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Transient Biventricular Dysfunction Following Pericardiocentesis for Cardiac Tamponade

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Pericardial decompression syndrome (PDS) is an unusual, potentially fatal complication that occurs after pericardial drainage for cardiac tamponade. either by needle pericardiocentesis or surgical pericardiostomy. It manifests with paradoxical hemodynamic deterioration and/or pulmonary edema, commonly associated with ventricular dysfunction. PDS usually begins after initial clinical amelioration after pericardiocentesis. It is largely under-reported and may be neglected in clinical practice. While the precise mechanisms behind PDS are not well understood, it seems to be strongly related to patients with preexisting ventricular dysfunction. Doctors who perform pericardial drainage should be mindful of the associated high-risk factors for the intervention, taking into consideration the uncommon possibility of PDS formation.

Keywords: Pericardial decompression syndrome; cardiac tamponade; post-pericardial drainage low cardiac output syndrome; paradoxical hemodynamic instability.

1. INTRODUCTION

Cardiac tamponade is a medical emergency that restricts the filling of the heart chambers and alters normal hemodynamic conditions, resulting in cardiogenic shock and hypotension. It happens after a quick or overflow of fluid into the pericardium cavity [1].

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Pericardial decompression syndrome also known as post-pericardial drainage low cardiac output syndrome is defined as a paradoxical worsening of hemodynamics and development of pulmonary edema after uneventful pericardial drainage in patients with cardiac tamponade [2].

Pericardial drainage, by either pericardiocentesis or pericardiotomy, is vital in making patients hemodynamically stable, but has been associated with numerous complications, including one very rare, underappreciated, and life-threatening complication known as pericardial decompression syndrome (PDS) [3].

PDS usually occurs after clinical improvement following pericardiocentesis. It is largely unrecognized and may go unnoticed in daily practice.

2. CASE REPORT

We report the case of a 38-year-old woman with no history of significant cardiovascular disease. She had a previous history of metastatic breast cancer and was treated with mastectomy for triple-negative SBR II invasive ductal carcinoma. Six months later she presented to the emergency department with dyspnea that had been progressively worsening over the previous two weeks and discomfort in the inferior chest.

Her physical checkup showed jugular venous distension, hypotension (blood pressure 80/50 mmHg), tachycardia (heart rate 172 beats/min), and dull heart sounds.

Her electrocardiogram revealed sinus tachycardia with diffuse low-complexity electrical alternation and QRS voltage, and flat T waves (Fig. 1). Her chest x-ray demonstrated an expanded cardiac silhouette with a right pleural effusion. Transthoracic echocardiography showed a widespread pericardial effusion (53 mm in the posterior wall, 31 mm in the anterior wall) with a fluttering heart. Global and regional contractility was maintained and left ventricular ejection fraction (LVEF) was preserved (FigS 2-3-4).

An emergency pericardiocentesis was done and 1500 ml of serosanguinous fluid was pulled out.

The pericardial drainage catheter was left in place for 1 day. The pericardial fluid was exudative. Cytological examination of the pericardial fluid detected malignant cells. The post pericardiocentesis TTE demonstrated no remaining pericardial fluid effusion with left ventricular systolic dysfunction. Her postpericardiocentesis EKG demonstrated normal sinus rhythm with no electrical alternations. Eight hours after the procedure, the patient became suddenly breathless with tachycardia, with no chest pain. TTE revealed a small pericardial effusion with no hemodynamic compromise, although we noticed that both ventricles had systolic dysfunction with an LVEF of 30%.

