

Reversible Sepsis-Induced Myocardial Dysfunction in Term Pregnancy: A Case Report of a Rare Medical Condition

Bedih Balkan ¹, Nazli Gonulduru ¹, Ebru Kaya ¹, Gulseren Yilmaz ²

Review began 03/30/2026

Review ended 04/08/2026

Published 04/12/2026

© Copyright 2026

Balkan et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.106877

1. Intensive Care Unit, Kanuni Sultan Suleyman Training and Research Hospital, University of Health Sciences, Istanbul, TUR 2. Anesthesiology and Reanimation, Kanuni Sultan Suleyman Training and Research Hospital, University of Health Sciences, Istanbul, TUR

Corresponding author: Nazli Gonulduru, nzlayan@gmail.com

Abstract

Sepsis-induced myocardial dysfunction (SIMD) is a reversible heart failure that occurs during sepsis or septic shock. Although rare in pregnancy, its clinical presentation can be very similar to other peripartum heart complications, making diagnosis difficult. We report a previously healthy 28-year-old primigravida woman at 38 weeks of gestation who presented with an acute onset of dyspnea (New York Heart Association Class IV). The patient had developed upper respiratory tract symptoms approximately seven days prior to admission, including fever and malaise, which progressively worsened. A nonspecific, blanching maculopapular rash on the trunk and extremities was noted on physical examination. An emergency cesarean section was performed due to clinical deterioration of the mother and fetal distress. Postnatal echocardiography showed a left ventricular ejection fraction (LVEF) of 38% and global hypokinesia, but no apical ballooning or regional wall motion abnormalities. Radiological evidence of pneumonia, in combination with elevated inflammatory markers (high-sensitivity C-reactive protein, procalcitonin, leukocytosis) and increased cardiac biomarkers (troponin, N-terminal pro-B-type natriuretic peptide), supported the diagnosis of SIMD. Targeted antibiotic therapy, cautious fluid management, and low-dose norepinephrine were administered. Rapid improvement in heart function was observed after infection control, with LVEF returning to 55% before discharge. In cases of acute heart failure associated with sepsis during pregnancy, SIMD should always be considered in the differential diagnosis. Early detection, appropriate infection treatment, and prompt hemodynamic support can greatly improve maternal and neonatal outcomes.

Categories: Other, Obstetrics/Gynecology, Cardiology

Keywords: acute sepsis, heart disease in pregnancy, reversible heart failure, sepsis and septic shock, sepsis-induced myocardial dysfunction

Introduction

Pregnancy is a complex physiological process that causes extensive cardiovascular, hormonal, and immunological adaptations. These changes are essential for fetoplacental circulation and preparing the body for delivery; however, modifications in the maternal immune response may increase susceptibility to infections [1-3]. Increased immune tolerance, higher cardiac preload and stroke volume, and decreased systemic vascular resistance may predispose pregnant women to rapid progression and severe symptoms of sepsis [2,3]. Cardiac dysfunction during sepsis is a key factor in determining prognosis. Sepsis-induced myocardial dysfunction (SIMD) is characterized by temporary but significant myocardial contractile depression and is linked with high mortality [4,5]. Proposed mechanisms include cytokine-driven inflammation, mitochondrial dysfunction, altered calcium regulation, and nitric oxide-induced myocardial depression [5,6]. Recent studies have shown that approximately 40%-50% of patients with severe sepsis or septic shock develop myocardial dysfunction. Early recognition and prompt initiation of hemodynamic support significantly reduce mortality [4-6]. Here, we report a case of reversible myocardial dysfunction secondary to pneumonia-induced sepsis in a term pregnancy. This case emphasizes the importance of distinguishing SIMD from peripartum cardiomyopathy (PPCM) and Takotsubo cardiomyopathy (TCM), which share overlapping clinical features but require different management approaches. Accurate differentiation is crucial for optimizing treatment strategies and enhancing maternal prognosis.

Case Presentation

A 28-year-old nulliparous woman at 38 weeks of gestation arrived at the emergency department with sudden and rapidly worsening shortness of breath, orthopnea, and malaise. She had no previous history of heart disease, high blood pressure, or diabetes. Her pregnancy had been uneventful until one week prior to admission, when she developed symptoms of an upper respiratory infection.

