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Case Report

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Findings of Severe Mitral Stenosis Complications by POCUS

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Abstract

Aim: To present the very late clinical and echographic outcomes of an untreated rheumatic mitral stenosis. Summary: Moroccan young man with no past medical history presented at emergency room with subacute dyspnoea, pitting edema, oppressive chest pain and chills. Physical exam revealed signs of hypervolemia, jaundice and intense pan-diastolic murmur. Laboratory tests showed mild pancytopenia, elevated liver enzymes and altered coagulation. Atrial fibrillation and bilateral Kerley B lines were described by ECG and X-ray. Bed side echocardiography showed an atrial dilation with severe rheumatic mitral stenosis and global systolic dysfunction. Two months after voluntary discharge, presented with exacerbated symptoms and shock signs. POCUS exam disclosed a giant left atrial thrombus, right ventricle overload and bilateral pulmonary consolidations that were described by CT as superinfected infarct areas with bilateral thromboembolism. Despite medical treatment, the patient finally expired few days later.

Conclusion: Massive intra-atrial thrombus in aneurysmal atrial dilation due to rheumatic mitral stenosis are exceptional findings with high impact in the management when promptly viewed under POCUS exam.

Case Presentation

A 43-year-old Moroccan man, settled in Spain thirteen years ago, was admitted to the Emergency Department (ED) presenting progressive dyspnoea, lower limbs edema, intermittent-oppressive chest pain, and ten days of chills without proved fever. Initial vital signs showed tachycardia of 135 bpm and physical exam revealed conjunctival jaundice, prominent jugular vein with hepatojugular reflux, and pitting edema. At auscultation, appeared a prominent apex pan-diastolic murmur, and palpable precordial thrill. Cabinet tests showed an atrial fibrillation (AF) by ECG, and mild pulmonary congestion images by chest X-ray (Figure 1). Laboratory findings were mild normocytic normochromic anaemia, elevated liver enzymes (total BR 42 µmol/l, ALT 391

UI/l, AST 113 U/l, GGT 77 UI/l, and ALT 83 UI/l), prolonged Prothrombin Time (PT), mild thrombocytopenia, and hyponatremia. Reactive C-protein was slightly above normal (66 mg/l), with normal lactate and procalcitonin levels. A trans-thoracic bedside echocardiography revealed severe Rheumatic Mitral Stenosis (RMS) with a valve area of 0,5 cm^2 and mild mitral regurgitation, atrial aneurysmal dilation of 73 mm, and severe functional tricuspid regurgitation, which allowed to measure a pulmonary artery pressure of 84 mmHg. Also was detected a global systolic dysfunction with an estimated ejection fraction of 32%. The patient was admitted at the Intensive Care Unit (ICU) initiating furosemide, enoxaparin and esmolol infusion under monitoring. An initial clinical improvement was observed but, after few hours, hemodynamic worsening occurred. The patient came up with hypotension and increasing heart rate, which caused oppressive chest pain recurrence. Cardiac surgery and percutaneous mitral valve replacement were proposed who rejected the procedure, even under the assistance of a translator explaining. He called for and signed an informed voluntary discharge, refusing to any medical treatment, too. Two months later, the patient was newly admitted at ED describing progressive dyspnoea, fever and daily sweating for ten days. At this time, the patient appeared obnubilated (GCS 9), tachypnoeic, hypervolemic, and poorly perfused with prolonged capillary refill, *livedo reticularis* and hemodynamically unstable (85/60 mmHg). The ECG (Figure 2) showed signs of right ventricular overload that were not observed previously. Laboratory tests revealed d-dimer elevation (1600 ng/ml), worsening of the all- preceding thrombocytopenia, PT and liver enzymes, as well as elevated sepsis parameters and lactic acidosis (pH 7,25 and lactate 3,5 mmol/l). Point of Care Ultrasound (POCUS) exam from apical four-chamber modified view demonstrated a very large left atrial thrombus, which occupied nearly 50% of this cavity and a thickened mitral valve (Video 1). From parasternal short axis view, indirect signs of pulmonary hypertension (Video 2) were seen.

Given the clinical suspicion of pulmonary embolism, a contrasted CT was performed, confirming the diagnosis (Figure 3). Despite efforts, including non-invasive mechanical ventilation, vasoactive drugs, anticoagulation and broad-spectrum antibiotics, the patient developed an adverse clinical outcome, and died two days after admission.

