Research Paper Medical Science

Peripartum breathlessness leading to Pulmonary Edema

*Dr. Urmila Palaria **Dr. D. C. Punera
Dr. Mohd. A. Rasheed * Dr. Santosh

* Associate Professor, GMC, Haldwani (Nainital)

** Assistant Professor, GMC, Haldwani (Nainital)

*** Assistant Professor, GMC, Haldwani (Nainital)

*** 2nd Year Student, Post Graduate in Anaesthesiology, GMC, Haldwani (Nainital)

ABSTRACT

The interrelationship of pregnancy related physiological changes with effects of various co-existing diseases and their medications are less well understood despite common occurrence of these conditions during pregnancy.
We present a patient in which pulmonary edema developed after caesarean delivery with past history of pulmonary tuberculosis with medications and was being treated with carbamazepine for seizure disorder during pregnancy.

Keywords : Pregnancy, Pulmonary tuberculosis, Seizure Disorder, Pulmonary Oedema

Introduction:
Respiratory problems are common in pregnancy and it is worth noting that in recent Confidential Enquiry into maternal death (1994-96), 53.7% of direct deaths were as a result of respiratory problems1. There may be pre-existing conditions e.g. TB, asthma, Chronic Obstructive Pulmonary Diseases or may be an acute illness e.g. pneumonia, pneumothorax or more serious conditions like pulmonary embolism or Acute Respiratory Distress Syndrome complicating pregnancy. The major presentation of Acute Respiratory Distress Syndrome is noncardiogenic pulmonary oedema. Pregnant women are known to develop pulmonary edema more frequently than nonpregnant women. The incidence of pulmonary edema in pregnancy estimated to be 80 per 100,000 of pregnancy2.

Breathlessness a common presentation of respiratory problem, may be due to physiological changes in respiratory system or may be due to pre-existing respiratory illness. About 70% of pregnant women will report some level of dyspnea2, for unknown reason but seldom it is of concern. Very rarely pulmonary edema develops during or after pregnancy.

Pregnancy induced physiological changes, which are supposed to be progesterone hormone mediated, do not cause pulmonary edema but predispose to and may exacerbate pulmonary edema, in the presence of an “inciting factor”. These physiologic changes are 20% decrease in colloid osmotic pressure, 50% increase in blood volume and cardiac output and decrease in FRC and residual volume2.

Seizure disorders complicate approximately 0.5% of pregnancies and are the most common neurological disorder encountered in parturients3. Various drugs are used to manage epilepsy. These drugs may be a cause of concern in presence of pregnancy.

If the mother required caesaren section for the delivery of baby, use of anaesthesia drugs and procedure has additional implications.

Case Report:
25 years old full term unbooked primigravida from low socio-economic group presented with labor pain and leaking per vagina since five hours. She had a history of breathlessness since last five months and an episode of convulsion seven months back. She was on oral carbamezepine 100mg twice daily since then. She suffered from tuberculosis two years earlier for which she had taken nine-month course of anti tuberculosis drugs, the records of which were not available.

On examination her respiratory rate was of twenty per minute. She had decreased air entry on the right lower zone of chest with dull percussion note. Her pulse rate was ninety-two per minute with a blood pressure of 118 /82 mm of Hg.

Decision for emergency caesarean section was made due to fetal distress revealed by cardiotocography after admission. She was immediately shifted to operation theatre. Pulse oximetry revealed Hb-saturation of 97% on room air. Preoperative X-ray chest was not done due to lack of time. After securing I.V line, monitoring was done with electrocardiography, non-invasive blood pressure & pulse oximetry.

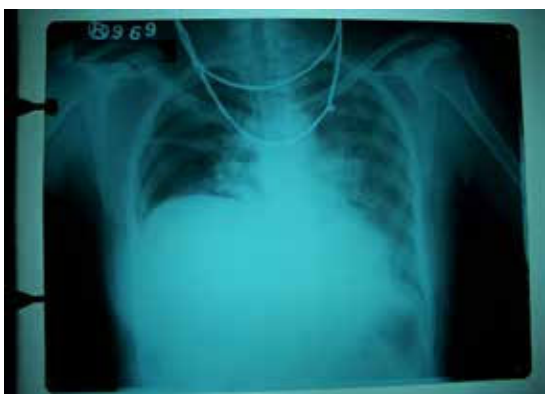
A preloading with 500 ml of Ringer lactate solution was done and spinal anesthesia was given with heavy bupivacaine, 2.5 ml in L3-L4 space in lateral position under aseptic precautions. Effect of spinal anesthesia was up to T6 level. A single live female baby weighing 2.5 kg was delivered after 15 minutes of spinal anesthesia. 10 units of injection oxytocin was given in infusion after delivery of baby. A total of 1700 ml of Ringer Lactate was transfused during operation. Total of 260 ml of urine came out intraoperatively.

The intraoperative bleeding and swings in haemodynamics were found within acceptable physiological ranges and no increase in breathlessness detected during operation. The whole operation lasted for seventy minutes and immediate postoperative condition was normal.

She was shifted to post anesthesia care unit and after about an hour shifted to her bed in ward. After 20 minutes of transfer in ward, patient complained increase in breathing difficulty and complained of shortness of breath. She was conscious but was not able to speak more than two words in a breath.

