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PERICARDIAL HEART DISEASE

CASE REPORT: HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE: CARDIO-OBSTETRICS 2023

Management of a Large Pericardial Effusion in Pregnancy

A Multidisciplinary Approach

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ABSTRACT

Pericardial effusions are rarely brought to clinical attention in pregnancy. When present, effusions are typically small, clinically silent, and related to fluid shifts in pregnancy. We present a case of a large pericardial effusion during the third trimester of pregnancy with management considerations for labor and delivery. (J Am Coll Cardiol Case Rep 2024;29:102225) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

CASE PRESENTATION

A 26-year-old gravida 1, para 0 at 38 weeks' gestation with history of sickle cell disease presented for an outpatient transthoracic echocardiogram (TTE) ordered by her obstetrician for follow-up of a pericardial effusion incidentally noted on a computed tomography angiogram during a recent admission for community-acquired pneumonia and an acute sickle cell pain crisis at 31 weeks' gestation. TTE on that admission showed a moderate, partially organized, circumferential pericardial effusion without any echocardiographic evidence for cardiac tamponade (**Figure 1**). She was discharged after completion of intravenous (IV) antibiotics, with plans for repeat echocardiography to monitor the pericardial effusion.

LEARNING OBJECTIVES

- To understand the importance of a multidisciplinary team approach in managing pericardial effusions during pregnancy.
- To recognize the hemodynamic changes during pregnancy that can influence the size and management of pericardial effusions.

Her follow up TTE at 38 weeks' gestation was significant for an interim increase in the pericardial effusion especially near the right atrium and right ventricle (RV). The largest fluid pocket measured 2.7 cm and was seen adjacent to the RV (**Figure 2**, Video 1). Her inferior vena cava was 1.2 cm with >50% collapse during inspiration (Video 2). The right atrium and RV appeared well-expanded throughout diastole. There were no significant respiratory variations in either mitral or tricuspid valve inflow velocities based on Doppler to suggest tamponade physiology (**Figure 2**). Due to rapid increase in size of pericardial effusion and impending delivery, she was admitted to our hospital under the antepartum service.

On admission, she was hemodynamically stable with blood pressure of 133/84, heart rate of 87, respiration rate of 16, normal oxygen saturation, and afebrile. She did not have any chest pain and was

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ABBREVIATIONS AND ACRONYMS

IV = intravenous

RV = right ventricle

TTE = transthoracic echocardiogram

otherwise asymptomatic. Physical examination was notable for a jugular venous pressure to the angle of the jaw, normal heart sounds, and warm extremities without edema. Laboratory test results were significant for anemia with hemoglobin of 8.2 mg/dL, which was at her baseline given her history of sickle cell disease. In addition, laboratory test results revealed a negative antinuclear antibody, and N-terminal prohormone brain natriuretic peptide, thyroid-stimulating hormone, erythrocyte sedimentation rate, and C-reactive protein were all within normal limits. Electrocardiogram was normal voltage, showed normal sinus rhythm, and T-wave inversion in V_2, without any evidence of electrical alternans. Chest radiography was significant for cardiomegaly without any evidence of central pulmonary vascular congestion.

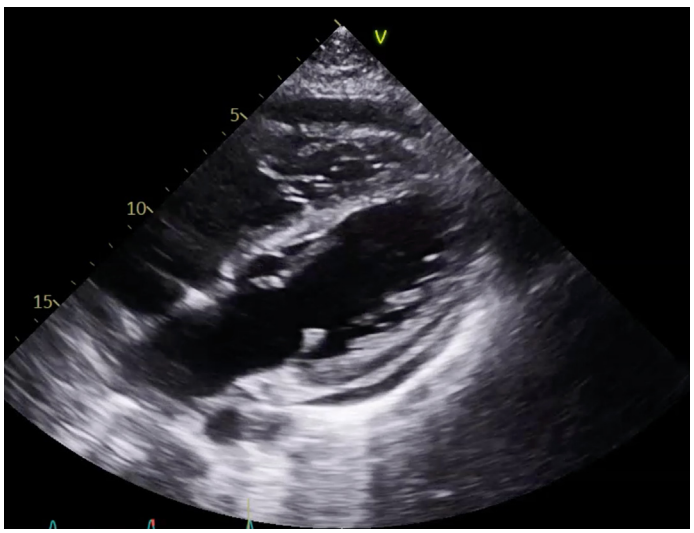
QUESTION 1: WHAT WERE THE NEXT STEPS IN MANAGEMENT OF THIS PATIENT'S PERICARDIAL EFFUSION?

