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Peer-Reviewed Case Report

Cardiac tamponade on ECPELLA: a case report of a unique hemodynamic picture

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Abstract

Extracorporeal membrane oxygenation is rapidly becoming a preferred therapy for short-term hemodynamic support in cardiogenic shock, along with the use of devices such as Impella (Abiomed, Andover, MA). The two together can create unique hemodynamics resulting in altered presentation of common hemodynamic conditions such as tamponade. We present a case of a patient with fulminant myocarditis requiring veno-arterial extracorporeal membrane oxygenation and Impella support. The patient later developed a pericardial effusion with atypical tamponade physiology, which masked the left ventricular systolic function recovery. We further highlight the complex hemodynamics of cardiac tamponade in patients with such mechanical circulatory support and its implications on echocardiography.

Background

Although extracorporeal membrane oxygenation (ECMO) has been available for over 40 years, used primarily in neonatology, over the past decade the technology has gained momentum in adult cardiology. ECMO is fast becoming a preferred device for short-term hemodynamic support in patients with cardiogenic shock, as seen by the exponential growth in the number of ECMO centers which has increased by 133% in the last decade.[1]

Early use of veno-arterial ECMO (V-A ECMO) has shown the promising trend of increased survival in patients with fulminant myocarditis presenting with cardiogenic shock .[2-5] A common drawback of this modality is an increase in left ventricular (LV) afterload, resulting in delayed myocardial recovery, pulmonary congestion, and other adverse sequalae. Several interventions can be used in conjugation with V-A ECMO to unload the LV, thereby avoiding complications .[6,7]

One of the options of venting the LV is facilitating the forward flow with another device such as Impella (Abiomed, Andover, MA). The combination of two means of mechanical circulatory support (MCS) creates new hemodynamic patterns which may alter the presentation of common hemodynamic conditions such as tamponade. We describe a case of a young patient presenting with fulminant myocarditis leading to cardiogenic shock who was treated with a combination of V-A ECMO and Impella (ECPELLA). The patient developed pericardial effusion with atypical tamponade physiology which masked the recovery of LV systolic function. We have also highlighted the complex hemodynamics of cardiac tamponade in a patient with dual MCS and its implication on echocardiographic diagnostic approach.

Case Report

A 25-year-old, previously healthy female presented to the emergency room with a four-day history of nausea, vomiting, abdominal and central chest pain, and a fever of up to 102 °F. She developed ventricular tachycardia, cardiac arrest, and underwent cardiopulmonary resuscitation with return of spontaneous circulation in less than two minutes. The patient was intubated for airway protection. Her echocardiogram showed an LV ejection fraction (LVEF) of less than 10%. As she remained in cardiogenic shock, she was placed on V-A ECMO and Impella CP support. The ECMO flow was 4.5 L/min at a speed of 3590 revolutions per minute, and the Impella flow was 2.5 L/min at P6. The patient was also on epinephrine (0.05 mcg/kg/min) and milrinone (0.25 mcg/kg/min).

On Day 2 of the patient's hospitalization, her echocardiogram showed moderate pericardial effusion without any echocardiographic features of cardiac tamponade such as right ventricular or right atrial collapse, plethoric inferior vena cava, or excessive mitral/tricuspid inflow variability (Figure 1).

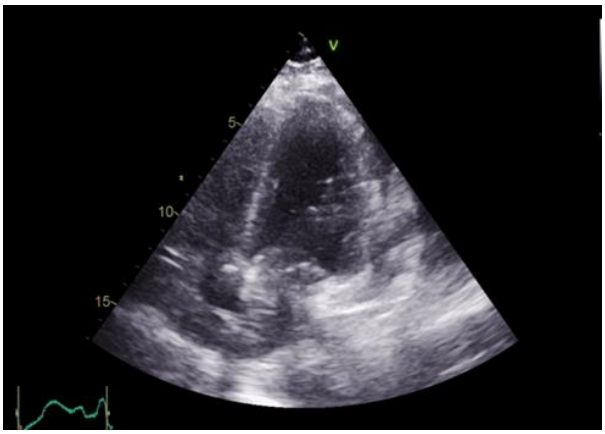

Figure 1: *Transthoracic echocardiogram 4-chamber view prior to pericardial drainage (Video 1).*

Although unclear, the etiology of pericardial effusion could be due to viral myopericarditis or ventricular perforation during placement of the Impella CP. The patient's blood pressure on Day 2 was 74/69 mmHg. The patient underwent right heart catheterization and endomyocardial biopsy. Percutaneous drainage of pericardial effusion was attempted, but unsuccessful.

The patient's right heart pressures were as follows: right atrium 8 mmHg, right ventricle 29/3/7 mmHg, pulmonary artery 21/12 mmHg, pulmonary capillary wedge pressure 11 mmHg. The patient's vasopressor requirement was increasing, with addition of vasopressin at 0.04 units/min. It was decided to drain the pericardial effusion surgically. The transesophageal echocardiography showed large pericardial effusion with floating of the heart with an estimated LVEF of around 15% (Figures 2). During the drainage there was an immediate gush of blood suggesting high intra pericardial pressure. The pericardial fluid was sanguineous, and a clot was also removed. Echocardiography showed a mild improvement of LV systolic function (Figures 3 and 4).

