethnicity, the severity of clinical presentation in preeclampsia exhibits considerable variability based on the patient's physiological status. Generally, outcomes are favorable when mild preeclampsia develops after the 36th week of gestation.[7] However, the risk of adverse maternal and perinatal outcomes significantly increases when preeclampsia develops before the 33rd week of gestation or occurs at any gestational age in individuals with preexisting medical conditions. Notably, outcomes tend to be less favorable for women residing in developing countries, irrespective of gestation or clinical presentation severity. This discrepancy in outcomes may be attributed to the challenges associated with limited access to modern medical care in those regions.[8]

Lactate dehydrogenase (LDH) is an intracellular enzyme, and an increase in serum LDH levels is indicative of cell injury or cell death. The pathogenesis of preeclampsia involves widespread activation of endothelial cells, triggering the release of oxidants that result in extensive cell injury and death. Depending on the nature of tissue injury, the enzyme can remain elevated in the bloodstream for up to seven days. This highlights the potential of serum LDH as a marker reflecting the ongoing cellular processes associated with preeclampsia pathogenesis.[9]

The increase in lactate dehydrogenase (LDH) is directly proportional to the degree of cell injury, making it a satisfactory marker for staging pathology, monitoring prognosis, assessing response to treatment, and evaluating body fluids other than blood. A decline in LDH levels during the course of treatment indicates a more favorable prognosis or a positive response to treatment in various conditions. Therefore, LDH can serve as an indicator for the severity of preeclampsia, offering valuable insights into the progression of the disease and the effectiveness of therapeutic interventions.[10,11]

This study holds paramount significance in addressing the pressing issues associated with hypertensive disorders during pregnancy, which constitute a challenging aspect of maternal health and are integral to the obstetric deadly triad alongside hemorrhage and infection. With eclampsia and preeclampsia emerging as increasingly prevalent causes of maternal mortality, especially in developing regions, there is a critical need for comprehensive monitoring strategies.

MATERIALS AND METHODS

Study design and setting: The study was a prospective comparative hospital-based study and was conducted in the department of obstetrics and gynecology, SDM College of Medical Sciences and Hospital, Dharwad, which is a tertiary care hospital, from the time period November 2019 to November 2020 on 130 antenatal cases attending the OPD or admitted as per the predetermined inclusion and exclusion criteria after obtaining an ethical clearance from the institution and informed consent of the patients. The inclusion criteria for this study encompass antenatal primigravida individuals with singleton pregnancies beyond 28 weeks. The participants are divided into two groups: Group 1 comprises healthy normal pregnant women serving as controls, and Group 2 consists of patients diagnosed with preeclampsia and eclampsia, who serve as subjects. Group 2 is further subdivided into two subgroups based on the severity of the condition: non-severe preeclampsia and severe preeclampsia. This categorization aims to provide a comprehensive understanding of the varying degrees of preeclampsia

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and eclampsia for the purpose of the study. Exclusion criteria for this study include individuals who are multigravida, those with twin gestation, women with chronic hypertension, individuals with medical disorders, women taking hepatotoxic drugs, those with renal disease, liver disorders, hypothyroidism, hyperthyroidism, and urinary tract infections. These criteria aim to ensure a specific focus on antenatal primigravida with singleton pregnancies beyond 28 weeks and to exclude potential confounding factors that may impact the study's objectives.

Subjects were again subdivided into three categories on the basis of LDH levels.[12]

A: < 600 IU/L

B: 600-800 IU/L

C: >800 IU/L

Sub group A – non severe preeclampsia

Pregnant women of > 20weeks of gestation with blood pressure >140/90mmhg and <160/110mmhg noted first time during pregnancy on >2 occasions at least 6 hours apart.

Proteinuria of >1+ (>300mg/dl) by dipstick method in a random urine sample, after excluding urinary tract infection.

