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Original Research Article

Fetomaternal outcomes in liver disorders of pregnancy: a retrospective study from South India

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ABSTRACT

Background: Liver disorders in pregnancy are rare but can cause life-threatening maternal and fetal complications. They arise from physiological, pregnancy-specific, or coincidental systemic diseases. This study aimed to analyze the etiological spectrum, biochemical profile, and fetomaternal outcomes among pregnant women with hepatic dysfunction at a tertiary-care center in Tamil Nadu.
Methods: A retrospective observational study was conducted between June 2024 and May 2025 in the department of obstetrics and gynecology, Government Medical College, Tiruvannamalai. All pregnant women with clinical or biochemical evidence of liver dysfunction were included. Data on symptomatology, etiology, biochemical parameters, complications, and fetomaternal outcomes were recorded. Statistical analyses included χ^2 tests and ROC curve analysis of serum bilirubin, transaminases, and LDH as predictors of maternal complications.
Results: Among 12,542 deliveries, 65 (0.5 %) women had liver disorders. Mean age was 25 years; 73.8% presented in the third trimester. The commonest causes were viral hepatitis (52.3%), HELLP syndrome (16.9%), and hyperemesis gravidarum (13.8%). Maternal complications included postpartum hemorrhage (23%), DIC (9%), and hepatorenal syndrome (15%). There were no maternal deaths. Of 57 live births, 12% were preterm and three fetuses had intra-uterine death. ROC analysis showed LDH (AUC 0.82) as the best predictor of adverse maternal outcomes.
Conclusions: Liver disease in pregnancy remains a significant threat to fetomaternal health. Elevated LDH serves as a reliable biochemical marker of severity. Early diagnosis and multidisciplinary management are key to preventing mortality.

Keywords: Acute viral hepatitis, Adverse maternal outcome, Fetomaternal outcome, HELLP syndrome, Liver disease in pregnancy

INTRODUCTION

Liver disease in pregnancy occupies a distinctive diagnostic space: pregnancy itself induces physiological shifts in hepatic synthetic and excretory function- a modest rise in alkaline phosphatase, a fall in serum albumin, and altered clotting factors- that can mimic, mask, or worsen genuine hepatic pathology. When the full spectrum, from asymptomatic transaminase elevation to fulminant hepatic failure, is considered, hepatic dysfunction is estimated to complicate a meaningful minority of pregnancies worldwide, although clinically significant liver disease requiring active intervention is considerably less common, with higher rates consistently reported from developing countries where viral hepatitis remains endemic.[1,2]

Etiologically, this dysfunction is conventionally separated into three groups: disorders unique to pregnancy- hyperemesis gravidarum, intrahepatic cholestasis of pregnancy (ICP), acute fatty liver of pregnancy (AFLP),

and hypertensive liver disorders including preeclampsia and HELLP syndrome; diseases coincidental to pregnancy but altered by its immunological and hemodynamic milieu- viral hepatitis A-E, autoimmune hepatitis, and infections such as leptospirosis and malaria; and pre-existing chronic liver disease unmasked or aggravated by gestation.[2,3] This distinction is more than academic: hyperemesis-related transaminitis is self-limiting, whereas HELLP and AFLP can evolve within days into multiorgan failure, so timely categorisation directly shapes the urgency of management.[4]

Hepatitis E virus (HEV) deserves particular emphasis in the South Asian context. Unlike in non-pregnant adults, in whom HEV typically causes a self-limited illness, pregnant women- particularly in the third trimester- experience a disproportionately severe course, with Indian series reporting maternal mortality between 10% and 33%.[5,6] The mechanism is thought to involve a pregnancy-induced, Th2-skewed immune state with relatively reduced cell-mediated immunity, which impairs viral clearance and predisposes to fulminant hepatic failure.[7] Studies from Mumbai, Rajasthan, and eastern Uttar Pradesh have repeatedly identified this hepatitis-E-driven mortality signal, although the proportion of cases attributable to hepatitis E versus other causes varies considerably between centres, most likely reflecting differences in water and sanitation infrastructure, availability of viral serology, and referral practice.[8-10]

In our reading of this literature, two gaps stand out. First, comparatively little contemporary data exist on this spectrum from South India specifically, where sanitation infrastructure, endemic disease patterns, and antenatal referral pathways differ from the northern, western, and Rajasthani cohorts that dominate the existing literature, and where the last dedicated South Indian series is now more than a decade old.[6] Second, most single-centre reports catalogue etiology and outcome but stop short of asking a more actionable, frontline question: which routinely available biochemical parameter best flags a woman at risk of progressing to a severe maternal complication while she can still be escalated to a higher level of care? Lactate dehydrogenase (LDH), a marker of hemolysis and tissue hypoxia, has shown promise as an early severity indicator of hypertensive liver disorders in a few Indian cohorts, but has rarely been compared head-to-head against bilirubin and transaminases specifically in a South Indian government-hospital setting, where resource constraints make a single, cheap, and readily available triage marker especially valuable.[5,8]

