# Cardiac tamponade with acute hypovolaemic shock induced by a right ventricular lesion during implantation of a PHILOS SR cardiostimulator and a SELOX ST 60 ventricular electrode – a case report



Tamponada serca z objawami ciężkiego wstrząsu krwotocznego po perforacji prawej komory serca w przebiegu implantacji kardiostymulatora PHILOS SR oraz elektrody komorowej SELOX ST 60 – opis przypadku

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## Abstract

A cardiac (pericardial) tamponade (PT) is an acute, life-threatening condition induced by compression of the cardiac chamber, caused by rapid blood accumulation in the pericardial sac, resulting in reduced atrial and ventricular filling and subsequently cardiac output. Severe acute pericardial blood pressure can cause a drop in the haemodynamics (blood pressure), cardiac shock, abnormal heart rhythms or death. In the following article we present the case report of an acute, life-threatening cardiac tamponade with acute hypovolaemic shock, in an 80year-old patient undergoing cardiostimulator and ventricular electrode implantation.

**Key words:** pericardial tamponade, hypovolaemic shock, invasive medical procedure.

## Introduction

An 80-year-old patient with a fixed atrial fibrillation in the history was admitted to the cardiology ward of a Łódź hospital due to heart rhythm disorders and fainting in the history. Atrial fibrillation in ECG, right bundle branch block, without fresh ischaemic changes. In 24-hour ECG monitoring with Holter method – atrial fibrillation, with a mean chamber activity of 77/mm, without bradycardia episodes, 606 additional ventricular beat, no changes in the ST section range.

#### Streszczenie

Tamponada serca jest ostrym stanem zagrożenia życia, wywołanym przez gwałtowne gromadzenie się krwi w worku osierdziowym. Powoduje wzrost ciśnienia śródosierdziowego, w następstwie czego upośledzeniu ulega napełnianie przedsionków i komór, co skutkuje zmniejszeniem rzutu minutowego serca, załamaniem się hemodynamiki układu krążenia (gwałtownym spadkiem ciśnienia tętniczego), rozwinięciem objawów wstrząsu kardiogennego, zaburzeniami rytmu serca lub śmiercią. W pracy przedstawiono przypadek zagrażającej życiu ostrej tamponady serca z objawami ciężkiego wstrząsu krwotocznego, która wystąpiła u 80-letniego pacjenta poddanego zabiegowi wszczepienia kardiostymulatora i elektrody komorowej.

**Słowa kluczowe:** tamponada serca, wstrząs krwotoczny, inwazyjne procedury lecznicze.

On 23 October, 2008, in a planned course, the patient was implanted with an artificial PHILOS SR cardiostimulator together with a SELOX ST-60 ventricular electrode. Several hours after the procedure the patient demonstrated arterial blood pressure drop to an undeterminable value. After fast transfusion of 500 ml of 10% HES, and 1000 ml of crystalloids the patient's haemodynamic condition remained unchanged. 3U of compatible packed red blood cells were ordered. In a heart USG performed at the bedside in the R ward, acute heart tamponade was diagnosed, with

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pericardium layers delamination up to 30 mm. Pericardiocentesis was performed by evacuating 200 ml of blood out of the pericardial sac. 1 U of erythrocyte concentrate and 500 ml of Ringer solution were transfused. In spite of treatment the patient's condition did not improve; in a subsequent heart USG rapid growth of fluid in the pericardial sac was observed. All considered, after phone consultation with the doctor on duty, the patient was transferred to the Cardiosurgery Clinic of the 1st Chair of Cardiology and Cardiosurgery in order to continue the treatment in emergency course, with securing of 2 U of packed red blood cells.

## **Description of the case**

The patient was admitted to the Anaesthesiology and Intensive Cardiological Therapy Clinic of the 1st Cardiology and Cardiosurgery Department on 23 October, 2008, with a diagnosis of cardiac tamponade (2000 ml of blood from a pericardial sac drainage), after right ventricular perforation during cardiostimulator implantation performed in a Łódź hospital. On arrival at the hospital, the patient was in serious haemorrhagic shock. Conscious, with limited contact, the patient's respiration at efficiency border, sinus tachycardia of 120/min, the pulse palpable on carotid arteries; after Levonor infusion systolic RR was 70 mg Hg, circulatory centralization, serious metabolic acidosis, Hgb-6.8, INR-2.5, aPTT-122 s. The patient was operated on in emergency. The site of haemorrhage from the heart right chamber was treated, 1000 ml of blood from the pericardial sac was evacuated.

During the operation 3 U of packed red blood cells, 3 U of frozen plasma, 1500 ml of colloids and crystalloids were transfused. After the operation (operational 24 hrs): CMV with FiO<sub>2</sub> 0.5, RR 100/60 mm Hg on Levonor infusion, metabolic acidosis, significant post-operative drainage (> 300 ml/h), in laboratory examinations – serious coagulative disorders: aPTT > 300 s, fibrinogen 0.7 g/l, INR 4, PLT 24 000, Hgb 5.5 g/dl. 4 U of packed red blood cells, 5 U of frozen plasma and 10 U of thrombocyte concentrate – thus achieving reduction of drainage dynamics and the gradual normalization of coagulation system parameters: aPTT 66 s, TT 25 s, PLT 112 000, fibrinogen 1.0 g/l. In the 24-hour period after the thoracotomy and the

treatment of haemorrhage from the damaged right ventricle, the patient's condition was still serious. Unconscious, lack of logical contact, CMV from FiO<sub>2</sub> 0.5, systolic RR 100–110 mm Hg supported by catecholamines infusion: adrenaline and Levonor in modified doses. Heart activity 90–100/min, secured by external pacemaker. Total drainage from operational 24 hours 3350 ml. Remarkable anaemia: Hgb 5.6 g/l – again, prolonged coagulation system parameters.