Subgroup B – severe preeclampsia

The presence of any of the following symptoms or indications:

- Systolic blood pressure measuring 160mmHg or higher, or diastolic blood pressure reaching 110mmHg or higher.
- Thrombocytopenia, characterized by a platelet count less than 100,000/microliter.
- Impaired liver function, as evidenced by abnormally elevated blood concentrations of liver enzymes (twice the normal concentration), severe and persistent right upper quadrant or epigastric pain unresponsive to medication and not explained by an alternative diagnosis, or both.
- Progressive renal insufficiency, indicated by a serum creatinine concentration exceeding 1.1mg/dL or a doubling of the serum concentration without other renal disease present.
- Pulmonary edema.
- Onset of new cerebral or visual disturbances.
- Spot urine protein–creatinine ratio greater than 0.3.

Procedure: Blood pressure measurements for women were conducted on the right upper limb in a semi-recumbent position, ensuring the arm was at heart level, using a mercury sphygmomanometer. Two-thirds of the arm were covered, and the arm cuff was positioned above the cubital crease by half an inch, with the tubes placed above the brachial artery. Systolic blood pressure was identified by the onset of tapping sounds (Korotkoff 1), while Korotkoff 5 (disappearance of the sound) was utilized to determine diastolic blood pressure.

The study involved two groups and subgroups categorized by age, gravidity, trimester, investigations, maternal and perinatal outcomes, complications, and follow-up. All participants were monitored from the time of enrollment until delivery, including the post-partum period, and newborns were followed until the neonatal period.

To estimate serum lactate dehydrogenase (LDH), plain blood samples were collected and analyzed using a fully automated biochemistry analyzer in the biochemistry laboratory. The method employed was based on the reduction of pyruvate to lactate in the presence of NADH by lactate dehydrogenase. The remaining pyruvate formed a complex with 2,4-dinitrophenylhydrazone, which was determined calorimetrically in an alkaline medium. The LDH level was calculated for all the study groups. Patients were followed until their delivery. Maternal outcome like mode of delivery, abruption placenta, PPH, renal failure, intra partum/post-partum eclampsia, HELLP syndrome, cerebral hemorrhage, pulmonary edema, maternal stay in the hospital, maternal death was also studied. In neonatal outcome gestational age, birth weight, APGAR, intra uterine growth restriction, birth asphyxia, fresh still birth, macerated still birth, NICU admission, early neonatal death was studied.

Statistical analysis: Following data collection, the information was entered into an Excel worksheet. Data analysis was conducted utilizing SPSS 18.0 and the R environment version 3.2.2. Quantitative data were presented using measures such as mean, standard deviation, median, and interquartile range. Analysis of variance (ANOVA) was employed to determine the significance of study parameters among three or more groups of patients. For categorical scale parameters between two or more groups, the Chi-square test or Fisher's exact test was utilized, especially in non-parametric settings for qualitative data analysis. Fisher's exact test was specifically applied when dealing with very small sample sizes.

Significant figures: suggestive significance (p value:$1.05<p<0.10$)

Moderately significant (p value: $0.01<p<0.05$)

strongly significant (p value: $p<0.01$).

RESULTS

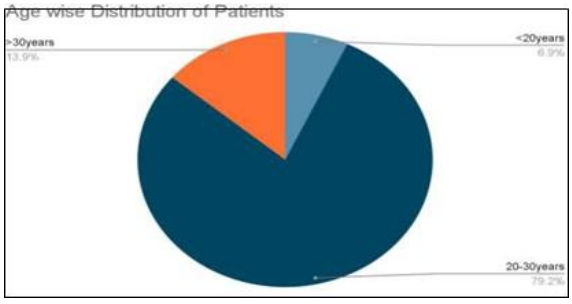

Figure 1: Age wise distribution of patients

The mean age of the Study population was 25.87±4.16years. Majority of the study population i.e. one hundred and three participants (79.23%) were aged between twenty-one and thirty years.

Out of the one hundred and thirty patients recruited in the study, thirty of them (23.08%) belonged to the

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normal group and thirty each (38.46%) belonged to SPE and NSPE group.

