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CASE REPORT

Liver Disease in Pregnancy

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ABSTRACT

Although jaundice does not occur frequently during pregnancy and associated with several liver disorders. The liver disorders that are specific to pregnancy including hyperemesis gravidarum, intrahepatic cholestasis of pregnancy, acute fatty liver of pregnancy, pre-eclcampsia, eclampsia, HELLP and hepatic rupture, may have a profound impact on the morbidity and mortality rates of mother and fetus. Although an unequivocal diagnosis is often difficult to make, It should be attempted in a timely manner so that optimal treatment can be determined. The maternal and fetal outcome are affected in an adverse manner if these conditions are left untreated.

Keywords: jaundice, liver disease, pregnancy

Introduction

Jaundice in pregnancy is related to hepatic disorders in pregnancy. The liver is one of many organs affected by the physiologic and hormonal changes that occur during pregnancy.[1] Abnormal liver tests occur in 3%-5% of pregnancies with many potential causes including coincidental liver (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: this fall into 2 main categories depending on its association with or without pre-clampsia.[2]

Case Report

A 38 years old G3P2 was admitted at 36 weeks of gestation, complaining about difficulty in respiration, decreasing fetal movement and generalized edema. A week ago she felt swollen, weak and icteric sclera. The urine's color was dark yellow but stool was normal. Two days ago she felt weak, swollen, nauseating, sometimes vomiting and experienced discomfort at epigastrium, difficulty in respiration and decreasing fetal movement. But she could still eat and had no fever. A day later her symptoms were worse and she was referred to this hospital.

She had two children, both with normal labor; 17 and 7 years ago. She visited antenatal-care 4 times and the gestation was 18th weeks at the first visit. The ultrasound showed alive twins pregnancy, 18 weeks of gestation, both male with a single placenta grade 0, normal amniotic fluid. All of her initial laboratory screening for ANC were normal. The ANC was shown in table 1. Upon admission BT

36.8°C, BP 130/90 mmHg., RR 22/min, PR 100/min, BW 70 Kg, and HT 160 cm. She had good consciousness icteric sclera, normal heart and lung findinds. The fundal height was 4/4 > umbilicus, cephalic presentation a both with fetal heart beat of 148 and 152/min.

Liver and spleen were not palpable. There were pitting edema 2+ in both legs and normal deep tendon reflex. The complete blood count, coagulogram, liver function test were shown in table 2, 3 and 4 Complete blood count, BUN, Cr and electrolytes were within normal limit. Urinalysis revealed trace glycemia and protienuria, bilirubin 2+ and normal microscopic. There were prolonged PT 25.3 min (11-13 min.) and PTT 44.6 min (25-32 min.) The LFT showed hypoprotienemia, high bilirubin, ALP and AST. The hepatic profile HBsAg, Anti HBc IgM, and Anti HCV were negative.

The ultrasound revealed living male twins, the estimated fetal weight 2410 and 2300 grams. The placenta was single, grade 3, attached to the anterior fundus. The membranes were 1.5 mm. thick and amniotic fluid was adequate. The provisional diagnosis was G3P2 36 weeks gestation with twins pregnancy and acute fatty liver of pregnancy, (pre-eclamptic liver diseases) (HELLP syndrome) or fulminant viral hepatitis. The planned management was cesarean section with tubal resection under general anesthesia.

There was 100 ml, ascites. The birth weight of twins A and B were 2240 and 2100 gm with APGAR score 7, 8 and 7, 7 respectively. The placenta was single with diamnion monochorion, weight 1000 gm. The estimated blood loss was 600 ml. The postoperative and postpartum were normal in general, except for icteric sclera. Fresh frozen plasma was given in order to correct coagulopathy. The follow up laboratories were shown in tables 2, 3 and 4. The patient could be discharged one week after delivering both children. The ultrasound of the liver showed increase liver parenchymatous echo suggesting liver parenchymatous disease.

Table 1. Summary of physical and obstetic exaninations at differenct gestational ages.

	25/9/2006	**30/10/2006**	**27/11/2006**	**18/12/2006**
GA (wks)	18+5	24	28	31
BW (kg)	55	57	58	60
BP (mmHg)	95/61	89/59	85/59	84/60
U.alb/sug	neg	neg	neg	Neg
FH	2/3> PS	26 cm	33 cm	34 cm
Presentation	-	-	Cephalic/transverse	Transverse/ transverse
FHS	-	+	+	+
Engagement	-	-	-	-
LAB	ultrasound	GCT = 138mg%	ultrasound	-
FMC	-	good	good	good
Appointment Date	30/10/2006	27/11/2006	18/12/2006	8/01/2006

Table 2. Results of complete blood count at different gestational ages.

