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**Case Report**

# Drug induced pulmonary artery hypertension in a parturient-diagnostic challenge

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## ABSTRACT

A 28-year-old gravida 2 abortion 1 at 40 weeks of gestation, Rh Negative non iso-immunized with no other obstetric or medical comorbidities and uneventful antenatal period was admitted for induction of labor on date. In view of pathological cardiotocography with fetal distress, she was taken up for emergency caesarean section under epidural anaesthesia. After delivery of the baby, she had postpartum haemorrhage (PPH) which was controlled with uterotonics. But immediately after administration of intramuscular carboprost, she had ectopic beats on echocardiogram (ECG) monitor, for which she was evaluated postoperatively and diagnosed as pulmonary artery hypertension. As diagnosis of exclusion, drug induced pulmonary artery hypertension was made. This case report insists on importance of continuous vitals and cardiac monitoring while treating PPH to predict and prevent the major complications associated with medications used for management of PPH.

**Keywords:** PPH, Pulmonary artery hypertension, Carboprost

## INTRODUCTION

Pulmonary artery hypertension is defined as a mean arterial pressure 25 mm Hg or more at rest as diagnosed by right heart catheterisation.[1] Postpartum pulmonary artery hypertension (PPPHTN) is a very rare disorder. Clinically may overlap with pulmonary embolism. Accurate diagnosis is crucial for management and as future pregnancies will be contraindicated.[2] We report a case of drug induced pulmonary arterial hypertension caused by intramuscular carboprost given within a maximal dose limit for management of atonic PPH during a caesarean section.

## CASE REPORT

A 28-year-old with body mass index of 26 kg/m$^2$, ASA II gravida 2 abortion 1 on date with no medical or obstetric comorbidities, in view of low bishop score, was induced with prostaglandins and labour augmented with artificial rupture of membranes and oxytocin. She was taken up for emergency caesarean section for pathological cardiotocography with fetal distress. In active labour, epidural analgesia was administered, which was uneventful. Her preoperative blood investigations and antepartum cardiac evaluation (ECG, ECHO) were all within normal limits.

Before the start of caesarean section, she was conscious, oriented with vitals of pulse rate 86/min, blood pressure 120/70 mmHg and oxygen saturation 99% at room air. Under epidural anaesthesia, surgery was started. After the baby got delivered, active management of third stage of labour was performed. Patient had PPH due to uterine atony, which did not respond to oxytocin infusion and prostaglandin E1. Methylergometrine was deferred as patient was Rh negative. Estimated blood loss amounting to 1.5 litres. As a second line agent for PPH management, prostaglandin F2-alpha-carboprost trometamol 0.25 mg was intramuscularly administered. Her PPH was controlled but we had noticed ectopic beats on ECG monitor. Her pulse rate was 110/min, blood pressure was 110/70 mmHg and oxygen saturation 98% on prophylactic 2 litres oxygen by face mask. She was asymptomatic,

conscious and oriented. Auscultation of the chest revealed normal vesicular breath sounds with no added sounds.

After completion of caesarean section, she was evaluated with ECG and was on continuous cardiac monitoring postoperatively in high dependency unit, which revealed persistent ectopic beats (Figure 1) with ECHO showing pulmonary artery hypertension with right ventricular systolic pressure of 50 mmHg, dilated right atrium and right ventricle and Ejection fraction of 72% (Figure 2).