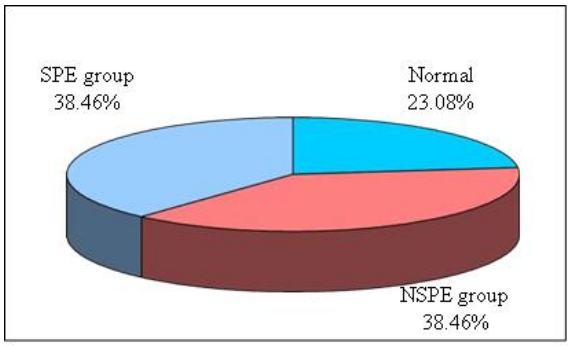

Figure 2: Group wise distribution of patients

In the SPE group, majority of them i.e. Thirty of them had a gestational age of above 37weeks, nine of them were having their period of gestation between34-37weeks and the remaining eleven were having their period of gestation between 28-34weeks. In the NSPE group also, the majority of them i.e. forty-four of them had their period of gestation above 37weeks of period of gestation, five of them had their period of gestation between 34-37weeks and one more participant had their period ofgestationof28-34weeks.In the normal group, all the thirty participants had their period of gestation above 37weeks. SPE was found in a greater number of patients at gestational age less than 37wks when compared to NSPE i.e. higher incidence of SPE was found at earlier gestational age than NSPE. [Table 1] All the thirty patients in the normal group had their systolic blood pressure (SBP) below 130 mm of hg, with a mean SBP of 122.70±1.99. Whereas in the NSPE group, out of the fifty participants, forty-nine of them had their SBP between 131-160mm oh hg and the remaining one participant had the SBP below 130mm of hg. The mean SBP in the NSPE group was 143.40±5.19 mm of hg. In the SPE group, the majority of them i.e. thirty-five of them had their SBP above 160 mm of hg and the remaining fifteen of them had their SBP between 131-160 mm of hg. the mean SBP in the SPE group was 148.24±19.04 mm of hg. This pattern of distribution of SBP was statistically highly significant with a p value of 0.0001. [Table 2]

In the Normal group, all the thirty patients had their diastolic blood pressure (DBP) below 90 mm of hg. The average DBP in the normal group was 86.67±88.17. In the NSPE group, all the fifty participants had their DBP between 91-110mm of hg. The average DBP was93.20±4.71mm of hg. In the SPE group, forty-nine of them had their diastolic blood pressure between 91-110 mm of hg and one participant had her DBP above 110mm of hg. The average DBP in the SPE group was 108.00±5.35 mm of hg. This pattern of distribution of diastolic blood pressure was statistically highly significant with a p value of 0.001. [Table 3]

In the normal group, all the thirty participants had their LDH below 600 units/L. In the NSPE group also, forty nine out of the fifty participants recruited in the study had their LDH levels below 600 units /L and the remaining one female had her LDH between 600-800 units/L. In the SPE group, sixteen of them had their LDH levels below 600 units/L, twenty-four of them had their LDH levels between 600-800 units/L and the remaining ten of them had their LDH above 800 units/L. This pattern of distribution of LDH levels was statistically significant with a p value of 0.0001. 34 out of 50 i.e. 68% in SPE group had LDH about 600 U/L. [Table 4]

Table 1: Comparison of three groups (Normal, NSPE and SPE) by gestational age in weeks

Gestational age in weeks	Normal	%	NSPE group	%	SPE group	%	Total	%
28.0-34.0	0	0.00	1	2.00	11	22.00	12	9.23
34.1-37.0	0	0.00	5	10.00	9	18.00	14	10.77
>=37.1	30	100.00	44	88.00	30	60.00	104	80.00
Mean	39.02		38.55		36.91		38.02	
SD	1.10		1.81		2.91		2.36	
Total	30	100.00	50	100.00	50	100.00	130	100.00
Chi-square=24.5190, p=0.0001, S								

