MATERNAL AND FETAL OUTCOME AMONG OBSTETRIC PATIENTS WITH DERANGED LIVER FUNCTION TESTS

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ABSTRACT: INTRODUCTION: OBJECTIVES: 1) To study maternal and fetal outcome among obstetric patients with deranged liver function tests. 2) To study the relationship between type of liver disease based on etiology and maternal and fetal outcome **METHODS**: 3 years prospective and retrospective study was carried out from Jan 2011 to Jan 2014 at rural medical Centre. 40 cases of pregnancy with deranged liver function tests were analyzed for incidence, maternal and fetal outcome. **RESULTS:** 1) Incidence of liver disease during pregnancy was 1.6% in our study. 2) Majority of causes of jaundice were pregnancy specific. 3) Common causes of deranged liver function tests in the pregnancy were pregnancy induced hypertension with HELLP syndrome (37%), acute fatty liver of pregnancy (37%) and viral hepatitis (20%). 4) Overall maternal and perinatal mortality found in our study were 17.5% and 35% respectively. 5) Majority cases of HELLP & acute fatty liver of pregnancy were young primes (20-30years) at 32-34 weeks of gestation. 5) All cases of HELLP were associated with pregnancy induced hypertension. 6) Disseminated intravascular coagulation was most common morbidity. 8) 14 cases (56%). 7) Maternal mortality was highest 35% in acute fatty liver of pregnancy which reduces to 18% if unbooked cases with uncorrected disseminated intravascular coagulation are excluded. 8) Perinatal mortality was highest with HELLP syndrome (50%). **CONCLUSION:** 1) Majority of causes of deranged liver function tests during ^{3rd} trimester were pregnancy induced. 2) Maternal and fetal outcome depends on gestational age of presentation, early recognition of the disease and tertiary care support.

KEYWORDS: HELLP Syndrome, Acute fatty liver of pregnancy, cholestasis, hepatitis.

INTRODUCTION: Deranged liver function tests during pregnancy are a commonly encountered problem, spectrum of which varies widely. Determining cause of liver disease can present a difficult challenge to the clinician. It is still a significant cause of maternal and perinatal mortality in India. Early diagnosis and timely treatment is a key to success³.Current study aims at evaluating spectrum of liver diseases in pregnancy and its relation to maternal and fetal outcome. 40 cases of pregnancy with deranged liver function tests during study period were analysed for maternal and fetal outcome.

MATERIALS AND METHODS: The present study was carried out from Jan 2012 to Jan 2014 at rural medical centre.

STUDY DESIGN: It was prospective and retrospective observational study.

SUBJECTS: 40 cases of pregnancy with deranged liver function tests were analysed for incidence, maternal and fetal outcome.

STUDY INSTRUMENTS: 1) Questionnaire-duration of disease, gastrointestinal or CNS symptoms or signs 2) Lab tests— CBP, LFT, kft, coagulation profile, electrolytes. 3) Special tests-viral markers,

hepatic sonography, occasionally endoscopy to rule out vary. Procedure of data collection-All patients attending obst. OPD, and patients admitted in delivery ward, and outside deliveries with acute complications admitted in acute medical care or acute surgical care or emergency ward with complaints of deranged liver function tests were contacted. Thorough history, clinical examination was carried out. Following investigations final diagnosis was made in consultation with physician. Patients were followed for maternal and fetal outcome. Different causes of deranged liver function tests during pregnancy are 1) Pregnancy specific liver disorders-1) PIH with HELLP syndrome-hypertention, proteinuria and oedema after 20 weeks of gestation with elevated transaminases and bilirubin and low platelets(<100000)) and haemolysis(suggestive peripheral smear with increased reticulocytosis). 2) Intrahepatic cholestasis -Pruritus without any local cause or systemic cause, with elevated transaminases, which disappears after delivery.

C) Hyperemesis gravidarum with liver dysfunction-Elevated transaminase or bilirubin in the presence of persistent vomiting for more than one week during 1st or 2nd trimester.

