

Case Report

Septicemia with Endotoxic Shock following Fetoreduction in a Triplet Pregnancy : A rare case of Multiorgan Failure and Successful Recovery

SHIFFIN RIJVI¹, SHREESHA NIRLA², MASUDA BEGUM³, NIAMUL KABIR KHAN SIDDIQUE⁴, MOHAMMAD ZAKIR HOSSAIN⁵

Abstract

Background: Sepsis during pregnancy, Particularly in the context of fetal demise or invasive obstetric procedure, can progress rapidly to endotoxic shock and multi-organ failure, posing a significant threat to both maternal and fetal outcomes. This report presents a challenging case of a 27-years-old woman with a triplet pregnancy, conceived through in-vitro fertilization, who developed septicemia with endotoxic shock, disseminated intravascular coagulation (DIC), and multi-organ failure following fetal demise and fetoreduction. The case emphasizes the importance of a multidisciplinary approach in the critical care management of obstetric emergencies to maximize the maternal and fetal outcomes.

Conclusion: This case underscores how sepsis and endotoxic shock should be considered as a top differential diagnosis in obstetric patients who come with circulatory collapse, fever, and organ failure. The mother recovered completely despite the initial life-threatening difficulties due to prompt identification of the infection (fetoreduction-associated septic focus), aggressive hemodynamic stabilization, and coordinated intensive care.

Keywords: Septicemia, Endotoxic Shock, Disseminated Intravascular Coagulation (DIC), Multi-Organ Failure, Triplet Pregnancy, Fetoreduction, Obstetric Sepsis

Introduction:

Maternal sepsis remains a significant cause of morbidity and mortality worldwide, accounting for approximately 10% of maternal death globally¹ The pathophysiology of Peripartum Cardiomyopathy (PPCM) involves a combination of hormonal and vascular factors, which may include elevated levels of prolactin, inflammatory markers, and genetic predispositions³. When left untreated, PPCM can lead

to severe complications, including septicemia, disseminated intravascular coagulation (DIC), and multi-organ failure. DIC, often triggered by severe infection or systemic inflammatory responses, is associated with a high risk of mortality, as it causes the uncontrolled activation of the coagulation system, leading to widespread clot formation and organ dysfunction². In cases of severe septicemia, the body's inflammatory response can exacerbate

1. Associate professor, Department of Obstetrics and Gynaecology, Anwer Khan Modern Medical College, Dhaka; Phone no: +8801736416335; E-mail: drshiffinrijvi74@gmail.com
2. Intern Doctor, Anwer Khan Modern Medical College, Dhaka; Phone no: +8801601211678; E-mail: shreesha.852.niraula@gmail.com
3. Professor, Department of Obstetrics and Gynaecology, Anwer Khan Modern Medical College, Dhaka; Phone no: +8801886576820; Email: drmasuda61@gmail.com
4. Assistant Professor and Consultant, Department of ICU, Anwer Khan Modern Medical College, Dhaka; Phone no: +8801720691806, +8801913464115; Email: drnksiddique@gmail.com
5. Associate professor, Department of Cardiology, Anwer Khan Modern Medical College, Dhaka; Phone no: +8801711709964; Email: jakir_gb@gmail.com

Address of Correspondence: Dr. Shiffin Rijvi, Associate Professor, Anwer Khan Modern Medical College, Dhaka, Bangladesh. Email: drshiffinrijvi74@gmail.com, Cell: +8801736416335

myocardial dysfunction, leading to heart failure [2]. Therefore, prompt diagnosis and a multidisciplinary treatment approach are crucial in managing septicemia induced Endotoxic Shock and preventing further complications, such as respiratory failure, renal impairment, and liver dysfunction [3].

Case Report

A 27-years-old unconscious primigravida from a rural area with hypothyroidism, presented to our emergency department (ED) with profuse per vaginal bleeding for 3 hours and lower abdominal pain for 1 day at 25+ weeks of pregnancy. She had undergone in-vitro fertilization and triplet pregnancy was conceived. At 12 weeks of gestation, selective fetal reduction was done by injecting a small amount of KCL directly into the fetal heart. Because of uterine contractions, she received intravenous tocolytic therapy and progesterone vaginally 400 mg twice daily for fifteen days. During examination in the ED, the patient was noted to be afebrile, blood pressure was unrecordable, pulse rate was 162 beats/min, oxygen saturation was 98% in 10-L oxygen, random blood sugar was 5.9 mmol/l, and arterial oxygen partial pressure (PaO₂) was 187.9 mm Hg on arterial blood gas analysis (ABG). She was started on an infusion plasmasol in the ED and shifted to the intensive care unit (ICU).