Table 2: Comparison of three groups (Normal, NSPE and SPE) by SBP in mm Hg

SBP (in mm Hg)	Normal	%	NSPE group	%	SPE group	%	Total	%
<=130	30	100.00	1	2.00	0	0.00	31	23.85
131-160	0	0.00	49	98.00	15	30.00	64	49.23
>=161	0	0.00	0	0.00	35	70.00	35	26.92
Mean	122.70		143.40		168.40		148.24	
SD	1.99		5.19		9.34		19.04	
Total	30	100.00	50	100.00	50	100.0	130	100.0
Chi-square=193.5720, p=0.0001, S								

Table 3: Comparison of three groups (Normal, NSPE and SPE) with DBP in mm Hg

DBP in mm Hg	Normal	%	NSPE group	%	SPE group	%	Total	%
<=90	30	100.00	0	0.00	0	0.00	30	23.08
91-110	0	0.00	50	100.00	49	98.00	99	76.15
>=111	0	0.00	0	0.00	1	2.00	1	0.77
Mean	86.67		93.20		108.00		97.38	
SD	88.17		4.71		5.35		9.87	

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Total	30	100.00	50	100.00	50	100.00	130	100.00
Chi-square=131.3130, p=0.001								

Table 4: Comparison of three groups (Normal, NSPE and SPE) by LDH

LDH (U/L)	Normal	%	NSPE group	%	SPE group	%	Total	%
<600	30	100.00	49	98.00	16	32.00	95	73.08
600-800	0	0.00	1	2.00	24	48.00	25	19.23
>800	0	0.00	0	0.00	10	20.00	10	7.69
Total	30	100.00	50	100.00	50	100.00	130	100.00
Chi-square=69.7790, p=0.0001, S								

Table 5: Comparison of three groups (Normal, NSPE and SPE) by mode of delivery

Mode of delivery	Normal	%	NSPE group	%	SPE group	%	Total	%
LSCS	8	26.67	27	54.00	35	70.00	70	53.85
VD	22	73.33	23	46.00	15	30.00	60	46.15
Total	30	100.0	50	100.00	50	100.00	130	100.0
Chi-square=14.1680, P=0.0010, S								

Table 6: Association between LDH levels and mode of delivery in NSPE+SPE group

LDH	LSCS	%	VD	%	Total	%
<600	34	52.31	31	47.69	65	65.00
600-800	21	84.00	4	16.00	25	25.00
>800	7	70.00	3	30.00	10	10.00
Total	62	62.00	38	38.00	100	100.00
Chi-square= 15.2730 P = 0.0001, S						

In the normal group, majority of them i.e. twenty-two of them (73.33%) underwent vaginal delivery and the remaining eight participants (26.67%) underwent LSCS. In the NSPE group, twenty-three of them underwent vaginal delivery and the remaining twenty-seven (54%) of them underwent LSCS. In the SPE group, only fifteen of them (30%) were delivered by VD and the remaining thirty-five of them (70%) underwent LSCS. This pattern of distribution of mode of delivery was statistically significant with a p value of 0.001. More number of women in SPE group (70%) underwent LSCS when compared to women in NSPE group (54%). [Table 5] Amongst those who had their LDH below 600 units/L, thirty-one of them (47.69%) had a vaginal delivery while the remaining thirty-four participants (52.31%) delivered by LSCS. Amongst those who had their LDH levels between 600-800 units/Liter, majority of them i.e. twenty-one (84%) delivered by LSCS and only four of them (16%) delivered by vaginal delivery. Amongst those who had their LDH more than 800 units/Liter, seven of them (70%) delivered by LSCS and the remaining three of them delivered by vaginal delivery. This pattern of distribution of mode of delivery in comparison to LDH levels was statistically highly significant with a p value of 0.0001. more number of patients with LDH more than 600U/L underwent LSCS when compared to vaginal delivery. [Table 6]