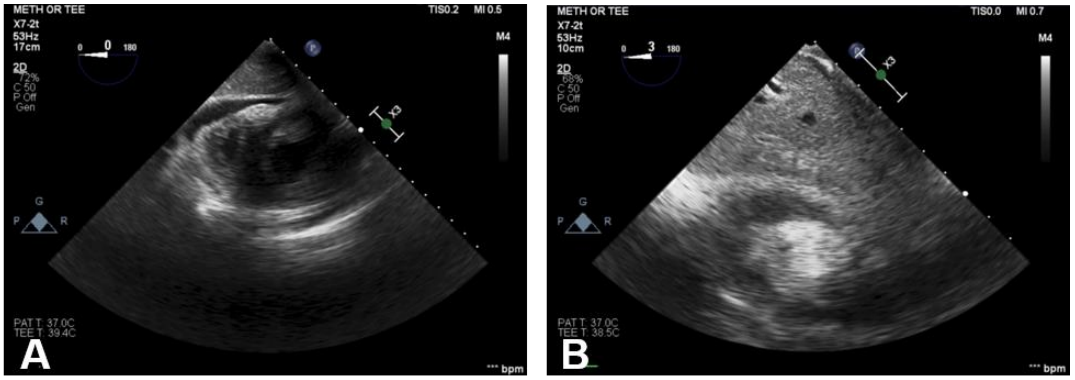

Figure 2. *Transesophageal echocardiogram (transgastric view) showing the significant pericardial effusion build-up. A. Papillary muscle level B (Video 2A). Apex of the heart (Video 2B).*

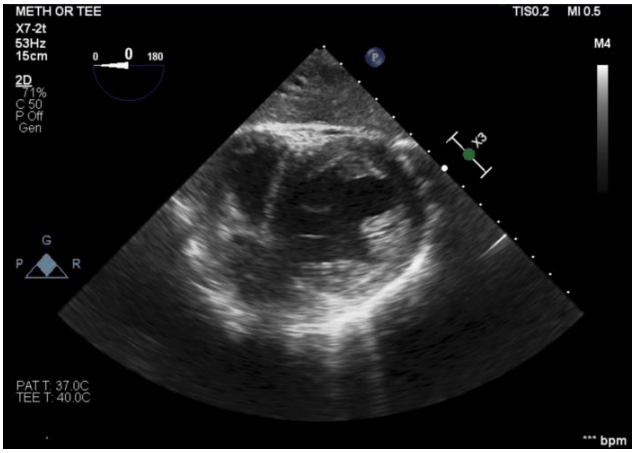

Figure 3. *Transesophageal echocardiogram showing slight improvement in left ventricular function immediately after pericardial effusion drainage (Video 3).*

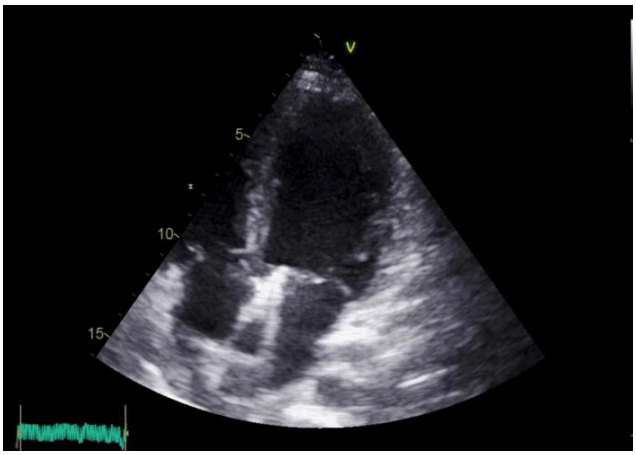

Figure 4. *Transthoracic echocardiogram the next day after pericardial effusion drainage, now with improving left ventricular function (Video 4).*

There was no immediate change in ECMO or Impella flow post-drainage; however, hemodynamics improved significantly with decreasing pressor requirements approximately 6 hours post-drainage. Epinephrine and vasopressin were reduced to 0.02 mcg/kg/min and 0.03 units/min, respectively. The patient was off vasopressin 24 hours post-drainage.

The pericardial fluid showed mesothelial and mixed inflammatory cells, blood, and fibrin. The patient's cardiac biopsy was consistent with acute lymphocytic myocarditis**.** The patient tested positive for SARS-CoV-2 and was treated with remdesivir (5-day course), high-dose methylprednisolone (3-day course), and convalescent plasma (2 doses). There was impressive recovery of LV function the next day. The patient made a complete recovery and was discharged home in less than 2 weeks.

Discussion

In this case report, we describe a patient who became hemodynamically unstable while being supported on both V-A ECMO and Impella, which lead to a suspicion of cardiac tamponade. Although no clinical or echocardiographic signs of tamponade were present, the drainage of pericardial fluid resulted in immediate hemodynamic stabilization, confirming the diagnosis of tamponade.