In the ICU, oxygen (40%) was administered via a simple face mask at a flow rate of 5L/min, urinary catheterization was done revealing oliguria, a Nasogastric tube, and a central venous (CV) line was inserted. Moreover, Noradrenaline (15mg), Vasopressin (3 ml), and Sodium bicarbonate (75 mg) were administered intravenously. However, the patient's condition progressively deteriorated. She was intubated and mechanical ventilation was initiated after refractory respiratory failure that could not be managed via noninvasive ventilator support. Her SpO₂ increased to 98% after intubation under the high positive end-expiratory pressure and a fraction of inspired oxygen (FiO₂). The complete blood count showed anemia (hemoglobin: 9.8gm/dl), leukocytosis ($25.21 \times 10^9/L$), and thrombocytopenia ($79 \times 10^9/L$). The blood chemistry revealed liver enzymes (alanine aminotransferase: 221 IU/L), azotemia (blood urea nitrogen: 59 mg/dL, creatinine: 2.6 mg/dL), and an increased level of inflammatory markers (C-reactive protein: 116 mm/hour, erythrocyte sedimentation rate: 46 mm/hour, procalcitonin: 50 ng/ml). The coagulation profiles showed a prolongation of activated partial

thrombin time (36 seconds) and D-dimer (10.0 mg/L). Therefore, we concluded that the patient had Acute kidney injury, septic shock, and disseminated intravascular coagulation (DIC).

Moreover, laboratory tests indicated pro-brain natriuretic peptide (pro-BNP) levels of 27066 pg/mL (normal range <125 pg/mL), whereas echocardiography indicated impairment of left ventricular systolic function, with an estimated left ventricular ejection fraction (LVEF) of 35%, grade 2 tricuspid regurgitation and mild pulmonary hypertension. The multidisciplinary team held urgent deliberations. Based on Medical history, absence of characteristic features, and particular laboratory findings, pregnancy-induced hypertension, long-standing cardiac disorders, Takotsubo cardiomyopathy, and pulmonary embolism were all ruled out. She was diagnosed with PPCM clinically due to her rapidly deteriorating heart failure. Per vaginal examination (P/V/E) revealed the cervix was fully dilated and there was excessive per vaginal bleeding. Two babies were spontaneously delivered vaginally 24 hours after admission. Placental cord membranes were delivered completely. Both babies were immediately taken to the NICU and confirmed dead. The uterus was well contracted and postpartum bleeding was adequately controlled. But the urine output was scanty (10ml) and reddish in color.

Intravenous antibiotics, diuretics, and inotropic agents, including dopamine and noradrenaline were infused continuously. She received three units of whole blood, 100 ml of 25% human albumin and 1 unit of aphaeretic platelet concentrate in ICU. On the 4th postnatal day, the patient's NG tube was removed, oral feeding commenced, they were extubated, and maintained 98% oxygen saturation on 10 L oxygen via facemask, indicating significant clinical improvement. On the seventh postnatal day, the patient's vital signs were stabilized, the subjective symptoms of the patient were improved, respiratory failure markedly improved and her renal function gradually improved with creatinine levels on a downward trend. Other laboratory reports also dramatically improved, and the echocardiogram showed recovery of LV systolic function with an ejection fraction of 55%. She was then transferred to the general female ward for observation and to complete the dose of antibiotics.

On the 15th postnatal day, the CV line was omitted and the patient was discharged from the hospital with the instruction to continue levothyroxine.

On the subsequent follow-up visit at OPD, she appeared to be in good condition.

Discussion

Pregnancy-related sepsis is a life-threatening illness that needs to be identified and treated quickly. In this instance, intrauterine infection after fetoreduction was the most likely cause. Septicemia with endotoxic shock and DIC were indicated by the clinical picture of hypotension, increased inflammatory markers, thrombocytopenia, and coagulopathy [4].

In this case, the patient presented with a severely reduced EF of 35%, suggesting advanced myocardial dysfunction. PPCM typically occurs in the final month of pregnancy or the early postpartum period, with the pathogenesis being largely idiopathic but believed to be influenced by several factors. Hormonal changes, particularly elevated prolactin levels, are thought to be a key contributor, as the 16 kDa prolactin fragment has been implicated in endothelial injury and myocardial dysfunction through oxidative stress [6]. Additionally, inflammatory mediators, such as tumor necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6), are elevated during pregnancy and contributed to myocardial inflammation, leading to impaired cardiac contractility and remodeling [7]. Genetic predispositions, especially mutations in genes encoding sarcomeric proteins like titin, have also been linked to PPCM in certain patients, highlighting a potential hereditary component [5].

Disseminated intravascular coagulation (DIC) is characterized by a widespread activation of the coagulation system, and this results in thrombotic complications that are due to intravascular formation of fibrin; there can be diffuse hemorrhage as well that is due to the consumption of platelets and coagulation factors [8]. The diagnosis of DIC in this case was supported by thrombocytopenia, prolonged coagulation times, and increased D-dimer. Platelet and fresh frozen plasma transfusions, careful coagulation parameter monitoring, and treatment of the underlying infection were all necessary.

