Fetal and Maternal Outcome in Patients with Impaired Liver Function at Third Trimester of Pregnancy

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Abstract

Background: Jaundice during pregnancy is a deadly combination resulting in a very high perinatal as well as maternal morbidity and mortality. Jaundice in last trimester can lead to coagulation defects, postpartum haemorrhage, organ failure and high maternal mortality and poor outcomes of their newborns such as intrauterine fetal death, still births, neonatal deaths.

Objectives: To find out the clinical profile and feto-maternal outcome in pregnancy with jaundice.

Methods: This descriptive cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, Mymensingh Medical College Hospital, Mymensingh, Bangladesh from August 2015 to February 2016.

Fifty pregnant women beyond gestational age of 28 weeks with impaired liver function were included in this study. Patient having chronic liver disease or past history of jaundice were excluded.

Results: The mean age of the patients was 22.4 (SD ± 2.9) years. Primigravida constituted 31 patients (62.0%).The mean gestational age of the patients was 35.3 (SD ± 3.2) weeks. The causes of jaundice during pregnancy were viral hepatitis (86.0%), HELLP syndrome (8.0%) and cholestasis in pregnancy (6.0%). Hepatitis E was the most frequent causes of viral hepatitis [26 (52%)]. Serum billirubin<10mg% in 46% of the women, SGOT, SGPT and alkaline phosphatase were raised in most of the cases.. Most common maternal complications were post partum haemorrhage (62.0%) and encephalopathy (20.0%). Most of the patients survived well [43 (86.0%)] and maternal death was 7 (14.0%),38(76%) babies were born alive and among the live born babies, resuscitation was needed in 30 cases, admission to neonatal unit required in 15 cases. Ultimately 29(58%) babies were survived well and perinatal mortality was 42%.The causes of perinatal deaths were intrauterine death [7(14%)], fresh still born [5(10%)] and early neonatal death [9(18%)].

Conclusion: Jaundice during pregnancy is associated with an increase in maternal morbidity and mortality and obstetric complications;and poor fetal outcomes with high perinatal mortality.

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Introduction

Deranged liver function tests during pregnancy are a commonly encountered problem,spectrum of which varies greatly.Determining cause of liver disease can present a difficult challenge to the clinician.It is still a significant cause of maternal and perinatal mortality.Early diagnosis and timely treatment is a key to success.[1] Liver involvement in pregnancy is of three types,namely, liver diseases peculiar to pregnancy,liver diseases coincidental to pregnancy and pregnancy in patients with pre-existing liver diseas.[2]

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Abnormal liver tests occur in 3%-5% of pregnancies, with many potential causes, including coincidental liver disease (most commonly viral hepatitis or gallstones) and underlying chronic liver disease. However, most liver dysfunction in pregnancy is pregnancy-related and caused by 1 of the 5 liver diseases unique to the pregnant state: these fall into 2 main categories depending on their association with or without preeclampsia. The preeclampsia- associated liver diseases are preeclampsia itself, the hemolysis (H), elevated liver tests (EL), and low platelet count (LP) (HELLP) syndrome, and acute fatty liver of pregnancy. Hyperemesis gravidarum and intrahepatic cholestasis of pregnancy have no relationship to eclampsia.[3]

Causes of liver disease in 3rd trimester of pregnancy: Pregnancy-induced hypertension-related liver dysfunction, intrahepatic cholestasis of pregnancy, acute fatty liver of pregnancy, hepatitis E ,hepatitis B, hepatitis non A-E, others , diagnosis obscure.[2] Acute hepatitis A can occur during pregnancy but has no effect on the fetus. Chronic hepatitis B requires identification in pregnancy because of long-term health implications for the mother and the effectiveness of perinatal vaccination (with or without pre-delivery maternal antiviral therapy) in reducing neonatal acquisition of chronic hepatitis B. Maternal transmission of hepatitis C occurs in 1% of cases, and there is no convincing evidence that the mode of delivery affects this. Hepatitis E is reported to progress to acute liver failure much more commonly in pregnancy, with a 20% maternal mortality.[4]