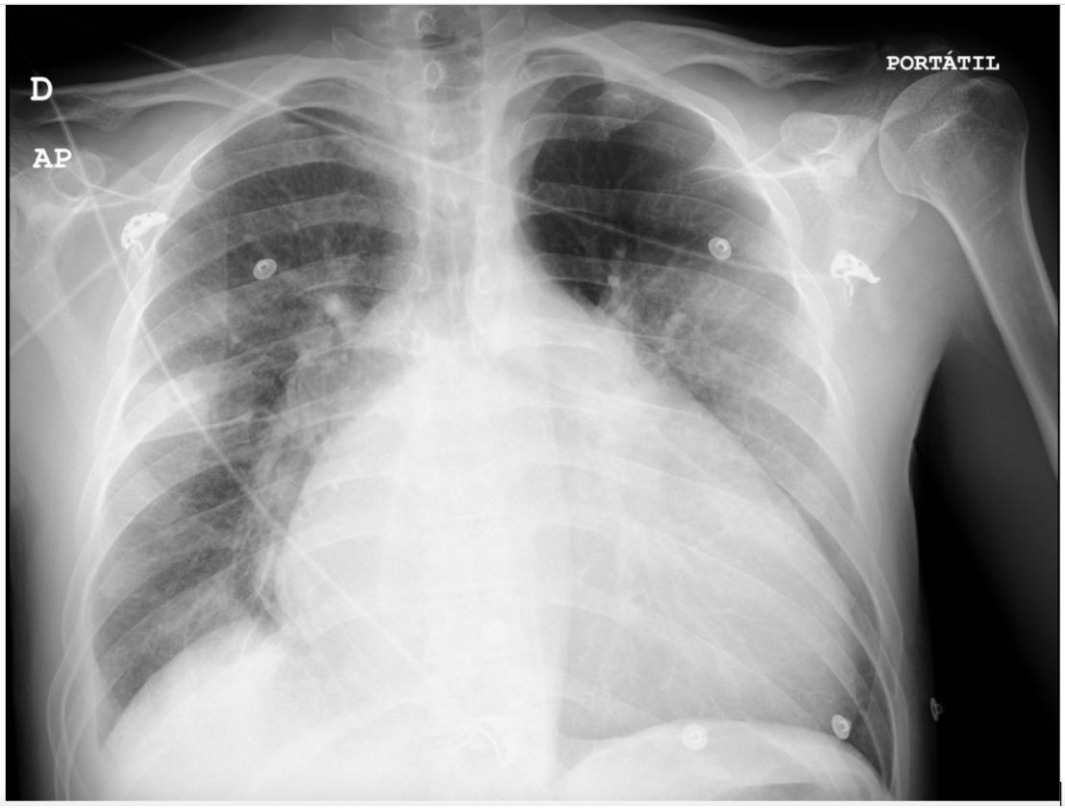


Figure 1: Chest X-ray: increased cardiothoracic ratio and vascular redistribution signs with Kerley B lines and upper lobes venous congestion.

