ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Maternal Serum Lactate Dehydrogenase andits Correlation with Maternal andFetal Outcomes in Women with Pregnancy Induced Hypertension-A Prospective Study

Cheena vaishnav[1], Sushma Mogri[2], Anchal Vaishnav[3*], Pragya Gupta[4], Neelam Toshniwal[5]

[1]Third year resident, Department of Obstetrics and Gynaecology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India
[2]Professor and unit head, Department of Obstetrics and Gynaecology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India
[3]Final year MBBS student, Pacific Medical College and Hospital, Udaipur, Rajasthan, India
[4]Resident Doctor, Department of Obstetrics and Gynaecology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India
[5]Assistant Professor, Department of Obstetrics and Gynaecology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India

***Corresponding author:**
Anchal Vaishnav, Final year MBBS student, Pacific Medical College and Hospital, Udaipur, Rajasthan, India

Abstract

Aims and objectives: In the present study,we evaluated the level of S. lactate dehydrogenase and its correlation with maternal and fetal outcomes in women with pregnancy induced hypertension.

Material and methods: This is a prospective study which was conducted in the department of Obstetrics and Gynecology in collaboration with the department of Biochemistry Geetanjali Medical College and Hospital, Udaipur for 12 months. Pregnant women with hypertension >20 weeks of pregnancyadmitted and maternity ward and women with PIH (pregnancy induced hypertension) delivering in emergency were enrolled in this study.

Results and conclusion: Increased LDH levels is associated with early intervention in preeclampsia and eclampsia patients –lowered gestational age. Increased LDH levels is associated with organ damage in preeclampsia and eclampsiapatients – high urine albumin levels. Increased LDH levels is associated with poor maternal outcomes in preeclampsia and eclampsia patients –high incidence of HELLP and abruption. Increased LDH levels is associated with poor perinatal outcomes in preeclampsia and eclampsia patients –high incidence of IUGR and IUD. Increased LDH levels is associated with low baby weight in preeclampsia andeclampsia patients. Increased LDH levels is associated with high emergency LSCS rates in preeclampsiaand eclampsia patients. In conclusion, LDH levels reflect the severity of preeclampsia and the occurrence ofcomplications.

1. INTRODUCTION

Pregnancy causes profound anatomical, physiological and metabolic changes in maternal tissue. These well-orchestrated changes can go wrong at some stage of pregnancy resulting in various feto-maternal complications. One of the commonest and most dreaded complications is hypertension (preeclampsia, gestational hypertension) which can further complicate to eclampsia. They still however continueto be a major cause of mortality in developing countries. Preeclampsia is defined as a pregnancy-specific multi-systemic syndrome of widespread endothelial malfunction and vasospasm developing after 20 weeks of gestation. It is a rapidly

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

progressive condition traditionally defined by increasedblood pressure (140/90 mm Hg), fluid retention and proteinuria. The ACOG (2013)revised guidelines define preeclampsia as a de-novo and abrupt onset persistent hypertension associated with proteinuria or pathological edema or thrombocytopeniaor impaired liver or kidney function or new onset of cerebral or visual disturbances. Globally, preeclampsia is a leading cause of maternal and infant illness and mortality claiming up to 76,000 maternal and 500,000 infant deaths per year, according to conservative estimates. In India, the incidence of preeclampsia is 8% to 10 % among pregnant women.

The major challenge posed by preeclampsia is its sudden and acute onset often withoutdefinitive symptoms. The major symptoms of preeclampsia, such as rise in blood pressure (BP), severe headache, nausea, vomiting, blurring of vision, light sensitivity, are highly nonspecific. Multifactorial risk factors like nulliparity, multifetal gestations, obesity, diabetes mellitus, maternal age above 35 years are associated withpreeclampsia. Hence risk assessment remains an enigma, and a delay in diagnosis often leads to severe maternal and neonatal complications encompassing IUD, foetal growth restriction, preterm birth, placental abruption, HELLP syndrome, eclampsia, maternal coma and even death.

Various causes that lead to these abnormalities have been proposed. These include immunological, genetic, and dietary causes, race, increased oxidative stress, and prostaglandin imbalance'. It carries substantial risks for both fetus and mother with a subsequent increase in the perinatal and maternal morbidity and mortality'.

Lactate dehydrogenase (LDH) is a cytoplasmic enzyme that is widely expressed in tissues and cells. LDH is an enzyme in the glycolytic pathway catalyzes the oxidationof L-lactate to pyruvate with the mediation of nicotinamide adenine dinucleotide (NAD+) as the hydrogen acceptor. This reaction is reversible and can be detected in the laboratory in serum samples by measuring LDH activity in terms of the rate of dihydronicotinamide adenine dinucleotide dehydrogenase (NADH) productiondetermined spectrophotometrically at 340 nm.

LDH is a critical serologic marker for diagnosis, staging/prognosis, and recurrence, and monitoring of germ cell tumors, as well as for multiple myeloma, another malignant disease wherein high LDH levels are associated with disease severity and poor prognosis [20-21]. Serum LDH levels increase in proportion to the clinical severity of idiopathic pulmonary arterial hypertension and have a strong, independent association with the long-term mortality of these patients. Assessing the potential roleof LDH as a biomarker and mediator involved in the pathogenesis of idiopathic arterial hypertension might be worthwhile [22]. LDH has had an exciting journey as a utility marker in different illnesses, but currently, its clinical utility has been relegatedto confirm hemolysis, as a tumor marker, and as a diagnostic biomarker of preeclampsia (PE). However, the findings of LDH concentrations taking reference values to healthy persons are not consistent when these are related to hypertensive disorders in pregnancy (HDP), mainly to begin symptoms or mild PE.

