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Case Report

Massive Ascites in a Patient with Severe Preeclampsia

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Abstract

Objective: *To present a rare case of massive ascites with severe preeclampsia.*

Case Report: *A 27-year-old, G2P1, woman presented at 37 weeks of gestation with severe preeclampsia with ascites with breech with intrauterine fetal growth restriction. A large amount of free fluid was noted intraperitoneally during caesarean section. Postoperatively ascites fluid continued to come through drain. Patient developed respiratory distress, hypertension and hypoproteinemia. Which were managed by multidisciplinary approach. These patients need intense surveillance to prevent complication.*

Introduction

Preeclampsia refers to the new onset of hypertension and proteinuria or hypertension and end-organ dysfunction with or without proteinuria after 20 weeks of gestation in a previously normotensive woman[1]. The pathophysiology of preeclampsia underlies maternal and fetal/ placental factors. Ascites can develop in preeclampsia[2], but massive ascites due to preeclampsia is a rare finding. Here a case of severe preeclampsia, who was presented at her 37weeks of pregnancy with massive ascites and was subsequently managed by multidisciplinary approach.

Case Report

A 27-year-old G2P1 lady came to a private clinic with severe preeclampsia at 37 weeks of gestation. She was in irregular and infrequent antenatal checkup. Her expected date of delivery according to her 2nd trimester USG corresponds with her LMP. But she noticed swelling of her feet from 28 weeks of gestation, and was on antihypertensive drug since 34 weeks of gestation. At first was on α-methyldopa. Then from 36weeks of gestation her BP was 160/120mmHg and Labetelol 200mg BD was added in addition to α-methyldopa 250mg 3 times per day. She was referred to tertiary care hospital. Then after development of severe headache and respiratory distress at her 37 weeks of gestation she was admitted in a private hospital. On admission her blood pressure was 180/ 120 mmHg and urinary albumin 2+. Abdominal examination revealed a growth restricted fetus with symphysio-fundal height of 30weeks size and shifting dullness was positive and abdominal girth was 100cm. On per vaginal examination cervix was soft, not effaced, posterior and with breech

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presentation. Baseline blood parameters such as CBC, blood sugar, S creatinine, S urea, and coagulation profile were sent and within normal limit. But USG revealed a severely intrauterine growth retarded fetus (fetal weight 1.3kg) with massive ascites. Cardiotocography showed late decelerations and she was diagnosed to have fetal distress. Prophylactic magnesium sulphate and antihypertensive drugs started and emergency cesarean section was performed. After opening abdomen there was huge ascites, about 3.5 liters of ascitic fluid drained and 1200 gm male fetus was delivered with APGAR score of 8/10. Abdomen was explored thoroughly to exclude any other intra-abdominal tumor/pathology. A drain kept in situ. Patient was intensely monitored during intraoperative and postoperative period. Postoperatively, ascitic fluid continued to come on 1st POD 2.5 liters, 2nd POD about 1.5liters. Her Blood Pressure was stable for first 24 hours on postoperative period then raised to 160/110mmHg and she was breathlessness and there was crepitations on both lungs field on auscultation. Postoperative urine output was normal. On postoperative period tab Bisoprolol and Inj Lasix were added. Her CBC, SGPT, PT, APTT, S creatinine, Serum electrolytes reports and echocardiography were within normal limit. but her urine R/M/E report shows RBC 12-16/HPF and protein +++, Urinary albumin to creatinine ratio (ACR)was 2.47 (normal <3 mg/mmol, her urinary creatinine was 2,890mg/24 hours (normal 955 to 2,936 milligrams (mg) per 24 hours), which indicates normal renal function, her urinary micro albumin 1167.4 and serum albumin was only 1.2gm/dl(normal 3.5 - 5.2 g/dl**)**; she was managed by multidisciplinary approach such as cardiologist, nephrologist and respiratory medicine specialist. Albumin was given by intravenous infusion daily for three days. Then subsequently her dressing was soaked with fluid on 3rd POD. But later on it became dry on next day. Gradually her Serum albumin was raised and breathlessness and hypertension was subsided. Her baby was in neonatal Intensive care unit (NICU) for 6 days the discharged. Both mother and baby discharged at 7th POD.

Discussion

A small amount of peritoneal fluid is often found during caesarean section of preeclampsia patient, but massive ascites is a rare finding. Though there is no clear definition of massive ascites in text, fluid collection of more then 2 liters is considered as massive ascites[2,3]. Preeclampsia is a multisystem disorder. Endothelial damage, release of vasoconstrictive agents, hyper permeability of the capillaries and microangiopathic hemolysis are the basic pathology of the disease. As a result of these pathology different complication such as hypertension, HELLP syndrome, ascites, plural effusion etc. develop. The recent concept of pathophysiology of preeclampsia involves widespread endothelial cell dysfunction.[4] It causes capillary leakage, which causes development of oedema, proteinuria and ascites. This subsequently causes low oncotic pressure and ascites. Atrial natriuretic peptide (ANP) secretion is more in patient with preeclampsia than normal pregnancy, it is released in maternal circulation in response to vasoconstriction and it causes increased volume load to cardiac atria. As compared to normal pregnancy renin, aldosterone and ANP all act as if to promote the natriuresis but due to unknown reasons natriuresis is impaired[5].

The reported case had hypoproteinemia, she had no history of known hepatic or cardiovascular or kidney disease. Her ECG, Echocardiogram, S bilirubin, SGPT, coagulation profile was normal. Massive ascites results in respiratory distress. In 10% of HELLP syndrome is associated with massive ascites[3]. Massive ascites usually develops in antenatal period but it in some cases also can develop in post-partum period [6]. The differential diagnoses include liver cirrhosis, tuberculosis, nephritic syndrome, Meigs syndrome, and malignant conditions of the ovary and other abdominal organs. In this patient after normalization of blood pressure gradually proteinuria subsided and then ascites and oedema gradually resolved.

Pradhan P et al., have reported massive ascites in antenatal period and development of pleural effusion in postpartum period [7]. There is role of steroid (Prednisolone) in severe ascites [8]. In a series of cases, reported requirement of early

termination of pregnancy in cases of women with severe ascites in pre-eclampsia[4].

Conclusion

Massive ascites in preeclampsia is a rare event. Management of theses patient needs multi-disciplinary involvement. These patient need more frequent maternal and fetal surveillance to avoid complications. Future studies may include hospitals where predominantly preeclampsia patients get admission to determine the prevalence and way of management of cases with massive ascites.

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