Case Report

Cirrhosis of Liver with Third Trimester of Pregnancy: First Report of two Cases From Bangladesh

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Summary

Pregnancy is uncommon in cirrhosis. This is however not related to the aetiology of cirrhosis and rather results from hepatic dysfunction, reduced hepatic blood flow and reduced end organ sensitivity to sex hormones in these patients. Pregnancy in cirrhosis is associated with high incidence of foetal loss as well as higher maternal mortality both during pregnancy and in the post-partum period. Here we report two cases of cirrhosis of liver with third trimester of pregnancy for the first time from Bangladesh

Introduction

Cirrhosis of liver with pregnancy is uncommon. The first report dates back to 1923 [1]. Since then till 1998, there are around only 100 reports of cirrhosis of liver with pregnancy in the literature [2, 3, 4, 5, 6]. Here we present the first two cases of cirrhosis of liver with pregnancy from Bangladesh.

Case Report

The first patient, a 29 year old house-wife coming from middle-class socio-economic background presented to us with 26th weeks of pregnancy. She was the mother of 2 children aged 7 and 5 years respectively. On examination, she had ascites and leucocychea. Investigations showed serum bilirubin 0.7 mg/dl, serum ALT 55 U/L, serum alkaline phosphatase 111 U/L, serum albumin 2.4 gm/dl, prothrombin time 17 sec (control 12 sec), HBsAg (ELISA) positive and anti-HCV (ELISA) negative. Her ultrasonography of whole abdomen showed coarse, irregular liver, spleenomegaly and huge ascites (Figures 1 & 2). She had multiple columns of grade III oesophageal varices on endoscopy of upper gastro-intestinal tract.

She was diagnosed as a case of hepatitis B virus related decompensated cirrhosis of liver with portal hypertension. The patient developed haematemesis and malaena while in hospital and despite adequate measures she could not be saved.

The second patient, a 22 year old house-wife also coming from middle-class socio-economic background presented to us with 31st weeks of pregnancy. This was her first pregnancy. On examination she had ascites. Her investigations showed haemoglobin 7.3 gm/dl, total count of WBC 5500/cmm, platelet count 200,000/cmm, mild microcytic hypochromic anaemia in peripheral blood film, serum sodium 137.3 mmol/L, serum potassium 3.43 mmol/L, serum bilirubin 0.8 mg/dl, serum ALT 40 U/L, serum albumin 39.6 gm/L, prothrombin time 14.4 sec (control 12 sec), both HBsAg and anti-HCV negative by ELISA, serum iron 36 micro gm/dl, serum ferritin 7.03 ngm/m.. Her ultrasonography of whole abdomen showed hepatomegaly, mild spleenomegaly, huge ascites and 31st weeks of pregnancy with breech presentation (Figures 3 & 4). She had multiple columns of grade III oesophageal varices with red signs and peptic ulcer disease on endoscopy of upper gastro-intestinal tract.

She was diagnosed as a case of cryptogenic decompensated cirrhosis of liver with portal hypertension. We did prophylactic oesophageal band ligation in this patient. The patient gave birth to a dead baby near term through normal vaginal delivery.

Discussion

Cirrhosis leads to infertility, which correlates with the degree of hepatic dysfunction [2]. This is because in cirrhosis, there is reduction of sex hormone receptors in the liver as well as reduced end organ sensitivity to these hormones. Irregular menses are common [7]. This has been attributed to dysfunction of hypothalamopituitry axis [8]. Besides malnutrition and poor hepatic metabolism of sex hormones also play role. Reduced hormonal metabolism is due to reduced hepatic blood flow and shunting of blood away from the liver [9].

Review of literature has shown that there is high incidence of foetal loss in cirrhotics [5]. There is 10-15% risk of

abortion in early pregnancy [2, 7]. Literature review shows that 45 out of 69 babies born to cirrhotics survived the neoatal period [5].

The outcome is also gloomy for mothers in pregnancy. Maternal prognosis depends more on the extent of hepatic dysfunction than the aetiology of cirrhosis although the outcome is best in well compensated cirrhotics resulting from primary biliary cirrhosis and Wilson's disease [10, 11, 12]. On the contrary, the outcome is poor in alcoholics with cirrhosis [13].

The most significant complication in cirrhotics with specially second and third trimester of pregnany remains oesophageal variceal bleeding [13]. During pregnancy, there is rise in circulatory volume, elevation of portal pressure and pressure of the gravid uterus on inferior vena cava causing diversion of venous blood to azygous venous system [5]. It has been shown that 38 patients with cirrhosis out of 160 bled during pregnancy [7]. Hepatic encephalopathy also remains a concern and cause of death in this setting [5].

In the post-partum period there is high incidence of gastrointestinal haemorrhage responsible for 5 out of 7 deaths during this period [5]. Other post-partum complications in these patients include uterine haemorrhage, hepatic coma and ascites [5].

Conclusion

Although uncommon, cirrhosis with pregnancy may be encountered in clinical practice. It warrants careful and coordinated approach as the outcome may be fatal for both mother and the baby.

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Figure 1: Ultrasonography showing coarse ecotexture of liver with ascites in the first patient.

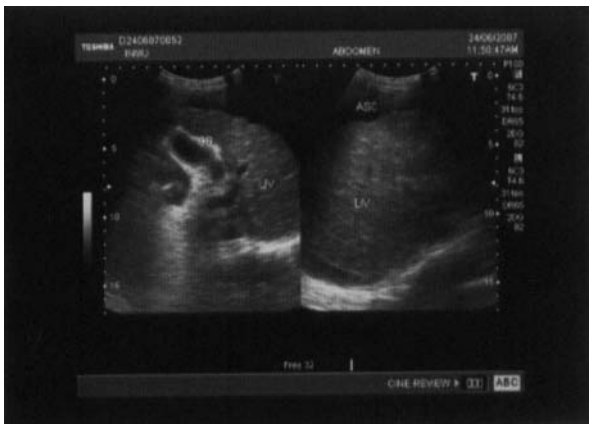


Figure 2: Ultrasonography showing gravid uterus in the first patient.