The classical Beck triad, described in 1935, remains the mainstay of our understanding and clinical diagnosis of pericardial tamponade. The triad consists of three medical signs including low blood pressure, distended neck veins, and muffled heart sounds, with the main pathophysiological phenomena of increased central venous pressure.[8] Paradoxically, in a patient where ECMO is working optimally, blood pressure is usually maintained or artificially supported by vasopressors, central venous pressure is within normal limits, and heart sounds are replaced by machinery hum. Pulsus paradoxus, another cornerstone of physical exam-based diagnosis of tamponade, is no longer present. Pulsus paradoxus reflects decreased systolic output on inspiration while the right ventricular filling increases because of increased interventricular dependence. In a patient supported by V-A ECMO and Impella, forward flow is much less dependent on the ventricular filling/contraction and is mostly determined by ECMO and Impella, both providing a continuous (non-pulsatile) flow. This unique physiology of a mechanically supported patient can mask the presentation of acute cardiac tamponade, which is difficult to diagnose or even suspect if the traditional bed side teaching principles are applied in an ECMO patient.

It is commonly said that tamponade is a clinical diagnosis; however, in reality, echocardiographic signs often precede the clinical signs. This is especially true in the intensive care setting in patients who are sedated, on a ventilator, and unable to report any symptoms. Echocardiography is the test of choice when the diagnosis of tamponade is clinically suspected. Significant progress and standardization has been made especially in Doppler interpretation of cardiac tamponade physiology, the hallmark of which is an exaggerated inspiratory decrease in mitral valve inflow. This is an echocardiographic equivalent of pulsus paradox. Echocardiographically this has been described as interventricular interdependence and interpretated as exaggerated changes in mitral and tricuspid Doppler inflow and M mode finding of enhanced reciprocal changes in ventricular dimensions.[9]

The physiology of a closed ECMO circuit is briefly reviewed for a better understanding of the hemodynamics of cardiac tamponade in patients with mechanical ventilator support. The V-A ECMO circuit withdraws deoxygenated blood from the right atrium or central vein with a non-pulsatile pump and directs it to the membrane oxygenator and then to a systemic artery via an outflow cannula, thus replacing the heart and lungs.[1] As seen with this circuit, the physiology of interventricular interdependence is replaced by a mechanical non-pulsatile pump and oxygenator, therefore making the diagnosis of cardiac tamponade on the basis classical signs and symptoms of pulsus paradox and interventricular interdependence unreliable.

The positive pressure ventilation is known to mask the pulsus paradoxus and in combination with ECMO support will further augment the masking of this

tamponade physiology. The LV Impella unloads the left ventricle and decreases the left ventricular end diastolic pressure, which intuitively should help in recognition of pulsus paradox. However, it also provides a continuous flow which masks any potential variability coming from the native left ventricular contractions.

It is important to understand that cardiac output in a patient on MCS consists of two parts: the support provided by the devices such as ECMO and Impella, and the output coming from the native heart. In our patient, who had a combined ECMO and Impella flow of 7 L/min, her own heart with a LVEF < 20% was only minimally contributing.

The hemodynamic performance of the LV in cardiogenic shock is best described by pressure volume loop (PV loop). Independent of underlying etiology, the LV contractility (reflected by Emax and defined as the maximum slope at the end systolic PV point) is reduced and LV end diastolic pressure is increased.[1] In this PV loop the exact hemodynamics on a patient on V-A ECMO, LV Impella, and mechanical ventilation has not been described before in our literature search.

At the molecular level, the phenomena of myocardial stunning in myocarditis has been described and is implicated due to increased reactive oxygen species production, with a decreased sensitivity of myofilaments to calcium and dysfunction of excitation coupling phenomena.[10] The immediate recovery of LV function after relief of pericardial tamponade in this patient may have some underlying stunning.

Pericardial tamponade has been described in pediatric and adult ECMO patients. However, this is limited to a few case reports.[11,12] Our case is unique, as pericardial tamponade in complex scenario such as the combined use of ECMO and Impella has not been described in the literature to our knowledge.

Conclusion

Several takeaway messages can be made from this case:

1. The patient on ECPELLA circuit shows atypical features of cardiac tamponade. Classical signs of pulsus paradox and interventricular interdependence are absent, which can delay the diagnosis of pericardial tamponade if existing clinical, hemodynamic, and echocardiographic criteria are applied.
2. Echocardiographically the use of Doppler criteria and inferior vena cava plethora can be misleading. The only findings could be demonstration of chamber invagination signs and large effusions with swinging motion which usually are seen late in the continuum of cardiac tamponade.
3. Invasive hemodynamics using right heart catheter cannot be completely relied upon, in view of the altered physiology in the MCS.
4. Limited echocardiography should be part of daily exams in patients on MSC, to assess the accumulation of pericardial fluid. The significant improvement in handheld devices can be utilized in intensive care unit settings as a standard protocol.

5. We recommend considering draining any substantial pericardial effusion in patients that are on advanced MCS. It is imperative to remember that the traditional signs of pericardial tamponade may not be reliable in patients supported on MCS.
6. Further research is needed in animal or computer models to assess the effect of tamponade physiology on PV loop in patients on these devices, with the potential to improve our understanding of treating these complex patients.

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