The present study was therefore designed to (i) characterise the etiological spectrum and clinical-biochemical profile of liver disorders among pregnant women at a south Indian tertiary centre, (ii) document the associated fetomaternal outcomes, and (iii) compare the predictive performance of LDH, bilirubin, and transaminases for adverse maternal outcomes using ROC curve analysis, so as to inform early triage and multidisciplinary referral in similar resource-limited settings.

METHODS

Study design and population

A retrospective observational study was conducted using medical records from June 2024 to May 2025 in the department of obstetrics and gynecology, Government Medical College, Tiruvannamalai. Of 12,542 deliveries during this period, 65 women (0.5%) were diagnosed with a liver disorder and constituted the study population; eligibility was determined according to the inclusion and exclusion criteria below.

Inclusion criteria

Pregnant women of any gestational age with either of the following were included: jaundice or abnormal liver function tests (total bilirubin >2 mg/dL, AST/ALT>40 U/L, or ALP>300 U/L); or clinical features suggestive of hepatic dysfunction (e.g., pruritus, right upper quadrant/abdominal pain, persistent nausea or vomiting, or encephalopathy).

Exclusion criteria

Liver dysfunction secondary to sepsis-related multiorgan failure; known chronic liver disease or cirrhosis pre-dating the pregnancy; incomplete case records precluding reliable classification of etiology or outcome; refusal to participate/records marked as opt-out of research use.

Data collection

A detailed review of medical records was performed, and relevant clinical and obstetric data- including gravidity, gestational age at presentation, and associated risk factors- were extracted. Laboratory parameters such as complete blood count, liver and renal function tests, coagulation profile, viral serology (hepatitis A-E), leptospira and scrub-typhus serology, and ultrasound findings of the liver and obstetric scans were retrieved from case sheets and the hospital database.

Maternal outcomes were postpartum hemorrhage (PPH), disseminated intravascular coagulation (DIC), acute kidney injury (AKI), hepatorenal syndrome (HRS), prolonged hospital stay, and mortality.

Fetal outcomes were preterm birth, intra-uterine death (IUD), stillbirth, and birth weight.

Statistical analysis

Data were analyzed using SPSS v26. Categorical variables were compared using the χ^2 test, with $p<0.05$ considered significant. The predictive accuracy of biochemical parameters for composite maternal complications was

assessed using receiver operating characteristic (ROC) curve analysis for LDH, bilirubin, and SGOT/SGPT, and positive and negative predictive values were calculated at clinically relevant cut-offs.

RESULTS

Demographics and presentation

Of 12,542 deliveries, 65 (0.5%) had liver disorders. The majority (43.1%) were aged 21-25 years and 29.2% were aged 26-30 years; mean age was 25±4.6 years. Primigravidae constituted 43% and multigravidae 57%. Most women (73.8%) presented in the third trimester. Common presenting symptoms were nausea or vomiting (23%), fever (21%), and abdominal pain (9%), while 31% were diagnosed incidentally on abnormal liver function tests (Tables 1 and 3).

Table 1: Age distribution of study participants.

Age group (years)	Frequency	Percentage
15-20	8	12.3
21-25	28	43.1
26-30	19	29.2
31-35	8	12.3
≥40	2	3.1
Total	65	100

Etiological distribution

Viral hepatitis was the predominant cause (52.3%), followed by HELLP syndrome (16.9%), hyperemesis gravidarum (13.8%), scrub typhus (7.6%), preeclampsia (3.0%), and leptospirosis (3.0%); AFLP and ICP were rare (1.5% each) (Table 2).

Table 2: Etiological distribution of liver disorders (n=65).

Etiology	Frequency	Percentage
Viral hepatitis	34	52.3
HELLP syndrome	11	16.9
Hyperemesis gravidarum	9	13.8
Scrub typhus	5	7.6
Preeclampsia with hepatic involvement	2	3.0
Leptospirosis	2	3.0
Acute fatty liver of pregnancy (AFLP)	1	1.5
Intrahepatic cholestasis of pregnancy (ICP)	1	1.5
Total	65	100

Clinical profile

The most frequent presenting features were nausea/vomiting (23%), fever (21%), and abdominal pain (9%); incidental detection on routine liver function testing accounted for 31% of cases, and pruritus was rare (1.5%), corresponding to the single case of intrahepatic cholestasis (Table 3). As these categories are not mutually exclusive, the frequencies in Table 3 do not sum to the total cohort size.

