Heart rupture as a first symptom of acute coronary syndrome

Pęknięcie serca pierwszym objawem ostrego zespołu wieńcowego

Aneta I. Gziut[1], Piotr Suwalski[2], Joanna Latek[1], Robert J. Gil[1], Kazimierz Suwalski[2]

[1]Department of Invasive Cardiology, Central Clinical Hospital of the Ministry of Internal Affairs, Warsaw, Poland
[2]Department of Cardiosurgery, Central Clinical Hospital of the Ministry of Internal Affairs, Warsaw, Poland

Abstract

A 67-year-old woman with symptoms of cardiogenic shock is reported. Heart rupture as a complication of acute coronary syndrome was diagnosed. Cardiorrhaphy was performed. In the next stage coronary angiography was performed. Subtotal stenosis of a small marginal branch was responsible for this incident.

Key words: heart rupture, acute coronary syndrome

Streszczenie

Przedstawiono przypadek 67-letniej chorej przyjętej z wstępnym rozpoznaniem wstrząsu kardiogennego. Rozpoznano pęknięcie serca jako powikłanie ostrego zespołu wieńcowego. Kardiochirurgicznie zszyto uszkodzony mięsień sercowy. W kolejnym etapie wykonano koronarografię. Ustalono, że za zaistniały epizod odpowiada subtotalnie zwężona mała gałąź marginalna.

Słowa kluczowe: pęknięcie serca, ostry zespół wieńcowy

Case report

A female patient was brought to the hospital's Emergency Department (ED) by an ambulance after losing consciousness, preceded by anginal pain (tightness in the chest) radiating to the jaw. Approximately 5 days before the presentation, the patient suffered from a nonspecific pain felt in the throat region, and mild shortness of breath, which she thought were signs of infection. Until the hospitalization, the patient was treated only for hypertension.

At admission to the ED (11:01), the patient was in a moderate general condition (blood pressure 70/40 mm Hg, HR 117/min, oxygen saturation 100%). She denied stenocardia, dyspnoea, and reported only thoracic spine pain. In laboratory tests performed at admission, the following abnormal results were found: elevated levels of cardiac necrosis markers (CK 181 IU/l, CKMB 32 IU/l, TnI 8.308 ng/l), elevated inflammation markers (WBC 14000, CRP 107 mg/l). Additionally, anaemia (RBC 4.83 million, haemoglobin 10.2 g/dl,

haematocrit 34.7%) and hypokalaemia (3.09 mmol/l) were found. An electrocardiography (ECG) made in the emergency department revealed upsloping ST-segment elevation of 1 mm in leads I and aVL, concave ST-segment elevation of 0.5 mm in leads II, III and aVF, and ST-segment depression of 1 mm in leads V_1-V_3. The echocardiographic study performed in the ED revealed no left ventricular systolic dysfunction. Pericardial effusion (up to 23 mm next to the diaphragmatic part of the right ventricle) and a large amount (up to 12 mm) of fibrin were found. Additionally, signs of right ventricular overload were observed [dilated inferior vena cava (30 mm) with a limited respiratory variation]. Aortic dissection was excluded on the basis of a computed tomography scan of the chest. For further diagnostic procedures and treatment, the patient was transferred to the Cardiac Intensive Care Unit.

At admission to the Cardiac Intensive Care Unit (14:51), the patient's general condition was described as severe.

Corresponding author/*Adres do korespondencji:*

Aneta I. Gziut, Departament of Invasive Cardiology, Central Clinical Hospital of the Ministra of Internal Affairs, 137 Wołoska, 02-507 Warsaw, Poland, e-mail: anetagziut@poczta.onet.pl

Physical examination revealed stasis in the pulmonary circulation (Killip class II), jugular vein congestion, blood pressure 80/40 mm Hg, HR 70/min. Laboratory tests revealed a significant anaemia (RBC 3.39 million, haemoglobin 7.8 g/dl, haematocrit 26.7%), and decreasing levels of cardiac necrosis markers (CK 119 IU/l, CKMB 32 IU/l, TnI 5.761 ng/l). An ECG revealed negative T-waves in leads I, aVL, V_{1-3}, and ST-segment depression of 1 mm in leads V_{1-3}. Initially, the patient was referred for urgent coronary angiography. During the preparation of the patient for the study, a sudden cardiac arrest occurred. Resuscitation procedures were undertaken (short external cardiac massage, ventilation with the Ambu bag). A return of patient's circulatory and respiratory function was achieved. The echocardiography showed signs of cardiac tamponade (increased amount of pericardial effusion, compression of the right ventricle and atrium).

