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Cardiogenic shock in a 29 weeks pregnant woman: A case report

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Abstract

Cardiogenic shock in pregnancy represents a rare clinical condition associated with high mortality. Its diagnosis and management pose a challenge for the medical team due to the hemodynamic changes induced physiologically by pregnancy, which contribute to a potentially rapidly evolving clinical course. In this article, we present the diagnostic and therapeutic approach of a 36-year-old woman pregnant at 29 weeks who presented to the hospital with fever, abdominal and chest pain, and anemia. Despite investigations and multiple specialist consultations, the etiology of symptoms remained elusive. Subsequent rapid hemodynamic deterioration led to severe cardiogenic shock, necessitating mechanical circulatory support and emergency caesarean section. In the postpartum, the patient exhibited rapid recovery and weaning from mechanical circulatory and respiratory support. Diagnostic hypotheses included fulminant post-viral myocarditis and reverse Takotsubo syndrome, but a definitive diagnosis was not achieved. Despite a not certain diagnosis, aggressive therapeutic interventions was successful in the management of this severe condition.

Keywords: Cardiogenic shock; Pregnancy; Caesarean section; Mechanical circulatory support; Hemodynamic instability.

Introduction

Background: Cardiovascular disease remains a leading cause of morbidity and mortality in pregnant women, accounting for over 25% of maternal deaths [1]. Although Peripartum Cardiomyopathy (PPCM) is a common aetiology, various other conditions such as acute myocarditis, Takotsubo syndrome, and acute coronary syndromes can lead to cardiogenic shock during pregnancy [2]. The intricate hemodynamic changes occurring during pregnancy, such as increased blood volume, physiological anemia, reduced vascular resistance and increased cardiac output [1], contribute to make the diagnosis and the therapy of cardiogenic shock a real challenge.

Long-term prognosis and subsequent pregnancies depend on resolution of cardiac dysfunction and need to be tailored on patient's cardiac function [3].

This case report aims to illustrate the clinical presentation and management of cardiogenic shock in pregnancy, a rare but potentially life-threatening condition for both the mother and the fetus, to aid in the identification of symptoms, the timing of delivery, and the therapeutic management both pharmacological and by invasive mechanical hemodynamic support.

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Case presentation

A 36-year-old pregnant woman was transferred after 4 days of hospitalization to our hospital (San Martino University Hospital of Genoa). She was currently at 29 weeks of gestation, with a physiological pregnancy. She was on acetylsalicylic acid 150 mg/day, for a previous preeclampsia and she had a family history of autoimmune diseases. She consulted the emergency service of the hospital for fever and asthenia, and after pneumological evaluation she was discharged with useless empirical antibiotic therapy (amoxicillin with clavulanic acid). Concurrently, her 3-year-old son presented with fever. After 5 days she was hospitalized for diffuse abdominal pain, asthenia, and fever. During the examination, her blood pressure was 90/60 mmHg, her pulse was 102 bpm, her body temperature was 38.3°C. The Electrocardiogram (ECG) revealed sinus tachycardia.

At blood test she had anemia (hemoglobin 8.9 g/dL), neutrophilic leukocytosis, and elevated levels of C-reactive protein.

Due to persistent fever, shaking chills, a new onset of chest pain radiating to the neck, with dry cough, the patient underwent different evaluations (pneumology, cardiology, infectious diseases, neurology, internal medicine) and various diagnostic examinations (abdominal ultrasound, chest X-ray, echocardiography, blood cultures, autoimmune panel, and viral and bacteriological tests). These investigations led to a suspicion of pericarditis/pleuro-pericarditis with normal troponin levels (4 ng/L). All other tests showed normal results. Antibiotic therapy was changed to a third-generation cephalosporin (ceftriaxone) and macrolide (azithromycin). After 4 days of symptoms persistence, she was transferred to our hospital.

