Clinical Case Reports International

Case Study
Published: 07 Nov, 2018

Peripartum Decompensation Uncovering Underlying Asymptomatic Cirrhosis and Portal Hypertension: An Unusual Case Report

Yadav P[1] and Tyagi S[2]*

[1]Department of Gynaecology and Obstetrics, Sant Parmanand Hospital, India

[2]Department of Gynaecology and Obstetrics, Lok Nayak Hospital, India

Abstract

Pregnancy is rarely encountered with liver cirrhosis, but carries a significant risk of complications with poor materno-fetal outcomes. Late second trimester and early third trimester is the most common period of hepatic decompensation when most of the complications are seen. We hereby describe an unusual case where the diagnosis of cirrhosis was unknown till the time of delivery due to its asymptomatic nature. However, the diagnosis was finally made with high index of suspicion and was further optimally managed to achieve a successful pregnancy outcome. The aspects of optimal management have been discussed.

Keywords: Cirrhosis; Portal Hypertension; Pregnancy

Introduction

Pregnancy associated with cirrhosis is a rare occurrence. Nevertheless, pregnancy in a patient with cirrhosis and portal hypertension is a unique problem which needs specialized care. Pregnant patient with cirrhosis and portal hypertension is at risk of significantly increased foetal loss, oesophageal variceal bleeding, splenic artery aneurysm rupture, hepato-renal syndrome and hepatic decompensation [1,2]. The complications and hepatic decompensation most commonly occur in the early third trimester (28 to 32 weeks of gestation). The present report describes a rare case with asymptomatic cirrhosis going undetected during pregnancy only to present with hepatic decompensation at the time of delivery and post-partum period.

Case Presentation

A 5th gravida multiparous 32 year old lady presented to emergency at 36 weeks and 5 days with complains of labour pains. She had been non-compliant with her antenatal visits with only two visits, one at 16th and another at 24th week of gestation. This was a precious pregnancy as she had two intrapartum still births associated with difficult delivery in the past. The patient had only 1 live issue who was 6 year old and was born of a caesarean section at term, done for meconium stained liquor in early labour. There was no history of any chronic medical or surgical illness.

On examination vitals were stable, with mild pallor and no icterus. Per abdomen uterus was term size with mild contractions and normal foetal heart rate. On per vaginal digital examination, cervix was 2 fingers loose and 30% to 40% effaced with intact membranes. The pelvis seemed to be inadequate for the baby. An emergency caesarean section was contemplated in view of anticipation of a difficult delivery. After shifting the patient to the operation theatre, spinal anaesthesia was given. Per operatively large dilated vessels (5 mm to 7 mm in diameter) were encountered subcutaneously and in the preperitoneal space. Some of them had to be secured with suture ties after being accidentally incised during surgery. A live male baby of 2.3 kg was delivered. Mild atonic post-partum haemorrhage occurred which could be controlled by uterine massage and uterotonics. Despite the best efforts, there was a blood loss of approximately 1.3 liters. An abdominal drain was inserted to prevent intra-abdominal collection. As the pre-operative haemoglobin was 7.7 gm % with a platelet count of 99,000/dL and an INR of 1.88, 1 unit of packed red blood cells and 4 units of fresh frozen plasma were transfused.

In the immediate post-operative period patient was comfortable, afebrile, maintained stable vitals and adequate urine output. However, the abdominal drain continued to produce an output

Correspondence:
Preeti Yadav, Department of Gynaecology and Obstetrics, Sant Parmanand Hospital, 27 Shanti Vihar, Karkardooma, New Delhi -110092, India, Tel: +91-9810995505; E-mail: yprity.04@gmail.com

Received Date: *25 Sep 2018*
Accepted Date: *03 Nov 2018*
Published Date: *07 Nov 2018*

Citation:
Yadav P, Tyagi S. Peripartum Decompensation Uncovering Underlying Asymptomatic Cirrhosis and Portal Hypertension: An Unusual Case Report. Clin Case Rep Int. 2018; 2: 1083.