Preeclampsia defined by the triad of hypertension, edema, and proteinuria. It affects about 5%-10% of all pregnant women and usually occurs late in the second trimester or in the third trimester. In preeclampsia, hypertension is defined as having a systolic pressure greater than 140 mmHg and a diastolic pressure greater than 90 mmHg on at least two occasions that are at least 4 to 6 h apart in a previously normotensive patient, and proteinuria is defined as equal to or greater than 300 mg of protein in a 24 h urine collection or 1+ protein or greater on urine dipstick testing of two random urine samples collected at least 4 to 6 h apart.[5]

Eclampsia involves all features of preeclampsia and includes neurologic symptoms such as headaches, visual disturbances, and seizures or coma. Risk factors for preeclampsia and eclampsia include nulliparity, extremes of maternal age, insulin resistance, obesity, and infection.[5, 6] Abnormal laboratory values include a 10- to 20-fold elevation in aminotransferases, elevations in alkaline phosphatase levels that exceed those normally observed in pregnancy, and bilirubin elevations of less than 5 mg/Dl.[7] Maternal mortality from preeclampsia/eclampsia is rare in developed countries, but may approach 15%-20% in developed countries.[5] Likewise, the fetal mortality rate is rare, occurring in 1%-2% of births. The only effective treatment for preeclampsia is delivery of the fetus and placenta. Pharmacological agents used in preeclampsia include antihypertensives such as calcium channel blockers and low-dose aspirin. Magnesium sulfate may be administered if eclampsia develops.[3]

HELLP syndrome is a multisystemic disorder of pregnancy involving hemolysis, elevated liver tests, and low platelets. About 70% of cases occur antenatally, during the last trimester of pregnancy.[8]Most patients present with right upper quadrant abdominal pain, nausea, vomiting, malaise, and edema with significant weight gain. Less

commonly associated conditions include renal failure (with increased uric acid), diabetes insipidus, and antiphospholipid syndrome. Other late findings of HELLP include disseminated intravascular coagulopathy (DIC), pulmonary edema, placental abruption, and retinal detachment. Laboratory findings include hemolysis with increased bilirubin levels (usually less than 5 mg/dL) and lactate dehydrogenase (LDH) levels greater than 600 IU/L, moderately elevated aspartate aminotransferase (AST) and ALT levels (200 IU/L to 700 IU/L), and thrombocytopenia (less than 100 000/mL). In early stages, prothrombin time and activated partial thromboplastin time are normal, but in later phases, DIC may be present with increased levels of fibrin degradation products and D-dimer, and thrombin- antithrombincomplexes.[3] HELLP syndrome is present in 3-10% of preeclamptic toxemia. It is associated with weight gain and edema in 60%, maternal mortality of 20%, DIC in 4-38%, neonatal mortality rate of 31%, and rupture and hematoma of the liver in 2%.[9]

Intrahepatic cholestasis of pregnancy (ICP), also known as obstetric cholestasis, is a rare pregnancy specific liver condition that occurs in the late second or third trimester and has a prevalence of about 1/1000 to 1/10 000. It is significantly more common in SouthAsia, South America (especially Chile), and Scandinavian countries.ICP is also more common in women of advanced maternal age, multiparous women, and in women with a personal history of cholestasis with oral contraceptive use.[10] The prognosis for women with ICP is usually good, but it is associated with increased fetal morbidity and mortality, particularly from chronic placental insufficiency, preterm labor, fetal distress, and intrauterine death.[11] The classic symptom is pruritus that usually begins in the second or third trimester. It usually occurs in the palms and soles and may progress to the rest of the body, and the pruritus is often worse at night. Pruritus may be severe but is usually relieved within 48 h after delivery of the fetus. Jaundice occurs in approximately 10%-25% of patients and may appear within the first four weeks of the onset of pruritus.[12]Cholelithiasis and cholecystitis have been observed to occur with greater frequency in women with ICP.[13] Abnormal laboratory findings include elevated total bile acid levels up to 10- to 25-fold, with an increase in cholic acid and a decrease in chenodeoxycholic acid leading to a marked elevation in the cholic/ chenodeoxycholic acid ratio. The glycine/taurine ratio is also reduced. AST and ALT levels rarely exceed two times the upper limits of normal, but may approach 10- to 20-fold elevations in rare cases. Bilirubin levels may be elevated, but are usually less than 6 mg/dL. Serum alkaline phosphatase levels may also be elevated, but this is usually less helpful to follow given typical alkaline phosphatase elevations seen in pregnancy.Liver biopsy is usually not required to make the diagnosis of ICP.[14]