Upon admission symptoms continued and new one developed, like dysphagia and fluctuating consciousness levels. Hemoglobin level decreased to 7.7 g/dL. Another pluri-specialistic evaluation was performed (infectious diseases, neurology, internal medicine, and otolaryngology). The etiology of anemia was assessed as secondary to inflammation, and she received a blood transfusion. A first dose of corticosteroids was given to induce fetal lung maturation.

After two days, a sudden worsening of chest pain and new-onset dyspnea developed. The ECG showed a junctional rhythm, with troponin levels initially within the normal range (26 ng/dL; normal range 3-53 ng/dL), that rose to 7060 ng/dL after four hours, along with an increase of pain intensity to a Numerical Rating Scale value of 10/10. Blood pressure was 150/100 mmHg, heart rate 120 bpm. On auscultation, the heart had a gallop rhythm with frequent extrasystoles and bi-basal crackles. Echocardiogram revealed a dilated left ventricle with normal thickness, akinesia of the entire septum and mid-basal portion of the inferior wall, a reduction of left ventricular Ejection Fraction (EF) to 30% and moderate functional mitral regurgitation. The right chambers and ascending aorta were normal, with no evidence of free pericardial effusion. The ECG revealed frequent ventricular extrasystoles, at times organized in pairs, supra ST elevations along with diffuse ST depression. A beta blocker (Labetalol i.v. 30 mg/h) was initiated. After 20 minutes ST-T abnormalities became less pronounced, but echocardiography findings remained unmodified. A second dose of corticosteroids (for fetal lung maturation) was injected and the patient was urgently transferred to the coronary intensive care unit.

Upon arrival, on physical examination, she exhibited dyspnea, tachycardia, cold sweats, and tendencies toward hypotension, along with signs of pulmonary congestion (evidenced by the presence of diffuse B lines on lung ultrasound). The ECG displayed sinus tachycardia with a heart rate of 103 bpm and diffuse nonspecific Ventricular Repolarization (VFR) abnormalities.

After two hours a subsequent echocardiographic assessment revealed a dilated, non-hypertrophic left ventricle with severe systolic dysfunction characterized by Left Ventricular Ejection Fraction (LVEF) 25%, marked diffuse akinesia of the mid-basal segments of all walls, localized apical hypercontractility, and severe functional insufficiency of the mitral valve. Arterial gas analysis indicated levels of lactates at 2.5 mmol/L (normal range 0.5-2 mmol/L).

Based on these assessments, a diagnosis of severe cardiogenic shock was made. Due to progressive hemodynamic instability, an Intra-Aortic Balloon Pump (IABP) was positioned to ensure an optimal flow rate and to conserve the maternal-fetal circulation. Patient's conditions continued to worsen, with the EF decreasing to 15%, persistent elevation of lactates, ongoing tachycardia, hypotension, and clinical signs of hypoperfusion.

Consequently, an emergency Caesarean section (C-section) at 30+1 weeks of gestational age was performed under general anesthesia. The newborn was viable and admitted at the Neonatal Intensive Care Unit (NICU). Mechanical circulatory support was implemented with peripheral veno-arterial Extracorporeal Membrane Oxygenator (ECMO) and vasoactive drugs (noradrenaline and dobutamine) were initiated to support hemodynamic circulation. Anticoagulant therapy with a continuous infusion of unfractionated heparin was started. Considering the hypothesis of an acute myocarditis due the acute onset and the progressive and rapid course of contractile dysfunction of the left ventricle, high-dose steroid therapy with methylprednisolone (125 mg i.v for 5 days) was initiated. Bromocriptine was started (2.5 mg every 12 hours for 7 days, and subsequently 2.5 mg per day for the following 7 days) accordingly to the 2018 European Society of Cardiology Guidelines for the management of cardiovascular diseases during pregnancy. Empirical antibiotic therapy with penicillin and beta-lactamase inhibitor (Piperacillin-Tazobactam), oxazolidinone (Linezolid), and macrolide (Azithromycin) was administered.