The HDP are among the leading causes of maternal and perinatal morbidity and mortality worldwide. The public classification system was adopted by the NationalHigh Blood Pressure Education Program (NHBPEP) Working Group in 1990 and subsequently endorsed by 46 medical organizations. The updated version in 2000 hasbecome a standard that the American College of Obstetrics and Gynecology (ACOG2016) follows. From the NHBPEP original reports, guidelines from international societies have emerged, each one with their evidence, although many with similar recommendations. The HDP should be classified as pre-existing hypertension, gestational hypertension, preeclampsia, or others hypertensive effects based on different diagnostic and therapeutic considerations. Hypertension in pregnancy is defined by systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90mmHg.

The link between abnormalities in trophoblast invasion and generalized maternal endothelial dysfunction seen in HDP, particularly in preeclampsia, maybe via release of placental factors,

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

such as syncytial knots, shedding of syncytiotrophoblast basement membrane fragments (STBM), leukocyte and platelet membrane particles, activated neutrophils, cytokines, growth factors, angiogenic factors, and hormones. These factors will interact with the maternal vascular endothelium, which may already be damaged and can cause maternal endothelial cell damages. The STBM mayalso damage the endothelium and activate neutrophils, and this may lead to endothelialdysfunction as part of the widespread intravascular inflammation. Evidence for endothelial dysfunction in preeclampsia includes reduced in-vitro endothelium- dependent dilatation of isolated vessels, increased vascular reactivity in response to vasoconstrictor stimuli, and elevated biomarker levels associated with endothelial activation and injury. Detection of high-risk patients with increased LDH levels, as a marker of endothelial damage by HDP, mandates close monitoring and correct management to decrease both maternal and fetal morbidities. In the present study,we evaluated the level of S. lactate dehydrogenase and its correlation with maternal and fetal outcomes in women with pregnancy induced hypertension.

2. MATERIALS AND METHOD

This is a prospective study which was conducted in the department of Obstetrics and Gynecology in collaboration with the department of Biochemistry Geetanjali Medical College and Hospital, Udaipur for 12 months

Study Design: Prospective cross sectional Study.

Study Period: Sept, 22 to Aug, 23.

Study Place: Outpatient Department and In Patient department of Geetanjali Medical College and Hospital, Udaipur.

Study Population: Pregnant women with hypertension >20 weeks of pregnancyadmitted and maternity ward and women with PIH (pregnancy induced hypertension) delivering in emergency were enrolled in this study.

Sample Collection: Blood samples were collected aseptically for analysis of Serum LDH along with routine blood investigations. All women were followed until deliveryand early postpartum period and babies till early neonatal period.

Sample size calculation: Prevalence of pregnancy induced hypertension is 6.9% among pregnant women. $Z\alpha$ = 1.96 at 95% confidence level. E = 6% be absolute error

$n=(Za+Z1-\beta)^2P(1-P)]E^2=140$ sample size **Inclusion Criteria**

- Pregnant women > 20weeks of gestation with hypertension.
- Pregnant women between 18-35 years.
- Singleton pregnancy.

Exclusion Criteria

- Essential hypertension as suggested by: (i) history or documentation of hypertensionin the pre-pregnant state; (ii) hypertensive present before 20 weeks of gestation.
- Renal diseases.
- Liver diseases.
- Thyroid disorder.
- Pre-existing diabetes mellitus.
- Coincidental seizures in pregnancy
- History or documentation of epilepsy in pre-pregnant state.
- Space occupying lesion in brain like tuberculoma, or brain tumor.
- Trauma to brain
- Hyperpyrexia.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

A prospective study in the department of obstetrics and gynaecology in collaboration with department of biochemistry in Geetanjali Medical College and Hospital, Udaipur,based on serum LDH levels in preeclampsia patients. informed consent was obtained from the patients. Detailed history including age, parity, previous medical disorders was elicited followed by physical examination including the measurement of blood pressure in the right arm, in sitting position with appropriate size cuff. patients with systolic blood pressure >140 mm Hg and diastolic blood pressure of >90 mm Hg and those with proteinuria, measured by urine dipstick ≥1+ /24-hour urinary protein ≥300mg/24hours were enrolled in the study based on inclusion and exclusion criteria 140 antenatal mothers with preeclampsia were subjected to the study. Then under aseptic precaution 3ml of blood sample were taken from the patients who were enrolledin the study and sent for serum lactate dehydrogenase assay.
The subjects were monitored from the time their serum LDH levels were assessed untilthe point of delivery and the early postpartum period. During this period, they were evaluated for the occurrence of complications such as eclampsia, HELLP syndrome, disseminated intravascular coagulation, placental abruption, cerebrovascular accidents,pulmonary edema. Additionally, the condition of the babies was closely observed throughout the early neonatal period, with a focus on outcomes including intrauterine death, intrauterine growth retardation, low APGAR scores, fetal distress, neonatal death, and premature birth.

