Massive Ascites Complicating Severe Preeclampsia : A Case Report

Vijayalakshmi S

Received: 13 December 2016 Accepted: 31 December 2016 Published: 15 January 2017

Abstract

Pre-eclampsia earlier known as Toxemia in pregnancy /pregnancy induced hypertension is primarily a endothelial disorder which is thought to be triggered by the placenta. 1 It affects about 4-8

Index terms—

1 Massive Ascites Complicating Severe

Preeclampsia : A Case Report Bharathi K.R ? , Vijayalakshmi S ? & Mahendra G ? I. Introduction re-eclampsia earlier known as Toxemia in pregnancy/pregnancy induced hypertension is primarily a endothelial disorder which is thought to be triggered by the placenta. 1 It affects about 4-8% of all pregnancies. Hypertensive disorders in pregnancy remains one of major component of the lethal triad in causing maternal mortality even in the era of modern obstetrics with the advent of newer management options and intensive care facilities being available. 2 The complications associated with pre-eclampsia are estimated to be around 20%. Complications like intracranial hemorrhage, cerebral edema, pulmonary edema, renal failure, global haemolytic tendency, HELLP syndrome and maternal ascites are all found in preeclampsia. The finding of presence of fluid in various body cavities is due to increased endothelial permeability and microvasular damage which is not unusual. 3,4 Some amount of fluid in peritoneal cavity is seen frequently seen in pre-eclamptic women but presence of massive ascites is a rare complication and needs cautious management.

2 II. Case Report

A 19year old primigravida with h/o 9 months of amenorrhea with good perception of fetal movements and regular antenatal checkups was referred from a private hospital to our antenatal OPD at AIMS, BG Nagara in view of high blood pressure readings. She gave h/o pedal edema and high blood pressure since 2 months for which she was on treatment with tab. Nicardipine 5mg thrice daily which has been increased to 10 mg thrice daily since 3 days. No h/o imminent symptoms. On examination, there was no pallor, icterus. Bilateral pitting type of pedal edema present. Pulse rate 94/min, BP was 160/120mmhg, respiratory rate 22/min, regular.

Cardiovascular & respiratory system examination were within normal limits. Abdominal examination showed abdominal wall edema, gross maternal ascites with fluid thrill, 28weeks size uterus, FHS couldnot be localised clinically but fetal cardiac activity and rate noted with the help of bedside scan. Vulval edema present. Vaginal examination revealed uneffaced and closed cervical os and diagnosis of 36weeks gestation with severe preeclampsia with severe IUGR with oligohydramnios with gross maternal ascites was made. Urine albumin was 4+. Serum uric acid 6mg/dl. Complete blood count, blood urea, serum creatinine, serum bilirubin, ALT, AST, LDH were within normal limits. Fundoscopy revealed grade I hypertensive retinopathy changes. She was given 10mg Nicardipine stat dose and 12mg betamethasone injection was given. Decision for caesarean section was taken in view of severe IUGR and oligohydramnios. Under spinal anesthesia, caesarean section was done and delivered a live preterm male baby weighing 1.5kg. 2litres of ascitic fluid drained intraoperatively, BP 140/100mmhg. Immediate postoperative period was uneventful. She was started on clear liquids orally after 12 hours, BP was 130/100mmhg, pulse rate 88/min. On 2 nd postoperative day patient developed abdominal distension with breathing difficulty. On examination pulse was 116/min, BP 130/100mmhg, with RR 24/min. Abdomen soft, nontender, ascites +. Ultrasonography revealed gross ascites and bilateral mild pleural effusion. Echocardiogram was normal. Serum albumin was 2.6g/dl, all other investigations were within normal limits. Patient was shifted

to intensive care unit. Ascitic fluid tapping done. Strict input/output charting maintained. Tab Nicardipine 10mg thrice daily continued. On 3 rd and 4 th post op day she continued to have tachycardia and abdominal distension. High protein diet started. From 5 th postop day the tachycardia started settling down and also the blood pressure with BP 140/90mmhg, abdominal distension also started reducing which was monitored by measuring abdominal girth. Complete hemogram, Blood urea, creatinine, AST, ALT, LDH, serum bilirubin were within normal limits. By 10 th day distension reduced completely. Review ultrasound revealed no pleural effusion but mild ascetic fluid. Nicardipine was stopped on 15 th day and BP monitoring was done which was within normal limits. She was discharged on 20 th day in stable condition with instructions for weekly follow up. Review follow up was uneventful with normal BP readings.

