An unusual presentation of left ventricular free wall rupture

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Left ventricular free wall rupture (LVFWR) is one of the most lethal complications following myocardial infarction. It accounts for approximately 12% to 21% of all in-hospital deaths following myocardial infarction. The majority of patients die shortly after LVFWR from instantaneous pericardial tamponade and hemodynamic collapse. However, up to one-third of cases are subacute in nature, allowing limited time for emergent surgical repair to prevent sudden death. A high index of suspicion and timely use of diagnostic tests are important in recognizing cases. The present report describes the case of a 69-year-old man who initially presented with acute pericardial tamponade and was subsequently diagnosed with LVFWR in the operating room as the cause of his hemopericardium. The pathology, diagnosis and management of LVFWR are reviewed.

Key Words: Echocardiography; Hemopericardium; Mechanical complications; Myocardial infarction

A 69-year-old male smoker with a history of polycythemia rubra vera presented with a three-day history of pleuritic chest pain and dyspnea. The patient was hypotensive with a systolic blood pressure of 80 mmHg and a heart rate of 110 beats/min. There was no evidence of a pulsus paradoxus. The cardiopulmonary examination was remarkable for an elevated jugular venous pressure of 8 cm to 9 cm above the sternal angle and decreased air entry bilaterally with minimal bibasilar crackles.

The complete blood count revealed a mildly elevated white blood cell count of $15.5 \times 10^9/L$. There was metabolic acidosis with a pH of 7.25 and an increased lactate level of 9.0 mmol/L. Troponin T was mildly elevated at 1.47 µg/L. A 12-lead electrocardiogram (ECG) demonstrated sinus tachycardia with nonspecific ST-T changes (Figure 1A). There was no evidence of an acute or recent myocardial infarction (MI) on the ECG. The chest x-ray revealed cardiomegaly with no evidence of pulmonary edema. Transthoracic echocardiography demonstrated normal left ventricular (LV) systolic function with no wall motion abnormalities. There was a loculated-appearing pericardial effusion, with echodense material within the pericardial space adjacent to the right ventricular free wall most consistent with fibrin or thrombus (Figure 1B). There was echocardiographic evidence of tamponade physiology with right atrial systolic collapse, right ventricular diastolic collapse, excessive respiration variation across the mitral inflow and a dilated inferior vena cava. Attempts at pericardiocentesis were unsuccessful, in which only 20 mL of serosanguinous fluid was removed. Open pericardial exploration was deemed to be indicated. Preoperative computed tomography of the chest revealed an ascending aorta of normal calibre and a moderate-to-large pericardial effusion that appeared hemorrhagic.

The operative findings demonstrated a clotted hemopericardium, which was evacuated with improvement of the tamponade physiology. Cardiopulmonary bypass was employed. The ascending aorta was intact with no obvious evidence of dissection. There was a small area in the epicardial surface of the midinferolateral LV wall that was hemorrhagic in nature, consistent with a recent MI. In this zone, a small, localized LV

La présentation inhabituelle d'une rupture de la paroi libre du ventricule gauche

La rupture de la paroi libre du ventricule gauche (RPLVG) est l'une des complications les plus mortelles après un infarctus du myocarde. Elle est responsable d'environ 12 % à 21 % de tous les décès en milieu hospitalier après un tel événement. La majorité des patients meurent peu après la RPLVG d'une tamponnade péricardique instantanée et d'un collapsus hémodynamique. Cependant, jusqu'au tiers des cas sont de nature subaiguë, ce qui donne une fenêtre limitée pour procéder à une réparation chirurgicale d'urgence afin de prévenir la mort subite. Il est important d'avoir un fort indice de présomption et d'utiliser rapidement les tests diagnostiques pour dépister les cas. Dans le présent rapport est décrit le cas d'un homme de 69 ans qui a d'abord consulté en raison d'une tamponnade péricardique aiguë, ensuite diagnostiquée en salle d'opération comme une RPLVG responsable de l'hémopéricarde. Les auteurs analysent la pathologie, le diagnostic et la prise en charge de la RPLVG.

rupture site was found as a punctate area of interrupted epicardium, confirming the anatomical diagnosis of LV free wall rupture (LVFWR). The rupture site was repaired using gelatin-resorcinol-formaldehyde glue and patched with bovine pericardium in a sutureless manner.

