Turkish Medical Journal, 2024; 9(1),32-36.

Olgu Sunumu / Case Report
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RESUSCITATIVE THORACOTOMY IN NON-TRAUMA PATIENT IN THE EMERGENCY DEPARTMENT

Acil Serviste Travma Dışı Hastada Resüsitatif Torakotomi

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Geliş / Received: 18.04.2024
Kabul / Accepted: 30.05.2024

Cite as:
Mutlu, H., Sert, T.E., Kokulu, K., Yazici, R. (2024) Resuscitative Thoracotomy in Non-Trauma Patient in The Emergency Department Turkish Medical Journal, 9(1),32-36.

ÖZET

Sol ventrikül serbest duvar rüptürü (SVSDR), tipik olarak kardiyak tamponad ve kardiyojenik şok ile kendini gösteren akut miyokard enfarktüsünün korkutucu ve ölümcül bir komplikasyonudur. SVSDR 'nin tedavisi cerrahidir. Bu yazıda, kardiyak tamponad bulguları ile acil servise başvuran ve tanı konularak resüsitatif torakotomi uygulanan 53 yaşında bir SVSDR olgusundan bahsedilmiştir.

Anahtar Kelimeler: Kardiyak tamponad, Resüsitatif torakotomi, Serbest duvar rüptürü

ABSTRACT

Left ventricular free wall rupture (LVFWR) is a fearful and mortal complication of acute myocardial infarction, which typically presents with cardiac tamponade and cardiogenic shock. The treatment of LVFWR is surgery. We mentioned about a 53-year-old case of LVFWR who was admitted to the emergency department with the findings of cardiac tamponade and diagnosed and underwent resuscitative thoracotomy.

Keywords: Cardiac tamponade, Free wall rupture, Resuscitative thoracotomy

INTRODUCTION

Left ventricular free-wall rupture (LVFWR) is the most serious mechanical complication of acute myocardial infarction (AMI) and usually leads to rapidly progressing cardiovascular collapse and sudden death(Cammalleri et al.,2020). Non-traumatic LVFWR is seen almost exclusively as a complication of myocardial infarction with acute ST elevation (Honda et al., 2014.). While the rate of occurrence of LVFWR is approximately 3% after AMI, the incidence of LVFWR decreases to approximately 1.7% after primary percutaneous coronary intervention (Wehrens & Doevendans ,2004). Risk factors for the development of post-AMI SVSDR include old age (i.e., > 65 years), being female, experiencing hypertension not accompanied by hypertrophy, first-time AMI and single-vessel disease (Usal et al.,1995). In this article, we discuss a case of LVFWR in which the patient was without any risk factors for the development of LVFWR; the patient was diagnosed with LVFWR in the emergency department (ED) and underwent resuscitative thoracotomy.

CASE REPORT

A 53-year-old male patient who was previously healthy was admitted to the ED with the complaint of being aggravated abruptly ,chest pain,and cold sweating. The patient's history revealed that his father and uncle had died at in their 50s, both to due to sudden cardiac death. In addition, the patient had a smoking history of 25 years. Relatives of the patient said that the patient had chest pain and sweating one week prior to visiting the ED buthad recovered after two hours. His vitalsat the time of admittance were as follows: afever of 36.2°C, heart rate of 103 beats/min, weak respiratory rate of 30 breaths/minute,blood pressure (BP) of 63/32 mmHg, and saturation of 90. Upon physical examination, the patient's Glaskov Coma Scale(GCS) score was9 (E2V3M4);heart sounds were deep in tone, and a jugular venous distension was present. We started intravenous (IV) fluid for hypotension. In the blood tests, the level of troponin T was 280.826 n/mL (reference range: 0–0.04 ng/mL), and the blood gas composition was as follows: pH of 7.32, partial pressure of carbon dioxide (PaCO2) of 35 mmHg, bicarbonate (HCO3) of 18 mmol/L,base excess (BE) of 6 mmol/L, and lactate of 8 mmol/L. An electrocardiogram (ECG) of the patient showed ST elevation and a right bundle branch block in the sinus rhythm and in D1, D2, aVL, V4, V5, and V6 (Figure 1).