Statistical analysis
The data obtained were entered in MS Excel software. Continuous variables were describe first and then compared with three groups using ANOVA and chi-square test. $P < 0.05$ was considered statistically significant. It was carried out by SPSS version 21.

3. OBSERVATIONS AND RESULT

Table: 1 Classification of study group

Study Groups	Number	Percentage
Control	64	32.00
Mild Pre-eclampsia	62	31.00
Severe Pre-eclampsia	60	30.00
Eclampsia	14	7.00
Total	200	100.00

In this study 64 (32%) were included in control group, 62 (31%) were in mild pre- eclampsia group, 60 (30%) were in severe eclampsia group and 14 were in eclampsia group.

Table: 3 Age distribution & Association in Mild Pre-Eclampsia, Severe Pre- Eclampsia, Eclampsia & Control group

Age Distribution	Mild Pre-Eclampsia	Severe Pre-Eclampsia	Eclampsia	Control
Mean	25.31	25.37	24.73	23.96
SD	3.41	3.67	2.79	3.32
P value ANOVA			257	

When analyzing the age distribution among the study patients, we observed that the majority of individuals in the mild pre-eclampsia group fell within the 21-30 years ageclass interval (n=45, 72.58%), followed by those aged ≤ 20 years (n=10, 16.13%), withan average age of 25.31 (±3.41) years.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

In the severe pre-eclampsia group, most study subjects also belonged to the 21-30 yearsage class interval (n=48, 80.00%), followed by the 31-40 years age class interval (n=6,10%), with an average age of 25.37 (±3.67) years.
For the eclampsia group, the majority of study subjects fell within the 21-30 years ageclass interval (n=10, 71.43%), followed by the 31-40 years age class interval (n=4, 28.57%), with an average age of 24.73 (±2.79) years.
In the control group, most study subjects were in the 21-30 years age class interval (n=60, 93.75%), followed by the 31-40 years age class interval (n=8, 12.50%), with anaverage age of 23.96 (±3.32) years.
Statistical analysis using a one-way ANOVA test did not reveal a statistically significant association between age distribution and study groups (p = 0.257).

Table: 4 Parity status & Association in Mild Pre-Eclampsia, Severe Pre- Eclampsia, Eclampsia & Control group

Parity Status	Mild Pre-Eclampsia N (%)	Severe Pre-Eclampsia N (%)	EclampsiaN (%)	ControlN (%)
Primigravida	40 (64.52)	34 (56.67)	11 (78.57)	36 (56.25)
Multigravida	22 (35.48)	26 (43.33)	3 (21.43)	28 (43.75)
Total	62 (100.00)	60 (100.00)	14 (100.00)	64 (100.00)
P value Chi Squared Test			0.456	

During the analysis of the parity status among the study patients, the followingobservations were made:
In the mild pre-eclampsia group, the majority of subjects were primigravida (n=40, 64.52%), while the remaining were multigravida (n=22, 35.48%).
For the severe pre-eclampsia group, a significant proportion of study subjects were categorized as primigravida (n=34, 56.67%), with the remaining being multigravida (n=26, 43.33%).
Similarly, in the eclampsia group, the majority of participants fell under the primigravida category (n=11, 78.57%), while a smaller proportion were multigravida (n=3, 21.43%).
In the control group, the majority of participants were classified as primigravida (n=36, 56.25%), while the rest were multigravida (n=28, 43.75%).
Statistical analysis using a chi-squared test revealed that there was no statistically significant association between parity status and the different study groups (p = 0.456).

Table: 6 Gestational Age Distribution & Association in Mild Pre- Eclampsia, Severe Pre-Eclampsia, Eclampsia & Control group

Gestational Age Distribution	Mild Pre-Eclampsia	Severe Pre-Eclampsia	Eclampsia	Control
Mean	36.83	35.47	34.90	37.61
SD	1.87	2.11	2.37	1.02
P value ANOVA – Single Factor			**0.001 (S)**	

During the analysis of gestational age distribution among the study patients, thefollowing trends were identified:
In the mild pre-eclampsia group, the majority of study subjects were in the 38-40 weeks gestational age range (n=36, 58.06%). A substantial number fell within the 34-37 weeks gestational age range (n=20, 32.26%). The mean gestational age for this group was 36.83

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

(±1.87) weeks.

In the severe pre-eclampsia group, most study subjects were clustered in the 34-37 weeks gestational age range (n=33, 55%). A smaller portion belonged to the 38-40 weeks gestational age range (n=20, 33.33%). The mean gestational age for this group was 35.33 (± 35.47) weeks. For the eclampsia group, the majority of study subjects had gestational ages falling within the 34-37 weeks range (n=10, 71.43%), while a minority were in the 38-40 weeks gestational age range (n=2, 14.29%). The mean gestational age for this group was 34.90 (±2.37) weeks.
In the control group, a significant majority of study subjects were in the 38-40 weeks gestational age range (n=50, 78.13%), with a smaller proportion in the 34-37 weeks gestational age range (n=10, 15.63%). The mean gestational age for this group was 37.61 (±1.02) weeks.

Statistical analysis using an ANOVA single-factor test revealed a statistically significant association between gestational age distribution and study groups ($p < 0.001$).

