Ultrasound Diagnosis of Cardiac Arrest in an 81-Year-Old Postoperative Patient

Jan Stassen, MD; Christophe Vandenbriele, MD, PhD; Guido Claessen, MD, PhD; Bart Jacobs, MD; Yves Debaveye, MD, PhD; and Tim Balthazar, MD

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An 81-year-old man had a witnessed in-hospital cardiac arrest. He had been hospitalized on the gastroenterology ward for 3 weeks, recovering from a partial hepatectomy with bile duct resection and hepaticojejunostomy for a cholangiocarcinoma. His medical history also included well-controlled arterial hypertension and hypercholesterolemia.

Bystander CPR was started immediately at the ward, followed by advanced cardiac life support (ACLS) at the arrival of the crash team. Pulseless electrical activity (PEA) was observed; the patient was intubated, and 1 mg of epinephrine was administered while CPR was continued. Return of spontaneous circulation was achieved after 2 minutes of ACLS. During transferal to ICU, however, PEA recurred, and ACLS was restarted and continued at arrival on the ICU. After 12 minutes of CPR and administration of 3 mg of epinephrine, return of spontaneous circulation was sustained.

At subsequent clinical evaluation chest expansion and lung auscultation were symmetric. The peripheral saturation was 83% with FiO₂ of 100%. Mean arterial pressure was 50 mm Hg, and there was a sinus tachycardia of 140 beats per minute. Arterial blood gas analysis confirmed severe hypoxemia and revealed severe lactate acidosis (maximum lactate, 18 mM). Hemoglobin was 7.4 g/dL, compared with 8.2 g/dL 20 hours earlier. Because the patient remained hypotensive despite fluid resuscitation, pharmacologic support with norepinephrine (maximum dose, 0.6 μg/kg/min) was initiated.

Point-of-care ultrasound scan of the abdomen was negative for free fluid. Postresuscitation ECG, however, showed pronounced ST-segment elevation in the inferior leads (Fig 1) and, subsequently, a transesophageal echocardiography (TEE) was performed (Video 1).
Question: Based on the patients’ clinical presentation and the ultrasound findings, what is the most likely diagnosis?

A. Acute coronary syndrome  
B. Pericardial tamponade  
C. Left ventricular outflow tract obstruction  
D. Aortic dissection

Figure 1 – Postresuscitation ECG shows ST-segment elevation in the inferior leads.
Answer: Left ventricular outflow tract obstruction

TEE revealed a systolic anterior motion (SAM) of the anterior mitral leaflet (AML) and subvalvular apparatus, resulting in left ventricular outflow tract (LVOT) obstruction (LVOTO) and eccentric mitral regurgitation. Furthermore, a hyperdynamic left ventricle with a small cavity was seen, which suggests underlying hypovolemia as a possible explanation for SAM. A small pericardial effusion surrounding the inferior right and left ventricular wall was noticed, but there was no atrial or ventricular collapse. The context and location suggest that this is a consequence of CPR, rather than the cause of the collapse. The right ventricle was mildly hypokinetic; however, it was not dilated, and there were no signs of right ventricle pressure overload (ie, no D-shaping, no bulging of the right atrial septum), which would be expected if pulmonary embolism, another possible cause of postoperative cardiac arrest, was the cause of the patient’s PEA and collapse. Right ventricle hypokinesia is observed in roughly 50% of patients in the postresuscitation period, independent of the underlying disease (Video 2).

Despite findings of inferior ST-segment elevation on the ECG, no regional wall motion abnormalities were noted on the initial TEE. Furthermore, cardiac arrest due to acute coronary syndrome mostly presents with ventricular tachycardia or ventricular fibrillation, rather than PEA, in the absence of mechanical complications. These observations make an acute coronary event unlikely. In this case, ST elevation that indicated myocardial ischemia can be explained by low flow due to LVOTO as a consequence of hypovolemia in combination with elevated myocardial oxygen use (and maybe coronary spasm) after administration of epinephrine. A second ECG confirmed spontaneous resolution of the ST elevation in the inferior leads.

Shortly after bedside ultrasound scanning was performed, severe hematemesis developed, which confirmed the suspicion that hypovolemia tipped this patient into SAM with LVOTO, which led to his cardiac arrest. Urgent gastroscopy could not identify the source of bleeding, but the presence of fresh blood in the duodenum suggested a bleeding at the hepaticojejunostomy. Considering the hemodynamic instability and ongoing bleeding, the patient was transferred urgently to the catheterization laboratory. Angiography confirmed active bleeding at the coeliac trunk, for which a successful coiling procedure was performed, followed by hemodynamic stabilization. Despite successful coiling and maximum supportive care, the patient died 2 days later due to multiorgan failure.

Discussion

SAM is defined as displacement of the AML towards the LVOT during systole, which can result in mitral regurgitation and LVOTO. Although typically observed in hypertrophic cardiomyopathy, it is uncommon in a structurally normal heart. The prevalence of SAM in patients without preexisting cardiac disease is not well-studied but is thought to be <1%. In these patients, SAM is more likely to occur when the heart is subjected to permissive physiologic conditions that provoke SAM: reduced preload, increased inotropic state, and decreased afterload. All these conditions are present in hypovolemic shock and during septic shock in instances when LVOTO might be an underestimated phenomenon.

The mechanism of SAM in a patient who is hypovolemic is explained by the small and hyperdynamic left ventricle generating high flow velocities in the LVOT, therefore creating a Venturi effect that pulls the mitral valve leaflet toward the septum, obstructing the LVOT. This can be detected easily by echocardiography. An elongated AML and hypertrophic appearance of the basal septum are predisposing anatomic factors, both of which were present in this patient.

The hemodynamic consequences of SAM with mitral regurgitation and LVOTO include elevated pulmonary capillary wedge pressures and a prolongation of systolic ejection phase with reduced stroke volume.

The clinical presentation ranges from symptoms like dyspnea (because of pulmonary congestion) to severe hemodynamic collapse due to LVOTO. Echocardiography enables early diagnosis of SAM and therefore is essential to guide resuscitative efforts. Doppler imaging easily can also localize and quantify the severity of LVOTO. Because LVOTO is associated with increased mortality rates, early identification is crucial in a critically ill patient. Because the most commonly used vasopressors in the ICU also have inotropic effects, it is crucial to correct the intravascular volume deficit as much as possible to avoid increased contractility in a volume-depleted and small left ventricle. Vasopressor agents with selective alpha agonist properties or vasopressin are preferred because this will not enhance
cardiac contractility as much, therefore relieving LVOTO. Inotropic agents should be avoided preferably.

Reverberations
1. Point-of-care echocardiography can provide valuable diagnostic information to differentiate between different types of shock.
2. The visualization of a small, hyperdynamic left ventricle with SAM of the AML and turbulent flow in the LVOT should raise suspicion for LVOTO.
3. Contribution of LVOTO to shock can be underestimated easily in conditions in which hypovolemia, enhanced cardiac contractility, and vasoplegia come together.
4. Norepinephrine can increase SAM by its inotropic properties.

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

References