

Massive ascites in Severe Pre- eclampsia : A rare complication.

P Pradhan, K Sherpa, A Joshi and S Pathak

Department of Obstetrics and Gynaecology, Nepal Medical College Teaching Hospital, Kathmandu Nepal

Corresponding Author: Prof. Pradhan Pramila FRCOG (London), Department of Obstetrics and Gynaecology, Nepal Medical College Teaching Hospital, Jorpati, Kathmandu, Nepal, email: drpramilapradhan@gmail.com

ABSTRACT

We report a rare case of massive maternal ascites complicating severe pre- eclampsia toxemia (PET) seen in April 2013. This complication developed in association with the rise of blood pressure of 160/110 mmHg or more, worsening of proteinuria and hyperuricaemia. The onset of massive ascites caused respiratory compromise to the patient, thus necessitating immediate termination of pregnancy.

Keywords: Ascites, pre-eclampsia, pregnancy induced hypertension.

Pre-eclampsia toxemia is primarily a vascular endothelial disorder triggered by the human placenta.¹ Pre-eclampsia complicates approximately 4-6% of all pregnancy affecting over 8 million women worldwide and is one of the important causes of maternal and perinatal morbidity and mortality. Surveillance, appropriate therapeutic strategies to minimize the complication of pre-eclampsia and delivery of the fetus remain the mainstay of management of the disease.² Some amount of peritoneal fluid is frequently seen during caesarean section of normal and hypertensive gravid women but massive ascites such as the presence of 3 litres or more of massive fluid is a rare finding in PET patients.³

CASE REPORT

A nineteen year old primigravida at 33 weeks gestation attended antenatal clinic at Nepal Medical College Teaching Hospital (NMCTH) on 28/04/2013 on examination. She was pale with bilateral pedal edema. The blood pressure measured 170/140 mmHg on both limbs on lying position. The urine protein was 3+ with dipstick method. She was diagnosed severe pre-eclampsia (PET) at 33 weeks gestation and admitted for management of severe PET and monitoring of fetal and maternal well being. Upon admission her daily urinary output of 1-1.5 litre was adequate. Serum biochemical parameters revealed Haemoglobin 9.3 gm%, severe hypoproteinaemia, total protein 4.1 gm%, albumin/ globulin ratio (<1.5) and hyperuricaemia (serum uric acid 7gm/dl), serum LDH 1140 U/L with other parameters being normal. Ophthalmoscopy demonstrated grade 1 hypertensive changes. Ultrasonography (USG) showed a single live fetus of 32 weeks gestation with small amount of maternal ascites. She was recommended bed rest and started on Nifedipine 30mg / day, Methylodopa 1.5 gm/day in divided doses. Maternal and fetal

surveillance was done in the form of estimation of BP 4 hrly, urine protein and output daily, fetal biophysical score (BPS) and Doppler on alternate days and measurement of weight, fundal height and abdominal girth, haematological, biochemical and urinary investigations weekly. She was started on high protein diet (1.5/kg) she was administered 2 doses of Betamethasone 12 mg at an interval of 12 hr to achieve fetal lung maturity. She was given prophylactic Magnesium Sulphate by Pritchard method. Patient felt comfortable with lower BP over few days with occasional increase to 160/110 mmHg, but proteinuria increased to 3 grams/ day and abdominal girth gradually increasing. Her weight gain after admission was 2 kg and daily urine output was 1 litre. On the ninth day of admission there was increase in abdominal distension with maternal respiratory distress and orthopnoea. Her Jugular Venous Pressure (JVP) was normal. There were no lung crepitations and no dullness on percussion over the chest wall. An urgent USG showed a live fetus of 34 weeks with no distress and ascites has slightly increased compared to previous level. Arterial blood gas (ABG) analysis showed respiratory acidosis with hypoxia. At this time her haematocrit was 0.46, serum creatinine was 1.2, Uric acid 7.5mg/dl and platelets count and LFT were normal. It was decided to terminate the pregnancy by an emergency lower segment caesarean section (LSCS) in view of maternal hypoxia and potential fetal jeopardy. On opening the abdomen approximately 3 litre of straw colored fluid was drained from the peritoneal cavity. At LSCS the fetus was in breech presentation and an alive male fetus was delivered by breech extraction. The APGAR score was satisfactory and the fetal weighed 1500 grams. There were no other abnormalities in uterus or abdominal cavity and organs. Patient was cared in intensive care unit for low PO2 saturation .She