Table: 12 LDH comparison in Mild Pre-Eclampsia, Severe Pre-Eclampsia,Eclampsia & Control group

Lactate Dehydrogenase	Mild Pre-Eclampsia N (%)	Severe Pre-Eclampsia N (%)	EclampsiaN (%)	ControlN (%)
≤ 600 IU	50 (80.65)	13 (21.67)	0 (0.00)	64 (100.00)
601-800 IU	12 (19.35)	18 (30.00)	3 (21.42)	0 (0.00)
801-1000 IU	0 (0.00)	11 (18.33)	2 (14.29)	0 (0.00)
> 1000 IU	0 (0.00)	18 (30.00)	9 (64.29)	0 (0.00)
Total	62 (100.00)	60 (100.00)	14 (100.00)	64 (100.00)

Table: 13 LDH distribution *& Association in Mild Pre-Eclampsia, Severe Pre-Eclampsia, Eclampsia & Control group*

Lactate Dehydrogenase Distribution	Mild Pre-Eclampsia	Severe Pre-Eclampsia	Eclampsia	Control
Mean	398.92	855.93	1310.32	225.16
SD	151.23	196.34	243.43	79.23
P value ANOVA			**0.001 (S)**	

During the analysis of lactate dehydrogenase (LDH) distribution among the study participants, the following trends were identified:
In the mild pre-eclampsia group, the majority of study subjects had LDH levels withinthe ≤ 600 IU range (n=50, 80.65%). A smaller proportion fell into the 601-800 IU LDHrange (n=12, 19.35%). The mean LDH level for this group was 398.92 (±151.23) IU.
For the severe pre-eclampsia group, most study subjects had LDH levels in the 601-800IU & >1000 IU range (n=18, 30.00%). A notable portion belonged to the ≤ 600 IU LDHrange (n=13, 21.67%). The mean LDH level for this group was 855.93 (±196.34) IU.
In the eclampsia group, the majority of study subjects exhibited LDH levels greater than 1000 IU (n=9, 64.29%), while a minority were in the 601-800 IU LDH range (n=3,21.42%). The mean LDH level for this group was 1310.32 (±243.43) IU.
In the control group, all study subjects had LDH levels within the ≤ 600 IU range (n=64, 100.00%). The mean LDH level for this group was 225.16 (±79.23) IU.
Statistical analysis using an ANOVA single-factor test indicated a statistically significant association between LDH distribution and study groups ($p < 0.001$).

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Table: 14 Maternal outcome & Association in Mild Pre-Eclampsia,Severe Pre-Eclampsia, Eclampsia & Control group

MaternalOutcome	Mild Pre-Eclampsia (N=62) N (%)	Severe Pre-Eclampsia (N=60) N (%)	Eclampsia (N=14) N (%)	Control N (%)	P value
HELLP	0 (0.00)	4 (6.67)	5 (35.71)	0 (0.00)	**0.001 (S)**
Abruption	3 (4.80)	16 (26.70)	2 (14.30)	0 (0.00)	**0.001 (S)**
PulmonaryEdema	0 (0.00)	1 (1.70)	2 (14.30)	0 (0.00)	NA
Maternal Death	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	NA

During the analysis of maternal outcome status among the study participants, the following results were observed:

No cases of HELLP syndrome were observed in the mild pre-eclampsia or control groups, while it occurred in 6.67% of severe pre-eclampsia cases and 35.71% of eclampsia cases ($P < 0.001$). Abruption was seen in 4.80% of mild pre-eclampsia cases, 26.70% of severe pre-eclampsia cases, and 14.30% of eclampsia cases, with nooccurrences in the control group ($P < 0.001$). Pulmonary edema was absent in the mildpre-eclampsia and control groups but occurred in 1.70% of severe pre-eclampsia casesand 14.30% of eclampsia cases. Notably, there were no maternal deaths reported acrossany groups.

Table: 15 Perinatal outcome & Association in Mild Pre-Eclampsia, Severe Pre-Eclampsia, Eclampsia & Control group

Perinatal Outcome	Mild Pre-Eclampsia (N=62) N (%)	Severe Pre-Eclampsia (N=60) N (%)	Eclampsia (N=14) N (%)	Control N (%)	P value
IUGR	3 (4.84)	28 (46.67)	8 (57.14)	2 (3.13)	**0.001 (S)**
IUD	0 (0.00)	3 (5.00)	1 (7.14)	0 (0.00)	**0.001 (S)**

In the analysis of prenatal outcome status among the study participants, the following results were observed:

In the mild pre-eclampsia group, the majority of study subjects experienced intrauterinegrowth restriction (IUGR) as an adverse perinatal outcome (n=3, 4.84%).

For the severe pre-eclampsia group, a significant majority of study subjects had IUGRas an adverse perinatal outcome (n=28, 46.67%), while a few experienced intrauterinedeath (IUD) (n=3, 5%).

Within the eclampsia group, the majority of study subjects had IUGR as an adverse perinatal outcome (n=8, 57.14%), and a smaller number experienced intrauterine death(IUD) (n=1, 7.14%).

In the control group, a small proportion of study subjects experienced intrauterine growth restriction (IUGR) as an adverse perinatal outcome (n=2, 3.13%).

Statistical analysis using a chi-squared test demonstrated a statistically significant association between perinatal outcome status, specifically IUGR and IUD, and the study groups ($p < 0.001$).