On admission, vital signs showed significant hemodynamic instability: blood pressure 80/60 mmHg, heart rate 130 bpm, respiratory rate 28 breaths/minute, body temperature 38.2°C, and oxygen saturation 86% on

How to cite this article

Balkan B, Gonulduru N, Kaya E, et al. (April 12, 2026) Reversible Sepsis-Induced Myocardial Dysfunction in Term Pregnancy: A Case Report of a Rare Medical Condition. Cureus 18(4): e106877. DOI 10.7759/cureus.106877

room air, and physical examination revealed jugular venous distention, bilateral basal crackles, and a nonspecific blanching maculopapular rash on the trunk and extremities; in the presence of markedly elevated inflammatory markers (C-reactive protein (CRP), procalcitonin, and leukocytosis), these findings were suggestive of a systemic inflammatory response consistent with bacterial infection.

Due to worsening respiratory distress and fetal bradycardia, an emergency cesarean section was performed under general anesthesia, resulting in the delivery of a healthy neonate. In the immediate postoperative period, the patient’s condition further deteriorated, and arterial blood gas analysis revealed a pH of 7.30, a partial pressure of carbon dioxide of 30 mmHg, and an arterial oxygen partial pressure (PaO2) of 88 mmHg, corresponding to a PaO2/fractional inspired oxygen ratio of 220, consistent with acute lung injury. This clinical deterioration necessitated endotracheal intubation and admission to the intensive care unit (ICU) for advanced hemodynamic and respiratory support.

Laboratory results showed severe systemic inflammation: CRP was 280 mg/L, procalcitonin was 15 ng/mL, and leukocytes were 18,000/μL. Cardiac biomarkers were elevated, with troponin I at 0.8 ng/mL and N-terminal pro-B-type natriuretic peptide at 600 pg/mL. Serum lactate was 4.8 mmol/L, indicating tissue hypoperfusion, and creatinine was mildly elevated at 1.5 mg/dL. Arterial blood gas analysis revealed metabolic acidosis with a pH of 7.21. Blood and sputum cultures grew Klebsiella pneumoniae. The Pneumonia Severity Index was calculated as 108 (Class IV), indicating high-risk pneumonia. The laboratory parameters at different time points are summarized in Table 1.

Laboratory parameters	Reference range	Day 1	Day 3	Day 12
C-reactive protein	<5 mg/L	280 mg/L	72 mg/L	3.8 mg/L
Procalcitonin	<0.5 ng/mL	15 ng/mL	4 ng/mL	0.21 ng/mL
Leukocytes	3.8-10 × 10³/μL	18 × 10³/μL	12 × 10³/μL	6.7 × 10³/μL
Troponin I	<0.014 ng/mL	0.8 ng/mL	0.31 ng/mL	0.007 ng/mL
NT-proBNP	<125 pg/mL	600 pg/mL	211 pg/mL	80 pg/mL
Lactate	0.5-2 mmol/L	4.8 mmol/L	0.8 mmol/L	0.5 mmol/L
Creatinine	0.50-0.90 mg/dL	1.5 mg/dL	1.2 mg/dL	0.59 mg/dL
pH	7.35-7.45	7.21	7.31	7.40

TABLE 1: Laboratory parameters at different time points during the clinical course

NT-proBNP: N-terminal pro-B-type natriuretic peptide

Chest radiography revealed consolidation in the right lower lobe, indicating pneumonia, as shown in Figure 1. Transthoracic echocardiography (TTE) showed global hypokinesia with a left ventricular ejection fraction (LVEF) of 38% (Simpson’s biplane method), as shown in Figure 2. Although pericardial thickening was suspected in a single echocardiographic view, it was not consistently observed throughout the full examination. The absence of apical ballooning or regional wall motion abnormalities ruled out TCM.

