importance of addressing left atrial reduction prior to valve replacement in such patients and the need for accurate assessment of prosthetic valve dysfunction. Proper diagnosis and monitoring are critical to prevent severe complications like thromboembolism and stroke in high-risk patients.

APCU 43 ACUTE CORONARY SYNDROME MASQUERADE: A CASE OF PRIMARY CARDIAC TUMOUR

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Introduction Typical angina pain characterised by central chest pain, which is associated with autonomic symptoms, is commonly associated with acute coronary syndrome (ACS). However, a detailed clinical examination should be done to exclude other differential diagnoses. Herein, we report a case of a patient diagnosed with ACS who was later found to have a primary cardiac tumour.

Case Presentation A 63-year-old woman with hypertension and spondylosis presented with central chest pain radiating to her back and right shoulder, associated with perioral numbness and breathing difficulty. Examination revealed blood pressure of 156/99 mmHg, pulse rate of 93 bpm, and respiratory rate of 22 breaths per minute, but no hypoxia. ECG showed right bundle branch block, premature ventricular contractions, and Q waves in leads III and AVF. Chest X-ray and cardiac enzymes were normal. She was diagnosed with unstable angina and treated with dual antiplatelet therapy and anticoagulants. Transthoracic echocardiography revealed a mass in the interventricular septum with good left ventricular function, while coronary angiography showed mild coronary artery disease. CT coronary angiography revealed no significant atherosclerosis but identified a right ventricular mass, likely a lipoma. Cardiac MRI confirmed a basal-mid anteroseptal mass, measuring 1.54 cm × 1.57 cm × 2.18 cm, likely a primary tumour. She is not keen on invasive surgery and is currently under regular transthoracic echocardiography follow-up.

Discussion Cardiac tumours are a rare but important cause of chest pain, often mimicking more common conditions such as acute coronary syndrome. In this case, the patient initially presented with chest pain radiating to the back and shoulder, which raised suspicion for coronary ischemia. Cardiac tumours can induce chest pain through direct compression of myocardial structures, leading to impaired coronary blood flow and ischaemia, or by interfering with the mechanical function of the heart, causing angina-like symptoms. Previous case reports have similarly described chest pain from cardiac tumours due to obstruction, compression, or infiltration of myocardial tissue. The diagnosis in such cases relies on advanced imaging modalities like cardiac MRI, which provides detailed characterisation of the mass. Small, benign tumours generally have a good prognosis with conservative management, whereas malignant tumours require aggressive interventions, including complete surgical resection and adjuvant chemotherapy.

Conclusion This case illustrates the challenge of diagnosing central chest pain when initial treatment addresses acute coronary syndrome, but other diagnoses must still be considered. Primary cardiac tumours are rare and require advanced imaging for an accurate diagnosis. Timely detection and intervention are crucial for effective patient management and prognosis.

APCU 44 NON-IMMUNE HEMOLYSIS AND RECURRENCE OF MITRAL REGURGITATION AFTER VALVE REPAIR

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Introduction Non-immune haemolysis following native mitral regurgitation is extremely rare. Typically, mechanical complications are directly associated with non-immune haemolysis in cases of native mitral regurgitation. We present a case of primary native mitral regurgitation complicated by non-immune haemolysis.

Case Presentation We report a case of a 30-year-old woman with history of primary mitral regurgitation due to chronic rheumatic heart disease for the past 10 years. Over the last two years, she had a decline in functional status and developed unexplained iron deficiency anaemia. In 2021, she underwent mitral valve repair with neo chord implantation. Intraoperatively, the A/3 was redundant and prolapse with ruptured primary chordae, and a small vegetation attached to it. 2 × CV4 neo chordae were placed at A2 and A3. A size 30 Medtronic CG Future was implanted. Despite this, she continued to suffer from recurrent, unexplained iron deficiency anaemia, requiring multiple episodes of intravenous iron supplementation to maintain her haemoglobin levels. Her baseline haemoglobin prior to the repair was 7.4 g/dL, which dropped to 5.5 g/dL, with hypochromic microcytic anaemia and very low serum ferritin levels, consistent with iron deficiency anaemia. Serum haptoglobin reduced and LDH elevated. A repeat echocardiogram revealed severe mitral regurgitation with an effective regurgitant orifice (ERO) of 0.3 cm^2 and a maximum velocity of 6 m/s. Transoesophageal echocardiography showed a very tight neo chord with non-coapting of the mitral leaflets. Redo mitral valve replacement done. Intraoperatively, dense adhesion at anterior surface of the heart. The AMVL was thickened and retracted at A3 with no coaptation of P3. Post operatively, patient had significant clinical improvement with restoration of baseline haemoglobin.

Discussion Non-immune mechanical haemolysis is related to shear stress on red blood cells as they pass through the valve, leading to crenation. In cases of native mitral regurgitation, this phenomenon is often related to multiple factors that contribute to the high amplitude, velocity, and magnitude of the regurgitant jet, resulting in haemolysis. In this case, the persistent anaemia is key point to the diagnosis. The IDA is caused by mechanical haemolysis. It is due to neo chord implantation in restricted way, and over time, its elongation lead to a recurrence of the prolapse. Ultimately, this patient experienced a recurrence of mitral regurgitation, necessitating mitral valve replacement.