The CT scan of the hearth revealed patent coronary arteries without stenosis. There was diffuse and severe global left-ventricular hypo-akinesia, with relative sparing of the apical segments and apex, accompanied by a severe reduction in biventricular systolic function with Left Ventricular Ejection Fraction (LVEF) of 16%.

Active vascular bleeding from the abdominal wall suture, required a surgical revision of the Caesarean section wound.

After 2 additional days, support with vasoactive drugs was reduced, and a cycle of Levosimendan was administered (bolus of 12-24 mcg/kg, followed by a continuous infusion of 0.1 mcg/kg/minute for 24 hours). Following the rapid improvement of cardiac function (LVEF of 40%) after only three days of hemodynamic support, the patient was successfully weaned from ECMA. On the same day, anti-inflammatory therapy with

ibuprofen (400 mg/day) was initiated. Upon echocardiographic re-evaluation, the EF increased to 60%, with mild hypokinesia of the basal septum and hyperkinesia of the apical portions. Consequently, the patient was extubated after 4 days and the IABP was removed after 6 days in the intensive care unit. Blood pressure tended toward hypertension, for which nitroprusside was initiated, subsequently supplemented with nitroglycerin and labetalol.

Upon the return to a normal level of consciousness, the patient exhibited amnesia starting from the day of admission to our hospital.

After only 7 days from admission in the intensive care unit, the patient was discharged and transferred to the cardiac surgery ward. Upon admission, the patient started a therapy with colchicine (0.5 mg/day) to improve the inflammatory response; she also underwent a Magnetic Resonance Imaging (MRI) which revealed systolic function at the lower limits, particularly in correspondence with the hypokinetic segments, notably the basal and mid-ventricular anterior and inferior interventricular septum with LVEF of 53%. The MRI findings, suggestive of a possible sub-acute inflammatory genesis, was compatible with the previous hypothesis of myocarditis but could not exclude the hypothesis of reverse Tako-tsubo syndrome in the process of resolution.

Antihypertensive therapy was modified to include a beta-blocker plus an Angiotensin-Converting Enzyme (ACE) inhibitor and transdermal alpha-agonist (clonidine). After one week of hospitalization in the cardiac surgery ward, blood chemistry tests showed increase platelet concentration (760 10^9/L), and an antiaggregant (cardioaspirin) was initiated.

The patient was discharged from the cardiac surgery ward after two weeks of hospitalization with an echocardiogram indicating a left ventricle no longer dilated and with normal wall thickness. There was only mild akinesia of the mid-basal segments of all walls with preserved global systolic function (55%). Minimal mitral and tricuspid valve regurgitation was noted.

All autoimmunity tests conducted during hospitalization yielded negative results.

When discharged, the patient's general conditions were good; she was euphonic, apyretic, and her blood pressure was under control. Fifteen days after discharge, Ibuprofen 400 mg/day was deescalated to Ibuprofen 200 mg/day, and after another 15 days stopped without an increase in markers of inflammation.

The cardiac MRI performed during the follow-up visit after discharge showed improvement in the global systolic function of the left ventricle (LVEF = 58% vs. 53% from the previous assessment two months earlier), with no segmental alterations in kinetics. There was a reduction in wall thickness and myocardial mass related to the resolution of edema, along with normalization of T1 and T2 mapping values of the overall myocardium. Presence of thin and faint sub-epicardial/intramural late gadolinium enhancement (non-ischemic pattern) at the mid-basal level, as described above, was suggestive of the previous processes. No pleuro-pericardial effusion was noted.

The patient is now on antihypertensive therapy with a beta blocker (Bisoprolol 2.5 mg/twice a day) and an ACE-inhibitor (Ramipril 2.5 mg/day), with optimal blood pressure control.