DISCUSSION

Hypertensive disorders of pregnancy affect roughly 6-8% of all pregnancies, with preeclampsia and eclampsia posing the severe issues. Lactate Dehydrogenase (LDH) is an enzyme found within cells.[13] In recent times, LDH has been proposed as a potential marker for predicting the severity of pre-eclampsia. Consequently, the current study aimed to establish correlations between maternal and perinatal outcomes and serum LDH levels. The primary objective was to evaluate serum LDH as an indicator of the severity of preeclampsia.[14]

The mean age of the recruited samples in our study was 25.87±4.16 years. Majority of the study population 103/130 (79.23%) were aged between twenty-one to thirty years. Out of the one hundred and thirty patients recruited in the study, thirty of them (23.08%) belonged to the normal group and fifty each (38.46%) belonged to SPE and NSPE groups. Similar to our study, the majority of the study sample in Gupta A et al were also aged between 25 to 30 years in both the groups.[15] Even Jaiwer SP et al also did not find any statistical difference in the demographic details.[12] Tessema GA et al., had reported women over 35 years old have 4, 5-fold risk of suffering preeclampsia compared to women aged 25-29 years, based on their observation.[16] Tyas BD et al., also found the higher prevalence of preeclampsia, cesarean delivery and the poor neonatal outcome in advanced maternal age group (p <0.004) compared to the women aged between 21- to 29-year-old. We could not find such correlation in our study.[16]

In our study, the majority of the participants in both groups were presented with a gestational age of above 37 weeks.

In Qublan HS et al confirmed in their study that the mean LDH levels in normal group was 299 ± 79 IU/L, whereas the women with mild preeclampsia was 348 ± 76 IU/l and in severe preeclampsia was 774 ± 69.61 IU/L.[17]

All the thirty patients in the normal group had their systolic blood pressure (SBP) below 130 mm of hg, with a mean SBP of 122.70±1.99. Whereas in the NSPE group, the majority of them had SBP between

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141-160mm of Hg with the mean SBP in the NSPE group was 143.40±5.19 mm of Hg. Whereas in the SPE group, the 35/50 were presented with SBP >160 mm of Hg, with the mean SBP of the group being 148.24±19.04 mm of hg. This pattern of distribution of SBP was statistically highly significant with a p value of 0.0001. Also, in Jaiswar SP et al, the majority of the patients with LDH <600 IU/l, had normal SBP.[12] Patients with LDH between 600 and 800 IU/l, majority (61.54%) of them were presented with SBP>160. In the remaining 36 patients with LDH levels >800 IU/l, 61.11% had systolic BP 160 and above.

In our study, all the samples in the normal group had their diastolic blood pressure (DBP) below 90 mm of Hg with the mean DBP of 86.67±88.17. In the NSPE group, all the fifty participants had their DBP between 91-110mm of Hg with the average DBP of 93.20±4.71mm of Hg. In the SPE group, 49/50 patients had their DBP between 91-110 mm of Hg and one participant had her DBP above 110mm of hg. The average DBP in the SPE group was 108.00±5.35 mm of hg. This pattern of distribution of diastolic blood pressure was statistically highly significant with a p value of 0.001. Even the Jaiswar SP et al also observed the similar findings.[12] Bhandari N et al also observed significantly increased blood pressure among patients with increased LDH levels than a normotensive.[18]

Based on our observations, none of the patients in the normal group had albuminuria. In the NSPE group, 76% had a urine albumin of 1+ and 22% were found with traces. Only one had a urine albumin of 2+. In the SPE group, only 4% had a urine albumin of 1+, 34% of them had a urine albumin of 2+, 32% found with 3+ and the remaining 30% of them had a urine albumin of 4+. This pattern of distribution of albuminuria was statistically highly significant with a p value of 0.0001. Even Dong X et al also reported increased severity of preeclampsia after the albumin level reaches more than 0.3g/L, significantly increases. Additionally, they found that birth weights were significantly lower in patients with proteinuria >3g/L. There was a significant increase in the frequency of stillbirth or fetal development restriction in patients with proteinuria more than 5g/L.[18]