Acute fatty liver of pregnancy (AFLP) is a rare but serious maternal illness that occurs in the third trimester of pregnancy. With an incidence of 1 in 10 000 to 1 in 15 000 pregnancies, it has a maternal mortality rate of 18% and a fetal mortality rate of 23%.[7,15] This is more common in twin and first pregnancies, and may arise more frequently when the fetus is male. It typically presents between 31 and 38 weeks of pregnancy with vomiting and abdominal pain followed by jaundice. In severe cases, this may be followed by lactic acidosis, coagulopathy, encephalopathy and renal failure. Hypoglycaemia can also occur. The features are characteristic of a defect in beta-oxidation of fatty acids in the mitochondria that leads to the formation of small fat

droplets in liver cells (known as micro-vesicular fatty liver). Differentiation from toxaemia of pregnancy (which is more common) can be achieved by the finding of high serum uric acid le) vels and the absence of haemolysis. Overlap between acute fatty liver of pregnancy, HELLP andtoxaemia of pregnancy can occur. Early diagnosis, specialist care and delivery of the fetus have led to a fall in maternal and perinatal mortality to 1% and 7% respectively.[4] As with most pregnancy-associated liver diseases, the treatment of AFLP involves delivery of the fetus. In rare cases, patients will progress to fulminant hepatic failure with need for liver transplantation.[16] careful attention should also be paid to the infant given the increased risk of cardiomyopathy, neuropathy, myopathy, nonketotic hypoglycemia, hepatic failure, and death associated with fatty acid oxidation defects in newborns.[3]

Methods

This study was a cross-sectional observational study which was carried out in the Department of Gynae and Obs, Mymensingh Medical College Hospital, Mymensingh. The study was carried out from 25th August, 2015 to 24th February, 2016.Total 50 Patients of abnormal liver functions in third trimester of pregnancy admitted in dept of Gynae, Mymensingh medical college Hospital were included in this study. Patients having medical illness e.g: DM, Bronchial Asthma, heart diseases, thyroid dysfunction, patients who could not be investigated properly and any diagnostic dilemma are excluded from this study. Cases were interviewed by the investigator himself pointing to a meticulous history of present illness, past illness, family, personal, drug history. Clinical examination including general, systemic and obstetric examination were carried out. Investigations including liver function tests, viral markers, PBF, CBC with platelet count and other relevant investigations were carried out as and when required . All necessary information were recorded in pre -designed structured case record form and finally all the relevant data were processed and presented in tabulated method in the form of frequency table and contingency table/cross table.

Results

The age of the patients ranged from 18 to 34 years with the mean age of 22.4(SD+2.9) years. Most of the patients [34(68%)] werein the age group of 20-24 years (Table I)**.** Figure -1 presented the distribution of patients according to parity.In this study, 31(62%) patients were primigravida. Regarding the distribution of special biochemical markers amongpatients serum bilirubin was raised <10mg% in 20 (40.0%) cases, 10-15 % in 12(24%)cases, >15-20mg% in7(14%) cases, >20 mg% in 1(2%) cases .Serum SGPT And SOPT was found <100IU/ml in 17(34%) cases, 100-500 lU/ml in 23(46%)cases, >500IU/ml in 10(20%)cases. Alkaline phosphatase raised in 18(36%) cases. Prothrombin time 12-14second in 30(60%) cases>14-l8 second in!4 (28%) cases and >1 8-22 second in 6(12%) cases.(Table II). This study showed the causes of impaired liver function during pregnancy were viral hepatitis in 35 patients (70.0%), HELLP syndrome in 4 patients (8.0%), cholestasis in pregnancy in 3 patients (6.0%),Preeclampsia in 7(14%),Acute fatty liver in pregnancy in 1(2%). Hepatitis-E was the most frequent causes of viral hepatitis [26 (52.0%)] patients (Table III)**.** Table IV showed the most common obstetric complication was post partum haemorrhage (PPH) in 31 (62.0%) cases, followed by premature rupture of membrane (PROM) in 12(24.0%)cases, preterm labour in 13(26.0%) cases, ante partum haemorrhage