An interdisciplinary team meeting involving general cardiology, interventional cardiology, obstetrics/gynecology, maternofetal-medicine, anesthesiology, and cardiothoracic surgery was held to review her clinical status and potential hemodynamic impacts on labor and delivery. Because she demonstrated no signs of hemodynamic compromise by clinical examination or echocardiography, the decision was made to proceed with vaginal delivery under close supervision in a controlled setting.

Subsequently, an arterial line was placed for more accurate hemodynamic assessment before induction of labor and she was transferred to the cardiac intensive care unit for closer monitoring. An early epidural catheter was placed to minimize the cardiac effects of catecholamine release in response to pain. She was started on IV oxytocin for induction of labor as recommended by the obstetrics team. Electronic fetal monitoring was used for continuous assessment of fetal heart rate and progression of contractions. Serial beside echocardiograms were done by cardiology to monitor size of pericardial effusion and for detection of any tamponade physiology using echocardiographic parameters. As labor progressed, the patient's pain was not adequately controlled; therefore, she underwent pudendal nerve block by anesthesia. The decision was made to proceed with a vacuum-assisted second stage of labor to minimize preload disruptions due to the large pericardial effusion. She delivered a healthy 6 lb 4.5 oz male infant. Thereafter, the third stage of labor was uncomplicated with no hemodynamic alterations observed.

On postpartum day 2, her course was complicated by pre-eclampsia without severe features, with laboratory test results significant for elevated urine protein/creatinine ratio, elevated creatinine to 1.51 mg/dL, and elevated aspartate aminotransferase to 52 U/L, for which she received IV magnesium and nifedipine. The pericardial effusion remained large but stable, and, therefore, no intervention was performed. Throughout her inpatient postpartum care, the patient continued to be followed up closely by the multidisciplinary team. She was discharged in stable condition after 10 days of hospital care. A repeat TTE 2 months postpartum revealed near full resolution of her pericardial effusion (**Figure 3**, Video 3).