Table: 17 Baby Weight distribution & Association in Mild Pre-Eclampsia, SeverePre-Eclampsia, Eclampsia & Control group

Baby Weight Distribution	Mild Pre-Eclampsia	Severe Pre-Eclampsia	Eclampsia	Control

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Mean	2.18	2.79	1.62	2.62
SD	0.21	0.49	0.31	0.19

P value=0.001 (S)

During the analysis of baby weight distribution among the study participants, the following trends were identified:

In the mild pre-eclampsia group, the majority of study subjects had baby weights fallingwithin the 2.1-3.0 kgs range (n=45, 72.58%), with a smaller proportion in the 3.1-4.0 kgs range (n=11, 17.74%). The mean baby weight for this group was 2.18 (±0.21) kgs.

For the severe pre-eclampsia group, most study subjects had baby weights in the rangeof 1.1-2.0 kgs (n=30, 50%), followed by those in the 2.1-3.0 kgs range (n=24, 40%). The mean baby weight for this group was 2.79 (±0.49) kgs.

In the eclampsia group, the majority of study subjects had baby weights in the range of1.1-2.0 kgs (n=10, 71.43%), with a smaller portion in the 2.1-3.0 kgs range (n=4, 28.57%). The mean baby weight for this group was 1.62 (±0.31) kgs.

In the control group, a significant majority of study subjects had baby weights in the range of 2.1-3.0 kgs (n=45, 70.31%), followed by those in the 3.1-4.0 kgs range (n=17,26.56%). The mean baby weight for this group was 2.62 (±0.19) kgs.

An ANOVA single-factor test was conducted on the data, revealing a statistically significant association between baby weight distribution and study groups ($p < 0.001$).

Table: 18 Mode of delivery comparison in Mild Pre-Eclampsia, Severe Pre-Eclampsia, Eclampsia & Control group

Mode of Delivery	Mild Pre-Eclampsia N (%)	Severe Pre-Eclampsia N (%)	EclampsiaN (%)	ControlN (%)
LN	21 (33.87)	11 (18.33)	1 (7.14)	43 (67.19)
Emergency LSCS	33 (52.23)	44 (73.33)	13 (92.86)	12 (18.75)
nergencyRepeatLSCS	4 (6.45)	5 (8.34)	0 (0.00)	9 (14.06)
Elective LSCS	2 (3.23)	0 (0.00)	0 (0.00)	0 (0.00)
ElectiveRepeatLSCS	2 (3.23)	0 (0.00)	0 (0.00)	0 (0.00)
Total	62 (100.00)	60 (100.00)	14 (100.00)	64 (100.00)

P value=0.001 (S)

During the analysis of the mode of delivery among the study participants, the followingpatterns were identified:

In the mild pre-eclampsia group, the majority of study subjects underwent emergency lower segment cesarean section (LSCS) as their mode of delivery (n=33, 52.23%), withnatural labor being the next most common mode (n=21, 33.87%).

For the severe pre-eclampsia group, a significant majority of study subjects also had emergency LSCS as their mode of delivery (n=44, 73.33%), followed by natural labor(n=11, 18.33%).

Within the eclampsia group, the majority of study subjects had emergency LSCS as their mode of delivery (n=13, 92.86%), with only a small proportion deliveringnaturally (n=1, 7.14%).

In the control group, a significant majority of study subjects delivered naturally (n=43,67.19%), followed by emergency lower segment cesarean section (LSCS) (n=12, 18.75%).

Statistical analysis using a chi-squared test indicated a statistically significant association between the mode of delivery and study groups ($p < 0.001$).

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Table: 20 Maternal outcome & its association with severity of LDH level in study population

Maternal OutcomeVersus LDH Levels	≤ 600 IU N=127N (%)	601-800 IUN=33 N (%)	> 800 IUN=40 N (%)
HELLP	0 (0.00)	0 (0.00)	9 (22.5)
Abruption	3 (2.36)	7 (21.21)	11 (27.50)
lmonary Edema	0 (0.00)	0 (0.00)	3 (7.50)
aternal Death	0 (0.00)	0 (0.00)	0 (0.00)

Table: 21 Maternal outcome & its association with mean values of LDH level

Maternal OutcomeVersus LDH Levels	Present		Absent		P value Unpairedt Test
	Mean	SD	Mean	SD	
HELLP - LDH Levels	1122.30	102.71	523.78	354.97	0.001 (S)
Abruption – LDH Levels	342.32	511.18	516.67	342.43	0.001 (S)
Pulmonary Edema -LDH levels	1730.23	1.34	554.98	354.87	NA
al Death - LDHLevels	-	-	-	-	-

The analysis of maternal outcomes in relation to lactate dehydrogenase (LDH) levels reveals significant correlations. In patients with LDH levels ≤ 600 IU, there were no cases of HELLP syndrome or pulmonary edema, and only 2.36% experienced abruption. Among those with LDH levels between 601-800 IU, there were no cases ofHELLP or pulmonary edema, but 21.21% experienced abruption. In contrast, patients with LDH levels > 800 IU had notably higher rates of adverse outcomes: 22.5% developed HELLP syndrome, 27.50% experienced abruption, and 7.50% had pulmonary edema. There were no maternal deaths across all groups. The relationship between LDH levels and maternal outcomes shows significant differences in mean LDH levels for patients with and without specific complications. For HELLP syndrome, the mean LDH level in affected patients was 1122.30 IU (SD =102.71) compared to 523.78 IU (SD = 354.97) in those without HELLP, with a significant P value (<0.001). For patients experiencing abruption, the mean LDH levelwas 342.32 IU (SD = 511.18), compared to 516.67 IU (SD = 342.43) in those without,also showing a significant P value (<0.001). Pulmonary edema cases had a markedly high mean LDH level of 1730.23 IU (SD = 1.34), whereas those without edema had a mean of 554.98 IU (SD = 354.87). No data were available for maternal death.