(APH) in 9(18.0%)cases, intrauterine death in7(14%)cases and eclampsia in 4(8%) cases. The most common maternal complication was post partum haemorrhage (PPH) in 31 patients (62.0%), followed by encephalopathy in 10 patients (20.0%), eclampsia in 4 patient(8%) (Table V)**.** Most of the patients were survived well [43 (86.0%) and there were 7 maternal deaths (14.0%). The causes of maternal deaths were fulminant hepatic failure in 6(12.0%) patients and uncontrolled post partum haemorrhage (PPH) in (2.0%) patients (Table VI)**.** Regarding fetal outcome,38 babies (76.0%) were born alive and 12 babies (76.0%) were born dead. Among the live born babies, resuscitation was needed in 30 cases, admission to neonatal unit required in 15 cases and early neonatal death in 9 cases. Among the dead born babies, intrauterine death was in 7 cases and still born 5 cases. Ultimately 29 (58.0%) babies were survived well and perinatal death was 21 (42.0%) (Table VII).

Table I: Distribution of patients on the basis of age group

Age	Frequency	Percent (%)
15-19	8	16
20-24	34	68
25-29	5	10
30-34	3	6
T0tal	50	100

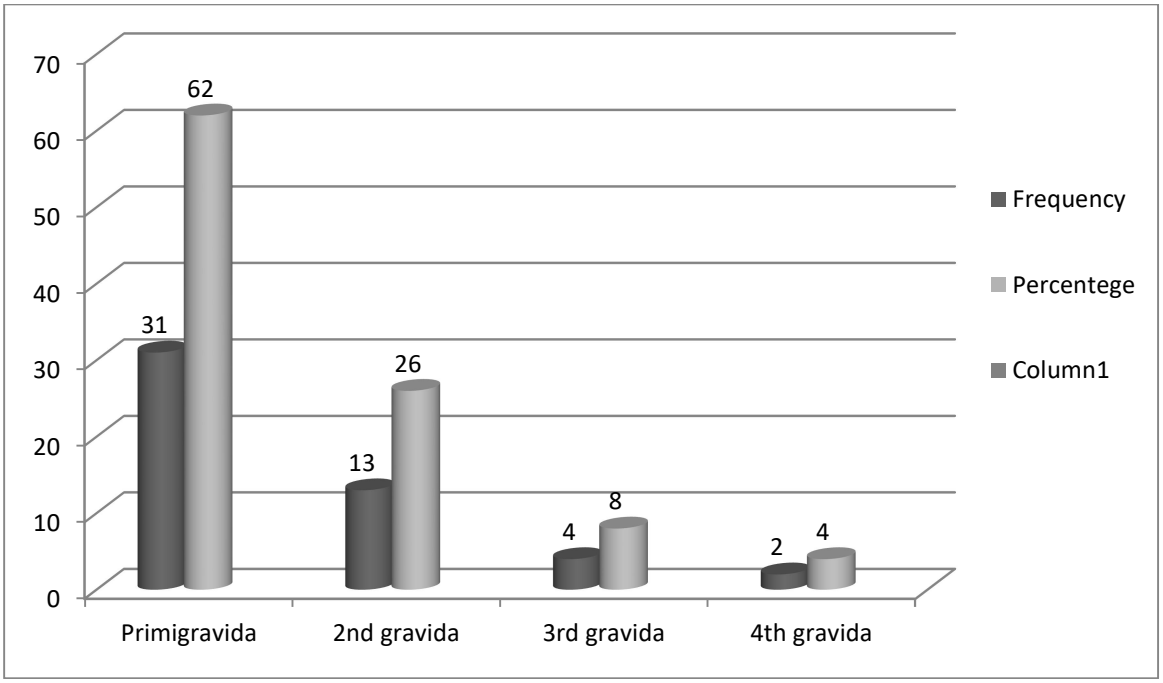

Figure 1. Distribution of patients according to parity(n=50)

Table II: Distribution of special biochemical markers among patients

Special biochemical markers	Frequency	Percentage
Serum Bilirubin		
<10mg%	22	44
10-15mg%	14	28
>15-20mg%	8	16
>20 mg%	1	2
SGPT and SGOT		
<100 IU/ml	17	34
lOO-500 IU/ml	23	46
>500-1000 IU/ml	10	20
Alkaline Phosphatase Raised	18	36
Prothrombin time		
12-14sec	30	60
>14-l8sec	14	28
>18-22sec	6	12
Activated partial thromboplastin time raised	4	8

Table III: Distribution of patients according to causes ofimpaired liver function during pregnancy

Causes	Frequency	Percentage
Viral Hepatitis	35	70.0
Hepatitis-A	0	0.0
Hepatitis-B	14	28.0
Hepatitis-C	3	6.0
Hepatitis-E	18	36.0
HELLP syndrome	4	8.0
Cholestasis in pregnancy	3	6.0
Preeclampsia	7	14.0
Acute fatty liver in pregnancy	1	2.0

Table IV: Distribution of obstetric complications among patients

Obstetric complications	Frequency	Percentage
Ante partum haemorrhage	9	18.0
Premature rupture of membrane	12	24.0