In our study, in the normal group, all the participants had their LDH <600 units/L. In the NSPE group 98% participants recruited in the study had their LDH levels <600 units /L. Whereas in the SPE group, 32% were with LDH levels <600 units/L, 48% of them had their LDH levels between 600-800 units/L and the remaining 20% of them had their LDH above 800 units/L, which was statistically significant with a p value of 0.0001. Gupta A et al also observed higher levels of LDH in pregnant women with preeclampsia, which was about 627.38±230.04 IU/l and 224.43±116.61 IU/l among the normal pregnant women.[15]

It was discovered that 26.67% of deliveries in the normal group were made via LSCS, while 73.33% of deliveries took place vaginally (VD). Of the NSPE group, VD delivered 46% and LSCS delivered 54% of the babies. In contrast, only 30% of the SPE group had VD, with the remaining 70% receiving LSCS. With a p value of 0.001, it was statistically significant that 70% of the women in the SPE group and 54% of the women in the NSPE group had undergone LSCS. In the research by Gupta A et al., there was a noteworthy correlation found between the manner of delivery and LDH levels in preeclamptic women. In other words, of women having LDH levels <600 IU/L, 73.58% gave birth vaginally, whereas of those having LDH levels >600 IU/L, 65.9% had a caesarean section and the rest gave birth vaginally. There was a statistically significant connection ($p < 0.001$) between the rate of caesarean sections and LDH levels >600 IU/L.[15]

In the normal group of our study, the majority of the participants 80% delivered a baby with weight >2.5kg and 20% with a baby weighing 1.5-2.5kgs. In the NSPE group, 68% delivered a baby weighing >2.5kg and 30% delivered a baby weighing 1.5-2.5kgs and the remaining 2% delivered a baby weighing <1.5kgs. Whereas in the SPE group, 36% delivered a baby weighing >2.5kgs and 50% delivered a baby weighing between 1.5 and 2.5kgs. 14% had delivered a baby weighing <1.5kgs. A higher proportion of the babies were born with a lesser birth weight in the SPE group when compared to the NSPE group, statistically significant with a p value of 0.0001.

No complications were seen among the patients in the normal group whereas in the SPE group, 16% had Acute Kidney Injury (AKI), 14% each had antepartum and postpartum, other 14% needed ICU admission, 6% each had PPH and HELLP, 2 had abruption (4%) and 1 case had pulmonary edema (2%) while in the non SPE group only one patient had PPH.

We found that IUGR, prematurity, NICU admission, RDS were the important problems in the newborn which also contributed to the prolonged hospital stay. IUD occurred in 4 patients and Neonatal death in 2 cases.

There were no neonate related complications found among the normal group. In the NSPE group, IUGR and preterm births occurred in 14% of the samples each. Three of the babies had respiratory distress at birth, six neonates required NICU admission, four babies required prolonged hospital admission. The most common neonatal morbidity found in the SPE group was intrauterine growth restriction (IUGR) in 84% of the babies, followed by preterm delivery in 36% of them; NICU admission in fourteen of them, twelve of the babies had respiratory distress, four intrauterine deaths and two neonatal deaths. More neonatal problems including IUD were encountered in the SPE group when compared to the NSPE group. 47.69% amongst those who had their LDH <600 units/L had vaginal delivery while the remaining 52.31% delivered by LSCS. Amongst those who had their LDH levels between 600-800 units/Liter, 84%

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delivered by LSCS and only 16% delivered by VD. Amongst those who had their LDH >800 units/Liter, 70% had delivered by LSCS and the remaining three of them delivered by VD. More patients with LDH >600 units/L underwent LSCS when compared to vaginal delivery with a statistically significant p value of 0.0001. In Kharb S et al.'s study, it was observed that 66% of patients in the mild preeclampsia group had LDH levels of <600 IU/L, while 30.8% had LDH levels >600 IU/L. Among the 49 patients with severe preeclampsia, 62 had LDH levels >600 IU/L. The analysis of this data indicated a rise in LDH levels corresponding to the increasing severity of preeclampsia. Similar to our study, Bhandari et al also observed increased morbidity among the patients with >800 IU/L with statistically significant p value of <0.05. Whereas Vyas NP et al observed the significant difference between HELLP syndrome, DIC, Pulmonary edema and the raised LDH.[19]