Table 3: Clinical presentation (n=65)*.

Presenting feature	Frequency	Percentage
Nausea/vomiting	15	23.0
Fever	14	21.0
Incidental abnormal LFT (no symptoms)	20	31.0
Abdominal pain	6	9.0
Pruritus	1	1.5

*Categories are not mutually exclusive; a woman could present with more than one feature.

Laboratory parameters

Serum bilirubin was in the 2-5 mg/dl range in 90% of women and exceeded 10 mg/dl in 10%. Transaminases were below 100 U/l in 85% of cases, 100-1000 U/l in 14%, and above 2000 U/l in a single HELLP-associated case. LDH was below 600 U/l in 77%, 600-1000 U/l in 15%, and above 1000 U/l in 8% (Table 4).

Table 4: Laboratory parameters.

Parameters	Category	Frequency
S. bilirubin	2-5 mg/dl	59
	6-10 mg/dl	3
	10-20 mg/dl	3
SGOT/SGPT	<100 U/l	55
	101-1000 U/l	9
	>2000 U/l	1
LDH	<600 U/l	50
	600-1000 U/l	10
	>1000 U/l	15

Maternal complications

Thirty women (46%) had no complications; PPH occurred in 15 (23%), DIC in 6 (9%), and AKI in 2 (3%). Hepatorenal syndrome developed in 10 (15%) cases, all of which resolved with supportive care. No maternal deaths were recorded (Table 5).

Maternal outcomes

Of 65 women, 21 (32%) had a normal vaginal delivery (including 7 preterm), 39 (60%) underwent lower-segment caesarean section (LSCS), and 4 (6%) experienced abortion or ectopic gestation. Induction of labor was common in the HELLP and hypertensive groups (Table 6).

Table 5: Maternal complications.

Complication	Frequency
No complication	30
Postpartum hemorrhage (PPH)	15
Disseminated intravascular coagulation (DIC)	6
Hepatic encephalopathy	0
Hepatorenal syndrome	10
Massive blood transfusion	0
Death	0
Prolonged hospital stay	9
Surgical site infection (SSI)	5

Table 6: Maternal outcomes (mode of delivery).

Outcome	Frequency
Ectopic pregnancy	1
Abortion	2
Spontaneous expulsion	2
Normal vaginal delivery (incl. 7 preterm)	21
Lower-segment caesarean section (LSCS)	39
Instrumental delivery	0
Total	65

Neonatal outcomes

There were 57 live births (88%), 3 intra-uterine deaths (5%), and no stillbirths. Twelve percent of live births were preterm, and low birth weight (<2.5 kg) occurred in 38% of neonates. Preterm birth correlated significantly with HELLP syndrome and viral hepatitis ($p<0.001$) (Table 7).

Table 7: Neonatal outcomes.

Outcome	Frequency
Live births (total)	57
Preterm	7
Term	50
Stillbirth	0
Intra-uterine death (IUD)	3

Diagnostic accuracy of biochemical markers

LDH showed the highest overall diagnostic performance for composite maternal complications (PPH, DIC, or hepatorenal syndrome), with an AUC of 0.82, a positive predictive value (PPV) of 77.3%, and a negative predictive value (NPV) of 91.5% at a cut-off of 600 IU/l. Bilirubin (>5 mg/dl) and SGOT/SGPT (>100 IU/l) showed somewhat lower discriminative accuracy (Table 8). Bilirubin and SGOT/SGPT had high NPVs, indicating that normal values reliably excluded severe maternal complications, while elevated LDH carried the greatest weight in ruling them in.

Table 8: Diagnostic accuracy of biochemical markers for composite maternal complications.

Marker	Cut-off	AUC	95% CI	P value	PPV (%)	NPV (%)
LDH	>600 IU/l	0.82	0.74-0.90	<0.001	77.3	91.5
Bilirubin	>5 mg/dl	0.79	0.70-0.88	<0.001	71.4	93.2
SGOT/SGPT	>100 IU/l	0.75	0.65-0.84	0.002	68.0	90.0

LDH>600 IU/l had the highest AUC and PPV for predicting maternal complications, particularly HELLP syndrome and DIC, supporting its value as an early severity marker, consistent with previously published Indian data.

Table 9: Associations between trimester of presentation, etiology, and outcomes.