Preterm labour	13	26.0
Post partum haemorrhage	31	62.0
Intrauterine death	7	14.0
Eclampsia	4	8.0

Table V: Distribution of maternal complications among patients

Maternal complications	Frequency	Percentage
Encephalopathy	10	20.0
Disseminated intra vascular coagulation	1	2.0
Renal failure	2	4.0
Post partumhaemorrhage	31	62.0
Eclampsia	4	8.0
Shock	2	4.0
Death	7	14

Table VI: Distribution of patients according to maternal outcome (n=50)

Maternal outcome	Frequency	Percentage
Survives well	43	86.0
Maternal death		
Fulminant hepatic failure	6	12.0
Uncontrolled PPH	1	2.0

Table VII: Distribution of patients in term of fetal outcome

Fetal outcome	Frequency	Percentage
Born alive	38	76.0
Need resuscitation	30	60.0
Admission to neonatal unit	15	30.0
Early neonatal death	9	18.0
Born dead	12	24.0
Intrauterine death	7	14.0
Stillborn	5	10.0

Discussion

This descriptive cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, Mymensingh Medical College Hospital, Mymensingh, Bangladesh during the period from August 2015 to February 2016 with a view to find out fetal and maternal outcome in patients presenting with abnormal liver functions in third trimester of pregnancy . For this purpose 50 pregnant women were purposively selected according to inclusion and exclusion.

In this study the age of the patients ranged from 18 to 32 years with the mean age of 22.4 (SD ± 2.9) years. This result was supported by Shukla et al.[17] that among their 100 pregnant women with jaundice the mean age was 23.85±2.5 years. Majority of the study population (68.0%) were in the age group of 20-24 years, which was in accordance with the study of Siddiqui and Rashid,[18] that majority of the intrapartum jaundice patients (52.5%) were from 21-25 age group.

This study showed majority of the study population were either primigravida (62%) or 2^{nd} gravida (26%). This result *was* supported by Siddiqui and Rashid,[18] where most of the patients of pregnancy with jaundice were either primi (35%) or 2^{nd} gravida (40%). Shuklaet al.[17]found primigravidae (41%) constituted the largest group of pregnant population with jaundice.