D) Acute fatty liver of pregnancy-malaise, anorexia, epigastric pain and progressive jaundice hypoglycemia, Bilirubin 3-10 mg)dl, Enzymes <1000 IU, thrombocytopenia, elevated white blood cell count, DIC renal impairment, coma. 2)Pregnancy associated liver disorders-a) Acute viral hepatitis, A, B, C, D, E. b) herpes simplex infection c)Chronic liver diseases. Outcome measures- Mothers will be followed for 1)mortality 2)morbidity which can be 1) Disseminated intravascular coagulopathy-It presents with deranged coagulation profile with clinical evidence of bleeding in form of postpartum haemorrage, intraabdominal clot collection, rectus sheath haematoma, vulval haematoma or hepatic infarcts. 2) Renal failure-Presents with urine output less than 30ml/hr, blood urea >20mg%, serum creatinine >0.9mg%. 3) Acute liver failure-Occurrence of encephalopathy within 4 weeks of onset of symptoms in absence of pre-existing liver disease, resulting in altered mental status, vasodilatation, renal and pulmonary failure Fetal outcome will be studied which can be 1) Intrauterine growth retardation. 2) Perinatal Mortality.

Data analysis-spss version 19 will be used in data analysis. el data will be entered in M S Excel & Data will be presented as percentages in categories. Appropriate statistical tests will be used which include chi sqare and other tests.

RESULTS:

- 1) During study period 40 cases were found to have liver disease. As there were 2400 deliveries incidence of liver disease in pregnancy was found to be around 1.6%
- 2) Different aetiologies' found behind liver disease were as shown in Figure no 1.
- 3) Majority of causes of jaundice were pregnancy specific 31)40 case. (77.5%).
- 4) Overall maternal and perinatal mortality found in our study were 17.5% and 35% respectively.
- 5) Figure 2 is showing comparative study of maternal and perinatal mortality in liver disease depending on aetiologies'.
- 6) As seen in figure 3-Demographic Data of HELLP cases shows: 1) Majority of cases of HELLP are between age group 20-30 years with gestational age less than 34 weeks. 2) Typical presenting features of case of pregnancy induced hypertension without any complaints suggestive of jaundice and laboratory reports usually reveal raised liver enzymes with raised bilirubin up to 3 mg% and thrombocytopenia was seen in 80% of cases. 3) Pregnancy induced hypertension was associated with all cases. 4) Incidence of lower segment caesarean section was 50%.

Indications were foetal distress, severe oligohydramnios and failed induction. 5) As seen in figure 3. MATERNAL mortality was seen in 1) 15 (12%) case. This was a prime patient with onset of pregnancy induced hypertension at 26weeks of gestation. Patient was on antihypertensive drugs since 2 months. Patient underwent caesarean section at 36 weeks for fetal distress. This was followed by sudden postpartum collapse on third postoperative day for which she was referred. After investigations cause found was dilated cardiomyopathy (Figure 3) for which pt was in cardiac care unit but did not respond and succumbed. Baby survived well. 6) morbidities seen in HELLP cases in our case series were as follows i) disseminated intravascular coagulation 2) 15(14%) -- a) rectus sheath haematoma -this was an outside abdominal delivery followed by massive oozing from abdominal incision. b) atonic postpartum haemorrhage 2) Pulmonary oedema-1 case 3/ Severe metabolic acidosis -1case.

6/Acute fatty liver of pregnancy—1/Majority of cases were prime with age group between 20-30 years 2) all cases presented after 34 weeks of gestation.4)Presenting features of nausea, vomiting,pain in abdomen and yellowish discoloration of sclera was seen in 8 (56%) of cases.6 (42%) were brought in critically ill condition of uncorrected DIC leading to hemorrhagic shock indicating symptoms were neglected by concerned patients and)or practioners. 5)Raised bilirubin(>5mg%) with lesser elevation of enzyme40-400IU), leucocytosis, hypoglycemia, and evidence of early renal failure and thrombocytopenia was in 12 (84%) cases.