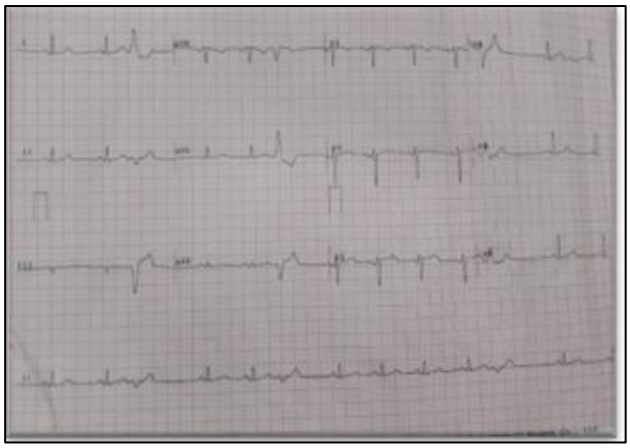

**Figure 1: ECG showing ectopic beats.**

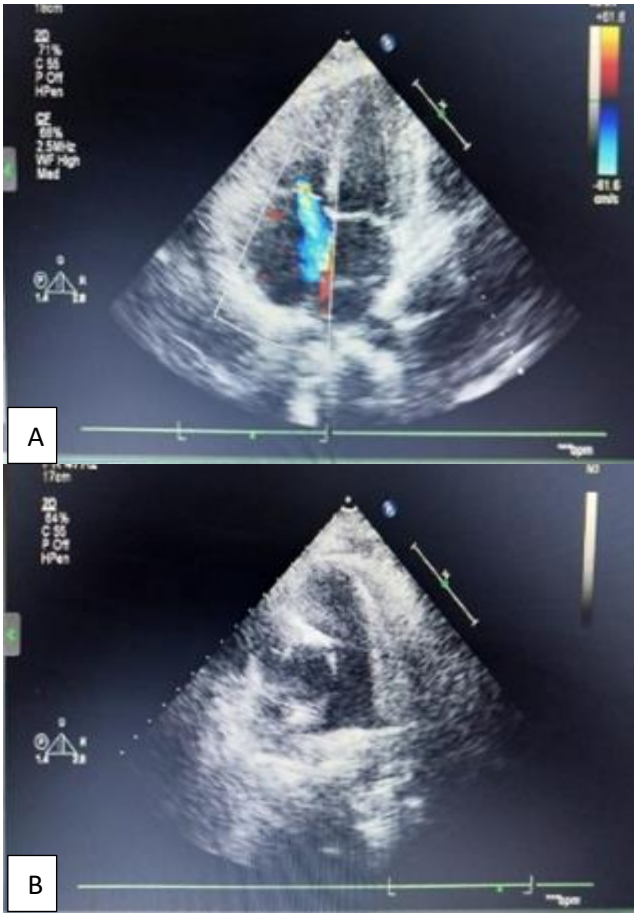


**Figure 2 (A and B): Postoperative echocardiogram.**

She was asymptomatic. Her pulse rate was between 80-90 per min throughout with normal blood pressure recordings. Blood investigations showed normal serum electrolytes levels. Cardiologist opinion was sought and she was started on anticoagulation with prophylactic low molecular weight heparin, suspecting pulmonary thromboembolism. On postoperative day 1, High resolution computed tomography and pulmonary angiogram was done, which showed no evidence of pulmonary thromboembolism. Bilateral lower limb doppler ultrasonogram had been done and deep venous thrombosis was ruled out. Autoimmune workup was normal. Prophylactic anticoagulation has been stopped. Patient was asymptomatic and was under vigilant monitoring for 72 hours and repeat ECG on postoperative day 2 and 3 was normal (Figure 3).

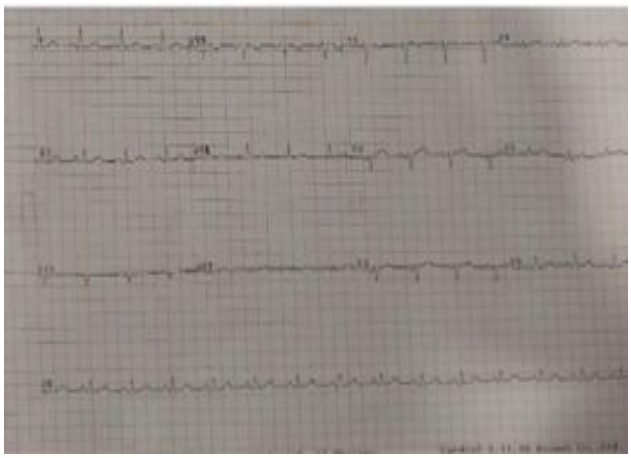

**Figure 3: ECG on postoperative day 3.**

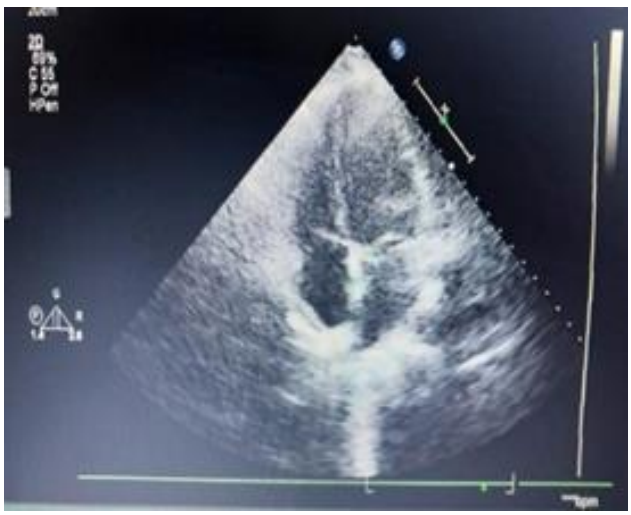

**Figure 4: ECHO-at 1 week postpartum.**

With suspicion of reversible causes of pulmonary artery hypertension, as diagnosis of exclusion, diagnosis of drug induced pulmonary artery hypertension was made, which was confirmed by reverting of ECHO findings in the first postoperative week (Figure 4) and on follow up, patient was asymptomatic and hemodynamically stable on postpartum 6 weeks.

## DISCUSSION

Globally, nearly one quarter of all maternal mortality are associated with PPH. The use of an effective uterotonic for the prevention of PPH during the third stage of labour is recommended for all births. To effectively prevent PPH, only one of the following uterotonics should be used: oxytocin, carbetocin, misoprostol, ergometrine or oxytocin and ergometrine fixed-dose combination. If the bleeding does not respond to first line agents, intramuscular injection of prostaglandin F2-alpha can be used with apparently dramatic effects.[3]

Carboprost (15-methyl PG F2-alpha) is a smooth muscle stimulant. It is an analog of PG F2-alpha with 15-60 minutes to peak plasma concentration and half-life of 8 minutes. It is administered by deep intramuscular injection or direct intramyometrial route at the dose of 250 mcg. 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 which include nausea, vomiting, diarrhoea, bronchospasm, and systemic hypertension.[4]

Secher et al found a 40% increase in cardiac output in pregnant anesthetized patient who was administered PG F2-alpha, showed an increase in pulmonary arterial pressure, doubling of pulmonary vascular resistance and increase in airway resistance.[5]

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. Cardiovascular collapse along with left heart failure has been reported with overdose of intramyometrial carboprost, the possible aetiology being a combination of acute pulmonary hypertension with decreased left ventricular end-diastolic pressure and decreased cardiac output.[6]

In our case, the cause of pulmonary artery hypertension could be either idiopathic or due to the administration of carboprost within a maximal dose limit. Patients with pulmonary arterial hypertension should be monitored in a critical care setting during postpartum period. Fluid status should be monitored, anticoagulation therapy should be initiated.

## CONCLUSION

Prostaglandin analogs whenever used for management of postpartum haemorrhage should be used carefully, along with vigilant cardiac monitoring to prevent major complications.

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