Pleural Effusion - A Rare Complication of Severe Pre-Eclampsia: A Case Report

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Pleural effusion is a very rare complication of severe pre-eclampsia, may be reason for low plasma colloid osmotic pressure (PCOP) and deterioration of renal filtration function. It is defined as accumulation of serous fluid within the pleural space is termed as pleural effusion. A twenty seven years old primigravida admitted in BIRDEM at her 28 weeks pregnancy with sign symptoms of severe pre-eclampsia. We treated her with multiple antihypertensive drugs and prophylactic anticonvulsant therapy (MgSO4).After counseling of her condition, cesarean section was done. On 2nd POD patient developed respiratory distress. So X-ray chest P/A view was done, which showed massive pleural effusion on right side.

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Key words: Pleural effusion, pre-eclampsia

Introduction

Pre-eclampsia is a multi-system disorder of unknown etiology characterized by development of hypertension to the extent of 140/90 mmHg or more with proteinuria (>300 mg/24h) of the twenty weeks pregnancy in a previously normotensive and non protienuric women.[1] There are several theories to explain its cause.[2] The common factor seems to be an anomaly of the stages of invasion of the cytotrophoblast on the wall of the uterine spiral arterioles causing turbulence, hypoperfusion and ischaemia of the sinusoidal spaces. The release of various substances of placental origin to maternal circulation causes arteriolar and capillary endothelial damage, basement membrane fragmentation and increased permeability that favour the phenomenon of capillary leakage of water, solutes and macromolecules(albumin) to interstices.[2,3] Thus, the balance of forces described by Starling in the microcirculation is altered from reduction in plasma colloid osmotic pressure(PCOP) of proteins and the relative increase in capillary hydrostatic pressure, which justifies the presence of local and then generalized edema.[4-8] In Preeclamtic patients, capillary leakage may be associated abnormal collection of fluid in pleura or peritoneum. The most important factor for its development seems to be reduced PCOP of proteins.[8] However, it has been documented that increased in mean arterial pressure,[5] extensive structural damage of the microvasculature in patients complicated by HELLP syndrome.[9] Intracapillary coagulopathy,[10] platelecount<100000/mm,[3,11] proteinuria >5g/24h,[11] serum creatinine>120mOsm/L,[11] portal hypertension,[9] presence of acute cardiogenic pulmonary edema,[9,10] uncontrolled systolic arterial pressure(SAP) and adult respiratory insufficiency syndrome(ARIS)[9,10] may favour their formation. Pre-Eclampsia are two types -

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Mild preeclamsia is characterised by sustained rise of blood pressure more than 140/90 mmHg but less than 160/110 mmHg without significant proteinuria. Severe Preeclampsia is characterized by systolicBP≥160mmHg and DiastolicBP≥110mmHg Proteinuriai >5gm/24hours. Riskfactorsare: Family history, primigravida, obesity, preexisting vascular disease, Placental abnormalities such as twin pregnancy, molar pregnancy.[12]

Pleural effusion is defined as accumulation of serous fluid within the pleural space is termed as pleural effusion. Common causes are - pneumonia, tuberculosis, pulmonary infarction, malignant disease, cardiac failure. Uncommon is hypoproteinemia.[13]

Case Report

A 27 years old primigravida admitted to BIRDEM hospital at her 28 weeks pregnancy with headache and blurring of vision and generalized body swelling for 6 days. She was a regularly menstruating woman. Her pregnancy was confirmed by early USG of lower abdomen. Her pregnancy was uneventful until the last 6 days before her admission. When she suddenly developed headache, blurring of vision and generalized swelling, she checked her BP at home which was raised. So she took Tab. Methyldopa 1 tab stat but BP was still raised. So, she got admitted to a private clinic with BP-160/120 mmHg and bed side urine albumin was (+++). She was diagnosed as a case of severe pre-eclampsia and advised her bed rest and gave her multiple antihypertensive drugs and anticonvulsant drug (Inj.MgSO4) and referred to BIRDEM.