The patient was urgently transferred to the operating theatre of the Department of Cardiac Surgery. During the preparation of the patient for surgery, the patient again experienced cardiac arrest (electromechanical dissociation). The resuscitation procedures were continued. The chest was opened immediately through a longitudinal sternotomy. After the incision of the pericardium, more than 600 ml of partially clotted blood was evacuated (blood flow under pressure). Signs of inferoposterior wall myocardial infarction were seen macroscopically. Within this area, two sites of myocardial rupture were seen. The sites were sutured. Additionally, considering the bleeding from the tissue surrounding the rupture sites and the myocardial infarction zone, the Mikulicz surgical towels were placed inside the pericardial sac. After the bleeding was limited, they were removed, and the myocardial zone was wrapped around with absorbable haemostatic material. Thereafter,

drainage tubes and temporary epicardial wire electrodes were placed, and the chest was closed. The patient was transferred in a stable condition to the Intensive Postoperative Care Unit of the Department of Cardiac Surgery. After about an hour after the surgery, the patient was extubated. After the waking up, the patient was in full verbal and logical contact, with no neurological disorders. In the third day after the surgery, the patient underwent a control echocardiographic examination. It revealed wall motion abnormalities within the mid and apical segment of the laterobasal wall. No significant amount of pericardial effusion was found. During the consultation of cardiologists and cardiac surgeons, the patient was scheduled for coronary angiography. The procedure was scheduled at least 14 days after surgery. The patients received aspirin, bisoprolol, sartan. In the 8th day after surgery, the patient was discharged from the Department of Cardiac Surgery.

At admission to the Department of Invasive Cardiology, the patient was in a good condition (blood pressure 145/85 mm Hg, heart rate 90/min) and denied stenocardia, shortness of breath, reduced exercise capacity, and the occurrence of arrhythmias. An ECG revealed negative T-waves in leads I, II, aVL, aVF and V_{5-6}. Echocardiography showed hypokinesis of the mid and apical segments of the inferior wall, and ejection fraction of 55%. Coronary angiography revealed (Figures 1, 2) 50% stenosis in the distal segment of the circumflex branch spreading to the ostium of the subtotally occluded second marginal branch. Additionally, the study revealed intermediate (40-50%) stenosis in the proximal segment of the left anterior descending artery, and the distal segment of the right coronary artery. The proximal and mid segments of the right coronary artery showed aneurysmal enlargement, in which there was turbulent flow of a contrast

Fig. 1 A-B. Angiogram of left coronary artery
Ryc. 1 A–B. Angiografia lewej tętnicy wieńcowej

medium. Slow flow and residual contrast medium were also observed in the left anterior descending artery. Because of the small diameter of the vessel with significant stenosis (the marginal branch), the patient was qualified for conservative treatment. Given the abnormal flow in the coronary arteries, we decided to use clopidogrel, statin, eplerenone, and an increased dose of aspirin. In addition, we increased the dose of β-blocker and sartan. The patient was discharged home in a good condition with a recommendation of continuous outpatient cardiology care.

Discussion

One of the most dangerous complications that can occur in patients with acute coronary syndromes is a heart rupture (HR). According to the classification by Becker *et al.*, two types are distinguished: free wall rupture, and interventricular septum rupture [1]. The classification of types of HR is shown in Figure 3.