A major factor in this case was septicemia, which was brought on by a serious systemic infection and further complicated the patient's clinical trajectory. Multiorgan failure is the result of endothelial dysfunction, vasodilation, and poor perfusion brought on by the systemic inflammatory response to infection. Elevated markers such as CRP and procalcitonin

indicated severe sepsis, while leukocytosis and metabolic derangements reflected the body's inflammatory state [9]. Sepsis-associated myocardial dysfunction, a well-documented phenomenon, exacerbated PPCM by increasing myocardial oxygen demand and reducing cardiac output. The integration of vasopressors, broad-spectrum antibiotics, and aggressive fluid resuscitation was pivotal in stabilizing the patient [1].

This patient's AKI was probably caused by a confluence of DIC-induced microvascular thrombosis and septic shock. Oliguria and increased creatinine levels are signs of further impaired renal function caused by renal hypoperfusion and inflammation. Early detection is essential in preventing long-term renal damage from acute kidney injury (AKI), a frequent consequence in critically unwell individuals with sepsis. Supportive interventions including fluid therapy and careful monitoring of renal function helped the patient recover gradually in this case, highlighting the significance of prompt intervention. The recovery of this patient emphasizes how important a multidisciplinary team is. To deliver comprehensive care, obstetricians, cardiologists, intensivists, nephrologists, and hematologists worked together. Stabilizing cardiac and respiratory functions required early intubation, mechanical ventilation, and inotropic support. Antibiotics and blood transfusions assisted in the treatment of DIC and septicemia, and ongoing observation made sure that organ function gradually improved.

By the seventh postpartum day, her cardiac function dramatically improved (LVEF 55%), indicating that PPCM may be reversible with prompt treatment. Because of the high likelihood of PPCM recurrence, long-term follow-up is essential, including counseling for future pregnancies.

Management of such critically ill obstetric patients requires a multidisciplinary approach involving obstetricians, intensivists, nephrologists, and cardiologists. Early initiation of antibiotics, mechanical ventilation, hemodynamic support, and transfusions contributed to the favorable outcome.

Conclusion

This case demonstrates the quick progression of maternal sepsis into endotoxic shock and multi-organ failure, especially when linked to invasive procedures such as fetoreduction. PPCM may be mistaken with

reversible myocardial dysfunction, which is sepsis-induced cardiomyopathy. To improve results, early detection, vigorous resuscitation, source control, and interdisciplinary cooperation are essential.

Conflict of interest : No

Acknowledgement: we are thankful to the patient for their consent to report the case. Also, we thank the staffs who helped us to manage the patients during the emergencies.

Patients consent:

We have obtained informed written consent for the publication of the details relating to the patient in this report. All possible steps have been taken to safeguard the identity of the patient.

Reference

1. Bonet M, Nogueira Pileggi V, Rijken MJ, et al. Towards a consensus definition of maternal sepsis: results of a systematic review and expert consultation. *Reprod Health*. 2017;14(1):67. doi:10.1186/s12978-017-0321-6. PMID: 28558733
2. Arany Z. Peripartum Cardiomyopathy. *N Engl J Med*. 2024 Jan 11;390(2):154-164. doi: 10.1056/NEJMra2306667. PMID: 38197818.
3. Pyatt JR, Dubey G. Peripartum cardiomyopathy: current understanding, comprehensive management review and new developments. *Postgrad Med J*. 2011 Jan;87(1023):34-9. doi: 10.1136/pgmj.2009.096594. Epub 2010 Oct 10. PMID: 20935342.
4. Cohen J. The immunopathogenesis of sepsis. *Nature*. 2002;420(6917):885–891. doi:10.1038/nature01326. PMID: 12490963
5. Bhattacharyya A, Basra SS, Sen P, Kar B. Peripartum cardiomyopathy: a review. *Tex Heart Inst J*. 2012;39(1):8-16. PMID: 22412221; PMCID: PMC3298938.
6. Leonard RB, Schwartz E, Allen DA, Alson RL. Peripartum cardiomyopathy: a case report. *J Emerg Med*. 1992 Mar-Apr;10(2):157-61. doi: 10.1016/0736-4679(92)90210-k. PMID: 1607622.
7. Dr. Sara Guleria, Dr. Suman Thakur and Dr. Kushla Pathania. Peripartum cardiomyopathy: A case report. *Int. J. Gynaecology Sci*. 2023;5(2):07-08
8. Kim KH, Jeong MH, Chung IJ, Cho JG, Song TB, Park JC, Kang JC. A case of septic shock and disseminated intravascular coagulation complicated by acute myocardial infarction following amniocentesis. *Korean J Intern Med*. 2005 Dec;20(4):325-9. doi: 10.3904/kjim.2005.20.4.325. PMID: 16491831; PMCID: PMC3891079.
9. Chirillo F, Baritussio A, Cucchini U, Toniolli E, Polo A, Iavernaro A. Challenges in the diagnosis of peripartum cardiomyopathy: a case series. *Eur Heart J Case Rep*. 2021 Feb 16;5(2):ytab001. doi: 10.1093/ehjcr/ytab001. PMID: 33738415; PMCID: PMC7954255.
10. Wang M. Peripartum cardiomyopathy: case reports. *Perm J*. 2009 Fall;13(4):42-5. doi: 10.7812/TPP/08-079. PMID: 20740101; PMCID: PMC2911830.