5/ Figure 3 Maternal mortality was seen in 5 out of 15 cases. Causes of maternal mortality were as follows:

- 1) unbooked cases with outside deliveries with uncorrected disseminated intravascular coagulation and haemorragic shock—3 cases
- 2) Pulmonary oedema-1 case
- Severe metabolic acidosis -1case Maternal mortality was highest 35% in acute fatty liver of pregnancy which reduces to 18% if unbooked with uncorrected disseminated intravascular coagulation are excluded. (Figure 6).
- 6/ Maternal morbidities seen were:
- DIC was most common morbidity in 8 cases (56%) out of which 5 cases died and 3 cases survived. Out of 3 cases which survived.1 needed laparotomy followed by evacuation of clots, & internal iliac ligation. Second case needed internal iliac ligation and obst hytrectomy. Third case needed evacuation of valval haematoma which developed within 12 hours of normal delivery. 2).renal failure needing dialysis in 1 case (7%) 3) septicemia in 1 case (7%) were other morbidities.
- 7) Figure 3 showing maternal and fetal Outcome: Cholestasis of pregnancy -There was no maternal mortality. Morbidities were preterm labour and eclampsia each. Infective hepatitis 1) hepatitis A +ve 2 cases -1) Sudden intrauterine death -1 case during active phase of disease 2) Preterm delivery with – postpartum haemorrage in 1 case. 2) hepatitis B +ve - 3cases were uneventful. Babies needed active and passive immunisation. 3) Hepatitis E +ve - 2 cases -one case was uneventful. Second case was G2 with acute liver failure with HEV +ve. Patient gave History of- nausea vomiting, slurring of speech, drowsiness since 2days before admission.

Her laboratory reports were as follows LFT– sr bilirubin-17.7mg% (D-10.4, ID-7.3) Coagulation profile - PT INR -5.23 Pt had full term vaginal delivery. AS DIC was well corrected with use of. Fresh frozen plasma and packed cell transfusion patient had no postpartum haemorrhage but her encephalopathy worsened and pt succumbed due to diffuse cerebral oedema on 5th day of delivery

DISCUSSION: Incidence of liver disease in pregnancy varies from 0.9% to 3%.^{1,2} In our study incidence was 1.6%.

Literature shows^{3,4,5} majority of causes were related to pregnancy specific conditions (57.6%). Most episodes of abnormal LFT occurred in the third trimester (59.2%). Hyperemesis gravidarum (55.8%) and viral hepatitis (47%) were the most common causes of abnormal LFT in the first and second trimesters respectively. HELLP (28.3%) and AFLP (14.8%) were the most common causes of abnormal LFT in the third trimester. Pregnancy-related liver disease is the most frequent cause of liver dysfunction in pregnancy and provides a real threat to fetal and maternal survival. A rapid diagnosis differentiating between liver disease related and unrelated to pregnancy is required in women who present with liver dysfunction during pregnancy⁶ We found incidence of pregnancy specific liver dysfunction in77% cases.

Different etiologies behind deranged liver function tests with pregnancy in our study were as shown in table no 1.

Maternal and perinatal mortality found in different studies are as follows:

Author	Maternal mortality	Perinatal mortality
Pareira ⁷	13%	9%
Harish ³	20.2%	24.6%
Umang Rathi ¹	20%	35%
Our study	17.5%	35%

BEACHER⁸ Defined HELLP by the association of hemolysis, hepatic dysfunction and thrombocytopenia and stated that Hemolysis, Elevated Liver enzyme, Low Platelets (HELLP) syndrome can complicate preeclampsia and worsen maternal and fetal prognosis. Majority are diagnosed antenataly but may be diagnosed in the immediate postpartum (30%) or in the absence of preeclampsia (10-20%). Clinical diagnosis can be difficult because there is no specific symptom. Abdominal pain or vomiting during the third trimester must lead to think about this diagnosis. Biological criteria are well defined: hemolysis by the presence of schistocytes, increased serum total bilirubin >12 mg)L or LDH >600 IU)L, hepatic dysfunction by increased transaminases and thrombocytopenia by a platelet count <100,000)microL.