Discussion and conclusion

The case describes the onset progression and resolution of a severe cardiogenic shock during pregnancy. A definitive diagnosis of the causes could not be reached, as a cardiac biopsy was not performed during the initial diagnostic assessment, due to the patient's precarious clinical conditions. Nevertheless, the primary diagnostic hypotheses include fulminant post-viral myocarditis, supported by the clinical context of viral infection in the days preceding admission and the rapid decline in the patient's clinical condition [4]. Contrary to this diagnostic hypothesis is the extremely rapid recovery time following the initiation of mechanical circulatory support and the swift weaning from mechanical cardiovascular support [5]. The second proposed diagnostic hypothesis is reverse Takotsubo syndrome, an acute but often reversible Left Ventricular (LV) dysfunction mainly triggered by emotional or physical stress. This variant is characterised by basal akinesis/hypokinesis associated with apical hyperkinesis [6] which resolves spontaneously. These characteristics were consistent with the ultrasound picture presented by our patient; however, this diagnosis was considered less likely due to the clinical picture preceding the onset of cardiogenic shock. Nevertheless, it was not possible to rule it out.

PPCM was considered as a possible differential diagnosis. However, PPCM is a diagnosis of exclusion. Due to the ultrasound suspicion of pericarditis preceding cardiogenic shock we were able to rule it out.

During cardiogenic shock there is a profound depression of myocardial contractility: this can lead to reduced cardiac output which causes low blood pressure. The reduction in blood pressure worsens the coronary ischemia, therefore an additional reduction in myocardial contractility occurs [7].

This paradigm also includes compensatory responses. The first phase of compensatory responses to the reduction in ventricular contraction attempts to maintain arterial pressure. The process begins when baroreceptors in the great vessels recognize the blood pressure reduction, activating efferent autonomic nerve fibers directed to cardiac and vascular structures and also stimulating catecholamines release from the adrenal gland. As a result, heart rate and - to a lesser extent - contractility, increase. The catecholamine-induced vasoconstriction leads to increased total peripheral resistance.

As a consequence of this vasoconstriction, the blood shifts from splanchnic circulation (high capacitance reservoir) to low capacitance vessels, leading to an increase in circulating blood volume which raises central venous and pulmonary venous pressures. Cardiogenic shock develops when cardiac output and arterial pressure decrease, leading to systemic hypoperfusion, vasoconstriction and end-organ damage [1].

Cardiogenic shock needs intensive use of medical therapy and, if insufficient, mechanical circulatory support. Before delivery medical therapy consists of standard guideline-directed medical therapy for heart failure avoiding medications that are contraindicated in pregnancy.

Beta blockers, hydralazine, loop diuretics, and in some cases, thiazide diuretics, may be considered. In particular, beta-1 selective agents, such as metoprolol, prevent ventricular arrhythmias and sudden death and prevent remodeling after cardiac injury. Beta-2 receptor blockade should be avoided as it may have an anti-tocolytic effect. Vasodilators, such as a hydralazine-nitrate combination, are safe in pregnancy and should be

considered when a patient is fluid overloaded. Thiazide diuretics and mineralocorticoids may be utilized to treat fluid congestion [3]. Digoxine may safely be used for symptomatic heart failure. Nytroglicerine is appropriate vasodilator to use.In patients presenting with cardiogenic shock the use of inotropes should be considered for clinical stabilization; they are considered safe, even if in a retrospective study of 27 patients it was observed that patient treated with dobutamine had irreversible heart failure requiring Left Ventricular Assist Devices (LVADs) or heart transplantation [1].

PPCM experimental treatment includes bromocriptine, a dopamine antagonist and a suppressor of prolactin release. Its use together with standard HF therapy has been associated with greater improvement in LV ejection fraction in comparison to who was treated only with standard therapy [2]. Before starting the therapy with bromocriptine we should inform the woman about the impossibility of breastfeeding due to this therapy. There is limited data on use of bromocriptine in case of cardiogenic shock.

