CASE REPORT

Cardiovascular Collapse Following Intramyometrial Injection of Prostaglandin

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Abstract

A 21 year old female gravida 2, para 1, live 1 with previous lower segment cesaerian section (LSCS), hypothyroid on tablet Eltroxin 12.5mcg, was posted for LSCS. Spinal anaesthesia was given and the effect was patchy, hence general anaesthesia was supplemented. Intramyometrial prostaglandin was given by surgeon for persisting uterine atony following which patient developed hypertension, tachycardia, pulmonary edema and later on cardiac arrest.
Key words: *Cardiovascular collapse, Intramyometrial Injectionection, Prostaglandin, Pulmonary edema.*

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Introduction

For treating uterine atony or established postpartum haemorrhage during caesarean section the administration of oxytocic drugs is an important intervention nowadays. It is an established fact that these agents have a narrow therapeutic range. A detailed knowledge of optimal doses and their side effects is therefore required by anaesthetists. Oxytocin remains the first line agent. Second line agents namely ergot alkaloids and prostaglandins may be required when there is receptor desensitization, Takagi[1] in 1976 first described the use of intramyometrial prostaglandin F2α for atonic Post Partum Haemorrhage. There are a number of ongoing anaesthesia and obstetric audits and research into these drugs with their narrow therapeutic range[2]. This should improve the anaesthetist's ability to limit obstetric haemorrhage during caesarean section in future, while at the same time also focusing on reduction in unpleasant or dangerous maternal side effects [3].

Case Report

A 21 year old, G2 P1 L1 A0, with previous LSCS was posted for emergency LSCS. She was a known hypothyroid, on tablet Eltroxin 12.5mcg, once daily. There was no history of pregnancy induced hypertension, asthma, cardiac diseases or convulsions or any other significant medical illness. Preanaesthetic checkup was done and following things noted. Moderately built and nourished female with Mallampatti class I airway and good dentition and normal physical parameters from head to toe. Systemic examination revealed bilateral good air entry, S1, S2 normal heart sounds, no murmurs. No neurological deficit. Her investigations revealed hemoglobin of 12.5gm%, white blood cells of 7500 cells/cumm, platelets were 2 lakhs/cumm, blood urea was 40mg%, serum creatinine was 0.8, clotting time and bleeding time were normal. Electrocardiogram was within normal limits.

After obtaining written consent, patient was taken up for LSCS. Routine monitoring done using a pulse oximeter, Noninvasive blood pressure recording and electrocardiography monitor connected. Preoperative vitals were BP 120/70mmHg, right arm supine, pulse rate: 76/min, regular, SPO2 98% at room air. ECG showed normal sinus rhythm. Under aseptic precautions spinal anaesthesia was given using 0.5% heavy bupivacaine; 2.1cc (Injection into Lumbar 3-Lumbar 4 subarachnoid space) in the

sitting position using 25 Guage spinal needle. effect was patchy. Hence general anaesthesia was administered as per the standard protocols of induction and anaesthesia. Surgery proceeded. A live male child with APGAR 8 was delivered. After baby delivery Injection oxytocin 10 units intravenous in drip was started and 10 units repeated again via drip. Injection Fortwin 18 mg iv was given. As uterine atony was present, Injection Prostaglandin 0.25mg was given intramuscularly. Still uterine atony was persisting and now the surgeon gave 0.25mg intramyometrial prostaglandin. Immediately the uterus retracted well.

