

# Prosthetic Mitral Valve Thrombosis Presented as a Pulmonary Embolism with Obstructive Shock in Emergency Department

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**Abstract:** We are describing a case of acute prosthetic valve thrombosis, presented in emergency department with clinical findings of pulmonary embolism. This case, underlines the best diagnostic accuracy of the chest CT scan, comparison to the trans thoracic echocardiogram for the differential diagnosis.

**Keywords:** Pulmonary embolism, MIP CT scan, trans thoracic echocardiogram, prosthetic valve thrombosis, emergency management.

## INTRODUCTION

Mechanical prosthetic valve thrombosis is a life threatening complication of artificial valve insertion, which need an immediate intervention. The presenting signs and symptoms are somewhat variable, to avoid catastrophic complications, valve replacement or debridement, or thrombolysis in the correct setting, must be immediately performed.

## CASE

A 74-year-old woman presented with progressive dyspnoea, asthenia, and abdominal pain for two days, in addition to nausea and vomiting. She complained permanent atrial fibrillation and was on Acenocumarol, Furosemide, Ramipril, and Bisoprolol.

Ten years earlier, the patient had undergone mitral valve replacement with a St. Jude bileaflet mechanical prosthesis.

Four months before, an acute thrombosis of the left arm occurred, due to phlebitis. During hospitalization, her chest radiography was performed, which showed cardiac enlargement and no pleural effusions. CT chest scan and trans thoracic echocardiogram confirmed a free mediastinum, left ventricle enlargement and a mild reduction of LV systolic function (EF 45%); the prosthetic valve was inserted correctly and was working in good condition, with no insufficiency or stenosis, and no pericardial effusions.

## CLINICAL FINDINGS

On admission in ED, her vitals were as follows: she was afebrile (36,2° Celcius); tachycardiac (HF 126 beats per minutes), with severe hypopnea (systolic brachial pressure 60mmHg); respiratory rate 30 breaths per minutes; non invasive oxygen saturation 86% (FiO<sub>2</sub> 21%). Electrocardiogram

showed atrial fibrillation, right axis deviation, left ventricle hypertrophy. The physical examination revealed clinical signs of RV overload (swollen jugular), pale skin, peripheral cyanosis with delayed capillary refilling and cold extremities, abolition of the pulmonary sounds in right base.

Irregular cardiac rate rhythm at the cardiac auscultation, padded tones, pauses not evaluable, soft abdomen, non distended, non tender, epigastric pain at palpation, focal and not radiate, normal bowel sounds. No hepato-splenomegaly, no flanks pain, no pitting edema and no signs of deep venous thrombosis.

