

## Sudden cardiac arrest due to Carboprost administration, Case report

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### Abstract:

The purpose of this report is to share a valuable experience of refractory significant bradycardia that progressed to asystole associated to general anesthesia for Caesarean section.

After activation of emergency Caesarean section, spinal block was induced, however it was inadequate. So, general anesthesia was instituted. We present the case of sudden extreme bradycardia and asystole. Code blue was activated immediately. Chest compression Initiated as high-quality chest compression (at least 100 to 120 per minute), compressing at least 5 cm [2 inches] with each down stroke) and ventilation (two ventilations at 10-12 breath per min). Epinephrine 1 mg was given and second dose was given after 3 minutes and the patient achieved return of spontaneous circulation within three minutes as spontaneous circulation was restored. The mother (patient) was shifted to intensive care unit for further monitoring and care.

The patient has restored spontaneous sinus rhythm without cardioversion. Common comorbidities were found, such as preeclampsia, diabetes mellitus, sepsis and the case occurred during COVID-19 era. The administration of Carboprost induced bradycardia due to effects on chemosensitive ventricular mechanoreceptors and coronary baroreceptors. The vasoactive effects of prostaglandin with reflex bradycardia and hypotension were due to stimulation of cardiac receptors predominantly in the inferoposterior wall of the left ventricle. Vagal bradycardia, as effect of prostaglandins due to activation of cardiac reflex sensitivity to myocardial ischemia, has been described

A comprehensive plan for the handling of cardiovascular complications must be available wherever general anesthesia and Carboprost are administered.

**Keywords:** Cardiac arrest; Carboprost

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### I. Introduction:

Sudden cardiac arrest (SCA) in pregnancy has a high impact on both the mother and the fetus. The incidence of sudden cardiac arrest in pregnant women varies from 1 in 20,000 to 1 in 50,000 ongoing pregnancies.<sup>1</sup> Based on the availability and adequacy of advanced care equipment, management requires rapid multidisciplinary approach.<sup>2</sup>

### II. Case Report:

A 39-year-female, 78 kg, body mass index 30, G8P4+3, admitted for emergency CS because of tender scar of previous CS. Covid-19 test was pending. Obstetric history revealed previous 4 CS, one ectopic pregnancy with blood transfusion, ERPC in AD premature rupture of membrane since two days, Oligohydramnios, she has not been eating or drinking adequately over the past few days. The patient recently was diagnosed with preeclampsia and diabetic type 2, non-compliant with the given medications in other hospital. Given these findings, the patient was informed and consented at operation theater for combined spinal-epidural (CSE). Induction of anesthesia by CSE insertion was smooth.

Surgery started; however, the spinal block was inadequate, so general anesthesia was instituted with Propofol 200 mg, Fentanyl 100 mcg and Rocuronium 50 mg. the Mallampati score was 2 with limited neck

movement, so iGel was inserted; size 4. Pressure-controlled ventilation with Sevoflurane for maintenance in 50% O<sub>2</sub>. Intraoperative course was smooth but uterus remained relaxed. Therefore, the induction of labor with intramuscular Carbocin and two doses of intramural Carboprost (PGF<sub>2a</sub>) was given. Blood loss about 500 ml and urine output was 200 ml. At the end of surgery at emergence from anesthesia, the patient went into significant bradycardia that progressed to asystole. Code blue was announced immediately. Chest compressions started and epinephrine given 1 mg and second dose after 3 minutes and she achieved return of spontaneous circulation (ROSC) within 3 minutes. She was intubated and shifted to ICU. Arterial line inserted in the OT before shifting and monitored at 04:25 am. GCS was 10 prior to intubation (E4, Vt, M5).

Epidural started later in the ICU. Epidural catheter was removed at 11:30 am pre-Enoxaparin dose in the ICU. While in the ICU, CT scan and X-ray were performed right after the end of surgery which did not show any signs of any complications. However, CT angio-chest was negative for pulmonary embolism, but slight basal infiltration was reported. Echocardiography reported good myocardial contractility and was normal, no pericardial fluid, ejection fraction 60 %. The patient hemodynamically stables on sedation Fentanyl 100 mcg hourly and Midazolam 3 mg per hour and expressed good ventilator parameters with Glasgow Coma Scale (GCS) of 15/15. The arterial blood gases (ABGs) were (pH: 7.30, PCO<sub>2</sub>: 36.1, HCO<sub>3</sub>: 18.4). After intensive monitoring for all vital parameters, which were all unremarkable, the patient was extubated and discharged with recommendation for follow-up.