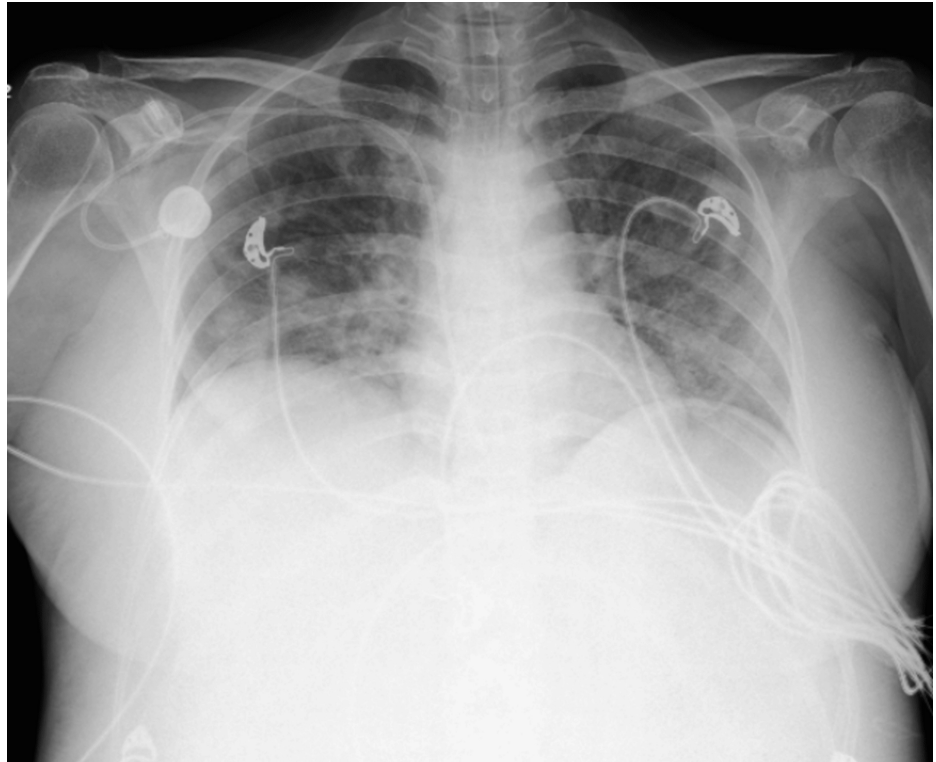


FIGURE 1: Chest X-ray demonstrating right lower lobe consolidation

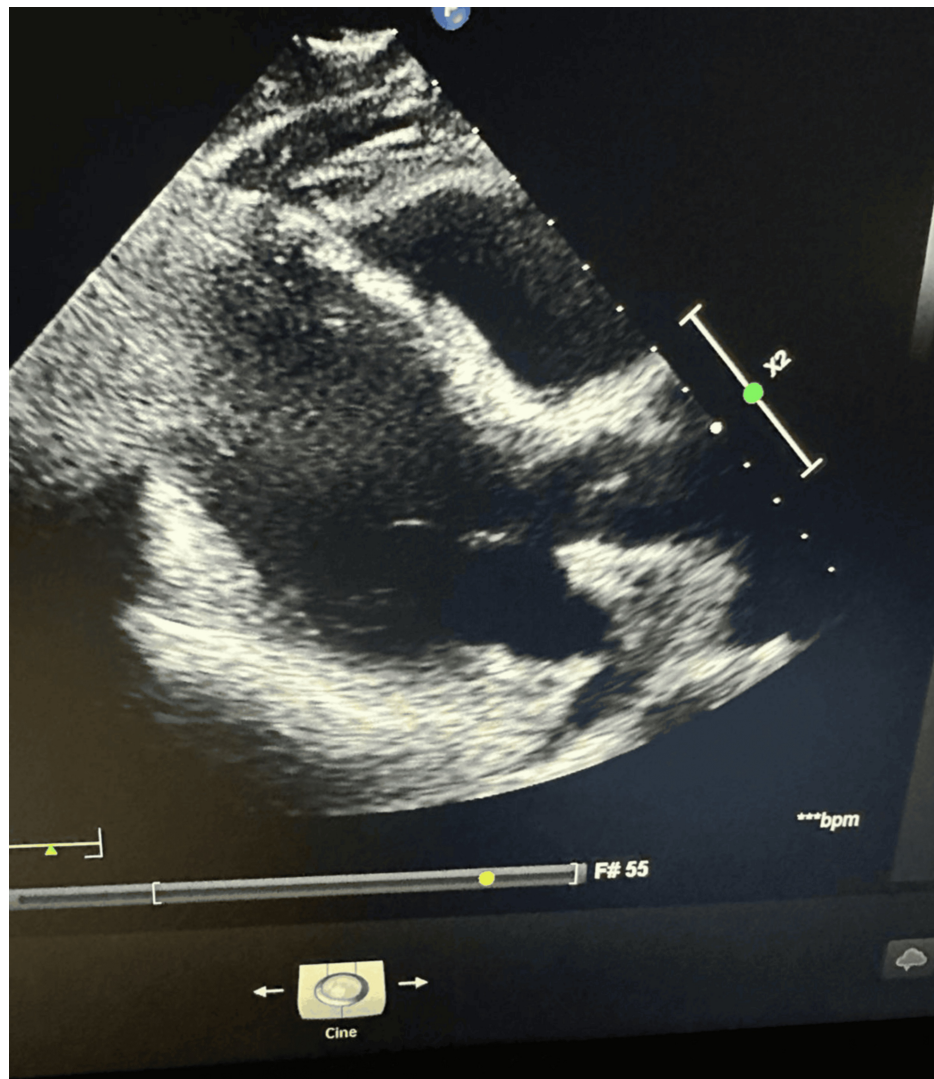


FIGURE 2: Transthoracic echocardiographic image demonstrating global hypokinesia with a left ventricular ejection fraction of 38% (Simpson's biplane method)

