Spontaneous Echo Contrast Mimicking Left Ventricular Thrombus in a Patient on Extracorporeal Membrane Oxygenation Support

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A 54-year-old man presented to our clinic with a 6-month history of dyspnea and angina. The patient was diagnosed with obstructive coronary artery disease and severe mitral regurgitation. Preoperative transthoracic echocardiography (TTE) showed regional wall motion abnormalities and a regurgitant area of 8 cm². The left ventricular ejection fraction was 22%. Myocardial perfusion imaging by single-photon emission CT detected approximately 30% viable myocardium in the left ventricle (LV). The patient underwent successful coronary artery bypass grafting and mitral valve repair. Peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) was initiated after unsuccessful attempts to wean the patient from cardiopulmonary bypass. Cannulation was performed with use of a right femoral venous drainage cannula (21F) and a right femoral arterial reinfusion cannula (15F). Because the VA-ECMO created LV distension, a venous cannula (16F) was inserted into the right superior pulmonary vein and connected to the femoral venous drainage cannula to reduce LV filling pressures.

On the first day of VA-ECMO support, the images of routine TTE and subsequent transesophageal echocardiography (TEE) are shown in Videos 1 and 2.

Question: On reviewing images of echocardiography, which is the most likely diagnosis in this clinical context? Based on the clinical information and the echocardiography, what would you recommend as the next step?

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Answer: TEE findings included absence of blood flow, severe blood stasis, and thrombus formation in the LV (Videos 1 and 2). The most likely diagnosis in the clinical context that we have described was severe spontaneous echo contrast (SEC) and severe blood stasis, rather than LV thrombus. The primary goals are to open up the aortic valve and reverse severe blood stasis in the LV.

Discussion

VA-ECMO is a well-established form of short-term mechanical circulatory support for patients in severe cardiogenic shock. In such cases, VA-ECMO can fully replace the functions of the heart and lungs, maintaining systemic blood circulation and pulmonary gas exchange to ensure adequate organ perfusion and tissue oxygenation. During peripheral femoral VA-ECMO, blood is extracted from the right atrium via the femoral vein and returned to the arterial systemic circulation via the femoral artery. The retrograde infusion of arterialized blood into the thoracic aorta has been shown to significantly increase LV afterload and reduce the LV stroke volume simultaneously.1

Cardiogenic shock is characterized by a low ejection fraction, decreased stroke volume, low pressure generation, and high LV end-diastolic pressure. The main hemodynamic effect triggered by the increasing VA-ECMO flow is an increase in LV afterload.1 Ostadal et al2 showed that decreasing VA-ECMO flow resulted in improvements in several parameters of LV performance in animals in cardiogenic shock. When LV systolic function was severely compromised, increases in afterload and the bronchial arterial circulation further contributed to the LV overload. If the LV cannot compensate for the increasing afterload through the Frank-Starling mechanism, the aortic valve does not open, and the blood accumulates in the LV. In another study, coronary angiography showed that patients with severe cardiac dysfunction had extremely slow flow even in the nonoccluded coronary arteries after extracorporeal CPR.3 Thus, severe blood stasis in the LV and severe cardiac dysfunction may predict poor recovery of LV function in patients who receive VA-ECMO.

SEC is an echogenic, swirling pattern of blood flow that can develop in any cardiac chamber if the velocity of blood flow is extremely low. SEC might increase risk for thrombogenesis.4 In patients who are receiving peripheral femoral VA-ECMO, SEC is often associated with reduced aortic valve opening and low pulsatility. Additionally, SEC may suggest increased afterload and LV distension. Presence of SEC increases the risk for impaired recovery of LV function by inducing subendocardial ischemia, increased myocardial oxygen consumption, and subsequent pulmonary edema.5

The goal is to facilitate aortic valve opening and, thus, resolve the blood stasis in the LV. In our center, when absence of opening of the aortic valve and LV blood stasis are detected in a patient, the ECMO flow is gradually decreased to improve the ejection fraction. If the ECMO flow point at which the aortic valve reopens is too low for adequate organ perfusion, we reduce the ECMO flow rate significantly to achieve aortic valve opening within a short period (approximately 0.5 minute) until the blood stasis resolves completely (Videos 3, 4). Subsequently, the flow rate is restored to the original setting. If the aortic valve fails to open, severe blood stasis can be observed again within minutes (Videos 5, 6). We typically decrease the pump flow rate gradually, every 1 to 2 hours, to ensure intermittent disruption of the thrombogenesis in the LV. To date, no LV or aortic root thrombi have been detected in any patients in our center during the implementation of this protocol.6

LV decompression is necessary to prevent myocardial ischemia and facilitate ventricular recovery. In our hospital, the unloading of the LV is typically achieved by placing a venous cannula into the left atrium through the right upper pulmonary vein. A large-bore cannula can ensure effective and secure LV unloading in most cases. However, in the case described herein, the intact mitral valve prevented the stagnating blood in the LV from draining through the right upper pulmonary venous cannula. The right superior pulmonary venous cannula was surgically repositioned to the LV apex, and the femoral VA-ECMO cannulas were left in place to ensure the effective unloading of the LV filling pressure and to inhibit thrombus formation. No macroscopic thrombus was observed during the surgical repositioning of the cannula. TTE obtained after this surgical procedure confirmed that there was no blood stasis or sedimentation of blood particles in the LV (Video 7).

Making a clear echocardiographic distinction between severe blood stasis and a firm thrombus in patients who are receiving VA-ECMO support is often a challenging task, even for experienced physicians. Severe blood stasis...
seems to be reversible after the aortic valve reopens. In a recent case series, TEE showed suspected thrombi that adhered to the aortic root, which were not observed by the surgeons. In conclusion, absence of aortic valve opening and the appearance of SEC on echocardiography do not confirm the presence of clinically relevant thrombi in patients who are receiving VA-ECMO, especially during the early days after ECMO is initiated. However, clinicians must be aware that long-term blood stasis will increase the risk for thrombosis.

Our case suggests that unloading the LV is an effective method of opening the aortic valve during peripheral femoral VA-ECMO. An intermittent decrease in the ECMO flow can easily achieve opening of the aortic valve. Serial echocardiography provides real-time visualization of the cardiac function. However, adequate interpretation of the echocardiographic images is a key step toward improving patient management and outcomes.

Reverberations

1. Echocardiography is an essential tool for monitoring patients who are receiving ECMO support.

2. Echocardiography can reveal severe LV dysfunction associated with severe blood stasis and SEC during peripheral VA-ECMO. Adequate interpretation of the echocardiographic images is necessary to distinguish between SEC and other entities and to guide management in such cases.

3. Unloading of the LV is a key step toward ventricular recovery. Serial echocardiography can be used to monitor the outcomes of different LV decompression methods.

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Additional information: To analyze this case with the videos, see the online article.

References