Surgery was completed in 15 minutes after the administration of intramyometrial Prostaglandin. After the surgery, patient was checked for bleeding per vagina which was not significant. Within 15 min after Injection of intramyometrial prostaglandin, Blood Pressure increased to 160/90mm Hg, Heart rate increased to 160/min with normal sinus rhythm. She was closely being monitored. Patient developed spontaneous respirations, and became conscious. Patient was reversed with neostigmine 2.5mg and glycopyrolate 0.4mg iv. Patient suddenly developed pulmonary edema [4] which was evident as pink frothy secretions from endotracheal tube. Hence patient was not extubated. Intermittent Positive Pressure Ventilation + Positive End Expiratory Pressure of 5cm H_20 with 100% Oxygen was continued. Injection Lasix 40 mg iv was given twice; Injection Morphine 9 mg iv was given. Antitrendelenberg's position was given. Tachycardia persisted. There were coarse crepts bilaterally. BP was 150/100 mm Hg. Injection Lasix 40 mg was again repeated iv. SPO2 decreased to 95%-90%-88% with 100 % oxygen support. Systolic BP was ranging from 120-->100-->80mm Hg.

Pulmonary edema was cleared, but patient developed Supraventricular tachycardia for which Injection Adenosine 6 mg was given IV bolus after a carotid massage. After few minutes patient developed cardiac arrest [5]. Cardiopulmonary resuscitation was done under standard Advanced Cardiac Life Support protocols. Patient was revived. Cardiologist was called for opinion. Bed side 2D echocardiogram was done which showed dilated Inferior Vena Cava, Ejection Fraction was 25% which gradually further decreased to 15%, decreased Left Ventricle function, moderate Tricuspid Regurgitation & Mitral Regurgitation.

BP further decreased to 70 systolic. Ionotrope support was started. Patient had cardiac arrest second time and was revived. Patient was shifted to Critical Care Unit where she was connected to ventilator. Central venous cannulation of Right Internal Juglar vein was done. Central Venous Pressure was 9cm H2O. Systolic BP was 90 mm Hg with Dopamine @ 10 mcg/min. PR: 96/min, Normal sinus rhythm , SPO2: 92% on Pressure Controlled Ventilation mode of ventilator [6].

Patient was unconscious, responding to deep pain. Pupils were sluggishly reacting to light. Patient developed convulsions. Injection Lorazepam, Injection Sodium valproate was given IV and Injection phenytoin drip was started. After 2 hrs convulsions were controlled and Injection phenytoin drip was stopped. Patient developed cardiac rhythm abnormalities. She went into Atrial Fibrillation (AF) with fast ventricular rhythm. Injection Amiodarone was given IV 150 mg bolus followed by ordering a continuous iv infusion at 1 mg/kg/min for first 6 hours followed by 0.5 mg/kg/min for next 18 hours. But the AF was refractory and persisted inspite of Amiodarone infusion. Injection Xylocard 50 mg was given IV. Still Bigeminy pattern persisted. Later patient had bradycardia and then cardiac arrest and was revived as per ACLS protocols. After 6 hours patient had another episode of cardiac arrest again, but this time she could not be revived.

Discussion

There are no significant associated risk factors in this case except mild hypothyroidism on tablet Eltroxin 12.5mcg. Case was taken for LSCS under standard techniques. As uterine atony was persisting as seen as deep pitting in the uterus and told by surgeon, patient was given intramuscular prostaglandin followed by intramyometrial prostaglandin which was administered by surgeon herself. Prostaglandin F2α when given intramyometrially reaches peak concentrations within a very short time[7]. It causes contraction of both pulmonary arteries and veins. Blood pressure may increase, cardiac output increases. There will be increased force

of contraction and increased heart rate. In a large measure, a reflex consequence of fall in total peripheral resistance can occur. Increased pulmonary arterial hypertension, decreased Left Ventricular End Diastolic Pressure producing pulmonary edema followed by cardiac arrest is reported. Increased airway resistance, bronchospasm in some patients and cardiovascular collapse along with left ventricular failure was reported with reference to previous studies done by others. Five patients required ventilatory support (Hankins et al).

Conclusion

PGF2α given intramyometrially especially when repeated after a short span of intramuscular Injection might produce pulmonary edema and cardiovascular collapse as was seen with our case. Hence intramyometrial prostaglandin should be used with great caution and as a last resort of treatment and is better to be avoided.

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