FIGURE 1 TTE at 31 Weeks' Gestation

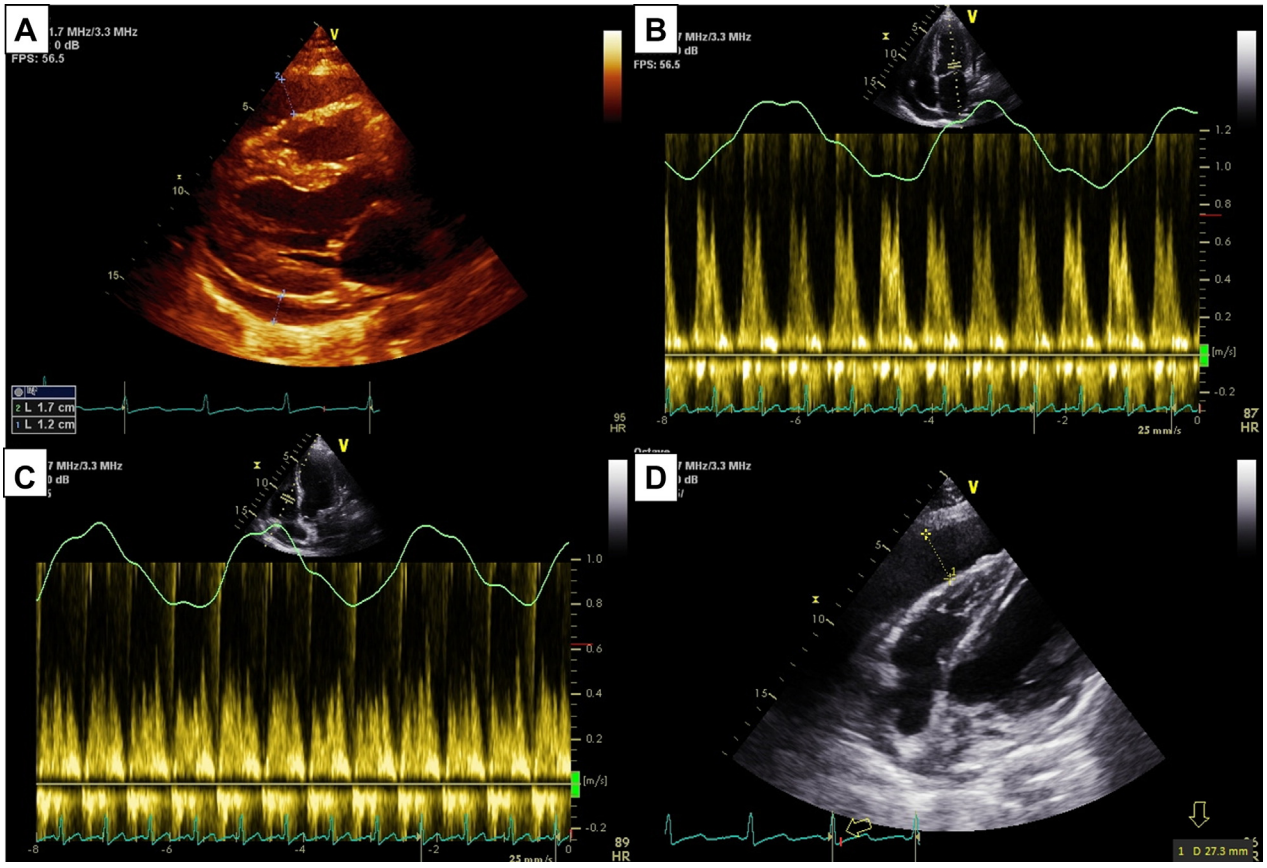


Subcostal view in diastole showing a moderate pericardial effusion with well-expanded RV. RV = right ventricle; TTE = transthoracic echocardiogram.

QUESTION 2: WHAT IS THE DIFFERENTIAL DIAGNOSIS FOR THIS PATIENT'S PERICARDIAL EFFUSION?

Pericardial effusion in pregnant patients with sickle cell disease presents a unique diagnostic and management challenge due to overlapping clinical features and potential complications associated with both conditions. Multiple differential diagnoses should be considered in this patient. First, acute chest syndrome, a common complication of sickle cell disease, can manifest with signs and symptoms similar to pericardial effusion, such as dyspnea,

FIGURE 2 **TTE at 38 Weeks' Gestation**



(A) Parasternal long-axis view showing large circumferential pericardial effusion; (B) pulse wave Doppler interrogation (apical 4-chamber view) showing no significant respiratory variation in mitral valve inflow velocities; (C) pulse wave Doppler interrogation (apical 4-chamber view) showing no significant respiratory variation in tricuspid valve inflow velocities; (D) subcostal view showing large pericardial effusion surrounding RV. Abbreviations as in Figure 1.

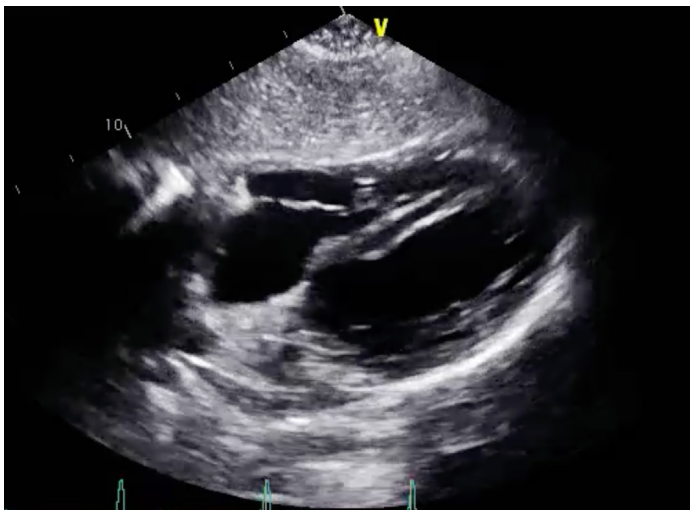
tachypnea, and chest pain. Second, pre-eclampsia, a hypertensive disorder commonly seen in pregnancy, can cause fluid retention leading to generalized edema, and rarely pericardial effusions. A study investigating cardiac function and hemodynamics using TTE in pregnant women with pre-eclampsia demonstrated the presence of pericardial effusions in 73% of the women.[1] Other potential but less likely causes include myocardial infarction, congestive heart failure, including possible peripartum cardiomyopathy, and pulmonary embolism, which can all present with similar symptoms.

On further inspection of prior medical records, our patient was found to have a small chronic pericardial effusion, noted on a TTE 5 years before her pregnancy. Given the partially organized nature of the effusion on the initial computed tomography of the chest, we suspect that this effusion was chronic. Therefore, we speculate that the etiology of her pericardial effusion was her underlying sickle cell disease and the hemodynamic changes of pregnancy exacerbated the existing pericardial effusion.

QUESTION 3: WHAT ARE THE HEMODYNAMIC CHANGES DURING PREGNANCY THAT CAN INFLUENCE THE SIZE OF PERICARDIAL EFFUSIONS?