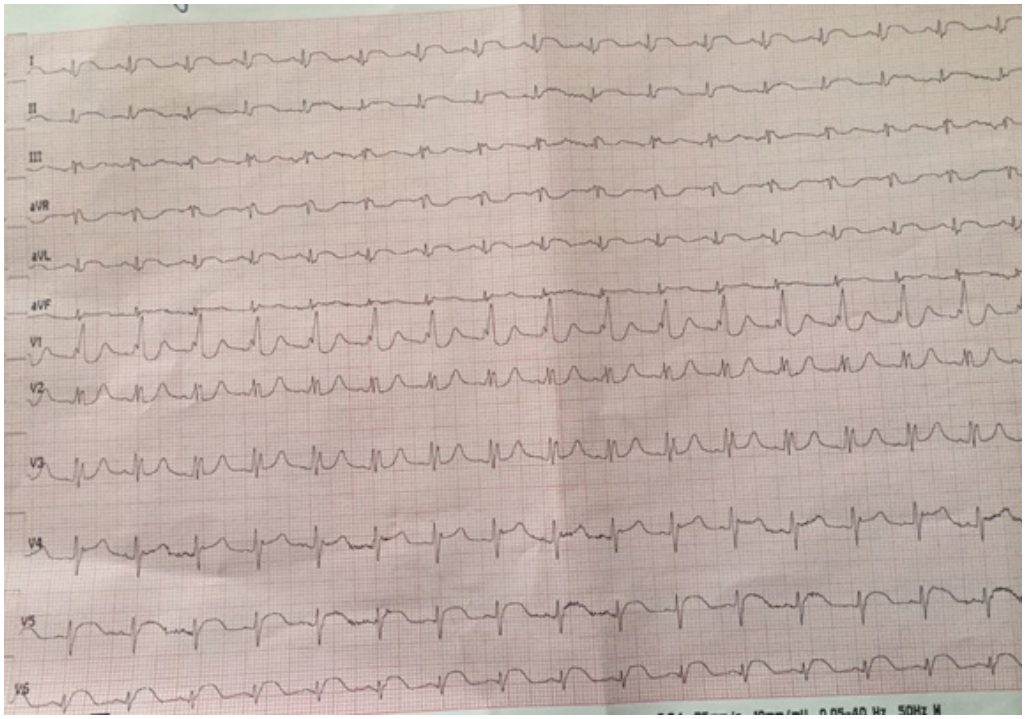

Figure 1: ST elevation in D1, D2, aVL, V4, V5, V6 and right bundle branch block.

In the bedside transthoracic echocardiography (ECHO), hypokinesia in the left ventricular anterior wall movements and pericardial effusion surrounding the heart and applying pressure to the right ventricle were detected. Computerized tomography (CT) was performed to exclude aortic dissection. Dissection was excluded in CT angiography, and there was pericardial effusion surrounding the heart evident in the CT scan (Figure 2).

We performed emergency pericardiocentesis with the diagnosis of cardiac tamponade. After approximately 150 cc of non-clotted blood was taken, the patient's BP became 113/78mmHg, pulse became 90 beats/min, GCS score became 15, and consciousness returned to normal. In the repeated ECHO, the pericardial fluid was shown to have begun wto decrease. The pericardiocentesis set was released in free drainage, and after 200 cc of incoming blood was administered, we removed the set upon the arrival of active bright blood. The patient then began to deteriorate again, with the BP falling to 53/22mmHg. We found that the ECHO-pericardial fluid increased and the patient then arrested. After 10 minutes of cardiopulmonary resuscitation (CPR), the return of spontaneous circulation (ROSC) was observed and an emergency thoracotomy was planned in ED;a diagnosis of free-wall rupture was reported, and cardiac surgery was required.We opened anterolateral emergency resuscitative thoracotomy in the supine position to the patient, who rearrested before being transferred to the operating room. We quickly opened the pericardium and discharged the accumulated blood. In the left ventricular anterior wall of the patient, diffuse LVFWR and diffuse necrosis were seen (Figure 3). Open cardiac massage was started. In the region with LVFWR, matrix suture was applied with 4-0 prolene. 50 minutes of open heart massage in the ED, but the patient died.