Table: 22 Perinatal l outcome & its association with severity of LDH level in study population

Perinatal OutcomeVersus LDH Level	≤ 600 IUN=127 N (%)	601-800 IUN=33 N (%)	> 800 IUN=40 N (%)
IUGR	5 (3.93)	11 (33.33)	25 (62.50)
IUD	0 (0.00)	2 (6.06)	2 (5.00)

Table: 23 Paternal outcome & its association with mean values of LDH level

Perinatal Outcome Versus LDH levels	Present		Absent		P value Unpairedt Test
	Mean	SD	Mean	SD	

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

IUGR	978.57	322.34	435.32	321.48	0.001 (S)
IUD	1123.88	312.49	544.34	384.34	0.03 (S)

During the cross-tabulation and analysis of perinatal outcomes and LDH (lactate dehydrogenase) levels among the study participants, the following trends were observed:
Patients with intrauterine growth restriction (IUGR) had a mean LDH level of 978.57 (±322.34) IU, whereas those without IUGR had a lower mean LDH level of 435.32 (±321.48) IU.
For patients with intrauterine death (IUD), the mean LDH level was 1123.88 (±312.49)IU, while those without IUD had a lower mean LDH level of 544.34 (±384.34) IU.
The statistical analysis, conducted using an unpaired t-test, revealed a statistically significant association between perinatal outcomes, specifically IUGR and IUD, and LDH levels among the study groups ($p < 0.001$).

4. DISCUSSION

This cross-sectional observational study, conducted at a tertiary care center in Udaipur,aimed to evaluate the maternal Serum Lactate Dehydrogenase (LDH) as a potential prognosticator and severity marker in pregnancy-induced hypertension (PIH). The rationale for this investigation stems from the recognition of preeclampsia as an idiopathic multisystem disorder specific to human pregnancy. Given that the preventionof severe preeclampsia and eclampsia represents a primary challenge in the realm of toxemia of pregnancy, early disease diagnosis becomes imperative. The commonly utilized triad of high blood pressure, edema, and albuminuria has proven neither specific nor sensitive enough, necessitating the quest for a more reliable marker. In thisstudy, LDH was systematically evaluated as a promising biochemical marker for discerning and predicting the severity of preeclampsia and eclampsia.
Preeclampsia, a significant pregnancy-related ailment, carries the potential for severe repercussions for both the mother and child. The onset of severe preeclampsia poses a substantial risk, leading to critical complications such as eclampsia, HELLP syndrome, abruption, and heightened perinatal mortality and morbidity.
In the current investigation, the age distribution of pregnancies revealed a predominant occurrence in the 21-30 age group, with a mean age of 25.32±2.15 across all three groups. Notably, this finding aligns with previous studies conducted by Talwar P et al.and Mary VP et al., where a majority of patients were situated in the younger age bracket. This consistency in age patterns underscores the importance of exploring age-related factors in the context of preeclampsia, providing valuable insights for futureresearch and clinical considerations.
In the present investigation, a noteworthy proportion of primigravida was observed, with 56.67% of patients in the severe preeclampsia group and 78.57% in the eclampsiagroup falling into this category. Similarly, 64.52% of those with mild preeclampsia and 56.25% in the normotensive group were primigravida, a finding of moderate significance. This aligns with prior research by Jain R et al, Gopinath S et al, and Umasatyasri Y et al, collectively indicating a predominance of primigravida among preeclamptic women. The elevated probability of developing preeclampsia during pregnancy in primigravida emphasizes the need for targeted monitoring and intervention strategies.
Upon data analysis in the present study, a discernible trend emerged, revealing a significant elevation in LDH levels with increasing disease severity ($P < 0.001$ – statistically significant). In the normotensive group, all individuals exhibited LDH levels below 600 IU/l, with a mean value of 225.16± 79.23 IU/l. The majority of patients in the mild preeclampsia group also had levels below 600 IU/l, while 19.35% had LDH in the range of 600–800 IU/l. In severe pre-eclampsia, the mean LDH level was calculated as 855.93±196.34 IU/l. Among the 60 cases of

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

severe preeclampsia, 21.67% had LDH levels below 600 IU/l, 30% had levels between 600 and 800 IU/l, and48.33% had levels above 800 IU/l. Importantly, this study revealed abnormal LDH levels in the majority of preeclamptic women, with a significant increase among midland severe preeclamptic women compared to normotensive controls. Notably, a highly significant increase in LDH levels was evident in women with severe preeclampsia compared to those with mild preeclampsia. These findings underscore thepotential utility of LDH as a valuable biomarker for assessing and stratifying the severity of preeclampsia.

