

Peri-operative management of pulmonary edema following oxytocin administration in a pregnant woman undergoing emergency lower segment caesarean section (LSCS) under spinal anaesthesia

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ABSTRACT

Pulmonary edema is an abnormal accumulation of fluid in the interstitial or alveolar spaces of the lung. Its etiology can be explained on the basis of the disturbance in the normal Starling equation. Water intoxication is not a common complication of oxytocin infusion. We have presented a case of 27 years old second gravida parturient.

The patient had developed acute severe pulmonary edema during emergency lower section caesarean section (LSCS) following oxytocin administration to prevent post-partum hemorrhage. Issues surrounding management of pulmonary edema and use of oxytocin therapy during pregnancy are briefly discussed.

Key words: Emergency LSCS, Oxytocin, Pulmonary edema, Spinal anaesthesia

Introduction

Oxytocin is a cyclic, nona-peptide posterior pituitary hormone. Because of its chemical similarity to vasopressin, oxytocin at higher doses has prominent antidiuretic effects[1]. Synthetic oxytocin has been widely used securely in clinical obstetrics for more than half a century. There are rare case reports about regarding water intoxication due to antidiuretic effects of the oxytocin preparations in the literature[2,3,4]. We describe here a case of pregnancy complicated with progressive pulmonary edema during subarachnoid block for LSCS following administration of oxytocin and vasopressor drugs.

Case report

A 27 year, ASA (American society of Anesthesiologists) grade 1 female, full term gravida 2, weighing 54 kg was taken up for emergency LSCS due to breech presentation. There was non-progression of labour and fetal distress. On pre-anaesthetic assessment her pulse rate was 86/min, and blood pressure 120/84 mmHg. Respiratory and cardiovascular system was normal. She was nondiabetic. Her blood group was O, Rh +ve and her Hemoglobin was 10.5 gm%, and all other investigations were within normal limits. The ultrasonographic report of abdomen revealed, single large size anencephalic fetus with breech presentation. In the operation theatre patient was kept in

left lateral position, two 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 2ml 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 15 units in 500 ml of dextrose 5% was started. Vitals were stable with pulse rate of 88/min and BP of 112/76 mmHg with urine output of 50 ml in half hour.

During surgery 5 units of bolus oxytocin was administered for increasing the uterine tone. At that time systolic blood pressure of the patient was dropped to 70 mmHg. Infusion of fluid was enhanced with polygeline (Haemaccel). Increments of mephentermine sulphate 10, 7.5, 5 (total 22.5mg) were given intravenously over a period of 10 minutes. Systolic blood pressure shot up to 100 mmHg. At the instance when rectus was being sutured, patient instantaneously started complaining of chest pain and cough along with difficulty in breathing with a drop in oxygen saturation to 95%. Oxygen was started at 5L/min, patient was repositioned and assured, but her SPO2

became 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. Patient was not considered to be over hydrated at any stage. 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 intensive care unit and was put on non-invasive ventilation (BIPAP) with IPAP 12cmH₂ o and epap 8cmH₂ o and backup rate of 8 breaths/min support. After 3 hours of continuous oxygenation on BIPAP, and diuretics, patient improved with SPO₂ of 95%, urine output of 750ml, and lung field become clear on auscultation. Vitals were within normal range with BP of 118/84 mmHg with mild tachycardia of 102/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 became stable with SPO₂ of 99% on room air and clear chest field. The patient was closely monitored throughout her stay in the intensive care unit and it was observed that the condition had improved and there was no residual effect.

Discussion

Oxytocin is primarily used at induction of the third stage of labour, principally to reduce post-partum hemorrhage[5]. The structure of oxytocin is very similar to that of vasopressin with a sequence different from oxytocin by 2 amino acids. The continuous intravenous infusion of 20 mU of oxytocin per minute usually produces a considerable decrease in urine flow. When the rate of infusion is increased to 40 mU/min, urine flow is significantly reduced. The antidiuretic effect of oxytocin has been observed in both non-pregnant and pregnant women. If electrolyte-free aqueous dextrose solutions are used in significant volume along with oxytocin, water intoxication can lead to pulmonary edema, convulsion, coma and death[6].

Hypotension during spinal anaesthesia occurs due to decrease in venous return, vasodilatation and decreased cardiac output. Bladder distention and vagal over activity may also contribute[7]. Standard preventive measures include preloading up to one liter of crystalloid, prevention of high levels of block, correction of hypervolemia, elevation of lower limbs, and use of vasopressors. In the patients with full term gravid uterus, the most important maneuver is left lateral tilt. Volume loading does not always guarantee arterial pressure and overloading can be harmful in patients

at the risk of pulmonary edema. Vasopressors have to be administered to maintain optimal level of arterial pressure.

In present case, usual measures were taken (foot end elevation, intravenous infusion of fluids was enhanced with polygeline, vasopressors administration). Incremental doses of mephentermine sulphate 10,7.5,5 (total 22.5 mg) was administered intravenously to correct hypotension[8]. Methylergotamine 0.2 mg I.V. stat and oxytocin 15 units were administered in infusion to ensure effective contraction of uterus. This reduced the incidence and extent of post-partum hemorrhage[9]. Both mephentermine sulphate and methylergotamine maleate are alfa agonists. Concomitant use of these two vasopressor agents may have caused severe widespread vasoconstriction causing marked increase in the afterload to heart. Clinch fist effect of mephentermine sulphate on heart with the markedly increased afterload prevent forward pumping action of heart resulting in pulmonary vascular leak and pulmonary edema[10]. Antidiuretic action of oxytocin might have been contributory[11,12].

Conclusion

In conclusion, clinicians must be aware that pregnant women, especially during labour, induction of labour and delivery, are at risk of pulmonary edema. Even very low doses of oxytocin (2–5 mU/min) are antidiuretic and can be associated with life-threatening complications such as acute severe pulmonary edema. To prevent this condition, rather than increasing the flow rate of a more diluted solution, oxytocin concentration should be increased. Moreover, fluid balance should be recorded carefully in every case. In critical patients who are prone to pulmonary edema, oxytocin and dextrose in fluids should be withheld. Until normalization, serum sodium concentration isotonic saline should be slowly infused. This should be followed by diuresis and, if necessary, furosemide administration. Hypertonic saline or Ringer's lactate would be other choices for oxytocin infusion.

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