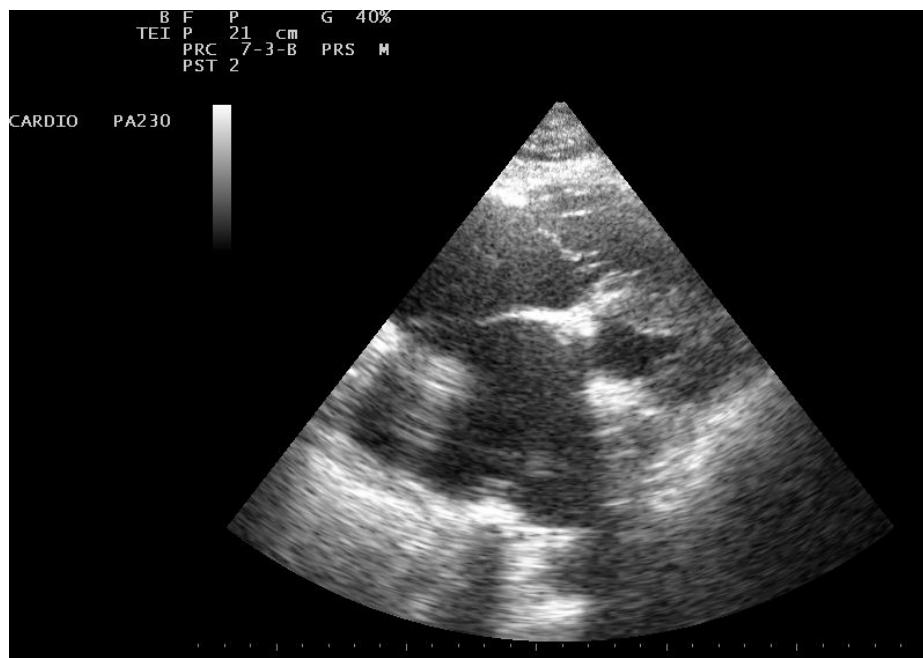
After her admission, she performed echocardiogram, which revealed a bi-atrial enlargement, an hyper echogenic, not well defined image in left atrium (Fig. 1), a prosthetic mitral valve with higher trans valvular gradient (50-55 mmHg), functional mitral valve area of 0,5 cm<sup>2</sup> (Fig. 2), left ventricular hypertrophy with normal cavity dimension, paradox movement of inter ventricular septum, right ventricle enlargement (30-40 mm), tricuspidal regurgitation, PAP'S 60 mmHg, mild pericardial effusion, and an enlargement of inferior caval vein (30 mm measured in front-posterior projection) with severe decrement of caval index (<5%). Pulmonary echography showed no wet findings into the pulmonary parenchyma.

## INVESTIGATION AND RESULTS

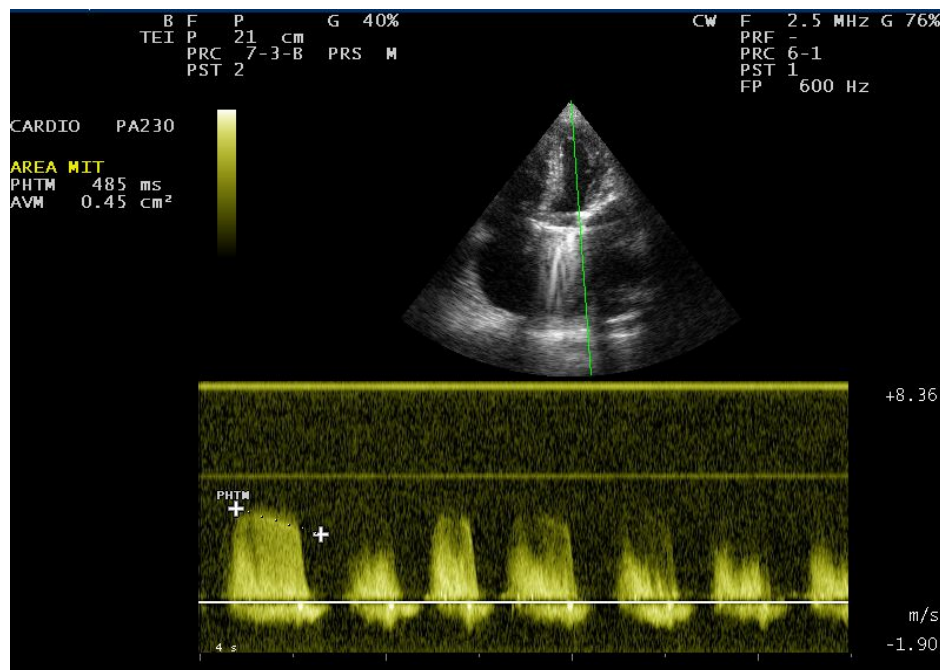
Laboratory investigation were as follows; white blood cell count (WBC) of 18,500 cells/mm<sup>3</sup>; haemoglobin, 11.4 mg/dl; hematocrit, 36%; INR, 2.3; D-Dymer, 1339ng/ml; BNP, 1500pg/ml; Troponin I, 0,29 ng/ml; CK-MB, 8,46 ng/ml; Myoglobin, 201 ng/ml; Emogas-analysis revealed ipoxemia, hypocapnia and metabolic acidosis (pH 7,19; PO<sub>2</sub> 60 mmHg; PCO<sub>2</sub> 27mmHg; HCO<sub>3</sub> 12,5 meq/l) with increased anion Gap (30) and lactate (>5 meq/l).