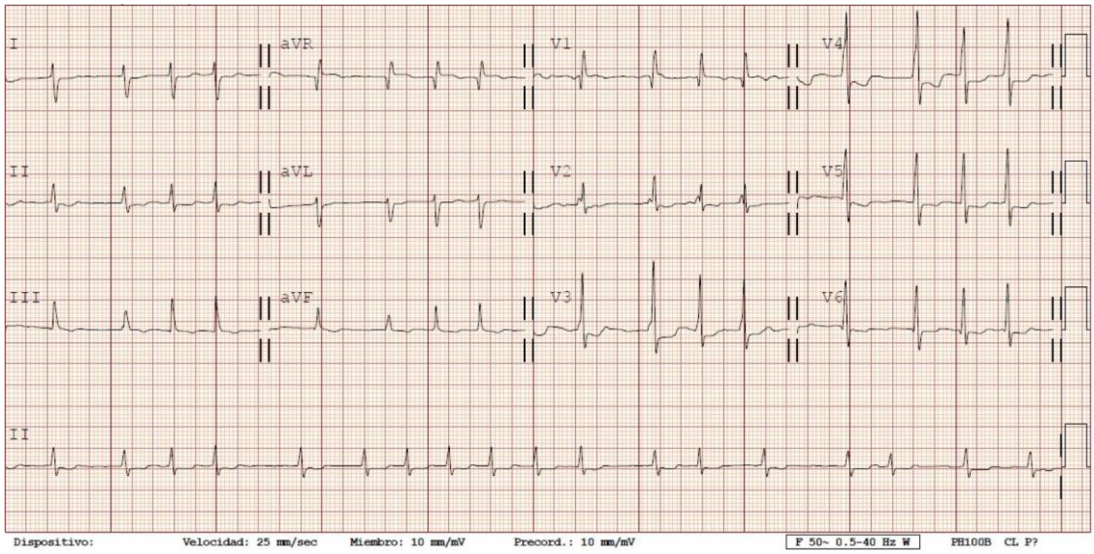
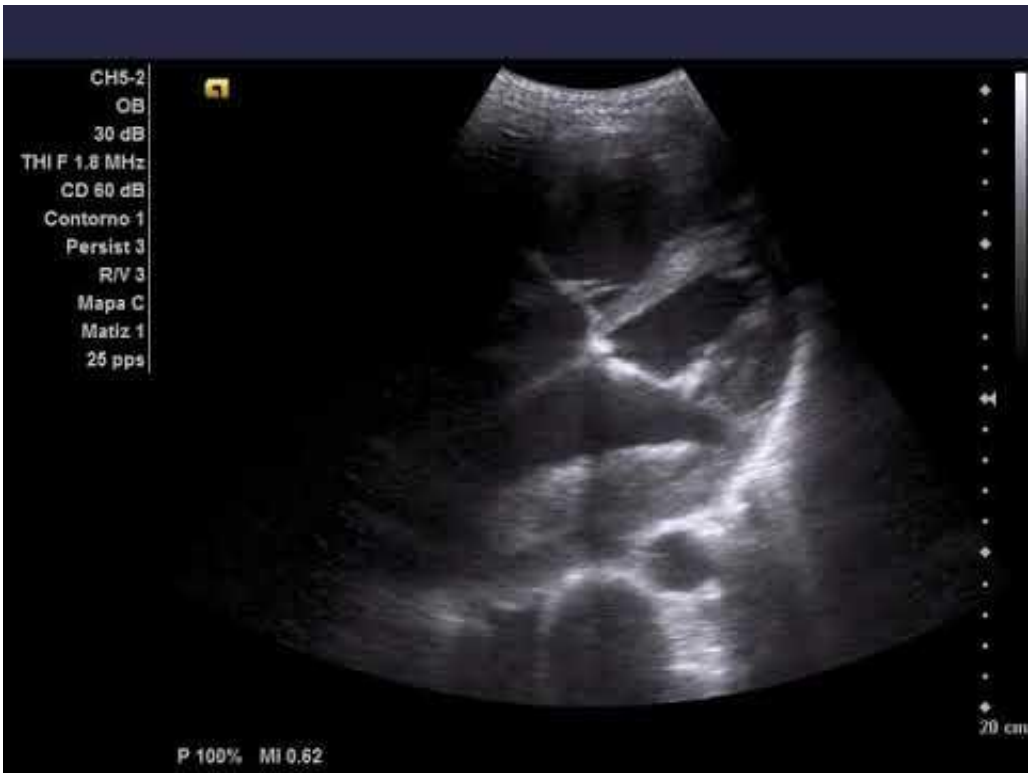