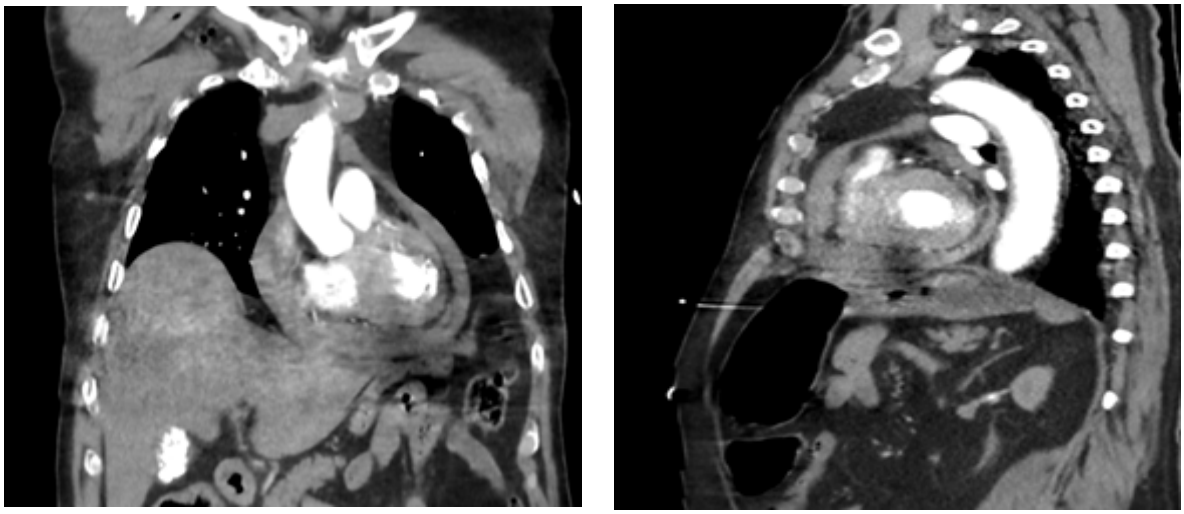

Figure 2: Image of pericardial effusion on CT.

DISCUSSION

In 85% of cases in which LVFWR is followed by AMI, the LVFWR occurs within the first week of AMI; however, it can be seen anywhere from the day of infarction to up to two weeks after infarction (Chetcuti, 2000). In this case, severe chest pain and perspiration about one week prior to admittance to the ED are evidence of rupture after AMI. Because LVFWR causes rapid cardiac tamponade and is fatal, these patients are usually diagnosed postmortem. Cardiac tamponade is an increase intrapericardial pressure due to intrapericardial fluid accumulation. The increase in intrapericardial pressure leads to decreased diastolic filling of the ventricles as well, causing decreased stroke volumes, decreased cardiac output, and poor tissue perfusion (Flounders,2003). Causes of LVFWR; include post-operative cardiac surgery, malignancies, AMI, pericarditis, bacterial infections, uremia, cardiac catheterization procedures, collagen

tissue diseases, chest trauma, hypothyroidism, and idiopathic factors (McMullan et al.2001).