In the era of treatment of myocardial infarction with percutaneous revascularization, HR is no longer a common complication of an acute coronary syndrome. However, in the so-called "pre-reperfusion" era, this complication occurred in up to 6% of patients with acute coronary syndromes. It was associated with in-hospital mortality of 30%. With the development of pharmacological treatment (thrombolysis, antiplatelet drugs), and other treat-

Fig. 2. Angiogram of right coronary artery
Ryc. 2. Angiografia prawej tętnicy wieńcowej

ment methods (percutaneous reperfusion), the incidence of HR significantly decreased. In a retrospective observation, Figueras *et al.* found that the incidence of HR was reduced from 6% in the period before the year 1982 to 3.2% in the years 2001-2006 [2]. However, in the population from the GRACE study, the incidence of HR was
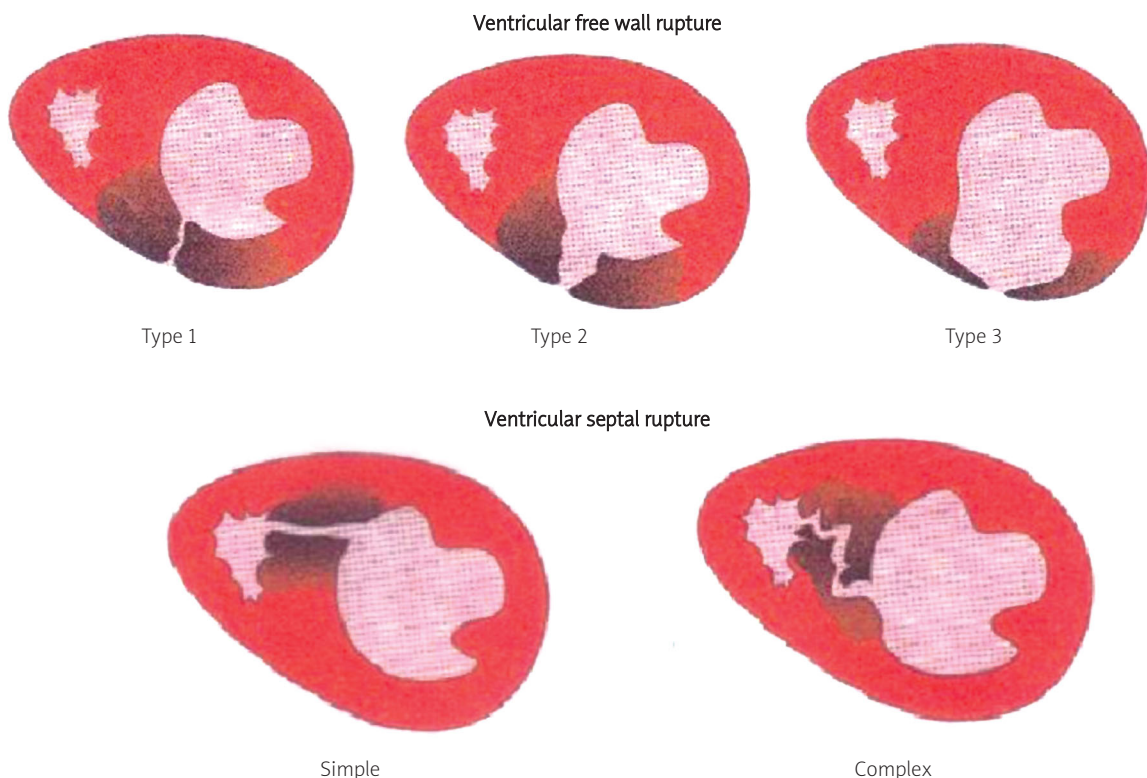

Ventricular free wall rupture

Type 1   Type 2   Type 3

Ventricular septal rupture

Simple   Complex

Fig. 3. Classification of heart rupture according to Becker
Ryc. 3. Podział pęknięcia mięśnia serca według klasyfikacji Beckera

Table 1. Incidence of heart rupture related to treatment strategy
Tabela 1. Częstość występowania pęknięcia serca w zależności od strategii leczenia

Variable		Incidence of HR (%)
Fibrinolysis (–), PCI (–)		1.2
PCI		0.7
Fibrinolysis		1.1
Time from onset of symptoms to fibrinolysis [h]	0-2	0.7
	2-4	1
	4-12	1.7
	> 12	2.2

approximately 1% [3]. Interestingly, it was found that its incidence depends on the type of acute coronary syndrome (0.9% in STEMI, 0.17% in NSTEMI, and 0.25% in unstable angina). However, despite the progress of diagnostic and surgical techniques, in-hospital mortality (5.6%) is high both in patients with cardiac free wall rupture (80%) and those with septum rupture (41%). In the multivariate analysis, the independent predictors of HR were STEMI, older age, female gender, previous stroke, tachycardia, hypotension, elevated markers of cardiac necrosis, and a high score on the GRACE Scale. In contrast, previous myocardial infarction and the use of β-blockers in the first 24 h after the onset of myocardial infarction were associated with a risk reduction [3].