### **III. Discussion:**

The prostaglandins are a group of 20- carbon unsaturated fatty acids synthesized from essential fatty acids. Bergstrom, Eliasson, Von Euler, et al. (1959) who first noted with two prostaglandins called prostaglandins E and F that there is a strong relation between some physiological and pathophysiological cardiovascular states and modifications of the prostaglandin and several prostaglandin purifiers and a synthesis of analogs, inhibitors, and basic antagonists of receptors of prostaglandin.<sup>3,4</sup>

Despite excellent preoperative evaluation and preparation. This case report presented a full stable hemodynamic patient who developed transient bradycardia that ended up with cardiac arrest after administration of two doses of Carboprost for around 30 minutes.

Several studies have discussed the effect of prostaglandin on cardiovascular system. Small doses of prostaglandin F<sub>2</sub> alpha applied to the level of origin of the coronary arteries resulted in marked reductions in heart and blood pressure, although there were no improvements to the ascending aorta in more distal positions following an injection of the same volume. These findings indicate that prostaglandin F<sub>2</sub> alpha induces bradycardia and patients' hypotension by activating receptors in the left heart or by operating on coronary arterial structures.<sup>5</sup> Moreover, since they are influencing chemosensitive ventricular mechanoreceptors and coronary baroreceptors, Hainsworth (1995) identified the potential mechanism for bradycardia and hypotension pathophysiology on bradykinin and prostaglandins.<sup>6</sup> Hintze, Kaley, and Panzenbeck (1984) concluded that the PGF<sub>2</sub> alpha reflex bradycardia is the result of the stimulation of the other prostaglandin synthesis, most probably PGI<sub>2</sub>, at least partly like arachidonic acid.<sup>7</sup>

The authors assumed the potential side effect of Carboprost on cardiovascular system causing bradycardia and a systole.<sup>8,9</sup> Administration of intramuscular intramural Carboprost followed by intramural Carboprost increases the uterus contractility and might lead to cardiac arrest as the result is consistent with the case report by.<sup>10</sup>

### **IV. Conclusion:**

During treatment with Carboprost for uterine induction, an immediate reduction in heart rate can occur. This progresses rapidly after the start of treatment, can be severe and is associated with cardiovascular complications, and is typically reversible after the discontinuity of Carboprost and effective cardiopulmonary resuscitation. Therefore, close monitoring of heart rate and blood pressure before, during and after treatment should be maintained as cardiac arrest should be suspected.

#### **Learning points/ Take home messages**

- As cardiac arrest should be suspected, close monitoring of heart rate and blood pressure should be maintained before, during, and after Carboprost therapy.

#### **Ethics approval and consent to participate**

This report was approved by the Institutional Review Board of Al-Balqa Applied University (Approval number: 8966/2/1/6/18).

#### **Availability of data and materials**

Not applicable

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### **Contributions**

M E and RM performed anesthetic management and wrote the manuscript. MR, MR, and AR helped design the case report and revise the manuscript. The authors read and approved the final manuscript.

### **Consent for publication**

Written informed consent was obtained from the patient for publication of this case.

### **Competing interests**

The authors declare that they have no competing interests.

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### **References**

- [1]. Baghirzada L, Balki M. Maternal cardiac arrest in a tertiary care centre during 1989-2011: a case series. *Can J Anesth Can d'anesthésie*. 2013;60(11):1077–84.
- [2]. Stuart Berger M. Sudden cardiac arrest and death in children. Up to date [Internet]. 2013; Available from: <http://www.ncbi.nlm.nih.gov/pubmed/22451713>
- [3]. Bergstrom S, Eliasson R, Von Euler U, et al. Some biological effects of two crystalline prostaglandin factors. *Acta Physiol Scand*. 1959 Mar 31; 45:133–144
- [4]. Stanley T, Greeley WJ, Leslie JB, et al. Prostaglandins and the cardiovascular system: A review and update. *J Cardiothorac Anesth*. 1987;1(4):331–49.
- [5]. Koss MC, Nakano J. Reflex bradycardia and hypotension produced by prostaglandin F<sub>2</sub>alpha in the cat. *Br J Pharmacol*. 1976;56(3):245.
- [6]. Hainsworth R. Cardiovascular reflexes from ventricular and coronary receptors. In: *Control of the Cardiovascular and Respiratory Systems in Health and Disease*. Springer; 1995. p. 157–74.
- [7]. Hintze TH, Kaley G, Panzenbeck MJ. Mechanisms of reflex bradycardia and hypotension by metabolites of arachidonic acid in the cat. *Br J Pharmacol*. 1984;82(1):117.
- [8]. Lampati L, Colantonio LB, Calderini E. Cardiac arrest during sulprostone administration—a case report. *Acta Anaesthesiol Scand*. 2013;57(3):395–7.
- [9]. Čerkez Habek J, Habek D, Gulin D. Refractory bradycardia—a rare complication of carboprost tromethamine for induction of abortion. *Acta Clin Croat*. 2016;55(2):323–5.
- [10]. Beerendonk CC, Massuger LF, Lucassen AM, et al. Circulatory arrest following sulprostone administration in postpartum hemorrhage. *Ned Tijdschr Geneesk*. 1998;142(4):195–7.

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