Renal function revealed a functional decrement, creatinin 4, BUN 115.

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**Fig. (1).** Sub-xiphoid echocardiography shows a poorly defined formation in left atrium.



**Fig. (2).** Four chamber trans thoracic echocardiography reveals a decrement in surface of mitral valve.

## MANAGEMENT

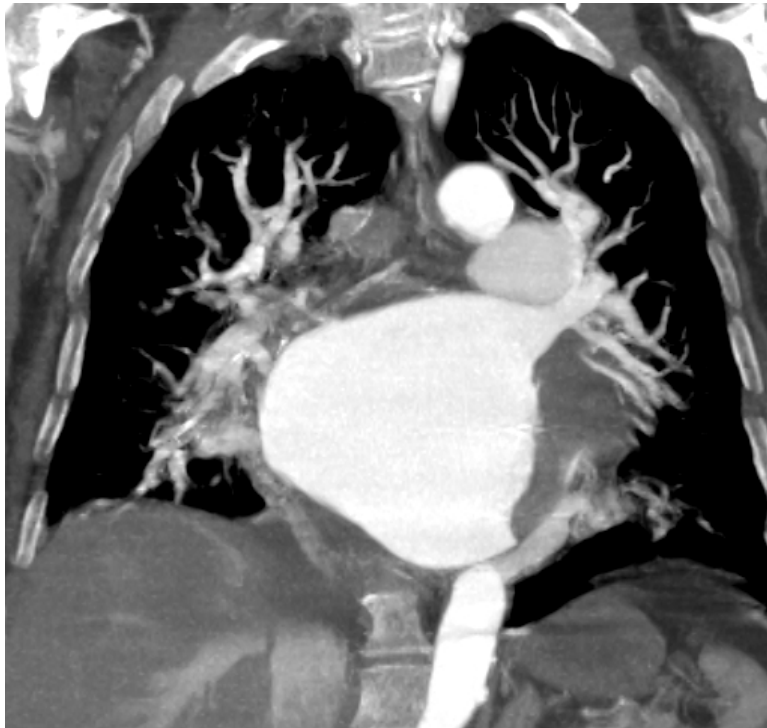
First line therapy was high-flux Oxygen and crystalloid solution (500 ml), such as ensuring adequate oxygenation and vascular perfusion of tissues.

An internal jugular central venous catheter has been placed, and all the infusions have been passed into this way.

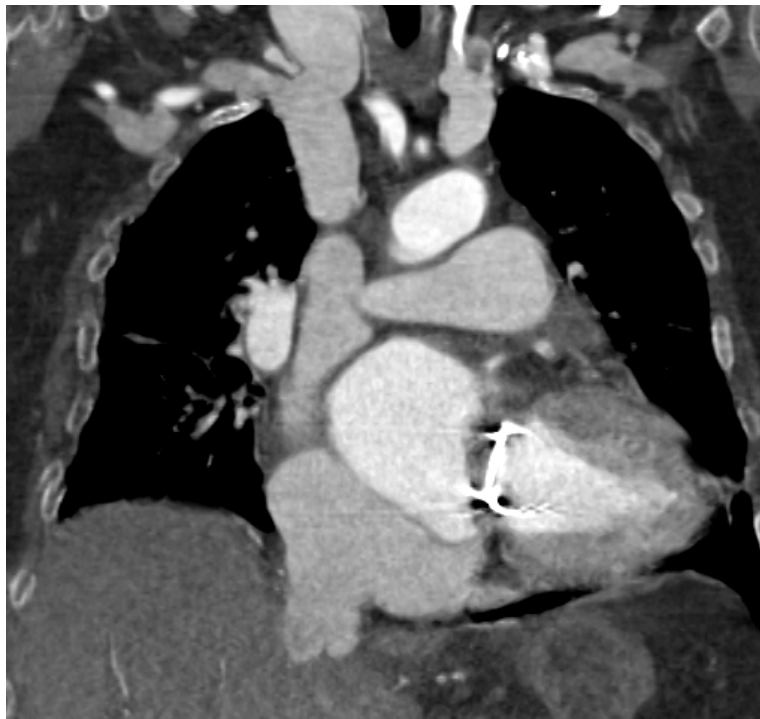
Before starting the thrombolytic therapy for massive pulmonary embolism with hemodynamic instability, a CT scan with contrast was performed [1].