In present study, majority of me cases the

causes of jaundice during pregnancy were viral hepatitis (70.0%), followed by Preeclampsia (14%) and HELLP syndrome (8.0%) and cholestasis in pregnancy (6.0%). Siddiqui and Rashid,[18] found viral hepatitis in 85% and HELLP Syndrome in 15% of cases with jaundice during pregnancy. Nearly similar result was observed in the study of Yi et al.[20]that majority of the cases the causes of jaundice during pregnancy were viral hepatitis (70.6%); HELLP syndrome constituted 5.9% and cholestasis in pregnancy constituted 15.9% cases.

In the current study, Hepatitis E was the most frequent causes of viral hepatitis (36%), followed by Hepatites B (28%) and Hepatitis C (6%). Siddiqui and Rashid,[18] found Hepatitis E being the major cause of infection (55,9%), followed by Hepatitis B in 20.6%,Hepatitis C in 5.9% cases. Yi et al.[20] found 29.4% patients of jaundice during pregnancy were due to Hepatitis B; 35.3% patients were due to Hepatitis E and 5.9%were due to Hepatitis A.

In the current study, raised serum billirubin level >10mg% in 46%of the cases. Majority of them (66%) had raised serum SGOT and SGPT above 100- >500 ill/ml. Alkaline phosphatase raised in 36% cases. Prothombm time raised in 40% cases and APTT raised in 8% cases. Tripti and Sarita.[19] found raised serum billirubin level >10mg% in48.48%o of the women.

In this study, the most common maternal complication was post partum haemorrhage (PPH) (62%), followed by encephalopathy (20%), eclampsia (8%), renal failure and shock (4%) and disseminated Intrauterine coagulation (2%). Tripii and Sarita,[19] reported that the most common maternal complication was encephalopathy(26.73%), followed by disseminated intravascular coagulation(21.88%) ,renal failure (19.45%), eclampsia (12.16%), shock (4.87%) ,post partum haemorrhage (PPH(4.87%) and pyrexia (4.87%).In this study, most of the patients survived well [43(86%)] and maternal mortality was 14%. The causes of maternal deaths were fulminant hepatic failure in 12% cases and uncontrolled post partum haemorrhage (PPH) in 2% patients. This result was similar to the study of Siddiqui and Rashid,[18] where 90% patients were improved well and maternal death was in 10% cases. Yi et al.[20] reported maternal mortality in 33.3% cases. Shuklaet al.[17] reported maternal mortality in 6.0% cases.

In the current study, 38 (76.0%) babies were born alive and 12 (24.0%) babies were born dead born. Among the live babies, resuscitation was needed in 30(60%) cases, admission to neonatal unit required in 15(30%) cases and early neonatal death in 9(18%) cases. Among the dead babies, intrauterine death was in 7 cases and fresh still born in 5 cases. Ultimately 58% babies were survived well and perinatal mortality was 42% which was supported by Siddiqui and Rashid,[18]where 23 (56.0%) babies survived well and there were perinatal death in 18 (44.0%) babies; the causes of perinatal death were intrauterine death in 5 (12.2%), still birth in 4 (9.8%) and early neonatal death in 9 (22.0%).

Conclusion

Impaired liver function during pregnancy is still a life threatening condition for mother and fetus. This study suggests that it is mostly limited to last trimester. The major cause of impaired liver function during pregnancy was viral hepatitis, Hepatitis E was the most frequent causes of viral hepatitis. Major complications were postpartum haemorrhage and encephalopathy. Most of the patients survived well and maternal death was

14.0%.The causes of maternal deaths were fulminant hepatic failure and uncontrolled post partum haemorrhage (PPH). Ultimately perinatal mortality was 42%. In conclusion, Impaired liver function in pregnancy is associated with an increase in maternal mortality and morbidity, obstetric complications, and poor fetal outcomes with high perinatal mortality.

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