developed bilateral pleural effusion of mild grade with bilateral lower zone pneumonia. The lungs problem was taken care by physicians and patient recovered well. The ascites resolved spontaneously over next 8-10 days. Antihypertensive drugs were slowly tapered and discharged home on Nefidipine retard 20 mg/day and metoprolol 25 mg/day, to be seen in OPD in 4 weeks time. However the baby expired on 7th day of admission at neonatal intensive care unit inspite of intensive treatment.

DISCUSSION

As early as 1949 Golden reported a case of ascites in a women seven months of pregnancy complicated

by eclampsia.⁴ He stated that the cause of ascites in pregnancy was either low concentration of protein with an altered albumin/globulin ratio, portal block or haemoconcentration in portal circulation. After Golden there have been only sporadic case reports of massive ascites complicating pre eclampsia.⁵⁻⁷

The cause of ascites in PET is obscure but the probable mechanism include renal retention of Sodium and fluid resulting in expansion of interstitial compartment.⁸ The most recent concept of pathophysiology of PET involves widespread endothelial cell dysfunction.⁹ Impairment of endothelial barrier function is suggested by generalized capillary leak which is responsible for odema, proteinuria and decreased colloid osmotic pressure.¹⁰ The low colloid osmotic pressure results in effusion such as ascites.^{10,11}

In a study comprising 23 pregnancy induced hypertension (PIH) patient with ascites, the incidence of ascites was found to be 21.6/1000 in severe PIH.¹² The clinical manifestations showed early onset of PIH at 29.4+4-8 weeks and ascites occurred at 32.4+2.4 weeks. It was concluded that ascites in severe PIH indicated termination of pregnancy as it cannot be cured by medical treatment. In other published case reports the usual gestational age of development of ascites was 27-31 weeks.^{5,6} In our study the incidence of ascites was 0.5/1000, it developed around 32 weeks of pregnancy and required premature termination of pregnancy. Our cases showed hypoproteinaemia and A/G ratio <1.5. The trigger for sudden and massive ascites may have been hypoproteinaemia. The patient needed premature emergency LSCS due to severe maternal respiratory compromise and potential or real fetal jeopardy. This patient had massive or large volume ascites as defined by amount equal to more than 2 litre of peritoneal fluid. Neither the text books nor the case reports available give a clear definition of massive ascite although they described massive

ascites in subjective terms of two or more litres.¹³ Therefore this definition of massive ascites is arbitrary but at the same time it is based on the clinical criteria of ascites causing significant respiratory distress to the mother and the ABG showing respiratory acidosis and maternal hypoxia (i.e. low oxygen pressure (PO₂)). Our patient did not suffer from post partum haemorrhage or poor wound healing but needed ICU admission for respiratory infection/ pneumonia of lower zones of both lungs. We did not keep any intraperitoneal drain in our patient and ascites resolved spontaneously. She received two units of blood transfusion post operatively. This patient did not have any evidence of haemolysis, thrombocytopenia and elevated liver enzymes. Wood *et al*¹³ have reported 10% incidence of large volume ascites with haemolysis. Elevated liver enzyme and low platelets (HELLP Syndrome).

We conclude that massive ascites developing in pre-eclampsia is an unusual complication and massive ascites leading to maternal respiratory compromise calls for active termination of pregnancy within 24-48 hrs. The paucity of reports in the literature may be due to under reporting and also to difficulty of recognizing this condition clinically as seen in our case. 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 pre-eclampsia might alert the obstetrician towards more intensive and more frequent maternal and fetal surveillance to avoid maternal and fetal hypoxia.

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