of serosanguineous fluid at approximately 50 to 100 cc/hr. The blood investigations were remarkable in terms of deranged complete blood count (haemoglobin 8.8 gm %, platelet count 81,000/dL, and total leukocyte count 15,100/dL) and deranged liver function tests (total bilirubin 1.8 mg/dL, direct bilirubin 0.9 mg/dL, aspartate aminotransferase/alanine aminotransferase-327/125 IU/L, total protein-4.4 gm % and serum albumin-2.3 gm %). On day 2, peritoneal drain could not be removed in view of persistent high output of 1500 ml to 1700 ml per day. Adequate amount of fluids were given keeping in mind the drain output, urine output and in sensible losses and peritoneal fluid became clear gradually. Ascitic fluid investigations revealed a white cell count of 1000/dL with 90% polymorphs, sugar-106 mg % and a protein level of 0.7 gm %, suggestive of infected transudative fluid. Ultrasound examination of the abdomen revealed lobulated 17.7 cm liver with coarsened echotexture, splenomegaly (17.9 cm), dilated portal vein (1.2 cm) and splenorenal collaterals, suggestive of cirrhosis with portal hypertension. However, there was no history of hematemesis, melena, recent jaundice or blood transfusion. And there were no stigmata of chronic liver disease that were evident.

Further investigations revealed negative tests for hepatitis B antigen, anti-hepatitis C antibody and autoimmune hepatitis antibody panel. Serum ceruloplasmin was done and Wilson's disease was ruled out. Iron profile was done and the patient was found to be iron deficient, for which iron replacement was initiated. The blood and the ascitic fluid cultures failed to show any microbiological growth, however the patient was already on antibiotics when the samples were collected. A liver biopsy was planned as per the gastroenterologist's opinion to ascertain aetiology of cirrhosis, but was not done as the patient declined consent. Final diagnosis of liver cirrhosis with portal hypertension and secondary bacterial peritonitis was made. Treatment was started with beta-blocker (propranolol), spironolactone, furosemide, intravenous albumin, stool softener and higher antibiotics in consultation with the gastroenterologist. Upper gastrointestinal endoscopy was also done and revealed grade 2 oesophageal varices for which endoscopic variceal ligation was done. The peritoneal drain output reduced gradually allowing its removal on day 12. Stitches were removed on post operative day 20 and was discharged next day.

Discussion

Pregnancy with portal hypertension secondary to cirrhosis is a scenario which is rarely encountered but commonly associated with serious complications leading to significant morbidity and poor prognosis. Cirrhosis is uncommon in reproductive age group with a reported incidence of less than 45 cases per 100,000 women [3]. Moreover, metabolic and hormonal derangements associated with cirrhosis lead to anovulation and hence infertility. In more recent times improvements in the treatment of chronic liver disease resulted in higher conception rates, making pregnancy with cirrhosis more common, however the exact incidence remains unknown. The diagnosis of cirrhosis usually surfaces in early pregnancy with routine evaluation or is known beforehand. Thus there is certain level of preparedness for screening and management of complications in the third trimester and peripartum period. Despite all measures, the pregnancy outcomes in cirrhosis remain dismal. The present case is unique in terms that a diagnosis of cirrhosis could be made only at the time of delivery. Even then, with appropriate timely recognition and management, a successful outcome was achieved.