Amongst the 7 babies which weighed <1.5kgs, 42.86% delivered by LSCS and the 57.14% were delivered by VD. Amongst the twenty-five babies weighing between 1.5 to 2.5kgs, 76% were delivered by LSCS and the remaining 24% were delivered by vaginal delivery. Whereas 72.2% of the neonates of >2.5kgs delivered by LSCS and the remaining five of them (27.78%) were delivered by vaginal delivery.

In both NSPE and SPE groups, 52/100 babies were>2.5kgs, 40 babies were between 1.5 and 2.5kgs and the remaining eight babies were less than 1.5kg.

Amongst those who had their LDH levels <600 units/L, only one patient had prolonged hospital admission. The patients with LDH levels between 600-800 units/L, eleven of them had prolonged hospital admission, followed by postpartum eclampsia in five of them. Amongst those who had their LDH >800 mg/dl, the most common morbidity was also prolonged hospital stays in nine of them followed by need for ICU admission and acute kidney injury in six of them each. Similar to our findings, when comparing severely preeclamptic women with LDH levels >800 IU/L to those with lower levels (<600 IU/L), Kharb S et al. found a significant increase in a number of maternal complications, including eclampsia, abruption, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelet count), disseminated intravascular coagulation, and transfer to the intensive care unit. On the other hand, there was no discernible rise in intrauterine growth restriction (IUGR) or premature labour associated with elevated LDH levels. Additionally, we observed a greater prevalence of maternal problems in our trial, with abruption and HELLP syndrome being much more common (P<0.001). Similarly, among patients with LDH >800 IU/L, abruption was the second most common consequence in Dev SV et al.'s research, HELLP syndrome being the most common. Rising blood LDH levels have also been linked to an increase in maternal problems, such as a greater risk of abruption, renal failure, HELLP syndrome, and pulmonary edema, according to studies by Umasatyasri et al. and Andrews L et al. Amongst those who had their LDH levels <600 units/L, the most common neonatal complication was IUGR in four of the babies followed by preterm delivery in three babies, IUD, prolonged hospital stays, need for NICU admission and respiratory distress in each. Amongst those who had LDH levels between 600-800mg/dl, the most common complications were IUGR and preterm delivery in thirteen babies each followed by need for NICU admission in nine of the babies, respiratory distress in seven babies, prolonged hospital stay in six babies, one IUD and one neonatal death. Whereas the patients with LDH levels >600 U/L, the most common complication was preterm delivery in nine of them, followed by IUGR in seven babies, respiratory distress, need for NICU admission and prolonged hospital stay in four babies each, IUD in two babies and one neonatal death.

While Kharb S et al. did not observe significant differences between subgroups of preeclampsia based on LDH levels concerning Apgar at 1 minute, birth asphyxia, NICU admission rates, and stillbirth, an overall significant difference was noted in terms of sepsis, mortality, and neonatal deaths (P< 0.01, <0.001, and <0.05, respectively). These findings align with our study. Similarly, Vyas NP et al. also reported statistically significant associations between increasing LDH levels and fetal complications, including Fetal Growth Restriction (FGR), NICU admission, and Apgar scores less than 7 at 5 minutes.[19] Even in Dev SV et al., the NICU admission rates and perinatal deaths were significantly higher with a p value of 0.001 in babies whose mothers had elevated LDH levels.[20] Andrew et al also confirmed the association of low birth weight of infants with the pregnant women with elevation in serum.[21]

CONCLUSION

LDH levels are significantly correlated with both the maternal and foetal prognosis, as well as the severity of preeclampsia. Patients with LDH levels more than 600 IU/L had a higher frequency of maternal and foetal problems. Because serum LDH correctly reflects the problems faced by the mother and the fate of the foetus, it can thus be a helpful biochemical diagnostic to predict the severity of preeclampsia.

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