3 III. Discussion

Pre-eclampsia is a multisystem disorder characterised by the involvement of global microvascular system associated with endothelial dysfunction. Out of all the complications known to occur in pre-eclampsia, ascites is a rare complication. Fluid in the peritoneal cavity is a frequent finding in preeclamptic women but massive ascites is very rare and only few cases have been reported in the literature. Incidence of ascites in pre-eclampsia is 1 in 1000 patients. Fluid in multiple body cavities in pre-eclampsia is thought to be due to increased capillary permeability (capillary leak syndrome), portal hypertension, hypoproteinemia due to proteinuria and altered albumin/ globulin ratio. 5 Cong and Wang conducted a study in 23 patients with pre-eclampsia and ascites where they found albumin/globulin ratio <1.5 in all patients. 6 The finding of altered albumin/globulin ratio was thought to reduce the intravascular oncotic pressure leading to transudation of serum to various body cavities predominantly peritoneal cavity. 7 A case of massive ascites in pre-eclampsia with bilateral hydrothorax reported by Ashmore et al reveals the use of intraabdominal drain to monitor the ascetic fluid production. 8 A case series conducted by Woo et al in pregnancy with severe-eclampsia and ascites reported rapid deterioration in maternal condition with rise in the blood pressure associated with increasing proteinuria requiring early termination of pregnancy. 9

4 IV. Conclusion

Ascites in preeclampsia is a rare complication which need cautious management to avoid complications of massive ascites and hydrothorax like respiratory distress and cardiac compromise. Patients need to be monitored well in intensive care set ups and decision regarding termination of pregnancy should be taken based on the clinical scenario rather than making a decision for early termination of pregnancy. [1] [2]

[1] © 2017 Global Journals Inc. (US)Year 2017

[2] Massive Ascites Complicating Severe Preeclampsia : A Case Report

[Woods et al. ()] 'Ascites : a portent of cardiopulmonary complications in the preeclamptic patient with the syndrome of hemolysis, elevated liver enzymes and low platelets'. J B Woods , P G Blake , K G Perry . *Obstet Gynecol* 1992. 80 p. .

[Calvin et al. ()] 'Characterization of ascites present at Caesarean Section'. S Calvin , M Silva , L Weinstein . *Amer J Perinatal* 1991. 8 p. .

[Cong and Wang ()] 'Complications of ascites in pregnancy induced hypertension'. K J Cong , T T Wang . *Zhonghua Fu Chan Ke Za Zhi* 1994. 29 (1) p. .

[Ashmore J and Attapattu ()] 'Massive ascites and bilateral hydrothorax complicating severe preeclampsia'. Ashmore J , J A Attapattu . *Ceylon Med J* 1998. 43 (4) p. .

[Foreman ()] 'Massive ascites as a complication of severe pre-eclampsia -a case report'. C S Foreman . *J Reprod Med* 1989. 34 p. .

[Ko et al. ()] 'Massive ascites complicating pre-eclampsia'. Lee Ko , M Huang , L W Chang , JZ . *Taiwanese J Obstet Gynecol* 2005. 44 (3) p. .

[Vaijyanath et al. ()] 'Massive ascites in severe pre-eclampsia : a rare complication'. A M Vaijyanath , B Nayar , N Malhotra . *J Obstet Gynecol Res* 2002. 28 p. .

[Jimmy and Damar ()] 'The unique presentation of massive ascites complicating severe preeclampsia and HELLP syndrome'. P W Jimmy , P Damar . *Indonesia J Obstet and Gynecol* 2011. 35 (2) p. .

[Woo et al. ()] 'Ultrasonic detection of maternal ascites in patients with severe preeclampsia'. J S Woo , S T Liang , V T Wong . *Aust NZ J Obstet Gynecol* 1982. 22 p. .