Maternal mortality varies from s 1.1%⁹ TO4. %¹⁰ Careful cardiorespiratory evaluation is must in cases of pregnancy induced hypertension with HELLP syndrome. Dilated cardiomyopathy was cause of death in our series of HELLP patients.

Bienias P¹¹ has reported case of Peripartum cardiomyopathy and preeclampsia complicated with HELLP syndrome.

Patient was a case of prime at 35 weeks of gestation first time in her life symptoms of severe heart failure (HF) in NYHA class III)IV. In 37th Hbd the diagnosis of Peripartum cardiomyopathy was established based on clinical status and echocardiographic examination, which demonstrated a

dilatation of heart chambers and impaired left ventricular systolic function with decreased ejection fraction (EF) 17%. In 37th Hbd she developed symptoms of preeclampsia complicated with HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) and further a DIC syndrome as well. Because the patient was in critical condition and the foetus' life was threatened the pregnancy was terminated with urgent cesarean section. Then the patient developed shock, respiratory insufficiency and increasing renal failure.

Sabai⁹ has described development of HELLP at < 27 weeks in 11% and 18% at term. 70%) of the cases occurred ante partum and 133 (30%) post-partum.

Serious maternal morbidity included disseminated intravascular coagulation (21%), abruptio placentae (16%), acute renal failure (7.7%), pulmonary edema (6%), subcapsular liver hematoma (0.9%), and retinal detachment (0.9%). Women with postpartum HELLP syndrome had significantly higher incidences of pulmonary edema and renal failure.

Romero¹⁰ 170 cases were analyzed, 156 (92%), occurred ante partum and 14 (8%) postpartum; 15 cases (9%) developed at < 27 weeks, 112 (66%) between 28 to 36 weeks of gestational age and 43 (25%) at term. Maternal morbidity included acute renal failure (13.5%), abruptio placentae (6.6%), pneumonia (3%), subcapsular liver hematoma (2.3%), pulmonary edema (2.3%), disseminated intravascular coagulopathy (1.7%) and cerebral hemorrhage (1.2%).

Tsukahara E¹² HAS DESCRIBED case of HELLP SYNDROMME in disseminated coagulation who underwent surgical removal of abdominal haematoma.

Batashki I¹³ has described case of HELLP syndrome developed in 30 gestational week, complicated with DIC syndrome.

Wei Q¹⁴ found all cases of acute fatty liver of pregnancy presented with a prodrome of nausea, vomiting, malaise and jaundice. Raised transaminases and serum bilirubin were found in all patients (100%), hypoglycemia was found in two patients (18.2%) and hypoproteinemia was found in five patients (45.5%) Except for one patient whose labor was induced with oxytocin because of fetal death before admission, all other pregnancies were terminated by cesarean section within 24 h of definitive diagnosis. All patients and neonates survived delivery. One (9.1%) maternal death and one (7.2%) fetal death occurred.

Yang¹⁵ The mean gestational age at onset was 34 +)- 2 weeks. All cases were primigravida. In the early stages, all patients presented malaise, nausea, vomiting and epigastric distress followed by jaundice in the third trimester of pregnancy. Laboratory findings: all had raised transaminases and serum bilirubin (32.5-510.8 mumol)L), hypoalbuminemia (22.4-30.0 g)L), hypofibrinogenemia (< 180 mg)dl), prolonged prothrombin time and prolonged partial thromboplastin time. Maternal complication included hepatic encephalopathy (6 cases), ascites (6), hypoglycemia (5), hematemesis (2), and postpartum hemorrhage (5) and preeclampsia (4). Emergency cesarean section was performed in 3 cases. One mother died of fulminant hepatic failure with increasing awareness, especially in the early recognition of milder cases, and prompt progressive management including early termination of pregnancy by cesarean section and large dose infusion of fresh frozen plasma and albumin alternately, the prognosis of AFLP can be improved. One mother died of fulminant hepatic failure and

Steingrub¹⁶: Preeclampsia, HELLP syndrome, and AFLP form a spectrum of disease that ranges from involving mild symptoms to severe life-threatening multi organ system dysfunction. They have been shown to be the primary causes of severe hepatic dysfunction during pregnancy.