	27/01/2007	28/01/2007	29/01/2007	30/01/2007	31/01/2007	1/02/2007	5/02/2007
WBC	10,250	11,020	13,200	9,200	7,940	12,250	6,990
Neutrophil	78.6	85.5	90.7	80.4	75.0	86.8	79.5
Lymphocyte	15.8	9.9	6.1	11.5	15.1	6.8	13.2
Hb	14.5	11.4	9.8	10.1	10.3	12.2	11
Hct	42.1	34.4	29.8	30.0	31.1	36.4	33.1
MCV	92.5	93	82.3	81.3	81	81.1	82.5
Platelets	104,000	83,000	50,000	47,000	47,000	66,000	107,000

Table 3. Results of liver function tests at different gestational ages.

	27/1/2007	29/1/2007	30/1/2007	31/1/2007	1/2/2007	4/2/2007
Total protein	5.7	5.2	4.9	5.5	5.6	5.8
Albumin	2.1	2.5	2.1	2.4	2.4	2.5
Globulin	3.6	2.7	2.8	3.1	3.2	3.3
Total bilirubin	14.10	10.68	10.64	11.81	10.43	7.46
Direct bilirubin	12.24	8.98	8.83	9.87	8.37	5.12
ALP	441	169	137	171	191	173
AST	80	57	42	49	49	54
ALT	39	39	29	29	29	32

Table 4. Coagulograms at different gestational ages.

	27/1/2007	28/1/2007	29/1/2007	30/1/2007	31/1/2007	4/2/2007
PT	25.3	17.4	16.6	17.5	16.5	12.5
INR	2.09	1.44	1.38	1.45	1.32	1.02
PTT	44.6	31.7	30.2	31.1	28.1	25.8
	FFP 4 U	FFP 2 U q 8 hr	FFP 4 U			

Discussion

Jaundice in pregnancy is associated with hepatic disorders and alteration liver function particularly in serum enzymes.[3] The causes are HELLP syndrome, acute fatty liver of pregnancy and viral hepatitis.[4,5] Early intervention and appropriate diagnosis can substantially reduce the derangement of pregnancy.[5]

Viral hepatitis occurs any time during pregnancy. Infections are mostly subclinical and the disease is insidious onset. The clinically symptoms-nausea vomiting, headache, and malaise-may precede jaundice by 1 to 2 weeks.[6] When jaundice develops, symptoms usually improve. Serum transaminease levels vary, and their peaks do not correspond with disease severity. Peak levels that range from 400 to 4,000 U/L are usually reached by the time jaundice develops. Serum bilirubin typically continues to reach its peak at 5 to 20 mg/dL.[6] Usually there is complete clinical and biochemical recover within 1 to 2 months in all cases of hepatitis A and most cases of hepatitis B. Some cases develop fulminant hepatitis and liver failure with hypoglycemia, hepatic encephalopathy and coagulopathy. The serological test for hepatic profile (HBsAg, Anti HBc IgM. Anti-HAV IgM.) are simplified diagnosis approach in patients with hepatitis.[6]

HELLP syndrome a syndrome of hemolysis include elevated liver enzymes and low platelet count associated with preeclamsia.[6,7] The patients show hypertension, edema and protienuria. The liver enzyme is not elevated to more than 500 IU/L and serum biliruin is not high.[6] The presenting symptoms were headache, abdominal or epigastric pain and hematuria that present in sever pre-ecclampsia.

Acute fatty liver in pregnancy is an uncommon complication that has often proved fatal for both mother and fetus.[7] It is also called acute fatty metamorphosis or acute yellow atrophy.[6] Acute fatty almost always manifests late in pregnancy.[7] The most common presenting symptoms were malaise nausea and/or vomiting, abdominal pain and progressive jaundice.[8] About half of all women have hypertension, protienuria, and edema sign-suggestive of preeclampsia. There is usully severe liver dysfunction with hypofibrinogenemia, hypoalbuminemia, hypocholesterolemia and prolonged clotting times. Hyperbilirubinemnia is usually less than 10 mg./dL, and there are modestly elevated serum transminase levels.[6]

This case report can definitely exclude acute viral hepatitis but the clinical presentation, biochemical findings were not clearly distinguished between HELLP syndrome and acute fatty liver of pregnancy. This condition is overlapping syndrome and is initially managed in a similar manner.

The immediate termination of pregnancy preferably by cesarean section has been shown to improve both maternal and fetal outcomes.[4]

Conclusion

Early intervention and appropriate diagnosis can substantially reduce the morbidity and mortality associated with hepatic derangement of pregnancy. Early diagnosis and immediate delivery are essential for maternal and fetal survivals.

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