LV dysfunction is associated with an increased risk of thrombosis and death, and these risks are even higher in the peripartum period due to the woman's hypercoagulable state. Therefore, anticoagulation may play a role in treatment. As warfarin is not recommended in pregnancy, heparin and unfractionated heparin are first-line treatment [3]. Arrigo et al. recently proposed a label to group together essential therapies in PPMC, "Welcome on BOARD"- Bromocriptine, Oral heart failure therapies, Anticoagulants, vasorelaxing agents, and Diuretics [8]. Mechanical support of the circulation can be used in cases of refractory shock unresponsive to medical therapy and devices should be tailored to underlying cardiac pathophysiology and degree of biventricular involvement. An analysis by Banayan et al., of over 53 million peripartum hospitalizations in the USA between 2002 and 2013, demonstrated that women who received early mechanical circulatory support, defined as 6 days or less from the onset of cardiogenic shock, had a lower mortality rate than women who received delayed support (18% vs 38% respectively) [9].

Given the higher rates of improved systolic function in patients with PPCM compared with other cardiomyopathies, temporary mechanical circulatory support is often employed as a "bridge to recovery" or "bridge to durable mechanical circulatory support" [10]. Extreme action is represented by cardiac transplantation [10].

There are different forms of mechanical support, as stated by Sharma et al in 2019, the IABP provides the least amount of hemodynamic support and does not support oxygenation, as previously illustrated. In our patient this support didn't provide any improvement. If additional support is required (such as biventricular support, arrhythmia or hypoxia) ECMO provides both oxygenation and maximum circulatory support by providing a cardiopulmonary bypass [11]. The procedure requires anticoagulation monitoring for the risk of hemorrhage. The most common complication (intracranial and multiple site bleeding were associated with higher mortality rates) [1], and it elevates the levels of prolactin, with the possible need of its pharmacological suppression (bromocriptine up to 10 mg twice daily) [1].

In cases of persistently severe cardiac failure despite optimal medical and device therapy, cardiac transplantation should be considered [10]. There is limited data in the literature regarding the timing and method of delivery: There is no published data to guide decisions.

Pregnant patients who are clinically decompensating due to heart failure should be considered for delivery; management, timing and mode of delivery should be decided by a team of cardiologists and obstetricians [12]. Regarding the time, early delivery needs to be weighed against the risks to the newborn and should generally be reserved for cases of impending peril to mother or fetus.

Considering the mode, in absence of obstetric indications, vaginal delivery could be possible in stable heart failure, but if the patient is hemodynamically unstable an urgent Caesarean section is the best option, even if it is associated with higher incidence of hemorrhage, infection and thromboembolic complications [13]. ECMO has been successfully used also during delivery [14]. Labor induction with prostaglandin E analogues can cause systemic vasodilation and consequent tachycardia. Spinal-epidural anaesthesia should be preferred over general anesthesia to avoid systemic vasodilation and the myocardial depression by general anesthetics [1]. Right after delivery the management of fluid overload is crucial to reduce preload, improve pulmonary congestion, and reduce peripheral edema. Loop diuretics are the most used ones. However, aggressive fluid depletion in HF patients may result in kidney injury and inadequate perfusion, in fact the fluid status requires careful monitoring, especially if the delivery was complicated by bleeding. Once patients are stable, they should be started on standard heart failure therapy [3]. Counselling regarding subsequent pregnancies should be tailored on each patient because the safety of a subsequent pregnancy depends on the functional status of the heart. Women who recover with a LVEF > 50% have lower risk of complications during a subsequent pregnancy, but persist the risk of recurrent heart failure [2,15].

In conclusion, cardiogenic shock during pregnancy is a rare but life-threatening condition with limited literature background. In our experience what was crucial to reach a favorable outcome for both the patient and the newborn was not to underestimate aspecific symptoms arising during pregnancy and a multidisciplinary approach in patient management.

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