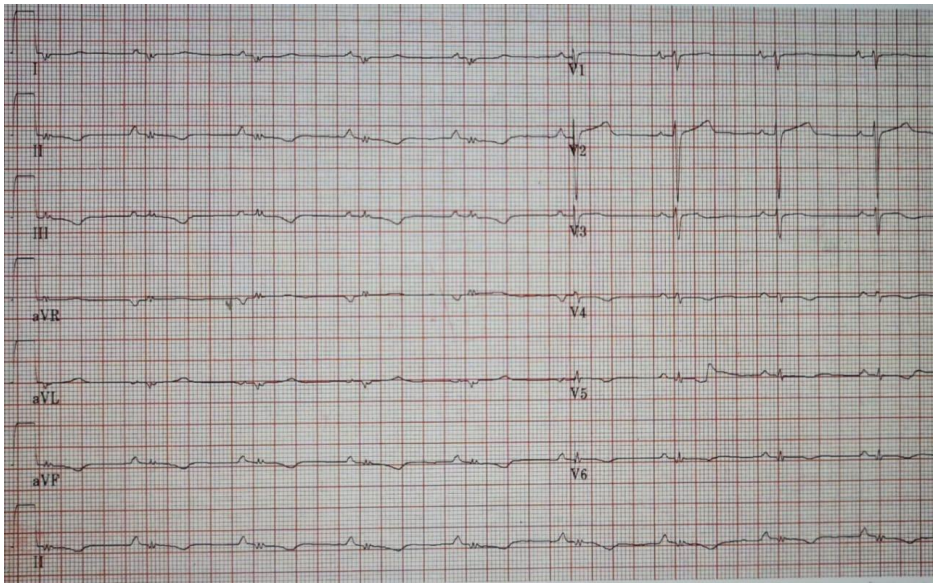

Fig. 1. Electrocardiogram showing low complex QRS voltage, and flat T waves

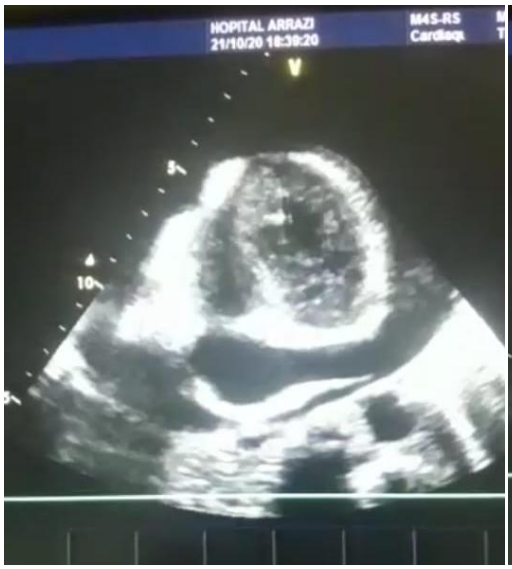

Fig. 2

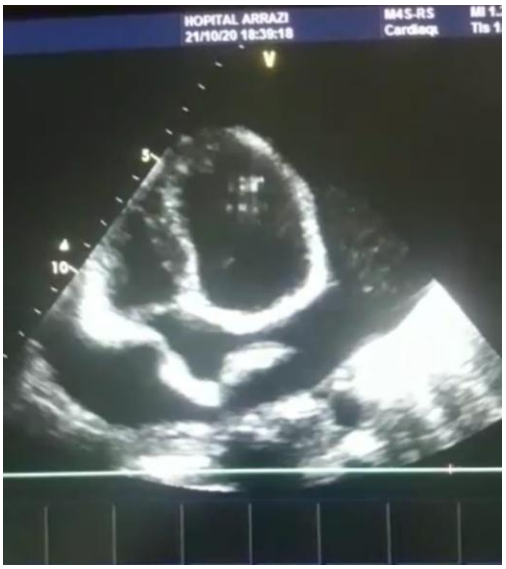

Fig. 3

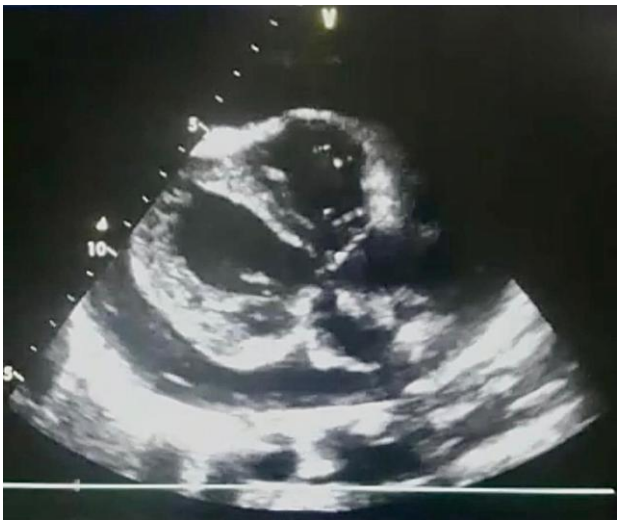

Fig. 4.

FigS. 2-3-4 : transthoracic echocardiography reveals a wide circumferential pericardial fluid effusion with evidence of right chamber compression in end-diastole

There was no physical evidence of myocardial perforation. The EKG manifested sinus tachycardia with diffuse low QRS voltage, negative T waves in the anterior leads. Troponin levels were normal. A repetitive renal workup did not show any renal failure and liver tests did show improvement in cytolysis. Instant i.v. therapy with dobutamine and furosemide 40mg daily was started with consistent clinical amelioration. Eleven days following admission, the patient was recuperating well in the cardiology department and was expressing a subjectively perceived good health. A subsequent echocardiogram showed spectacular enhancement of contractility in both ventricles. The ventricles had both regained normal size and LVEF was 55%. Pain medication was given and the Oncology team was prepared to begin the breast cancer chemotherapy.

3. DISCUSSION

The presence of pericardial tamponade, drainage, whether performed transcutaneously or surgically, allows in the vast majority of cases an improvement in hemodynamics. Apart from the

standard complications such as puncture of cardiac cavities, coronary artery injury, arrhythmia, pneumothorax, hemothorax, pneumopericardium or liver injury, this procedure may be accompanied by a paradoxical hemodynamic failure. This is a rare complication with an incidence of the disease is about 5 %, but it is severe with a mortality of about 16%. Mortality seems to be more important in the aftermath of surgical drainage 29% [3].

The first description of this clinical syndrome dates back to 1983 Vandyke et al. [4] in a patient of Caucasian origin with acute myeloid leukemia who benefited from a surgical drainage by pericardiocentesis of 500 ml of serosanguinous fluid, but it is only in 2010 that the term pericardial decompression syndrome was proposed.