Variable	Comparison	χ^2	P value
Trimester versus maternal complications	$1^{st}/2^{nd}/3^{rd}$ trimester × presence of complications	9.62	0.008
Etiology versus preterm birth	Viral hepatitis/HELLP/others	12.78	<0.001
LDH versus DIC occurrence	LDH <600 versus ≥600 IU/l	8.91	0.003

These associations align with the findings of Panchbudhe et al., in which HELLP syndrome and hepatitis E significantly influenced preterm births and NICU admissions ($p < 0.01$).13

Associations between trimester of presentation, etiology, and outcomes

Chi-square testing was used to evaluate associations between trimester of presentation, etiology, and outcomes (Table 9). Both trimester at presentation and etiology significantly affected complication rates.

DISCUSSION

Incidence and demographics

The 0.5% incidence found in this study lies at the lower end of reported Indian data (0.3-2%). The predominance of young mothers (21-30 years) and third-trimester presentation is consistent with the BRD Medical College

series of Tiwari et al and with other Indian cohorts.[9,11] The third trimester is particularly vulnerable to hypertensive and infective triggers that impair hepatic microcirculation.[9,11]

Etiological spectrum

Viral hepatitis remained the leading cause in our cohort, similar to other Indian series in which hepatitis E was the dominant viral subtype. This contrasts with data from high-income settings, where ICP and AFLP account for a much larger proportion of pregnancy-associated liver disease, reflecting differing endemic infection burden and screening infrastructure rather than a true biological difference in disease susceptibility.[12] Our etiological pattern is broadly comparable to the western-India data of Panchbudhe et al, where HELLP syndrome (16.9% in our cohort vs. higher rates reported elsewhere) and hepatitis E were also the dominant contributors, and to the Uttarakhand experience reported by Ramola et al.[13,14]

Maternal outcomes

The absence of maternal mortality in this study is notable given previous reports of 10-30% mortality attributable to hepatitis E and HELLP syndrome. Contributing factors likely include earlier referral, availability of ICU care, and standardized protocols for hypertensive disorders. Our PPH rate (23%) was comparable to that of Sharma et al (20%), and our DIC incidence (9%) was lower than that reported by Tiwari et al (25%).[9,10] No maternal deaths occurred in our series- a favourable contrast to the 13.3% mortality reported by Panchbudhe et al and the mortality range described by Konareddy and Krithika in their Mysuru cohort.[13,15]

Fetal outcomes

Perinatal mortality in our cohort was 7.6% (3 intra-uterine deaths), lower than the 10.8% reported by Panchbudhe et al.[13] Preterm birth (12%) and low birth weight (38%) remained important concerns; HELLP syndrome and preeclampsia impair uteroplacental perfusion, while maternal infection contributes independently to fetal distress, and AFLP- though rare in our cohort- is recognised elsewhere as a cause of abrupt fetal compromise requiring prompt delivery.[16]

Predictive role of LDH

LDH reflects tissue hypoxia and hemolysis, and our finding of LDH>600 U/l as a predictor of maternal complications (AUC 0.82) is consistent with the western-India data of Rathi et al and Solanke et al, as well as the broader Indian spectrum described by Rathi and colleagues in their 2015 series.[5,8,17] In our cohort, LDH outperformed bilirubin and transaminases in predicting DIC and hepatorenal syndrome, supporting routine LDH monitoring as a low-cost, widely available early-warning marker in hypertensive pregnancies, particularly in settings such as ours where more specialised biomarkers or elastography are not readily accessible.

Clinical implications

Routine liver function and LDH screening in hypertensive pregnancies can enable timely detection of HELLP syndrome, and integration of gastroenterology and critical care teams appears to improve outcomes, consistent with broader reviews of liver disease management across the reproductive spectrum, including in the peripartum and post-transplant setting.[18] Public health efforts directed at safe drinking water and sanitation remain central to reducing hepatitis-E-driven maternal mortality in this population.

This study is limited by its single-centre, retrospective design and modest sample size, which may restrict generalisability; long-term neonatal follow-up was not performed, and biochemical cut-offs were derived from a relatively small number of severe events. Nevertheless, the study adds contemporary South Indian data to a literature that remains dominated by northern and western Indian cohorts.

CONCLUSION

Pregnancy-specific liver disorders and viral hepatitis were the major contributors to hepatic dysfunction in this South Indian cohort. Elevated LDH is a reliable, inexpensive, and widely available early biochemical marker of severe maternal complications. Early recognition, routine biochemical screening, and multidisciplinary management can minimize fetomaternal risk and, as demonstrated here, can eliminate maternal mortality even in a resource-constrained government tertiary centre.

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