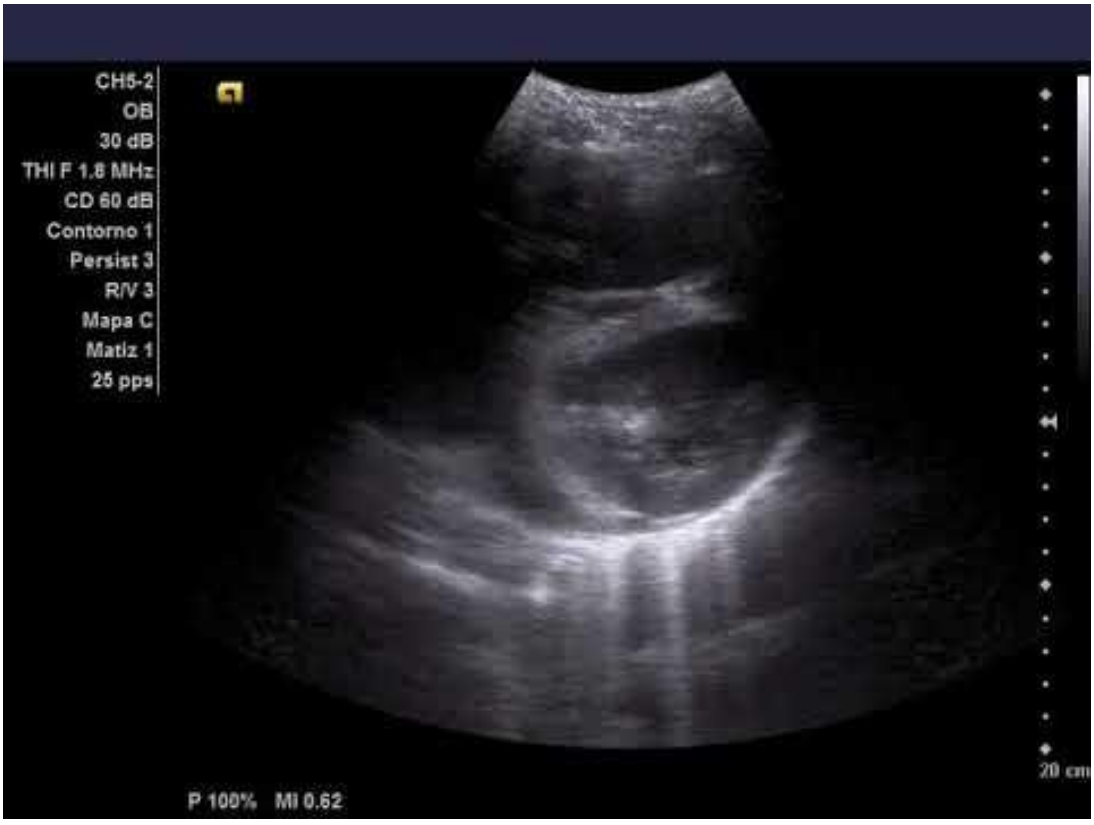