Cardiac catheterization performed one week later demonstrated proximal occlusion of a second obtuse marginal branch, with minimal coronary artery disease in the remaining vessels (Figure 1C). The patient recovered uneventfully and was discharged nine days after the emergent surgery.

DISCUSSION

The incidence of LVFWR is estimated at approximately 4% post-MI and accounts for 12% to 21% of in-hospital deaths following MI (1-3). The increased availability of bedside echocardiography has contributed to a progressive rise in the number of cases of LVFWR being diagnosed and reported. LVFWR should be suspected in any MI patient presenting with persistent or recurrent chest pain, nausea, clinical and hemodynamic evidence of cardiac tamponade, pulseless electrical activity or shock, and persistent ST segment elevation with evidence of infarct expansion or extension (1).

Risk factors for cardiac rupture that have been identified to date include anterior location of the infarct, large transmural infarct, age older than 70 years, female sex, and no history of previous angina or MI (1). The development of ST segment elevation or Q waves on the initial ECG and a peak creatine kinase-MB above 150 IU/L have also been associated with an increased risk of LVFWR (1-3). There is evidence that restoring the patency of the infarct-related artery may reduce the incidence of cardiac rupture, either through fibrinolytic therapy or percutaneous coronary intervention, although the potential benefits of late fibrinolysis are less clear (4). In patients older than 75 years of age, fibrinolytic therapy may actually increase the risk of free wall rupture (4).

Although there is no consensus regarding the classification system, LVFWR can be divided morphologically into four types (1). Type I

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Figure 1) A Sinus tachycardia with no evidence of acute or recent myocardial infarction. **B** Subxiphoid view on transthoracic echocardiography demonstrating a moderate-sized pericardial effusion with echogenic material lateral to the right ventricle (arrows) consistent with fibrin or thrombus. **C** Occluded second obtuse marginal vessel at the ostium with a short, tapered stump (arrow)

displays little dissection or infiltration of the myocardium. Type II has extensive myocardial dissection, often with a trajectory composed of multiple canaliculi. In type III LVFWR, the rupture site is protected either by a thrombus on the ventricular side or by pericardial adhesion. Type IV is considered incomplete because the trajectory does not traverse all layers of the myocardium. The basic mechanism of rupture is believed to be a continuous stretching of infarcted tissue and, thus, structurally compromised myocardium. Erosion of necrotic tissue, intramural hemorrhage and degradation of collagen struts act together to cause slippage and lengthening of individual myocytes.

The clinical presentation of LVFWR may include persistent or recurrent chest pain, particularly pericardial pain, nausea, restlessness and agitation, sudden hypotension, ECG features of localized or regional pericarditis, and/or sudden death (1). Echocardiography usually represents the first-line diagnostic tool for suspected myocardial rupture that is rapid and noninvasive. Colour flow imaging and pulsed Doppler may be useful in the assessment of flow characteristics at the presumed rupture site, and an intravenous echocardiographic contrast agent may be useful in identifying intrapericardial hemorrhage caused by myocardial rupture or development of ventricular pseudoaneurysm.

If the transthoracic echocardiography demonstrates pericardial fluid and the subsequent pericardiocentesis reveals blood, immediate surgery is indicated to repair the rupture site. Medical therapy directed toward hemodynamic stabilization, including fluids, inotropic support and vasopressors, should also be initiated. The most appropriate technique for surgical repair must be tailored to the clinical, hemodynamic and anatomical circumstances, including whether to implement cardiopulmonary bypass. In a blowout-type situation, suture repair with reinforcing prosthetic material is required. In a contained rupture or oozing situation, the relatively recent introduction of sutureless

techniques using glue and patch material, as in our patient, have been met with encouraging operative success, and fairly good early and midterm results (5).

In the present case, the clinical picture of pleuritic chest pain, pericardial effusion and nonspecific ST-T changes initially appeared to be most consistent with pericarditis and pericardial effusion, until the finding of hemorrhagic fluid on pericardiocentesis raised suspicion for a mechanical complication post-MI. Ischemia of the left circumflex territory can present as a relatively silent phenomenon on a traditional 12-lead ECG, with a serious complication of LVFWR, as illustrated in the present case. Because survival depends primarily on early diagnosis and expeditious relief of tamponade followed by emergency surgery, the first step toward improving the mortality risk from LVFWR is a high index of suspicion by the clinician.

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