FLORID IMMEDIATE POST-OPERATIVE PULMONARY OEDEMA WITH SEVERE HYPOTENSION AND COMA

(A Case Report)

by

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Introduction

Although it is often easy to look wise later and see in retrospect a clear cut diagnosis and its specific therapy, the actual encounter is not that straight forward as is illustrated in the following case report.

CASE REPORT

An obese, oedematous, 28 year old, full term, 3rd gravida underwent elective lower segment caesarean section under general anaesthesia. Previous obstetric history was uneventful with 2 full term normal deliveries, both females of birth weight 2.8 and 2.6 Kg respectively. During the only ante-natal visit 3 days prior to surgery, she was found to have cephalopelvic disproportion and severe pre-eclamptic toxaemia. A day prior to surgery, she was administered frusemide 80 mg, pethidine 100 mg and promethazine 25 mg.

Pre-anaesthetic findings included pulse rate 88 per minute, regular; BP 140/110 mm Hg; generalised oedema and no other systemic abnormality. Haemoglobin was 11.5 g/100 ml. Bleeding time, clotting time, serum electrolytes and ECG were within normal limits.

Following premedication with atropine 0.5 mg I V, anaesthetic sequence comprised of preoxygenation, induction with thiopentone 200 mg, intubation under suxamethonium and maintenance under controlled respiration with $N_2 + O_2$ and suxamethonium till delivery of normal male baby and intermittent d-tubocurare and trichloroethylene subsequently and was uneventful, till skin closure. Pulse and blood pressure had fluctuated around preanesthetic levels despite inj. methergin 0.4 mg I V to control brisk haemorrhage after delivery of the child. 5% dextrose 400 ml was the only fluid infused during the 1½ hour procedure. Spontaneous respiration and protective reflexes had returned following reversal with nostigmine 2.5 mg and atropine 1 mg, when she was extubated.

Immediately after extubation respiratory difficulty was noticed. Pharyngeal suction, oxygenation, ventilation with bag and mask and further reversal with inj. neostigmine 1 mg and atropine 0.5 mg repeated twice in next 5 minutes proved ineffective. She became deeply cyanosed, comatose and areflexic. Pulse and BP were unrecordable. With esophageal stethoscope a heart rate of 160 per minute could be counted. Bubbling sounds were heard all over chest. Pupils were of normal size, but fixed and central. Immediate reintubation, ventilation, oxygenation and frequent tracheobronchial suction were carried out. Tremendous resistance was felt while manually inflating the reservoir bag.

Then followed a pitched encounter between therapeutic measures and relentless outpouring of tracheobronchial secretions, which were mucoid initially, later turned watery and pinkish and finally, almost blood red in colour. Inter-

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mittent positive pressure ventilation (IPPV), first manually and then with Radcliffe ventilator was continued. The ventilatory parameters included respiratory rate 24 per minute to begin with, later reduced to 16 per minute; inspiratory pressure 25 cm H_2O initially and gradually reduced to 15 cm H_2O and expiratory tidal volume below 500 ml in the beginning, but gradually increased to 900 ml. Other measures included dexamethosone 16 mg IV repeated every 10-15 minutes and frusemide 80 mg I V initially and 40 mg repeated twice at 2 hour interval. For extreme hypotension, mephentermine 30 mg I V bolus and 450 mg in 5% dextrose drip was started. Sodabicarb 7.5% 50 ml was administered twice at 2 hour interval. Within an hour of frusemide administration, 500 ml of urine was collected when digoxin 0.5 mg IV was given under continuous ECG monitoring which showed sinus tachycardia. Chest X-ray revealed pulmonary oedema. (Fig. 1) Despite mephentermine drip having continued for two hours, pulse was still feeble and BP 60 mm Hg, when one unit of blood was started. An hour later, sensorium improved slightly, eye balls started rolling and urine output had averaged 400 ml per hour; but there was no change in hypotensive state and in outpouring pulmonary secretions. Morphine was given 3 mg IV stat and 1.5 mg repeated at 1-2 hour intervals. An infusion containing 20% glucose 200 ml. soluble insulin 10 units and potassium 20 ml was started.

After nearly 4 hours, patient regained consciousness fully; pulse was 140 per minute, BP 90 mm Hg, systolic, urine out put was 1500 ml and pulmonary secretions subsided. Under manual ventilatory support, she was shifted to the intensive care unit. There she started blowing out blood-red fluid several centimeters high in the air through the orotracheal tube. With continuation of the above measures the patient settled down in next half an hour. X-ray chest showed a marked improvement (Fig. II). Quiet, comfortable spontaneous respiration returned. BP was 120/70 mm Hg and pulse 120 per minute. She was extubated. On first post-operative day 2400 ml urine was passed. Subsequent recovery, except for transient mephentermine psychosis and thrombophebitis at the site of venesection, was uneventful.

Discussion

Although this patient presented with pre-eclamptic toxaemia, with preoperative preparation, she withstood anaesthesia and surgery, including intraoperative administration of methergin uneventfully. The respiratory embarrassment, which started on exturbation, did not respond to pharyngeal suction, ventilatory support and full reversal of muscle relaxant action. It worsened into cyanosis, coma, extreme hypotension and outpouring of pulmonary secretions. Conjectures crossed floor between obstetric and anaesthesia colleagues, hinting at Mendelson's syndrome and amniotic fluid embolism. There was however no wheeze or other auscultatory evidence of bronchospasm and ECG showed no evidence of right ventricular preponderance. Koshy reported presence of amniotic squamae in inferior venacaval blood of 5 per cent of patients undergoing caesarean section without any other evidence of amniotic embolism.

It must be admitted that the sudden simultaneous occurrence of deep cyanosis, coma, extreme hypotension and florid outpouring pulmonary secretions forced the urgent symptomatic rather than rational therapy. Accordingly, IPPV, oxygenation and frequent swift tracheobronchial suction were instituted. Massive steroids and frusemide were administered and for extreme hypotension, mephentermine, sodabicarb and later one unit of blood were given. This may not be in keeping with current evidence favouring reduction of preload by vasodilatation and application of tourniquet in pulmonary oedema. According to Fishman, hypotension with pulmonary oedema is a contraindication for use of tourniquet. Moreover, it was difficult to be dogmatic about the blood loss during caesarean section.

Fishman (1979) pointed out that when faced with pulmonary oedema, battery of

therapeutic measures is applied in rapid succession or simultaneously and that some of the measures may be more harmful than the disorder. We had never encountered simultaneous occurrence of pulmonary oedema, coma and extreme hypotension. Mephentermine was employed in a desparate bid to deal with the extreme hypotension. It did not produce the desired effect. Temptation to try other vasopressors was resisted in view of the pulmonary oedema. Tachycardia of 170 per minute precluded use of isoprenaline; digoxin was used instead. Urine output of 2400 ml on the first postoperative day, despite restricted fluid intake, pointed at the massive fluid overload of preeclamptic toxaemia, with which she had presented. Along with other measures, frusemide played a 'sheet anchor' role in the management.

References

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See Fig. on Art Paper I