There were transfused: 5 U of packed red blood cells, 5 U of frozen plasma, U of cryoprecipitate – thus achieving coagulative parameters: aPTT 56 s, TT 29 s, platelets 66 000, fibrinogen 1.2 g/l, INR 1.73, the level of Hgb 11.1 g/l, and a distinct stabilization of the circulatory system allowing for Levonor iv infusion. 24-hour diuresis (forced) 1000 ml, drainage 850 ml. In USG examination: generalized, slightly worse heart muscle contractility of left ventricle with EF 53%, IM I°, enlarged left ventricle, IT and /II°, an electrode in the enlarged right atrium and right ventricle, relaxation disorders, delamination of the pericardial sac up to 1.1 cm with precipitated fibrin in the apex 1.1 = 1.2 cm. On the second day after the operation, the patient's condition was still very serious. Lack of logical contact. CMV ventilation, RR 110/60 mm Hg, CVP 9 mm Hg, arrhythmic heart activity, periodical stimulation with the pacemaker. Htk 32%, PLT 46 000, INR 1.8, ALAT 1854 U/I, ASPAT 1973 U/I, urea 87 mg/dl, crea 2.8 mg/dl, potassium 5.9 mmol/l, diuresis 1050 ml. On the third day after thoracotomy and haemorrhage treatment for right ventricle damaged in the course of cardiostimulator implantation, the patient opens the eyes periodically, still there is no logical contact, remains on controlled respiration. RR 110-120/60-70 mm Hg, HR 130-90/ min - total arrhythmia. Cordarone iv infusion was added together with Mg<sup>2+</sup>. UREA 133 mg/dl, CREA 4.7 mg/dl, ALAT 1697 U/I, ASPAT 1031 U/I, INR 2.8, potassium 5.2-6.2 mmol/l – after Resonium dosage the reduction of potassium level to 4.87 mmol/l. Diuresis forced by furosemide infusion - 2400 ml in 24 hours.

During the next days after the operation: symptoms of kidney failure, increasing levels of UREA 212–241–173 mg/l, creatinine 6.8 = 7.4 mg/l, periodical hyperkalaemia = 5.5 mmol/l, hyponatraemia and hypochloraemia. Abundant diu-



Ryc. 1. TTE, apical projection. The arrows denote liquid



Ryc. 2. TTE, long axis, sternal projection. The arrows denote liquid

resis (5000–5500 ml/24 hrs) forced by a constant infusion of mannitol and furosemide. From the eighth day the patient was in logical contact. On the ninth day after the thoracotomy the patient was transferred to the Intensive Care Unit at the parent hospital in order to continue the treatment (dialysis therapy included).

## Discussion

Cardiac tamponade is a rapid, life-threatening condition, caused by sudden blood accumulation in the pericardial sac [1, 2]. Fast accumulation of blood leads to heart compliance lesion [1-3], acute right ventricular failure [2-4], drastic reduction in stroke volume [1, 2, 4], cardiac shock [1-4], haemodynamic disorders resulting in multiorgan failure [5] and death [1, 6, 7]. All conditions in the course of which acute blood extravasation into the pericardial sac occurs, i.e. stab wound of the heart walls [7, 8], rupture of a heart wall in myocardial infarction [9], ruptures with perforation to the pericardium of ascending aorta aneurysm [10], cardiac surgery complications [1, 4], and invasive diagnostic and medicinal procedures in the area of large blood and heart vessels [5, 11, 12], may be complicated by an acute heart tamponade [1, 5], often of a dramatic course. The factor directly affecting the survival and prognosis of patients with an acute heart tamponade is the time passing from its onset to the moment of intervention [4, 5, 8]. In the case of an injury penetrating the right ventricle it is an operation leading to surgical treatment of the haemorrhage site, and the whole diagnostic and medicinal process should be limited to the necessary minimum [4], and consist of diagnosis confirmed by a clinical examination: 2D echocardiography [13], transfusion of at least 2 U of packed red blood cells, decompression of pericardial sac [6], volume resuscitation (transfusion of colloids and crystalloids) [14] and the urgent transfer of such a patient to a suitable reference medical centre.

In the case report above the patient with an acute tamponade, after right ventricular perforation undergoing cardiostimulator implantation, at the time of admission to our clinic was in a critical condition with developed symptoms of acute hypovolaemic shock. Shock is a state in which the oxygen supply is highly insufficient to maintain proper activity of many organs, and the clinical process may be complicated by disseminated intravascular coagulation (DIC), the syndrome of acute respiratory distress of adults (ARDS), ischaemic myocardial dysfunction, and liver and kidney failure [15].

## Conclusion

In the case report described above, in spite of the immediate surgical intervention, intensive treatment, packed red blood cells, plasma, thrombocyte concentrate, cryoprecipitate, and colloids and crystalloids transfusion in the peri- and postoperative period, there were serious accompanying coagulative disorders, which finally were controlled, together with acute kidney failure, which was successfully managed with a necessary aggressive treatment by dialysis therapy.

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