The differential diagnosis included SIMD, PPCM, and TCM. The presence of diffuse hypokinesia, elevated inflammatory markers, and active infection favored SIMD. Given the moderate troponin elevation and lack of ischemic symptoms, coronary angiography was not indicated.

A multidisciplinary ICU team obtained microbiological samples, including blood, urine, and tracheal aspirate cultures, upon admission to the ICU, and initiated empirical broad-spectrum antibiotic therapy with meropenem (1 g every eight hours, IV) and vancomycin (1 g every 12 hours, IV) immediately thereafter. The patient did not receive antibiotic therapy intraoperatively. Fluid resuscitation was performed cautiously, and low-dose norepinephrine infusion was titrated to maintain a mean arterial pressure of at least 65 mmHg. Mechanical ventilation was continued using lung-protective strategies. Stress ulcer prophylaxis was provided with intravenous pantoprazole (40 mg once daily), and venous thromboembolism prophylaxis was administered using subcutaneous enoxaparin sodium (4,000 anti-Xa IU once daily).

By day 3, inflammatory markers and hemodynamics had significantly improved, and lactate levels had normalized. The patient was successfully extubated on day 8 and transitioned to spontaneous breathing. By day 12, she was hemodynamically stable and transferred to the obstetrics ward.

Guideline-directed medical therapy for acute decompensated heart failure was not initiated, as myocardial dysfunction was considered secondary to SIMD. Management was primarily focused on infection control and hemodynamic stabilization. Follow-up TTE before discharge showed normalization of left ventricular function (LVEF 55%), as demonstrated in Figure 3, further confirming the reversible nature of myocardial dysfunction. This case shows that SIMD in late pregnancy can resemble PPCM and TCM clinically, but with

prompt diagnosis and focused treatment, full cardiac recovery is possible. Written informed consent was obtained from the patient for publication of clinical details and accompanying images.