Approximately up to 50% of the pregnancies in cirrhotic patients are expected to face complications, the most common being variceal haemorrhage and liver failure [4,5]. Refractory ascites, hepatic encephalopathy, hepatorenal syndrome, spontaneous bacterial peritonitis, post-partum haemorrhage and adverse fetal outcome are other complications which may be encountered [6]. The risk of variceal haemorrhage is 400 times during pregnancy and remains one of the deadliest complications. Up to 80% patients with detectable varices are expected to bleed during the gestational period. The most common occurrence is during the late second and early third trimester, which is the period of maximum vasodilation [7]. Although uncommon, splenic artery aneurysms also rupture and bleed around the same time period. The importance of splenic artery aneurysms lie in the requirement of a high index of suspicion to reach a diagnosis and its association with high rates of materno-fetal mortality. Secondary to increased intra-abdominal pressure during pregnancy, extravasation of fluid from splanchnic vessels is hindered, making ascites relatively uncommon as a complication during pregnancy. The rates of fetal wastage are alarming in cirrhosis with estimates reported in the range of 10% to 66%. Even with live births, the risk of prematurity is significantly high. A case series reported a prematurity incidence of 25% amongst live births in pregnancies in cirrhotic patients [8]. It is important to recognize that the occurrence of complications in cirrhosis is often interlinked with one complication triggering the other. Variceal haemorrhage may precipitate hypotension, acute liver failure and thus hepatic encephalopathy. It also increases the risk of spontaneous bacterial peritonitis which in turn increases the risk of hepatorenal syndrome significantly. Thus prevention of complications in the first place remains an important goal.

Management of pregnancy in cirrhosis entails careful assessment of liver function, esophageal varices and screening of other complications followed by an individualistic approach for each case. The importance of maintaining hydration and avoiding hypotension cannot be overemphasized. Hypotension can precipitate hepatorenal syndrome and is a risk factor of hepatic decompensation and encephalopathy. The risk of hypoglycaemia is real and needs to be addressed on a regular basis with generous supplementation of simple sugars and fruit juices. Renal function should be monitored on a regular basis to pick up any dysfunction early. Regarding management of labour, choice of mode of delivery should be made as per obstetric indications. Underlying coagulopathy in cirrhosis often needs correction to prevent post-partum haemorrhage which is common and has been reported in 7% to 26% of the cases. Risk further increases with a caesarean section and blood products should be readily available during labour of such a patient. Regional anaesthesia should be preferred over general anaesthesia if required during operative delivery. And sedatives should be used minimally as they may precipitate hepatic encephalopathy.

In the present case, dilated pre-peritoneal veins and significant ascites amounting to continuous post-operative abdominal drain output were important markers towards portal hypertension. Since the patient had tolerated the course of gestation well without any symptoms, a chronic significant systemic illness was hardly suspected. However, to our surprise, an unexpected diagnosis of cirrhosis uncovered on further evaluation. The patient was successfully managed with beta blockers (propranolol 20 mg thrice daily), spironolactone (50 mg twice daily), antibiotics for secondary bacterial peritonitis and ligation of esophageal varices which were picked up before they bled on screening upper gastrointestinal tract endoscopy.

It was difficult to maintain fluid balance because of large drain output, but formed an important part of the management. Fortunately in this case, the outcome was good for both the baby and the mother.

To conclude, the possibility of portal hypertension and liver cirrhosis should be borne in mind when significant ascites and dilated pre-peritoneal vessels are noted on caesarean section, irrespective of the course of pregnancy. Further evaluation and management by a multidisciplinary effort from the obstetrician, gastroenterologist and neonatologist holds the key to a successful pregnancy outcome.

References

1. Tiribelli C, Rigato I. Liver cirrhosis and pregnancy. Ann Hepatol. 2006;5(3):201.
2. Cerqui AJ, Haran M, Brodribb R. Implications of liver cirrhosis in pregnancy. Aust N Z J Obstet Gynaecol. 1998;38(1):93-5.
3. Russell MA, Craigo SD. Cirrhosis and portal hypertension in pregnancy. Semin Perinatol. 1998;22(2):156-165.
4. Pajor A, Lehoczky D. Pregnancy and extrahepatic portal hypertension. Gynecol Obstet Invest. 1990;30(4):193-7.
5. Misra S, Sanyal AJ. Pregnancy in a patient with portal hypertension. Clin Liver Dis. 1999;3(1):147-162.
6. Tan J, Surti B, Saab S. Pregnancy and Cirrhosis. Liver Transpl. 2008;14:1081-91.
7. Lee WM. Pregnancy in patients with chronic liver disease. Gastroenterol Clin North Am. 1992; 21(4):889-903.
8. Pajor A, Lehoczky D. Pregnancy in liver cirrhosis. Gynecol Obstet Invest. 1994;38:45-50.