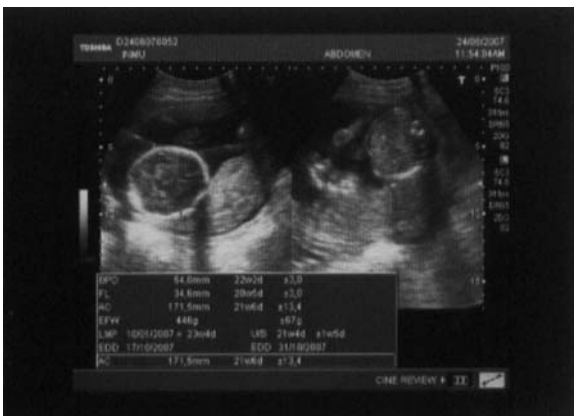


Figure 3: Ultrasonography showing coarse ecotexture of liver with ascites in the second patient.

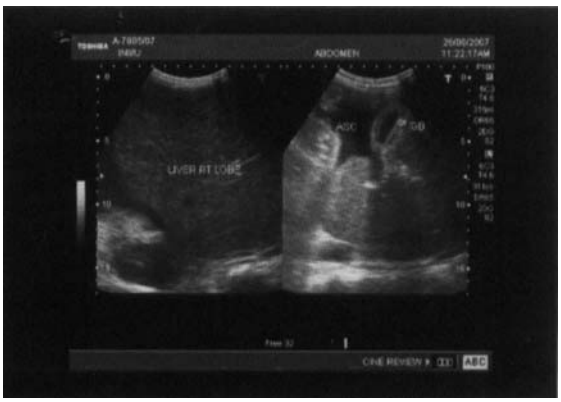
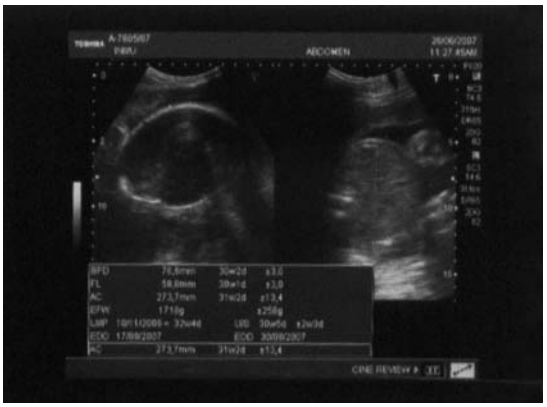


Figure 4: Ultrasonography showing gravid uterus with breech presentation in the second patient



Case Report

Case Report of Autoimmune Hepatitis from a Tertiary Centre in Bangladesh

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Abstract

Autoimmune hepatitis is defined as chronic liver disease of unknown aetiology with aberrant autoreactivity and genetic predisposition, characterized by female predominance, circulating auto-antibodies, hypergammaglobulinaemia and association with HLA DR3 and HLA DR4 [1].

We present two patients with autoimmune. The first patient is a young lady who was diagnosed with autoimmune chronic hepatitis. The second patient, on the other hand, is an elderly gentleman who presented to us with autoimmune hepatitis related decompensated cirrhosis of liver

Case Report

The first patient was a 20 year old house wife from an upper middle class family. She presented to us with unexplained raised serum ALT, detected incidentally on routine investigation.

Her investigations revealed serum ALT 74 U/L, serum AST 62 U/L, fasting blood sugar 4.6 mmol/L, triglyceride 170 mg/dl, HDL 39 mg/dl, TSH 1.44 µmol/L, insulin resistive index 0.5 and urinary copper <0.03 mg/L. Her CA 19.9 was 8.9 U/L. There was no KF ring on slit lamp ophthalmic examination. She tested negative for HBsAg, HBV DNA by PCR, anti-HCV, ASMA and AMA, but was positive for ANA at 1:120 dilution.

Her endoscopy of upper GIT was normal and so was the abdominal ultrasound. CT san of upper abdomen was also done and revealed inflammatory or focal fatty change in right lobe of liver. A liver biopsy was done which revealed necro-inflammation (HAI-NI) score of 7 and fibrosis (HAI-F) score 1. The diagnosis of type 1 autoimmune hepatitis was done made.

The second patient, a 72 year old gentleman, coming from upper middle class socio-economic background presented to us with ascites. He was a known diabetic for 20 years.

On examination he had stigmata of cirrhosis of liver. His liver panel showed serum bilirubin 16 µmol/L, ALT 66 U/L, AST 114 U/L, alkaline phosphatase 291 U/L, albumin 27 gm/L, prothrombin time 12 sec. (control 12 sec.) and AFP 1.5 ngm/ml. He tested negative for HBsAg, anti-HBs, anti-HBc total, HBV DNA by PCR, anti-HCV and HCV RNA by PCR. ANA was positive at 1:640 dilution, but tests for AMA, anti-M2, anti-LKM 1, anti-SLA and anti-LP antibodies were all negative. Ascitic fluid was examined and SAAG was calculated at 2.1 gm/L. Endoscopy of upper GIT revealed grade II oesophageal varices.

The patient's serum creatinine was 82 µmol/L, serum sodium 133 mmol/L, potassium 3.2 mmol/L, chloride 99 mmol/L and bicarbonate 27 mmol/L. His IgG was 74 gm/L. Ultrasonography of abdomen revealed coarse ecotexture of liver with irregular,