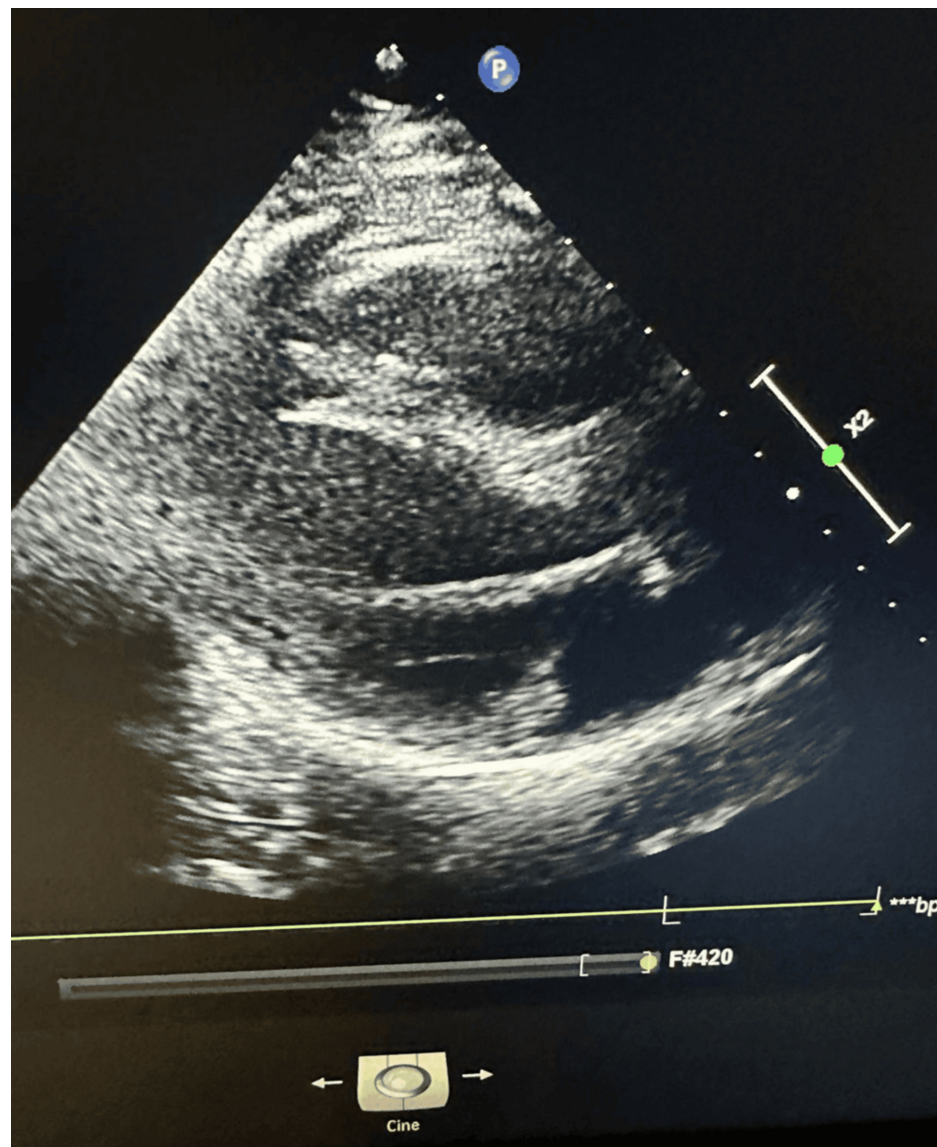


FIGURE 3: Follow-up transthoracic echocardiography showing normalized left ventricular systolic function (55%) with complete resolution of wall motion abnormalities

Discussion

SIMD is a common yet frequently underrecognized complication of sepsis and septic shock, marked by temporary, widespread ventricular contractile impairment [7]. Its pathophysiology involves several interacting mechanisms, including excessive proinflammatory cytokine release (cytokine storm), increased nitric oxide production, mitochondrial energy failure, and disturbances in calcium handling [8]. These processes cause reversible myocardial depression without lasting structural damage. Usually, cardiac function recovers within 7-10 days [9].

Clinically, SIMD presents with hypotension, tachycardia, increased requirement for inotropic support, and echocardiographic evidence of global hypokinesia [10]. The absence of coronary artery disease, moderate troponin elevation, and rapid functional recovery help distinguish SIMD from other acute cardiomyopathies, such as PPCM and TCM [11,12]. Importantly, infectious myocardial involvement may mimic acute coronary syndromes, as demonstrated in cases such as varicella-associated myopericarditis presenting with electrocardiographic and biochemical features suggestive of acute myocardial infarction despite the absence of obstructive coronary disease [13].

PPCM occurs within the last month of pregnancy or up to five months postpartum and is often associated with severely reduced LVEF, sometimes leading to persistent heart failure [14]. TCM is typically triggered by physical or emotional stress and characterized by apical ballooning on echocardiography [15]. In contrast, SIMD generally shows global rather than regional dysfunction, lacks apical ballooning, and demonstrates rapid improvement after infection control.

In our patient, pneumonia-related sepsis during the third trimester caused reversible global left ventricular dysfunction (LVEF 38%). The absence of apical ballooning, the low likelihood of coronary artery disease based on clinical and echocardiographic findings, and full recovery within weeks supported the diagnosis of SIMD.

Management emphasizes controlling the underlying infection and restoring hemodynamic stability [16]. Early empiric antibiotics, careful fluid management, and vasopressor support (preferably norepinephrine) are linked to better survival rates. In our case, early antibiotic use, conservative fluid therapy, and low-dose norepinephrine led to quick clinical stabilization and full recovery of cardiac function.

Conclusions

In cases of acute heart failure linked to sepsis during pregnancy, SIMD should always be considered in the differential diagnosis. Early identification, appropriate infection treatment, and prompt hemodynamic support can greatly improve maternal and neonatal outcomes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Nazli Gonulduru, Ebru Kaya, Gulseren Yilmaz

Critical review of the manuscript for important intellectual content: Nazli Gonulduru, Ebru Kaya, Gulseren Yilmaz