Upon scrutinizing the aforementioned data, a conspicuous pattern emerges, revealing a significant elevation in LDH levels correlating with the increasing severity of the disease ($P < 0.001$). This trend aligns with findings in studies conducted by Rajoria L et al, Dev SV et al, and Bhave NV et al. The robust consistency across studies underscores the reliability of LDH as a promising biomarker for assessing the severityof preeclampsia.

Furthermore, in the present study, a noteworthy association was established between the mean gestational age at delivery and increasing LDH levels ($P < 0.001$). This association signifies a heightened incidence of preterm deliveries in patients with elevated LDH levels, shedding light on the potential role of LDH as a predictive factorfor preterm birth.

The link between low birth weight and serum LDH levels was explored, drawing insights from the study by Hall et al, albeit in contrast to the findings of Qublan etal.[16] In our investigation, a significant association between low birth weight and increasing LDH levels ($P = 0.019$) was observed, potentially attributed to a higher prevalence of premature births within this cohort.

Moreover, our study delved into the correlation between serum albumin levels and theseverity of preeclampsia. The observed decline in serum albumin during pregnancy, attributed to the dilution effect of increased maternal plasma volume over total serum albumin, prompted an investigation into its role as a severity predictor. [88-90]In line with Brown et al.'s findings, low serum albumin levels were significantly associated with severe preeclampsia and perinatal mortality, though the results lacked universal confirmation. Gojnic et al. revealed a correlation between serum albumin levels and the severity of preeclampsia, emphasizing hypoalbuminemia as an early indicator of developing preeclampsia. Witlin et al. proposed that serum albumin levels (<3.0g/dl) could be considered as a predictor of eclampsia, albeit lacking significance in multivariate analysis. This nuanced exploration underscores the need for further research to elucidate the role of serum albumin as a prognostic marker in preeclampsia.

Within our investigation, we identified a positive correlation between elevated LDH levels and higher systolic (SBP) and diastolic (DBP) blood pressure. Noteworthy studies, such as the one conducted by AD Sonagra et al, highlighted mean LDH levels in preeclampsia (356.46±158.09), gestational hypertension (282.3±120.98), normal pregnancy (151.57±47.47), and controls (130.5±44.36.8). In our study, a statistically significant difference in mean serum LDH was observed between the control group (201.5 ± 125.9) and the study group (570.5 ± 270.9).

Umasatyasri et al[21] explored the prognostic significance of serum LDH values in the context of preeclampsia, revealing varying mean LDH levels in different conditions: normotensive (159.06 ± 41.93), mild preeclampsia (323.30 ± 77.40), severe preeclampsia (636.20 ± 132.29), and eclampsia (649.32 ± 153.53). Notably, severely pre-eclamptic women with LDH levels exceeding 800 IU/l experienced a substantial increase in complications, including eclampsia, abruption placenta, and other issues, as demonstrated in the study by Qublan et al.[16] High LDH levels (1,400IU/l) demonstrated a strong predictive value for significant maternal morbidity in the investigation by Martin et al. Additionally, Catanzerite et al. reported a subgroup of patients with elevated LDH levels associated with hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome, posing a heightened risk of maternal mortality.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Demir et al. concluded that a statistically significant relationship exists betweenmaternal complications and elevated LDH levels.
Our study further revealed that higher serum LDH levels were linked to an increased incidence of maternal complications, including abruption placenta, renal failure, HELLP syndrome, and cerebrovascular accidents. This correlation resulted in a significant rise in maternal morbidity with increasing serum LDH levels ($P < 0.001$). Notably, maternal mortality experienced a notable increase, reaching 13.8% in patientswith LDH levels surpassing 800 IU/l. This rise was statistically significant ($P = 0.006$),aligning with comparable findings in other studies.

CONCLUSION

It is concluded in our study that;
Lactate dehydrogenase is a useful biochemical marker that reflects the severity of pre-eclampsia and eclampsia. Since it is heavily associated with disease severity, it can be safely assumed that it isassociated with other factors associated with disease severity like. Increased LDH levels is associated with early intervention in preeclampsia andeclampsia patients – lowered gestational age. Increased LDH levels is associated with poor maternal outcomes in preeclampsia andeclampsia patients –high incidence of HELLP and abruption. Increased LDH levels is associated with poor perinatal outcomes in preeclampsia andeclampsia patients –high incidence of IUGR and IUD. Increased LDH levels is associated with low baby weight in preeclampsia andeclampsia patients. Increased LDH levels is associated with high emergency LSCS rates in preeclampsiaand eclampsia patients. In conclusion, LDH levels reflect the severity of preeclampsia and the occurrence ofcomplications.

