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Case Report

Massive ascites and bilateral pleural effusion causing respiratory embarrassment in a postnatal case of severe preeclampsia

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Case report

A 26-year-old female patient, second gravida, unbooked case at 32 weeks 3 days period of gestation with severe preeclampsia was admitted with blood pressure (BP) of 166/110 mmHg, urinary albumin of 2+ and generalized edema. She reported her weight gain to be 12 kg above her prepregnancy weight. Her daily urine output at 1–1.5 L was adequate throughout her pregnancy. 3 yrs back she had one previous full term normal delivery with no history of high blood pressure during pregnancy & postpartum. On admission, her hematological and biochemical parameters were within normal limits except serum uric acid levels of 7.2 mg%. Ophthalmoscopy demonstrated grade 1 hypertensive changes. Ultrasonography (USG) showed a single live fetus of 29 weeks gestation along with amniotic fluid index of 6.5 cm. She was recommended bed rest. Maternal and fetal surveillance was done in the form of estimation of BP 6-hourly, urine proteins and output daily, fetal biophysical score (BPS) and Doppler on alternate days and measurement of weight, fundal height and abdominal girth, hematologic, biochemical and urinary investigations twice weekly. She was started on tab labetalol 100 mg thrice daily. She was administered two doses of betamethasone 12 mg at an interval of 24 h to achieve fetal lung maturity. With this her blood pressure was controlled

Introduction

Hypertensive disorders complicate 5–10 percent of all pregnancies, and together they form one member of deadly triad, along with hemorrhage and infection, that contribute greatly to maternal morbidity and mortality rates.¹ Preeclampsia is primarily a vascular endothelial disorder triggered by the human placenta. The differentiation between nonsevere and severe preeclampsia can be misleading because what might be apparently mild disease may progress rapidly to severe disease. Some amount of peritoneal fluid is frequently seen during cesarean section of normal and hypertensive gravid women, but massive ascites, such as the presence of 2 L or more of ascitic fluid, is a rare finding in preeclampsia patients. We are presenting a case of massive ascites with bilateral

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and blood pressure remained between 130/80–140/96 mm of Hg. She went into spontaneous labor at 34 weeks 3 days period of gestation and delivered a male fetus weighing 1.6 kg with a good Apgar score.

Postdelivery tab labetolol was continued and on first postnatal day she developed ascites which gradually increased and on second postnatal day she had difficulty in breathing with SPO₂ of 82–88% on room air. X ray chest showed bilateral pleural effusion (Fig. 1) and USG showed massive ascites and bilateral pleural effusion. She was started on free flow oxygen by mask, antibiotics and diuretics. Ascitic tapping was done and 2.5 L of straw colored fluid was drained. With this treatment, her SPO₂ improved and breathing difficulty decreased. Ascitic fluid cytology was suggestive of transudate, no organisms were grown on culture and no acid fast bacilli were seen in Z–N stain. Her biochemical and hematological parameters were within limits including total protein, albumin and globulin levels.

She responded well and her ascites and pleural effusion resolved within a week and she was discharged on 12th postnatal day.

Discussion

The etiology of preeclampsia remains an obstetric enigma. Several etiologies have been proposed but most have not withstood the test of time. Some of these suggested causes include abnormal trophoblast invasion of uterine vessels, immunologic intolerance between fetoplacental and maternal tissues, maladaptation to cardiovascular changes, inflammatory changes of pregnancy, and genetic abnormalities.² Recently, there is substantial evidence suggesting that the pathophysiologic abnormalities of preeclampsia are caused by abnormal angiogenesis, particularly an imbalance in soluble fms-like tyrosine kinase 1 : prostaglandin growth factor (sFlt-1 : PLGF ratio).³

The cause of ascites in preeclampsia is obscure but the probable mechanisms include renal retention of sodium and fluid resulting in expansion of interstitial compartment. Preeclampsia involves widespread endothelial cell dysfunction which may be responsible for generalized capillary leak

and hence third space fluid collection in the form of massive ascites.⁴ The increased capillary leak may be due to a combination of increased postcapillary resistance and endothelial disruption resulting in disordered protein movement and reduced intravascular oncotic pressure.⁵ The low colloid osmotic pressure results in effusions such as ascites.

According to Cong the incidence of ascites was found to be 21.6/1000 in severe preeclampsia and it was concluded that ascites in severe preeclampsia indicated termination of pregnancy as it cannot be cured by medical treatment.⁶ Woods *et al.* have reported 10% incidence of large-volume ascites with hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome. Neither the obstetric text books nor the case reports available give a clear definition of massive ascites, although they describe massive ascites in subjective and semiojective terms of 2 or more liters.⁷

Vaijyanath and associates concluded that massive ascites developing in preeclampsia is an unusual complication and massive ascites leading to maternal respiratory compromise calls for active termination of pregnancy within 24–48 h. The paucity of reports in the literature may be due to under-reporting and also to the difficulty of recognizing this condition clinically. A high index of suspicion, careful clinical and ultrasonographic examination to detect ascites will help in elucidating the true incidence of this complication. The incorporation into clinical practice of evaluating the amount of ascites in preeclampsia might alert the obstetrician toward more intensive and more frequent maternal and fetal surveillance to avoid maternal and fetal hypoxia.⁸

All the studies found in literature regarding presence of ascites in severe preeclampsia were of antenatal period. Postnatal development of massive ascites with bilateral pleural effusion causing respiratory difficulty in severe preeclampsia is very rare. In our case hypoproteinemia was absent and ascitic fluid culture did not grow any organism. We conclude that there is a possibility of development of ascites and pleural effusion in cases of preeclampsia during the postpartum period also.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY. Pregnancy hypertension. In: *Williams Obstetrics: Obstetrical Complications*. 23rd ed. 2010:706–708.
2. Sibai B, Dekker G, Kupferminc M. Preeclampsia. *Lancet*. 2005;365:785–799.
3. Levine RJ, Thadhani R, Qian C, *et al.* Urinary placental growth factor and risk of preeclampsia. *JAMA*. 2005;293:77–85.
4. Brown MA, Zammit VC, Lowe SA. Capillary permeability and extracellular fluid volumes in pregnancy-induced hypertension. *Clin Sci*. 1989;77:599–604.
5. Sullivan JM. *Hypertension and Pregnancy*. Chicago: Year Book; 1986. 73–81.

Fig. 1 – Bilateral pleural effusion.

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6. Cong KJ, Wang TT. Complication of ascites in pregnancy induced hypertension. *Chung-Hua-Fu-Chan-Ko-Tsa-Chin*. 1994;29:7–9.
 7. Woods BJ, Blake PG, Perry KG, et al. Ascites: a portent of cardiopulmonary complications in the preeclamptic patient with the syndrome of hemolysis, elevated liver enzymes and low platelets. *Obstet Gynecol*. 1992;80:87–91.
 8. Vaijyanath AM, Nayar B, Malhotra N, Deka D. Massive ascitis in severe preeclampsia, a rare complication. *J Obstet Gynaecol Res*. 2002;28(4):199–202.