The radiologic examination with contrast revealed the pulmonary arteries free from embolism, an important right heart failure, enlargement of inferior caval vein and supra-hepatic veins reflux.

Left atrium was increased of volume with an hypo density within and expansion of the left auricula by a thrombus that dealt in 1/3 of the atrium and in the whole left auricula (Fig. 3 and Fig. 4). Furthermore, was detected bilateral pleural effusion, and no signs of aortic dissection.



**Fig. (3).** Coronal MIP Computer Tomography (CT scan) reveals the dilatation of left atrium and the thrombus in left auricula (arrow).



**Fig. (4).** Coronal CT scan shows the involvement of the mitral valve by the thrombus (arrow).

After the diagnosis of thrombosis on prosthesis, the patient was thus transferred to the cardiothoracic theatre for urgent surgery. She was put on extra body circulation, the thrombus was removed and a new prosthetic valve was successful placed. After the operation, the patient was transferred to cardio-thoracic intensive care unit and maintained circulatory support for three days.

#### CONCLUSION AND LESSON

This case highlights the difficulty in early diagnosis of acute thrombosis of mitral valve prosthesis in emergency department. The incidence of mitral valve thrombosis is low, ranging from 0.1% to 5.7% per patient per year, and accounts for approximately 14% of all valve re-operations.

Patients who receive inadequate anticoagulation, particularly with prosthetic mitral, have an increased risk for thrombus formation [2].

The risk of thrombosis remained consistent, despite the anticoagulant therapy is performed; in this case, the INR was not perfectly in range (should be maintained between 2.5 and 4.9) [3]. The first symptoms the patient complained, were abdominal pain, nausea and vomiting, caused indirectly by the right heart failure, hepatic congestion and hepatic capsule irritation [4].

The clinical feature of this case were very indicant for pulmonary embolism, the emergency physicians followed the pre-clinical Canadian (Well's/ Geneva) score to stratify the possibility to have a pulmonary embolism, in this case the probability was high and D-Dymer, BNP and cardiac biomarkers supported the diagnose [1].

Echocardiogram identified a right ventricular enlargement (end diastolic diameter >30 mm), hypokinesia of the free wall and Mc Connell sign (paradoxical inter-ventricular sept movement), the tricuspid incompetence was moderate-severe and the PAP'S was increased (>25 mmHg).

The emergency management of massive pulmonary embolism or sub-massive with hemodynamic instability consider the thrombolysis with Streptokinase or Urokinase [1]. after the evaluation of risk factors [5], but before examine the myocardial stretching (BNP), the presence of active co-

agulation pattern (D-Dymer), and right ventricle dysfunction (Echocardiography).

In this case the "wait and see" approach give the possibility to have a better diagnosis and an early surgical.

Trans-thoracic Echocardiography is the most widely used diagnostic tool in patients with suspected PHVT: the trans prosthetic pressure gradient can be estimated and a reduction of the effective area of the valve orifice detected. In most cases an echogenic mass is observed on the prosthetic valve surface at the site of stenosis [6]. Although it is the quickest and most practical diagnostic methodology to make a diagnosis of valve thrombosis, in this case, the heart CT scan has been the most sensitive and specific diagnostic approach.

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Received: November 05, 2009

Revised: February 05, 2010

Accepted: February 10, 2010

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