REFERENCES

1. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet 2010; 376(9741): 631–44
2. Totan AR, Greabu M. Effect of chronic hyperglycemia and vanadate treatment on erythrocyte Na/K-ATpase and Mg-ATpase in streptozotocin diabetic rats. Acta poloniae pharmaceutica. 2002; 59(4): 307–11. Epub 2002/10/31.
3. Preeclampsia Foundation. New Guidelines in Pree -clampsia Diagnosis and Care Include Revised Definition of Preeclampsia. USA. December 2013. Available at: https://www.preeclampsia.org/the-news/1-latestnews/299-new-guidelines-in-preeclampsia-diagnosisand-care-include-revised-definition-of-preeclampsia
4. National Health Portal of India: Preeclampsia. India. Zahid.2016 June 1. Available from: https://www.nhp.gov.in/disease/gynaecology-and-obstetrics/preeclampsia
5. Koc B, Erten V, Yilmaz MI, Sonmez A, Kocar IH. The relationship between red blood cell Na/K-ATPase activities and diabetic complications in patients with type 2 diabetes mellitus. Endocrine 2003; 21(3): 273–8
6. Page NM. The endocrinology of pre-eclampsia. Clin Endocrinol,2002; 57: 413–23
7. Norwitz ER, Hsu CD, Repke JT. Acute complications of preeclampsia. Clin Obstet Gynecol. 2002;45:308-93
8. Feron O. Pyruvate into lactate and back: from the Warburg effect to symbiotic energy fuel exchange in cancer cells. Radiother Oncol 2009;92: 329-333
9. Jialal I, Sokoll LJ. Clinical utility of lactate dehydrogenase: A historical perspective. Am J Clin Pathol 2015;143: 158-159
10. Vazquez-Rodriguez JG, Rios-Gutierrez CD, Paredes-Lozano EP, Garcia-Flores A. Frequency and maternal complications of the criteria of hemolysis in preeclamptic patients with HELLP syndrome treated in an intensive care unit. GinecolObstet Mex

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

2016;84: 19-26
11. Phipps E, Prasanna D, Brima W, Jim B. Preeclampsia: Updates in pathogenesis, definitions, and guidelines. Clin J Am Soc Nephrol 2016;11: 1102-1113
12. Magee LA, Helewa M, Moutquin JM, von Dadelszen P, Hypertension Guideline C et al. Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. J Obstet Gynaecol Can 2008;30:1-48
13. Redman CW, Sargent IL. Pre-eclampsia, the placenta and the maternal systemic inflammatory response--a review. Placenta 2003;24:21-27
14. Cockell AP, Learmont JG, Smarason AK, Redman CW, Sargent IL, et al. Human placental syncytiotrophoblast microvillous membranes impair maternal vascular endothelial function. Br J Obstet Gynaecol 1997;104: 235-240
15. Myatt L, Webster RP. Vascular biology of preeclampsia. J Thromb Haemost 2009;7:375-384
16. Qublan HS, Ammarin V, Bataineh O, Al-Shraideh Z, Tahat Y, et al. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. Med Sci Monit 2005;11: 393-397
17. Talwar P, Kondareddy T, Pranidha SCA. LDH as a prognostic marker in hypertensive pregnancy. Int J Reprod Contracept Obstet Gynecol 2017; 6:2444-6
18. Mary V P, 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
19. Jain R, Upadhyay C, Mehta L, Nayak B, Desai G. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia, Gujarat. Int J ReprodContracept Obstet Gynecol. 2017; 6 (8): 3418-22
20. Gopinath S, Shafi M, Ramegowda HK, Mehta PK. Outcome of referred obstetric emergencies at a tertiary centre. Int J Biol Med Res. 2016; 7(2): 5518-21
21. 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
22. Rajoria L, Nenkar S, Gidwani C. Lactic acid dehydrogenase and uric acid as prognostic markers for hypertensive disorders of pregnancy. IJSAR. 2018; 5 (2): 12-17
23. Bhave NV, Shah PK. A correlation of lactate dehydrogenase enzyme levels in pregnancy induced hypertensive disorders with severity of disease, maternal andperinatal outcome. Int J Reprod Contracept Obstet Gynecol. 2017; 6(10): 4302-08
24. Hall DR, Odendaal HJ, Kirsten GF, et al. Expectant management of early onset, severe preeclampsia perinatal outcome. BJOG. 2000; 107:1258–1264
25. von Dadelszen P, Magee LA, Devarakonda RM et al. The prediction of adverse maternal outcomes in preeclampsia. J Obstet Gynaecol Can 2004; 26: 871–879
26. Gojnic M, Petkovic S, Papic M et al. Plasma albumin level as an indicator of severity of preeclampsia. Clin Exp Obstet Gynecol 2004; 31: 209–210
27. Witlin AG, Saade GR, Mattar F, Sibai BM. Risk factors for abruptio placentae and eclampsia: analysis of 445 consecutively managed women with severe preeclampsia and eclampsia. Am J Obstet Gynecol 1999; 180: 1322–1329
28. AD Sonagra K Dattatreya JD Murthy Serum LDH, ALP and Uric Acid in hypertensive disorders of pregnancyInt J Pharm Bio Sci2012232019
29. Martin JN, Jr, May WL, Magann EF, et al. Early risk assessment of severe preeclampsia: admission battery of symptoms and laboratory tests to predict likelihoodof subsequent significant maternal morbidity. Am J Obstet Gynecol. 1999; 180:1407– 1414
30. Catanzerite VA, Steinberg SM, Mosley CA, et al. Severe preeclampsia with fulminant and extreme elevation of aspartate aminotransferase and lactate dehydrogenase levels.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Am J Perinatol. 1995; 12:310–313
31. Demir SC, Evruke C, Ozgunen FT, et al. Factors that influence morbidity, and mortalityin severe preeclampsia, eclampsia and HELLP syndrome. Saudi Med J. 2006; 27:1015–1018