Acute pulmonary edema after intramyometrial prostodin

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Abstract
A 25 year old, 68 kg, primigravida, was taken up for emergency caesarean section for meconium stained liquor and fetal distress. She was a known case of pre eclampsia and her blood pressure was controlled on methyl dopa. She was administered general anaesthesia. After delivery of baby she went into postpartum hemorrhage which was controlled with intramyometrial prostodin. But immediately after its administration she went into acute pulmonary edema.

Key words: Postpartum hemorrhage, intramyometrial, prostodin

Case Report
A 25-year-old, 68 kg, BMI 27.64 kg/m², ASA II primigravida, was taken up for emergency caesarean section for meconium-stained liquor and fetal distress. She was a known case of preeclampsia and her blood pressure was controlled on methyl dopa 250 mg PO thrice daily. Her preoperative hematologic investigations were all within normal limits (Hb, 10.8 g%; TLC, 5700/mm³; platelets, 2.2 million/mm³; S. bilirubin, 0.85 mg%; blood urea, 42 mg%; INR, 1.13; PT (test), 13 s; PT (control), 14 s; PTTK (test), 36 s; and PTTK (control), 35 s. Her urine was positive for albumin. Her chest X-ray and electrocardiogram did not reveal any abnormality. An informed consent was taken after explaining all anesthetic concerns to the patient and her relatives.

Before induction she was conscious, oriented with blood pressure 143/88 mmHg, pulse rate 101/min, and oxygen saturation 98% on room air. In view of acute fetal distress (urgency grade 1),[1] it was decided to administer general anesthesia for the caesarean section. She was preoxygenated with 100% O₂ for 3 min and rapid sequence intravenous induction performed with 250 mg thiopentone, 100 mg succinylcholine, using Sellick’s maneuver, and the surgery was started. Anesthesia was maintained with O₂, N₂O, and sevoflurane along with rocuronium bromide. After 7 min the baby was delivered after which inhalational agent was switched off. Patient was administered fentanyl 100 μg intravenously for analgesia. Oxytocin infusion was started at the rate of approximately 0.04 IU/min after a bolus of 5 IU. However, the patient went into postpartum hemorrhage (PPH) due to uterine atony, which did not even respond to an increase in the rate of oxytocin infusion. The patient lost about 1–1.5 L of blood.

The surgeons decided to administer prostaglandin F2-alpha—carboprost trometamol 0.25 mg (Prostodin, AstraZeneca, India) intramyometrial. Within 1–2 min of its administration airway pressures shot up to 55 cm and the oxygen saturation fell to 88%, although her PPH was now controlled. Her blood pressure fell to 92/54 mmHg with a pulse rate of 110/min but was successfully restored with the administration of a total volume of 3 L of crystalloids. Auscultation of the chest revealed bilateral widespread rhonchi. She was immediately administered hydrocortisone (200 mg) and deriphyllin (110 mg) and nebulised with salbutamol 2.5 mg diluted in 4 mL saline. As the PPH was controlled, the closure of uterine incision started. Auscultation of the chest now revealed minimal rhonchi but the appearance of fine crepitations, especially at the bases. A diagnosis of acute pulmonary edema was made and the patient was given frusemide 40 mg along with morphine 6 mg intravenous stat.

After completion of surgery, neuromuscular blockade was not reversed and the patient was shifted to ICU for further management. In the ICU she was nursed in the head
An important aspect of PPH treatment is uterotonic therapy. The most commonly used agents are injectible oxytocin and/or ergometrine. Parenteral prostaglandins have shown promise,[4] but their use has been limited by side effects. In dire situations, intramyometrial injection of prostaglandin F2-alpha has been used with apparently dramatic effects.[5] Intrauterine irrigation with PG F2-alpha has also been used.[6]

Prostaglandin (15-methyl PG F2-alpha) acts as a smooth muscle stimulant and is a recognized second-line agent for use in the management of postpartum uterine atony unresponsive to oxytocin/ergometrine. It is an analog of PG F2-alpha (dinoprost) with a longer duration of action than its parent compound, attributed to its resistance to inactivation by oxidation at the 15-position. Available in single-dose vials of 0.25 mg, it may be administered by deep intramuscular injection or, alternatively, by direct intramyometrial injection. The latter route of administration is achieved either under direct vision at caesarean section or trans-abdomen or trans-vaginal following vaginal delivery and has the advantage of a significantly quicker onset of action.[7] Peripheral intramuscular injection yields peak plasma concentrations at 15 min in contrast to less than 5 min for the intramyometrial route.

Side effects of PG F2-alpha are related to its effects on smooth muscles. They include nausea, vomiting, diarrhea, bronchospasm, and systemic hypertension. Hayashi et al. reported infrequent and mild side effects, including nausea, vomiting, and diarrhea.[8] Sudden collapse and death have been reported by Cares and Jordan[9] following the use of PG F2-alpha for abortion.

Secher et al. found a 40% increase in cardiac output in pregnant anesthetized women during infusion of 300 μg of PG F2-alpha with an increase in pulmonary arterial pressure, doubling of pulmonary vascular resistance and increase in airway resistance.[10] Hankins et al. reported the development of marked arterial desaturation within 5–10 min of the administration of 15 methyl PG F2-alpha, secondary to intrapulmonary shunt.[11] Five patients required ventilator support for variable durations. Cardiovascular collapse along with left heart failure has been reported with overdose of intramyometrial prostaglandin, the possible etiology being a combination of acute pulmonary hypertension with decreased left ventricular end-diastolic pressure and decreased cardiac output.[12]

In our case, the cause of pulmonary edema could be either fluid overload or the administration of prostodin. Three liters of crystalloids were transfused for a loss of approximately 1–1.5 L of blood. Development of pulmonary edema immediately after the injection of prostodin points toward it being responsible for the edema. Although pulmonary edema after the administration of prostaglandins is common in patients with pre-existing cardiac disease, it has also been reported in patients with normal heart.[13]

It may be prudent to use first-line uterotonics to control PPH as far as possible. Prostaglandin analogs, whenever used, should be used carefully, along with good monitoring and preferably under the cover of diuretics.

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