CASE REPORT

Stress-Induced Cardiomyopathy Following Cesarean Delivery Under General Anesthesia

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Case Report

A 34-year-old non-hypertensive, non-diabetic woman, second gravida, was admitted with 36 weeks' pregnancy complaining of abdominal pain and vaginal bleeding. Apart from the occasional wheeze which promptly settled with inhaled salbutamol, her past history was insignificant. Obstetric examination revealed abruptio placenta with profuse fresh bleeding. She was taken for emergency cesarean surgery under general anesthesia. Her preoperative pulse was 84 bpm, BP 110/80, and her cardiovascular and respiratory systems were normal. Basic investigations were normal including an electrocardiogram (ECG) (Fig. 1). The surgery was uneventful and a male child weighing 3.1 kg was delivered. Intraoperatively, she was given parenteral Glycopyrrolate, Propafol, Suxamethonium, Fentanyl, Inj Syntocin 10 U infusion, and Inj. Prostodin 125 mcg intramuscularly. 2 h after surgery, she complained of shortness of breath and left mammary pain. She was dyspneic and mildly desaturated (spO₂ 88 %). Clinical examination revealed sinus tachycardia (HR 110 bpm), BP of 100/70 mmHg, faint third heart sound, and basal rales

over the chest. With a provisional diagnosis of left ventricular failure, ECG, X-ray chest, and Echocardiogram were obtained. The ECG (Fig. 2) revealed subtle changes like loss of "r" wave in V2 and the echocardiogram revealed hypokinesia of the mid and apical left ventricle with moderate systolic dysfunction, and her ejection fraction was 38 %. X-ray chest (Fig. 3) showed interstitial edema in the basal zones of both lungs, more on the right. Qualitative troponin T was negative. She was managed with supplemental oxygen, a mild dose of diuretics, dobutamine infusion, and levocarnitine. She responded well, her symptoms abated rapidly, and clinically her lungs became clear and her blood pressure improved. 2 days later, a repeat echocardiogram revealed no wall motion abnormality with normal left ventricular function.

Discussion

Left ventricular failure after cesarean surgery in an apparently healthy woman is rare and could be due to hitherto unrecognized peripartum cardiomyopathy (PPCM) or stress-induced cardiomyopathy (SIC). However, other causes like valvular heart disease, metabolic and endocrine diseases, and rarely acute coronary syndrome should be considered and ruled out. Differentiation of PPCM and stress-induced cardiomyopathy could be difficult in this situation. The following Table lists out the essential differences between PPCM and SIC.

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Characteristic	PPCM	SIC [1]
Onset	Last trimester or immediate postpartum	Anytime
Duration of symptoms	Usually insidious and long	Acute
Recovery	May recover	Usually recover rapidly
Troponin T	Not elevated	Modest elevation
ECHO features	Dilated LV, global hypokinesia	Non-dilated LV, apical ballooning involving mid and apical segments
Stressors	Absent	Usually present

Our patient complained of chest pain and acute shortness of breath. Although troponin was not elevated, ECG showed subtle fresh changes. The Echocardiogram was diagnostic, revealing a wall motion abnormality typical of SIC. There were multiple stressors for our patient, namely, acute blood loss, general anesthesia [2], and prostodin injection. SIC has been reported after cesarean delivery under spinal anesthesia [3], after IV ergonovine injection

soon after cesarean delivery [4], and after intramuscular Prostodin to control postpartum hemorrhage [5].

SIC can be diagnosed with Mayo Clinic criteria [6]:

- Transient hypokinesia, dyskinesia, or akinesia of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always, present.
- The absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
- New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin level.
- The absence of pheochromocytoma or myocarditis.

The pathogenesis of SIC is multifactorial with several theories. Coronary vasospasm, endothelial dysfunction, and catecholamine injury [7] may all contribute.

There is no consensus or guideline for the management of SIC. Usually, the management consists of anti-failure measures; when there is evidence of sympathetic surge, beta blockers will be beneficial. SIC is usually reversible

Fig. 1 Preop ECG—within normal limits

Fig. 2 Sinus rhythm, loss of r and coved ST in lead V2

Fig. 3 Interstitial edema in the basal zones more on right side

without complications. However, rarely, multiple pulmonary emboli, arrhythmias, and very rarely death have been reported.

Conclusions

SIC, although rare following cesarean delivery, requires a high degree of suspicion and prompt recognition for proper management.

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