Wikström Shemer^{17,18} confirm an increased risk of preterm delivery, but not of stillbirth, in actively managed cholestasis of pregnancy. The high rates of gestational diabetes and pre-eclampsia are new findings, and need to be considered in the management of ICP pregnancies. Mirghani¹⁹ found Except for a higher incidence of pre-term birth, the outcome of pregnancy in the case group was not affected. It is concluded that pregnancy is a risk factor which increases the mortality of viral hepatitis and that viral hepatitis does not affect the outcome of pregnancy except for pre-term birth.

Nayak NC²⁰ Hepatitis B infections accounted for 17% of all cases in pregnant women compared with 45% in controls. Acute viral hepatitis in pregnancy had a poor outcome as assessed by maternal and) or fetal mortality (28.5%). The outcome was equally bad in hepatitis NANB and hepatitis B.

In our study patient infected with HEV progressed to fulminant hepatic failure and succumbed.Patra ²¹ found Infection with HEV caused acute viral hepatitis in 60% of included women. Fulminant hepatic failure was more common (relative risk, 2.7 [95% CI, 1.7 to 4.2]; P = 0.001) and maternal mortality was greater (relative risk, 6.0 [CI, 2.7 to 13.3]; P < 0.001) in HEV-infected women.

CONCLUSION:

- 1. Majority of causes of deranged liver function tests during ^{3rd} trimester were pregnancy induced.
- 2. Maternal and fetal outcome depends on gestational age of presentation, early recognition of the disease and tertiary care support.

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ABBREVIATIONS:

HELLP- Hymolysis-Liver enzymes (raised) low platelets. AFLP-Acute fatty liver of pregnancy. CP- Cholestasis of pregnanc.



LIVER DISEASE	MATERNAL MORTALITY	PERINATAL MORTALITY			
HELLP 15	1 (7%)	7 (50%)			
AFLP 15	5 (33%)	5 (33%)			
INFECTIVE HEPATITIS 8	1 (12%)	5 (14%)			
CHOLESTASIS	NIL	NIL			
CIRRHOSIS LIVER	NIL	NIL			
Figure 2					

E (1)=0.667. E (5)=0.33 CHI SQUARE(X2)=0.188. P=0.665. i.e.p>0.05. z) It is not significant.

	HELLP	AFLP	Cholestasis	INFECTIVE HEPATITIS 7 (20%) HAV HAB HEV
Incidence	15 (37%)	15 (37%)	2(5%)	2(28.5%) 3(42.8%) 2(28.5%)
Age group20-30	14 (90%)	10 (66.66%)		
Age group-30-40	1 (6.66%)	5 (33.33%)		
Gravida-1	13 (86%)	12 (80%)		
Gravid-2 &above	2 (13.33%)	3 (20%)		
Gestational age of presentation	28-32weeks-3 32-34 weeks12 (80%) >34weeks-0	>34 weeks—15 (100%)		
Typical presentation	12(80%)	8(56%)		
Association with PIH	15(100%)	9(60%)		
Incidence of caeserian section	8(53.33%)	8(53.33%)		

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Maternal mortality	1 (6.66%) cause- dilated cardiomyopathy with postpartum collapse	5(33.33%) FIGURE 6-/3-unbooked cases – haemorragic shock 2-pulmonary edema	nil	NIL NIL 1 (50%)
morbidities	DIC 2 (13.33) Pul edema1 (6.66%) Severe met acidosis1(6.66)	DIC 3 (20%) Renal failure1 (6.66%) Septicemia1 (6.66%)	eclamptia - 1 pretermlabo ur1	PPH 1 (50%) NIL Nil
		Figure 3		

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