When one analyses the risk factors for HR, it is worth considering the presence of coronary collateral circulation, the impact of fibrinolysis, and the time from the onset of symptoms to fibrinolysis. Undoubtedly, heart rupture is affected by a total occlusion of a coronary artery, without concomitant collateral circulation. This explains why HR is more frequent in patients with first myocardial infarction (especially STEMI). Therefore, it was thought that reperfusion of the infarct-related artery would result in reduction of this complication. However, the results of conducted studies were surprising (Table 1) [3]. They confirmed that early thrombolysis and reperfusion have a protective effect. However, delayed reperfusion is associated with an increased incidence of intramyocardial bleeding, and consequently with an increased risk of heart rupture. In addition, inadequate myocardial reperfusion occurs – increased blood flow in the already irreversibly damaged muscle.

Primary coronary angioplasty leads to restoration of coronary blood circulation more often than fibrinolysis, and the risk of bleeding is lower. This explains why mechanical complications occur less frequently after PCI than after fibrinolysis. However, if PCI is unsuccessful, the risk of HR increases.

Prompt diagnosis in patients with suspected cardiac free wall rupture and acute tamponade of unknown origin should lead to direct transfer of a patient to the operating room [4]. In such cases, performing diagnostic studies other than echocardiography (e.g. coronary angiography) is not recommended. The time to open the pericardial sac should be reduced to the necessary minimum. For this reason, only in centres with cardiology and cardiac surgery facilities are these patients likely to be effectively treated. In severe tamponade, pericardiocentesis can be the "last chance" manoeuvre before transport to the operating room. The use of the intra-aortic balloon pump (IABP) depends on the patient's condition, and can help in extreme cases. However, note that the use of IABP causes a significant delay of cardiac surgery procedures, and IABP does not affect the mechanism of tamponade.

Several treatment methods of acute free wall cardiac rupture are described in the literature [4-9]. However, because they are mainly reports of single cases, the decision about the choice of method depends on the experience of a cardiac surgeon. Undoubtedly, the following factors have an influence on the choice of type of surgery: patient's haemodynamic status, location of bleeding, the extent of myocardial infarction, and the presence of other comorbidities (such as ventricular septal defect). The available data indicate that in order to obtain significant reduction of bleeding, cardiopulmonary bypass should be avoided, and surgical suturing should be limited to the minimum. It is also worth using other haemostatic methods. All sutures should be strengthened with the use of a felt or pericardial pad. In the "classical" method, continuous horizontal "mattress" sutures on two felt strips are used. An important limitation of this method is the fragility of the heart muscle tissue [4]. The removal of necrotic tissue from the ventricle wall, and the use of single horizontal "mattress" sutures with felt or Dacron patches, reinforced by a second layer of monofilament sutures, have been reported primarily in patients undergoing closure of septal rupture [8, 9]. Núñez *et al.* described a method based on suturing of the rupture site with single horizontal sutures with felt or Dacron pads and subsequent suturing to the "healthy" epicardium (with a wide margin of healthy tissue) with a Teflon patch, bovine pericardium or the patient's own pericardium [7]. The researchers emphasized particularly good haemostatic properties of this method, especially when combined with the use of biological and non-biological tissue adhesives [9]. The reports of the successful use of fast-drying glues (cyanoacrylate adhesives), well known to all as the glues advertised as necessities in a household, together with felt or Teflon patches, are very interesting [8, 9].

Although the incidence of HR is currently negligible, the case of the present patient best shows that this problem still occurs. Undoubtedly, it is not typical. The present patient interpreted her symptoms as a respiratory infection and therefore the myocardial infarction was not diagnosed. It was a coincidence that a free wall heart rupture, and symptoms of tamponade, occurred in the hospital, which enabled the patient's survival. However, we have to admit that for the entire cardiological and cardiac surgery

team caring for the patient, the result of coronary angiography was surprising. We expected HR to have been caused by an occlusion of an artery with a much higher diameter.

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