Perioperative management of pulmonary oedema due to oxytocin in a case of caesarean section

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ABSTRACT

Pulmonary oedema develops acutely during perioperative and poses as a life-threatening complication. It is usually due to unknown cause especially if it occurs in an otherwise normal patient. Noncardiogenic pulmonary oedema may be caused by upper airway obstruction due to laryngospasm after general anaesthesia. Timely recognition and proper management is of paramount importance for successful outcome.

Key words: oxytocin, pulmonary oedema, non invasive ventilation

INTRODUCTION

Pulmonary oedema is the abnormal accumulation of fluid in the interstitial or alveolar spaces of the lung. It occurs for a number of reasons which can be explained on the basis of a disturbance in the normal Starling equation. It involves changes in hydrostatic or oncotic pressure across the alveolar membrane or in the permeability of the alveolar membrane such that fluid moves across from the capillaries into the alveolar space.

Case Report

A 26 year, ASA grade 1 female, full term gravida 2, weighing 50 kg was taken up for emergency LSCS due to breech presentation with non-progression of labour and fetal distress. On pre-anaesthetic assessment her pulse rate was 80/min, and blood pressure 126/84 mmHg. Respiratory and cardiovascular system was normal. Her Hb was 11.0gm%, and all other investigations were within normal limits. In the operation theatre patient was kept in left lateral position, intravenous lines were secured and premedication was given. Prior to block, she was preloaded with 500 ml of ringer lactate. With all aseptic precautions, the subarachnoid block was given with 2.5ml of bupivacaine at L3-L4 space using 25G spinal needle. Patient was positioned and after ensuring sensory block up to T4-T5, surgery was ensued. Baby was extracted within 3 minutes, and along with clamping of the umbilical cord, methylergometrine 0.2 mg was given in left deltoid intramuscularly. Oxytocin 10 units in 500 ml of dextrose 5% was started. Vitals were stable with pulse rate of 86/min and BP of 116/74 mmHg with urine output of 50 ml in half hour.

During surgery 5 units of bolus oxytocin given for increasing the uterine tone, at that time blood pressure fell to 90 mmHg systolic along with tachycardia of 110 beats/min. BP was maintained with mephenteramine bolus of 3 mg. When rectus was being stitched, patient suddenly started complaining of chest pain and cough along with difficulty in breathing with a drop in oxygen saturation to 95%. Oxygen started at 5L/min, patient was repositioned and assured, but her SPO2 become 85% with severe respiratory distress and on auscultation bilateral crepitations were present all over the lung fields. Patient was suspected of having pulmonary edema and immediately taken on bag and mask and 100% oxygen administered on spontaneous ventilation. Inj. Lasix 80 mg i.v. given as increments of 40 mg at an interval of 4-5 minutes. Patient was still conscious, so intubation was not planned. Meanwhile, surgery was completed and patient shifted to postoperative i.c.u. and put on non invasive ventilation (BIPAP) with IPAP 12cmH2o and EPAP 8cmH2o and backup rate of 6b/min support. After 3 hours of continuous oxygenation on BIPAP, and diuretics, patient improved with SPO2 of 95%,
urine output of 800ml, and lung field become clear on auscultation. Vitals were within normal range with BP of 110/74 mmHg with mild tachycardia of 104/min. An echocardiography was done, all the parameters were within normal range and there was no evidence of any cardiac lesion. After 24 hours, patient becomes stable with SPO2 of 99% on room air and clear chest field. On regular follow-up of the patient, there wasn’t any residual effect of the incident.

**DISCUSSION**

The administration of oxytocin, after uterine emptying is routine in obstetric anesthesia in parturient undergoing cesarean sections. It promotes the contraction of the uterine smooth muscle, reducing blood loss at the place of placental detachment. Therefore, both its prophylactic and therapeutic use is justified to decrease the incidence of postpartum hemorrhage. However, since it also has extra-uterine actions, the improper use of exogenous oxytocin may be deleterious.

Besides contraction of the uterine smooth muscle and myoepithelium that surrounds the alveolar ramifications in the breast, oxytocin has systemic effects, such as relaxation of the smooth muscle of the vessels, promoting vasodilation; this leads to a reduction in systolic blood pressure and, especially, the diastolic blood pressure, besides reflex tachycardia. This vasodilation, usually temporary, can be clinically significant when oxytocin is administered in bolus, and may lead to a reduction in coronary perfusion and cardiac collapse; these effects are more prominent in the presence of general anesthesia. The cardiovascular effects of oxytocin are known from studies that have been published. Although it has a wide therapeutic index, the administration of oxytocin may cause a significant reduction in peripheral vascular resistance and increase in cardiac output. Initially, it was believed that this increase was due to maternal self-transfusion after detachment of the placenta, but it has been shown that the cardiac output also increases when a bolus of oxytocin is administered in the beginning of the pregnancy (in uterine curettage, for example). It was then suggested that pure synthetic oxytocin had a b-stimulating action, increasing chronotropism and inotropism, and promoting peripheral vasodilation. More importantly, the cardiovascular effects seem a consequence of the excessive administration of oxytocin and not of its preservative.

The anti-diuretic action is also observed when high doses are administered, and cases of water intoxication with hyponatremia secondary to the retention of free water, resulting in pulmonary edema, seizures, coma, and death have been reported. However, it should be mentioned that when used in physiological doses, in the absence of increased volume status, pure synthetic oxytocin rarely has an anti-diuretic action.

**CONCLUSION**

We conclude that peripartum use of oxytocics can precipitate pulmonary edema, without any evidence of vascular overloading or any occult cardiac disease. High index of suspicion and prompt treatment is mandatory. We believe that oxytocin must be used judiciously.

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