The clinical presentation is variable, ranging from acute pulmonary oedema without shock to right, left or bi-ventricular failure, and can occur up to hours after the procedure. The main risk factors for mortality are neoplastic damage to the pericardium, post-radical damage, pericardial calcifications, previous damage to myocardial function or the need for circulatory support, whether medical or mechanical [5].

The pathophysiology is not fully understood at present, several hypotheses have been put forward (Fig 5).

- The first plausible hypothesis is paradoxical hemodynamic dysfunction based on a sudden increase in venous return following the lifting of the hemodynamic obstruction of the effusion, leading to an increase in afterload and dilation of the right ventricle which may lead to failure of the latter. The induced transmural pressure elevation of the right ventricle is further exacerbated by a negative in-trapericardial pressure related to drainage. Indeed, the physiological pressure of the intrapericardial space being almost zero, the latter becomes negative following the opening of this space and the implementation of the drainage. The dilation of the right ventricle also leads to a deviation of the interventricular septum towards the left and thus a decrease in cardiac output by virtue of the ventricular interdependence resulting in reduced volume and left ventricular output and results in decompensated left heart failure and/or pulmonary edema [6].
- Other researchers, have specified that the dysregulation could be due to the drop in coronary flow due to the compression of the epicardial coronary arteries by pericardial fluid and that ischemic heart disease could be one of the contributing factors [6].
- The last hypothesis suggests a neuro-vegetative origin with a sudden decrease in sympathetic activity after drainage, which may reveal a pre-existing ventricular dysfunction or lead to para-sympathetic hyperactivity.

