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
Patient developed cardiac rhythm abnormalities. Patient developed convulsions. Injection Sodium valproate 18 mg iv was given. As uterine atony was persisting as seen as deep pitting in uterus and told by surgeon, 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 Ektroxin 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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**References**


