Preeclampsia complicated by antenatal and postnatal ascitis

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Abstract—Hypertensive disorders of pregnancy are an important cause of severe acute feto-maternal morbidity, lasting disability and mortality. In Africa, about one tenth of all maternal deaths are associated with hypertensive disorders of pregnancy. The majority of deaths attributable to preeclampsia can be circumvented by instituting appropriate and effective care to women presenting with complications one of which is ascitis. Ascitis can lead to dehydration, respiratory failure, heart failure and thromboembolism

Index Terms— Preeclampsia, ascites, hypoalbuminaemia, heart failure .

I. INTRODUCTION

Preeclampsia poses management challenges for the obstetrician and it is a significant contributor to perinatal and maternal morbidity and mortality. The etiology of preeclampsia has lingered unresolved. Ascitis complicating preeclampsia is a rare complication and a manifestation of the multisystem organ exaggeration of preeclampsia. The occurrence of massive ascites causes respiratory distress and predisposes the patient to heart failure. Astute antenatal assessment, knowledge of its occurrence and course, is essential in management of this condition in the post vaginal delivery/Caesarean section patient so as to prevent unintended (re)laparotomy with its associated anaesthetic and surgical complications.

II. CASE REPORT

A 37 year old para 1⁺² lady, booked index pregnancy at 28 weeks gestation. Booking blood pressure was 110/60mmHg and her last confinement was 8 years prior to presentation. The antenatal period was uneventful until 37 weeks when the blood pressure was noted to be 167/98mmHg at routine antenatal visit. No family history of hypertension nor features of imminent eclampsia. Examination revealed, bilateral pitting edema up to the knee, clear lung fields, symphysio-fundal height was 34cm, the fetal heart rate was heard and regular. The Bishop score was 3, clean catch urine dipstick examination revealed 2+ proteinuria. Ultrasonogram revealed a foetus that was small for gestational age (EFW was 2.2kg), liquor volume was adequate and placenta was anteriorly sited. A moderate amount of ascitic fluid was seen intra-peritoneal. The patient was admitted and commenced on seizure prophylaxis with Mgso₄ using the Zuspan regimen. She was commenced on aldomet and nifedipine and she made adequate amount of urine over the next two hours. The laboratory results are shown in table 1 She had Caesarean section for severe preeclampsia with unfavourable cervix. Outcome was a live female infant that weighed 2.3kg with Apgar scores, 8 and 9 in 5 and 10 minutes respectively.

Approximately 0.6L of straw coloured fluid was drained from the peritoneal cavity. Estimated blood loss at surgery was 500ml. Exploration revealed no other intra-abdominal abnormalities. Magnesium sulphate was continued for 24 hours post-surgery and she had prophylactic antibiotics.

Eight (8) hours post section, the urine output was noticed to have reduced, averaging 21mls/hr with accompanying abdominal distension. Abdominal girth was 103cm and abdominal paracentesis revealed slightly bloody peritoneal collection. The pulse rate was 94 beats/minute and the blood pressure ranged from 146/85 to 168/102mmHg. Fluid intake was maintained at 3litres/24hours, hourly monitoring of urine output and antihypertensive (aldomet and nifedipine) was commenced. The abdominal girth increased to 106 cm on the 1st day post section and her hemogram was 8.2g/dl on the 2nd day post section. Urine output averaged 52ml/hour.

Table 1: LABORATORY RESULTS

	Day of C/S	1 st DPO	2 nd DPO	3 rd DPO	4 th DPO
Hemoglobin (g/dl)	9	—	8.3	—	10.3
Haematocrit (%)	27	—	25	—	31
WBC(mm ³)x100	153	—	168	—	169
Platelet count x 10 ³ (/L)	198	—	211	—	218
Na (mmol/L)	128	130	129	131	135
K (mmol/L)	3.8	3.0	3.4	3.2	3.3
Cl (mmol/L)	97	99	95	100	101
Creatinine(mg/dl)	0.9	—	0.8	0.7	0.6
Urea (mg/dl)	48	—	39	38	32
ALT(U/l)	28	—	22	—	—
AST(U/l)	18	—	15	—	—
Alk phosph. (U/l)	110	—	121	—	—
Tot. bilirubin(mg/dl)	2.7	—	2.2	—	—
Dir. bilirubin(mg/dl)	1.3	—	1.1	—	—
Total protein(g/l)	4.6	—	4.7	—	4.9
Albumin (g/L)	2.5	—	2.7	—	2.8

DPO Post-operative day, ALT: alanine transaminase, AST: Aspartase transaminase, Alk phosph: alkaline phosphatase, Dir: direct

She developed heart failure on the 3rd day post operation and she was co-managed with the physicians with blood transfusion (3 units of fresh whole blood), oxygen administration, spironolactone and frusemide. The abdominal girth which peaked on the 3rd day post-surgery at 109cm progressively regressed from the fifth day post-

surgery. The haemogram was 10.9g/dl on the 6th day post section. She was discharged on the 8th day post-surgery, the abdominal girth was 94cm, and the antihypertensives were subsequently tailed off and stopped 24 days postpartum. At the postnatal visit the blood pressure was normal at 100/60mmHg.