Her respiratory rate was thirty per minute and pulse rate of 112 per minute. SpO2 of Hb was 94% on room air. Auscultation of chest revealed presence of fine crepitations. Bedside chest X-ray PA view was done. X-ray of chest showed elevation of right diaphragm with a thin parallel rim of air gas shadow below it. Numerous soft fluffy infiltrations were seen bilaterally in lower and middle parts of lung shadow(Figure-1). A diagnosis of pulmonary edema was made and patient was immediately shifted to intensive care unit.

Figure Legend:
Figure -1: X-ray chest PA view showing features of pulmonary edema, high up right diaphragm and adhesions in right lobe.



Management of condition was done with oxygen inhalation with simple mask at a rate of 5 L/minute. Intravenous Inj. frusemide 40 mg, inj. deriphyllin 1 ampoule (etofylline 84.7mg and theophylline 25.3mg) and Inj. Hydrocortisone 100 mg was given.Ventilator was kept ready if required. After about thirty minutes her general condition started improving with improvement in oxygen saturation. Auscultatory finding also improved. Respiratory distress was decreased and patient became comfortable after about six hours. Ventilatory support was not required.

Ultrasound of thoracic cavity was done - elevation of diaphragm was confirmed and collapse and fibrosis of right lower lobe was reported. Investigations for reactivation of tuberculosis were done, which were found to be negative.

Her condition gradually improved with oxygen, rest and diuretics. No further investigation like bronchoscopy, was done due to lack of facility. Patient gradually improved and discharged after seven days with advice of regular follow up.

Discussion:
Pregnancy is associated with significant physiological changes in the respiratory system. Oxygen consumption and minute ventilation progressively increase due to increase in tidal volume and to a lesser extent, due to increase in respiratory rate. Oxygen consumption increases by about 20%, and minute ventilation by 50%. PaCO2 also decreases to about 32 mmHg, but respiratory alkalosis is prevented by a compensatory decrease in plasma bicarbonate concentration4. This increased depth of breathing is supposed to be progesterone stimulated. Diaphragm is raised from its normal position by term but this does not have significant effect on respiratory function because diaphragmatic excursion is unaltered. These changes are generally well tolerated by pregnant women.

In this case, the patient was complaining of breathlessness during second trimester, which continued till term. Neither preoperative X-ray nor proper investigation for breathlessness was done due to poverty, ignorance and erroneous thought of its occurrence normally during pregnancy. No proper antenatal visits were done. In addition to the normal physiological changes attributable to pregnancy, diaphragm on the right side was found to be higher up because of the right-sided lower lobe collapse. It was revealed only post operatively. This may be one of the reasons for aggravation of breathlessness. There was no active tuberculosis at that time, but it may be a sequel of past tuberculosis. In X-ray chest, on right side subdiaphragmatic very thin rim of gas shadow was found to be associated with postoperative changes.

After delivery of baby cardiac output rises with placental expulsion. The contracted uterus also contributes to auto transfusion, increasing circulating blood volume, by about 500 ml during the first 12 to 48 hrs postpartum5.

Cardiac output remains elevated for several hours after delivery. In normal pregnancy, plasma oncotic pressure falls significantly after delivery, reaching its nadir 6 hours postpartum. Intravenous fluid given during perioperative period and normalization of physiology after spinal anesthesia may contribute to further increase in preload. All these factors increase fatigability and cause respiratory distress.

The interrelationship of maternal epilepsy, metabolic and pharmacodynamic changes of various drugs is complex and has led to considerable controversy concerning epilepsy in pregnancy.Pregnancy induces several pharmacokinetic changes that may significantly alter the serum concentration of anticonvulsants.

Anti-epileptic drug, carbamazepine, acts by stimulating vasopressin release from neurohypophysis and has antidiuretic effects,. A late complication is retention of water with decreased osmolality & concentration of Na+ in plasma6. This may in turn predispose a compromised pregnant woman to develop pulmonary edema. Current recommendations are that ideal anti-epileptic concentration should be established for each patient before conception and monitoring of levels should be performed during each trimester till the last month of pregnancy7. In this case serum concentration of drug was not being monitored during the antinatal period.

The present case report highlights the risk of developing pulmonary edema in pregnant women with compromised chest conditions and taking anti epileptic medication. These kinds of patients should be cared with caution and appropriate preventive measures should be taken to prevent occurrence of this kind of life threatening situation.

REFERENCES

1. Idiopathic pulmonary fibrosis? Postgraduate Med J 2000(November); 76:683-89 | 2. D.K. James, P.J. Steer, Respiratory disease, High risk pregnancy, IIIrd Ed, pg. 828, | 852- 853. | 3. Mark C. Norris, Preoperative Evaluation of the Parturient with Coexisting Disease, | Obstetric Anesthesia, IInd Ed, pg. 70 | 4. G.Edward Morgan et al, Maternal & Fetal Physiology & Anesthssia, Clinical | Anesthesiology, IVth Ed, pg. 876 | 5. Bernard Wittles et al, Repeated episodes of respiratory distress in an obese | parturient after cesarean delivery, Anesthesia & Analgesia, April 2009, Vol.108, | 4:1247 | 6. Goodman & Gilman's, Drugs effective in the therapy of epilepsies. The | pharmacological basis of therapeutics, Xth Ed, pg. 534. | 7. Pennell PB, Anti epileptic drug pharmacokinetics during pregnancy and | lactation, Neurology 2003;61:S35-S42.