Right Atrial Mimicry: A Unique Case of Cardiac Tamponade Caused by a Loculated Right Pleural Effusion Following Coronary Artery Bypass Graft Surgery

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Pleural effusion is a common finding after coronary bypass graft surgery. Rarely, patients may develop loculated effusions, which may potentially lead to cardiac chamber compression. We report a rare case of loculated pleural effusion (LPE) that resulted in right atrial compression, leading to cardiac tamponade physiology, which resolved completely after emergent surgical drainage of the pleural effusion. This case underscores the need for early implementation of echocardiography in the postoperative management of hemodynamically unstable patients following cardiac surgery.

PATIENT DESCRIPTION

A 71-year-old male presented with non-ST-segment elevation myocardial infarction according to the fourth definition of myocardial infarction. The electrocardiogram did not reveal any ischemic abnormalities. Physical examination upon presentation was unremarkable. A coronary angiogram demonstrated multivessel coronary artery disease. Relevant medical history included diabetes, hypertension, and dyslipidemia. Initial two-dimensional transthoracic echocardiography (TTE) demonstrated normal left ventricle (LV) and right ventricle (RV) contraction without evidence of any valvular heart disease. The patient underwent coronary artery bypass graft surgery (CABG), which included a pedicled a left internal mammary artery (LIMA) to the left anterior descending artery and a T graft of a free right internal mammary artery from the LIMA. It also included a harvested saphenous vein graft to the posterior descending artery.

On postoperative day 1 (POD1), the patient developed diaphoresis and dyspnea. Physical examination was notable for increased heart rate of 100 bpm and hypotension of 80/40 mmHg. The patient appeared pale. Increased jugular venous pressure was noted. Because of refractory hypotension, vasopressors were administered. Chest X-ray revealed a large right-sided pleural effusion [Figure 1A]. An emergent TTE demonstrated an a right LPE compressing the adjacent free wall of the right atrium (RA), mimicking the RA cavity [Figure 1B], alongside accelerated tricuspid flow on color flow-Doppler (CFD) [Figure 1B, Figure 1E] and significant mitral inflow velocity variation consistent with pulsus paradoxus [Figure 1C].

Immediate transesophageal echocardiography (TEE) confirmed the presence of LPE compressing the RA cavity [Figure 1D, Figure 1E], resulting in a functional tricuspid stenosis with a mean gradient exceeding 9 mmHg. Given the patient's rapid deterioration, immediate surgical drainage was performed, leading to prompt hemodynamic improvement. Repeated chest X-ray demonstrated complete resolution of the pleural effusion. A follow-up echocardiogram performed the day after demonstrated normal LV and RV function with complete resolution of the LPE, completely expanded RA cavity without evidence of atrio-ventricular gradient across the tricuspid valve or a respiratory flow variation pattern. The patient achieved complete recovery within one week and was subsequently discharged home for an outpatient rehabilitation program.

COMMENT

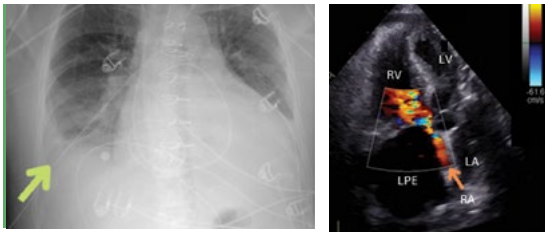
Pleural effusion following cardiac surgery is a common finding. At least 30% of patients develop a pleural effusion in the immediate postoperative period. In addition, patients with pleural effusions after cardiac surgery have an increased incidence of postoperative complications, delayed recovery, and rehospitalizations [1]. The etiology of pleural effusions often remains unclear. Nonetheless, pleural effusions

Figure 1. Radiographic and echocardiographic features of cardiac tamponade physiology from a loculated pleural effusion

LPE = loculated pleural effusion, PWD = pulsed-wave Doppler, RA = right atrial, TEE = transesophageal echocardiography, TTE = transthoracic echocardiogram

[A] Chest X-ray showing a moderate to large pleural effusion with blunting of the right costophrenic angle (green arrow)

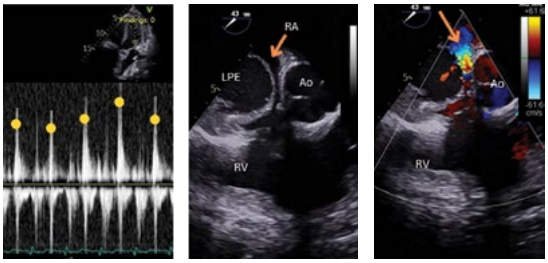
[B] A two-dimensional TTE in a four-chamber view illustrating a right LPE with compression of the RA cavity



[C] PWD assessment of the mitral valve flow indicating a notable respiratory flow variation (yellow dots)

[D] A two-dimensional TEE demonstrating a LPE compressing the RA cavity

[E] color-flow Doppler indicating accelerated velocity across the tricuspid valve (orange arrow)



may arise from a combination of inflammatory responses to pleural incision and postoperative oozing from the graft beds [2]. In our case, the latter is the most probable cause, given the rapid onset of LPE on POD1.

Most patients demonstrate small unilateral left-sided pleural effusions, with about 10% experiencing larger ones. These findings are categorized into early effusions, including hemorrhagic exudates with a high number of eosinophilic cells occurring within 30 days following surgery, as well as late effusions, which are characterized by lymphocytic exudates that manifest after 30 days post-surgery [3].

In our case, cytological analysis of the pleural effusion revealed hemorrhagic characteristics, characterized by a markedly elevated count of inflammatory cells, which suggested the presence of an early pleural effusion. The reported incidence of cardiac chamber collapse in patients with pleural effusion, without concomitant pericardial effusion, is 18% [4]. Pleural effusions may be loculated, thereby potentially resulting in compression of the cardiac chambers and consequently compromising the physiological function of the affected chamber. This, in turn, impairs ventricular filling, leading to a reduction in cardiac output and a clinical presentation consistent with cardiac tamponade physiology, as exemplified in our case.

Our patient demonstrated clinical signs consistent with cardiac tamponade physiology, characterized by hypotension, elevated venous pressure, and pulsus paradoxus, as indicated by the presence of a significant respiratory flow variation pattern [Figure 1C]. Therefore, prompt surgical drainage of the LPE by a pleural chest tube was performed, resulting in rapid hemodynamic improvement.

The morphology of the LPE may be incorrectly misidentified as the RA cavity by less experienced physicians, which could result in delays in diagnosis and treatment. In fact, in our case, the LPE resembled the RA cavity. However, given the absence of proper contractility of the cavity, the true RA appeared collapsed, indicating the presence of LPE. Because of the unusual presentation of LPE, a complementary TEE demonstrated greater efficacy in clarifying the anatomical and hemodynamic implications of the LRPE, with evidence of elevated right atrio-ventricular gradients and cardiac tamponade physiology. Hence, following cardiac surgery, a complementary TEE in cases of suspected postoperative pleural effusion may enhance diagnostic accuracy, particularly in identifying loculated effusions. Armstrong and colleagues [5] demonstrated that TEE had a superior diagnostic performance compared to TTE in detecting localized cardiac tamponade among 23 patients in the intensive care unit after cardiac surgery.

Our case underscores the critical importance of considering LPE in patients with hemodynamic instability after cardiac surgery, highlighting the need for timely recognition and intervention. Moreover, it advocates for the systematic incorporation of echocardiography in the postoperative evaluation and management of individuals following cardiac surgical procedures.

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