IV. DISCUSSION

Ascites has been reported in pregnancies¹ and at cesarean section delivery in pregnancies complicated by preeclampsia²⁻⁴. The pathophysiology of peritoneal fluid collection in preeclampsia has remained uncertain². However, the most contemporary concept implicates the release of vasoconstrictive mediators, and widespread endothelial cell injury. The endothelial damage at the capillary level, affects the capillary microcirculation resulting in unbridled permeability at the capillary end and microangiopathy.

Several researchers have reported abnormalities in serum proteins in preeclampsia complicated by ascites³⁻⁶. Albumin does not diffuse liberally through intact vascular endothelium. Hence, it is the chief protein that ensures the critical colloid osmotic or oncotic pressure that regulates the route, water and diffusible solutes pass through the capillaries.

Impairment of endothelial barrier function manifests as generalized capillary leak⁴ and albumin which comprises only about 50–60% of the total protein content of plasma, but is responsible for about 80% of intravascular colloid oncotic pressure, diffuses freely through the dysfunctional vascular endothelium⁵ and results in reduction of serum albumin. The import of hypoalbuminemia is the transfer of fluid from the intravascular compartment to the interstitial space, resulting in intravascular volume depletion and a consequent low colloid osmotic pressure. This invariably results in effusion causing ascites⁸. This may be the reason for the accumulation of fluid in the peritoneal cavity and the subsequent abdominal distension. After abdominal surgery there is always blood in the peritoneal cavity, this mixed with the peritoneal collection may give the erroneous belief of continuous intra-abdominal bleeding.

Albumin in this case ranged from 54.3% to 57.7% of the total protein. However, serum albumin (as shown in the table above) is below the normal range of 3.5 to 5g/dl but with the delivery of the fetus and placenta, there was a steady rise in the serum levels of albumin. At surgery no other cause of the ascites was ascertained by inspection and exploration of other intra-abdominal organs.

Due to the nature of the disease which affects all the systems in the body and in keeping with the recommendation by Calvin et al³ the renal, hematological and liver systems were all assessed for any abnormalities. The elevated levels of the transaminases and bilirubin is due to the effect of hypoxia on the liver in preeclamptic pregnancy which causes necrosis and degeneration of hepatocytes⁹. The total and direct bilirubin, platelet count and the electrolytes, urea and creatinine were all within normal limits.

With the delivery of the placenta which is the trigger for the development of preeclampsia, the endothelial cell dysfunction is gradually rectified, this coupled with the rising serum albumin, use of furosemide and spironolactone and control of the blood pressure, triggered the ascites to

resolve. No attempt at draining the peritoneal fluid in the post-operative period was made because of the risk of introducing infection. There was no manifest difficulty in breathing implying that the shift of fluid did not affect the pulmonary system. Transfusion of albumin to correct the hypoalbuminemia has been described and was effected in the management of ascites in this situation with favourable result^{7,8}. However, Ascites has been reported to persist for up to 12 days post-delivery of the fetus and the placenta¹.

The patient developed heart failure on the 3rd day post-delivery, Calvin et al³ and Woods et al¹⁰ reported that ascites, especially in large volumes, would cause respiratory compromise and increased incidence of heart failure. The patient susceptible to developing heart failure does so in the first 24 hours post partum¹⁰. She was managed with blood transfusion, diuretics (frusemide and spironolactone) and controlled fluid administration. The antihypertensives were tailed off to avoid rebound hypertension and the patient was normotensive subsequently.

IV. CONCLUSION

The actual etiology of ascites in preeclampsia is still murky, nevertheless, with the little understanding of the pathophysiology, significant management success have been achieved and reported. Ascites is an unusual and uncommon complication of preeclampsia, a detailed history, astute clinical examination and the use of ultrasonography is needed for prompt diagnosis so that appropriate multidisciplinary management can be instituted. The presence of fluid in the peritoneal cavity on ultrasound in a patient with preeclampsia is a telltale sign. Furthermore, following Caesarean section there will be some blood in the peritoneal cavity, this mixed with ascitic fluid in the face of abdominal distension may give the erroneous impression of continuous intraperitoneal bleeding. This may lead to unnecessary laparotomy with attendant increased morbidity and mortality. Therefore practitioners should have this in mind.

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