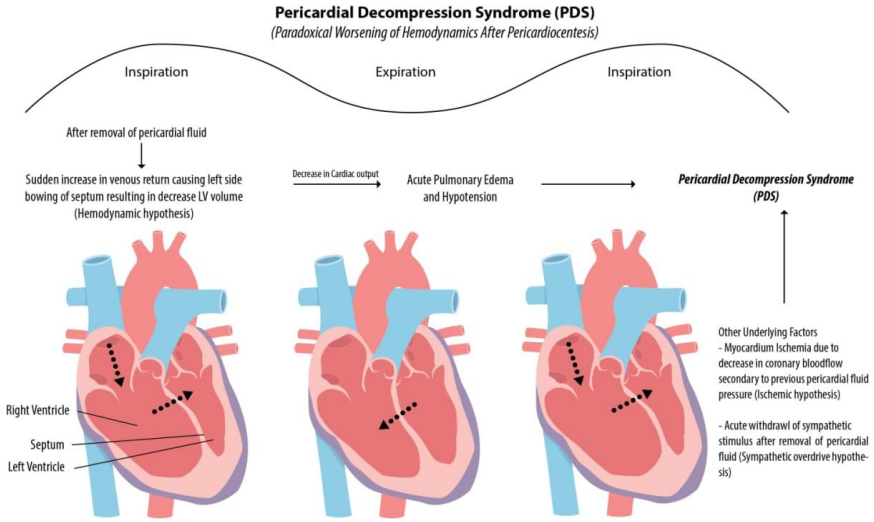

Fig. 5. Schematic Illustration of the pathophysiology of PDS [4]

Wolfe and Edelman noted that the abolition of the sympathetic stimulus after pericardiocentesis may result in the uncovery of a previously unrevealed background left ventricular dysfunction due to an inflated catecholaminogenic state. In addition, Martins and colleagues showed that while the administration of exogenous catecholamines raised coronary blood flow in the cardiac tamponade patients, there was no significant increase in the filling pressures and only a minor increase in the cardiac index [7].

They considered that because activation of the sympathetic nervous system was already occurring, high levels of endogenous catecholamines had no additional prospective benefit. Removal of the stimulus that leads to an enhanced sympathetic state may well give rise to the revelation of left ventricular dysfunction that was previously equilibrated by high levels of endogenous catecholamines [8].

Therefore, the sympathetic overdrive mechanism may play an integral part in the etiology of PDS, because abnormalities in left ventricular systolic function may occur after the described pericardiocentesis procedure [9].
As postpericardiocentesis ventricular dysfunction is not a common finding in the clinic, we believe that transient myocardial dysfunction after pericardial drainage is likely to happen more when a high volume of pericardial fluid is eliminated in a brief period of time, thereby suppressing rapid regulation of coronary resistance and the autonomic nervous system.

In the lack of specific treatment, the management is based mainly on an early diagnosis and the initiation of hemodynamic supportive therapy. The administration of vasopressors or inotropic therapy is usually required, and the most serious of these cases may need external circulatory support [7].

4. CONCLUSION

PDS is a phenomenon uncommon (incidence <5%) and can be observed after drainage of a pericardial effusion, regardless of the technique used.

To date, there are no clear evidence-based guidelines or recommendations for specifically preventing PDS. A reasonable approach, however, would be not to remove large amounts of pericardial fluid at one time if there is large pericardial effusion. The approach that is most manageable would be to remove the correct amount of pericardial fluid to resolve the physiology of the cardiac tamponade (which can be obtained easily by haemodynamic or echo-Doppler monitoring) and then to place extended pericardial drainage to obtain gradual, slow removal of the extra pericardial fluid.

The Cardiologist should be conscious of this condition and be ready to deal with it in a timely manner as this diagnosis leads to a high degree of mortality, particularly after prompt surgical decompression [10].

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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