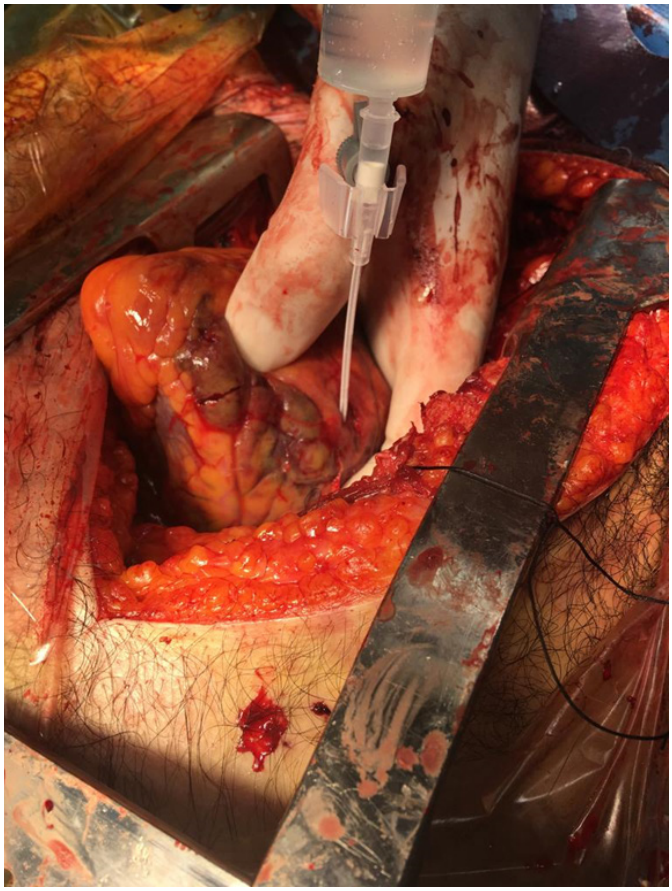

Figure 3: The line shows the necrotic areas. Arrows indicate LVFWR.

As cardiac tamponade is a life-threatening condition, it should be diagnosed and treated rapidly. The clinical condition is usually the same as that which was observed in our clinical case (e.g.,jugular venous distension, hypotension, and deep heart sounds). In our case, emergency pericardiocentesis was performed because the signs of tamponade developed suddenly. Patients presenting with LVFWR rarely have time for pericardiocentesis and surgery. Although emergency surgical treatment is accepted as the standard treatment for heart rupture, early mortality is still very high (Elhajj et al.,2020). Thoracotomy in the ED is performed after blunt or penetrant injuries, and the survival rate is between 1% and 14% (Hromalik et al.,2023). The highest survival rate inthe ED is in patients with penetrant cardiac wounds, especially if it these are associated with pericardial tamponade(-Fernandez et al.,2022; Hromalik et al.,2023) . The early recognition of cardiac tamponade, rapid pericardial compression, control of cardiac hemorrhage, and successful ED are key components inpatient survival (Yaginuma et al.,1997). Except for in cases of trauma, thoracotomy is not recommended in the ED; however, cases have been reported in which patients underwent bedside thoracotomy because of insufficient time to get to the operating room (Ito et al.,2000; Yaginuma et al.,1997). The fact that our patient's condition orsened suddenly and shiny blood was seen in the pericardiocentesis fluid suggested a diagnosis of LVFWR. As such, urgent thoracotomy was planned immediately, but the patient went into cardiac arrest and, accordingly, underwent emergency resuscitative thoracotomy. Following this,the pericardial fluid was drained, and a large number of infectious and ruptured areas were tried to be sutured; however, the patient died.

It should be kept in mind that there may cases in which LVFWR occurs with cardiactamponade. Pericardiocentesis should be performed without any delay in patients in whom cardiac tamponade has been found and LVFWR is being considered; if pericardiocentesis is insufficient, emergency thoracotomy and even bedside thoracotomy in the ED should performed.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication and images. Funding disclosure: The authors received no financial support for the research and/or authorship of this article. (If any, this must be declared.)

FINANCIAL SUPPORT

The authors received no financial support for the research and/or authorship of this article. (If any, it must be declared.)

CONFLICT OF INTEREST

There is no conflict of interest between the authors.

ACKNOWLEDGMENTS

We thank the patients and their relatives who agreed to participate in the study.

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