Acquisition, analysis, or interpretation of data: Bedih Balkan

Drafting of the manuscript: Bedih Balkan

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Walley KR: Sepsis-induced myocardial dysfunction. *Curr Opin Crit Care*. 2018, 24:292-9. [10.1097/MCC.0000000000000507](https://doi.org/10.1097/MCC.0000000000000507)
2. Lin YM, Lee MC, Toh HS, et al.: Association of sepsis-induced cardiomyopathy and mortality: a systematic review and meta-analysis. *Ann Intensive Care*. 2022, 12:112. [10.1186/s13613-022-01089-3](https://doi.org/10.1186/s13613-022-01089-3)
3. Shvilkina T, Shapiro N: Sepsis-induced myocardial dysfunction: heterogeneity of functional effects and clinical significance. *Front Cardiovasc Med*. 2023, 10:1200441. [10.3389/fcvm.2023.1200441](https://doi.org/10.3389/fcvm.2023.1200441)
4. Khalid N, Patel PD, Alghareeb R, Hussain A, Maheshwari MV: The effect of sepsis on myocardial function: a review of pathophysiology, diagnostic criteria, and treatment. *Cureus*. 2022, 14:e26178. [10.7759/cureus.26178](https://doi.org/10.7759/cureus.26178)
5. Lima MR, Silva D: Septic cardiomyopathy: a narrative review. *Rev Port Cardiol*. 2023, 42:471-81. [10.1016/j.repc.2021.05.020](https://doi.org/10.1016/j.repc.2021.05.020)
6. Carbone F, Liberale L, Preda A, Schindler TH, Montecucco F: Septic cardiomyopathy: from pathophysiology to the clinical setting. *Cells*. 2022, 11:2833. [10.3390/cells11182833](https://doi.org/10.3390/cells11182833)
7. Beesley SJ, Weber G, Sarge T, et al.: Septic cardiomyopathy. *Crit Care Med*. 2018, 46:625-34. [10.1097/CCM.0000000000002851](https://doi.org/10.1097/CCM.0000000000002851)
8. Ehrman RR, Sullivan AN, Favot MJ, Sherwin RL, Reynolds CA, Abidov A, Levy PD: Pathophysiology, echocardiographic evaluation, biomarker findings, and prognostic implications of septic cardiomyopathy: a review of the literature. *Crit Care*. 2018, 22:112. [10.1186/s13054-018-2043-8](https://doi.org/10.1186/s13054-018-2043-8)
9. Lukić I, Mihić D, Varžić SC, et al.: Septic cardiomyopathy. *Rev Cardiovasc Med*. 2024, 25:23.

10. [10.31083/j.rcm2501023](https://doi.org/10.31083/j.rcm2501023)
Orde SR, Pulido JN, Masaki M, Gillespie S, Spoon JN, Kane GC, Oh JK: Outcome prediction in sepsis: speckle tracking echocardiography based assessment of myocardial function. *Crit Care*. 2014, 18:R149.
11. [10.1186/cc13987](https://doi.org/10.1186/cc13987)
Hilfiker-Kleiner D, Haghikia A, Nonhoff J, Bauersachs J: Peripartum cardiomyopathy: current management and future perspectives. *Eur Heart J*. 2015, 36:1090-7. [10.1093/eurheartj/ehv009](https://doi.org/10.1093/eurheartj/ehv009)
12. Lv X, Wang H: Pathophysiology of sepsis-induced myocardial dysfunction. *Mil Med Res*. 2016, 3:30. [10.1186/s40779-016-0099-9](https://doi.org/10.1186/s40779-016-0099-9)
13. Chaudhari M, Sharma S, Jha RK, Ahuja RS, Bansal S: Varicella myopericarditis mimicking acute myocardial infarction with ARDS - a rare association in an immunocompetent young adult. *Indian Heart J*. 2016, 68:274-5. [10.1016/j.ihj.2015.08.026](https://doi.org/10.1016/j.ihj.2015.08.026)
14. Tani K, Shirakabe A, Kobayashi N, et al.: The prognostic impact of the serum heart-type fatty acid-binding protein level in patients with sepsis who were admitted to the non-surgical intensive-care unit. *Heart Vessels*. 2021, 36:1765-74. [10.1007/s00380-021-01865-4](https://doi.org/10.1007/s00380-021-01865-4)
15. Evans L, Rhodes A, Alhazzani W, et al.: Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. *Intensive Care Med*. 2021, 47:1181-247. [10.1007/s00134-021-06506-y](https://doi.org/10.1007/s00134-021-06506-y)
16. Cecconi M, De Backer D, Antonelli M, et al.: Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. *Intensive Care Med*. 2014, 40:1795-815. [10.1007/s00134-014-3525-z](https://doi.org/10.1007/s00134-014-3525-z)