After admission in BIRDEM she was treated conservatively and some investigations were done. USG report showed 26 weeks pregnancy with oligohydramnios (AFI-6cm) with moderate fluid collection in peritoneal cavity. Her S.total Protein-50.8 gm/L (Normal: 65-82 g/L), S. albumin-24.3 g/L (Normal: 35-55 gm/L), A:G- 0.91 :1 (Normal >1). Fundoscopy-Normal, FDP-80mg/L (Normal- <40 mg/L) APTT-38.4 (Normal: 27-35) PT-11.3 sec (Control-12) . Patient and her attendants were thoroughly counseled about her condition and advised the patient to terminate the pregnancy by caesarean section. Then caesarean section was done. Per operatively patient got 1 unit of fresh blood and 1 unit of fresh frozen plasma. Post operatively patient was managed with 1500ml I/V fluid, injectable antibiotics and analgesics, antihypertensives and Inj. frusemide 6 hourly. Inj. MgSO4 to prevent post partam eclampsia and maintain intake output chart. On 2nd post operative day patient complaints of breathlessness at that time breath sound was diminished. Nebulisation was done and also give Inj. frusemide. Then, chest X-ray P/A view was done, which reveals massive pleural effusion (fig 1). So we consulted with

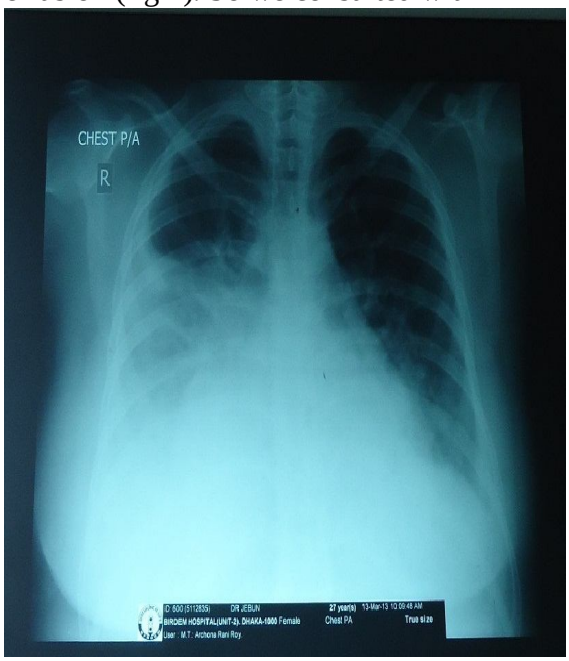

Figure 1. Chest X-ray P/A view shows massive pleural effusion

respiratory medicine department and shifted the patient in that department. At 12.00 AM patient complaints of severe shortness of breath, at that time O_2 Saturation-86% with 4-6 litre of O_2. Then patient shifted to ICU where 800cc pleural fluid was aspirated on 1st day and 400cc fluid was aspirated on next day. Patient gradually improved and discharged on 12th POD with antihypertensive drugs and follow up.

Discussion

During normal pregnancy the increase in circulating volume and hydrostatic pressure in the venous system favours the development of mild edema of the extremities and in 5% the appearance of a discrete pericardial effusion.[14] Ascites fluid and or pleural effusion are not common findings in women of normal pregnancy.[14] Durrelle et al[11] described the postpartum complication in 453 patients with Pre-eclamsia and HELLP syndrome and found that 16.77% (76 cases) had ascitic fluid and pleural effusion. Lilford et al [15] who described the case of preclamptic patient with massive ascites with pleural effusion. Forman[16] reporting on a patient with SP who has massive ascites (12litrs) without any other pathology to explain it.

In severe preeclampsia the reduced total PCOP ofprotein,imbalance(increase) in the hydrostatic pressure of the microcirculation are important , the involvement of structural factors such as endothelial injury, disruption of basal membrane and coagulation in situ[10,11] remain to be assessed , also impaired renal function may promote the development of generalized edema and thus the formation of abnormal fluid collection in the serosa in these types of patients.[11]

Conclusion

Pre-eclampsia is not a preventable condition. It needs regular ANC and intensive monitoring to diagnose early and also early intervention in an appropriate and well equipped hospital to decrease the morbidity and mortality rate of mother and fetus.

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