During pregnancy, physiological circulatory changes lead to volume expansion and vasodilation. These changes may alter the clinical manifestations of pericardial diseases. It is important to emphasize that, in most cases, pregnancy-associated pericardial effusions are small or moderate and, in the absence of trauma, aortic dissection, acute pericarditis, or active autoinflammatory diseases, such as systemic lupus

FIGURE 3 TTE 2 Months' Postpartum



Subcostal view in diastole without evidence of significant pericardial effusion. Abbreviations as in Figure 1.

erythematosus, they are well tolerated and have a benign course. Consequently, they rarely require any invasive intervention. However, it is crucial to highlight that hemodynamic alterations occurring to accommodate the developing fetus can have a direct impact on the occurrence and progression of pericardial effusions.

First, the progressive increase in total blood volume, plasma volume, and red blood cell mass necessary to meet the metabolic demands of pregnancy can potentially worsen cardiac compression if a pericardial effusion is present.[2] This occurs because the increased plasma volume may lead to elevated hydrostatic forces within blood vessels, promoting the leakage of fluid into the pericardial space. Second, in addition to the activation of the renin-angiotensin-aldosterone system, which maintains blood pressure and helps retain salt and water in pregnancy, relaxin (a peptide hormone produced by the corpus luteal phase) stimulates increased vasopressin secretion and thirst, resulting in increased water retention. Dysregulation in this hormonal axis can lead to excessive salt and water retention.[3] Third, hormonal and vascular changes during pregnancy lead to decreased systemic vascular resistance, which starts fairly early in pregnancy and can be as much as 30% from baseline. This allows increased blood flow to the placenta. Although this is beneficial for placental perfusion, it can reduce compensatory mechanisms that counteract a dramatic decrease in preload or diastolic filling. This may exacerbate the hemodynamic compromise associated with large pericardial effusions.[2,4]

It is essential to be aware of these dynamic hemodynamic changes and their potential impact when managing pregnant women with pericardial effusions. In our situation, the arterial line served a vital purpose, enabling us to closely monitor hemodynamic changes. This monitoring capability allows for the rapid assessment of pulsus paradoxus or narrowing in the pulse pressure that may indicate low stroke volume and impending tamponade. Although our patient was under intensive care observation, such monitoring may not always be obligatory. The decision regarding location of labor and delivery should be based on the resources, experience, and logistical considerations of the institution responsible for managing these patients.

QUESTION 4: HOW DOES PRESENCE OF A SIGNIFICANT PERICARDIAL EFFUSION AND RATE OF ACCUMULATION OF EFFUSIONS IN PREGNANCY INFLUENCE MANAGEMENT?

The hemodynamic effects of pericardial effusions regardless of size are the most important factor affecting management. It is also important to emphasize that, although evidence-based guidelines for managing pericardial diseases during pregnancy are lacking, it has been observed that pericardial effusions may be present in up to 44% of women in the third trimester.[5]

This highlights the significance of understanding the implications of effusion size for tailored, individualized care. It is imperative to avoid overreaction to pericardial effusions in the absence of features of tamponade. In most cases, avoidance of volume restriction strategies and diuretics is critical. Counteracting the physiological volume expansion in pregnancy can worsen the hemodynamic compromise but also lead to conditions such as oligohydramnios during pregnancy, further jeopardizing fetal viability. Small-to-moderate pericardial effusions, without any hemodynamic compromise, typically require close monitoring with serial echocardiography. This approach allows for the assessment of effusion progression and the detection of any changes in size or development of symptoms that may necessitate intervention.[6] Large or rapidly progressive pericardial effusions carry a higher risk of significant hemodynamic compromise, but do not frequently require aspiration because even large effusions are generally well tolerated and usually have a benign course.[4] In cases with evidence of cardiac tamponade, prompt intervention becomes necessary to relieve cardiac

compression and optimize maternal and fetal well-being. Options for intervention may include volume administration, pericardiocentesis, or a pericardial window.[6] Pericardial windows are rarely performed and should only be used in specific circumstances. A window to the pericardium should also not be done in cases where the effusion is expected to resolve. In severe cases where pericardial tamponade is imminent or when initial interventions fail, urgent pericardial intervention may be considered. In these situations, cesarean section is often the preferred mode of delivery, ensuring the safety of both the mother and the baby.

In general, pericardial interventions in pregnant women are not associated with increased maternal risk, but have been associated with increased risk of fetal complications. Thus understanding the impact of hemodynamics, size, and rate of accumulation of a pericardial effusion on management decisions is critical in providing effective care during pregnancy, where the well-being of both the mother and the fetus must be prioritized.

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APPENDIX For supplemental videos, please see the online version of this paper.