Figure 2: Twelve-lead ECG: irregularly irregular rhythm with right deviation axis (130º), and ST asymmetric depression in V3-V5 suggestive of right ventricle overload.



Video 1: Four apical chamber modified view: giant aneurysmatic Left atrium (LA) occupied by a large thrombus. A thickened and hyperechogenic mitral valve. Video link: https://www.youtube.com/watch?v=5lkhQm4pe1s



Video 2: Parasternal short axis view at mitral valve level: interventricular septum paradox movement "D-sign" which traduces right ventricle overload. Video link: https://www.youtube.com/watch?v=v3Ro5oIfIM

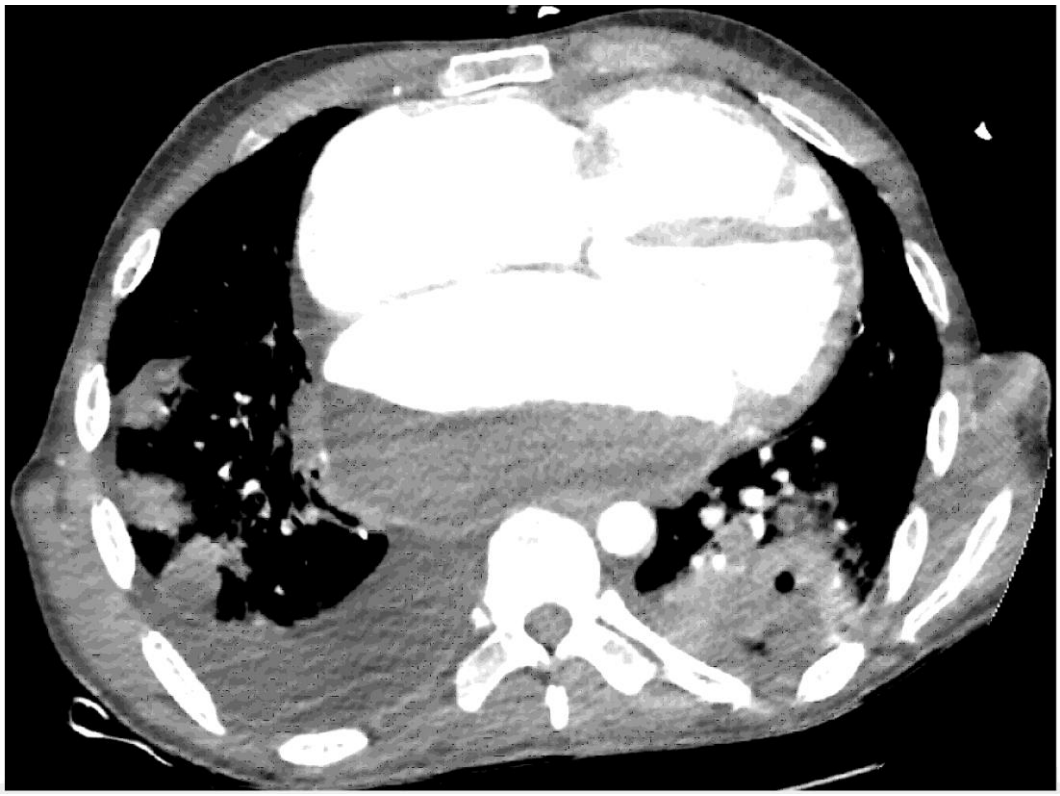


Figure 3: CT chest angiography: peripheral lung consolidations and repletion defects in both right and left inferior lobar arteries that confirms pulmonary thromboembolism and necrotizing bilateral consolidations. Large repletion defect in left atrium, which is consistent with an intracavitary thrombus.

Discussion

There are several causes of mitral stenosis (MS) such as degenerative, rheumatic, congenital deformities (e.g., parachute mitral valve), infiltrating (e.g., mucopolysaccharidosis) or systemic diseases (e.g., Fabry's disease, systemic lupus erythematous or rheumatoid arthritis). Among all causes, RMS is the most common, albeit only 50 – 70% of patients report a history of Rheumatic Fever (RF) **[1].** Repeated episodes of RF correlate with persistent valve damage that promote gradual progression of the valvulopathy, and haemodynamic injury that leads to atrial remodeling **[2].** In some cases, those morphological changes origin aneurysmatic left atrium that contributes to AF. Any condition that induces tachycardia can decompensate those patients because of the decrease on diastolic filling period **[3].** Furthermore, undiagnosed RMS with giant – aneurysmatic left atrium and non-anticoagulated AF, such as our patient, have high risk of thromboembolic complications which are the main cause of mortality **[4].** In a situation of hemodynamic instability, it becomes unsafe to transfer a patient from the ED room (e.g., to the CT scan room). In order to avoid that, POCUS exam is the fifth cornerstone of the physical examination and its extended fashion gives critical information in trained hands. The scope of this tool is: First, to identify immediately the life-threatening causes for hemodynamic failure (e.g., massive pulmonary embolism with right ventricle overload), categorization of shock state - initial management strategy and identification of concurrent processes **[5].** In conclusion, the role of POCUS is evolving becoming globally accepted as a beneficial complementary modality in care, treatment and monitoring **[6].**

References

1. Chandrashekhar Y, Stephen Westaby, Jagat Narula. Mitral stenosis: Seminar. Lancet. 2009;374(9697):1271-83.
2. El Maghraby A, Hajar R. Giant left atrium: a review. Heart Views. 2012;13(2):46-52.
3. Horstkotte D, Niehues R, Strauer BE. Pathomorphological aspects, aetiology and natural history of acquired mitral valve stenosis. Eur Heart J. 1991;12(B):55-60.
4. Rowe JC, Bland EF, Sprague HB, White PD. The course of mitral stenosis without surgery: ten- and twenty-year perspectives. Ann Intern Med. 1960;52:741-9.
5. Whitson MR, Mayo PH. Ultrasonography in the emergency department. Critical Care. 2016;20(1):227.
6. Walley Patricia E, Walley Keith R, Goodgame Ben, Punjabi Vivek, Sirounis Demetrios. A practical approach to goal-directed echocardiography in the critical care setting. Crit Care. 2014;18(6):681.

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