A Case of Extensive Myocardial Calcification

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ABSTRACT

Aortic valve and mitral annular calcification are commonly seen in elderly patients with chronic kidney disease and/ or altered calcium hemostasis. Extensive myocardial calcification is rare. We present a case of an elderly female who presented with sepsis and heart failure on a clinical background of chronic kidney disease and showed extensive myocardial calcification on multi-modality imaging, leading to severe impairment in systolic and diastolic function with restrictive physiology.

***Conclusion*:** Myocardial calcification is an under diagnosed entity that leads to considerable morbidity and mortality. Modern imaging methods allow a prompt diagnosis but a high index of suspicion has to be maintained for accurate detection of medical condition.

Keyword: Annular calcification, Myocardial calcification, Sepsis,

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INTRODUCTION

As for calcification elsewhere in the body, myocardial calcification can be categorized as dystrophic and metastatic.[1] Dystrophic calcification is due inflammation and cell membrane damage. Cell membrane damage leads to calcium being concentrated in and binding to phosphates, released from damaged membranous vesicles. Systemic hypercalcemia accelerates this process. In this case report we present a case of extensive myocardial calcification with multiple contributary factors.

CASE REPORT

An 84-year old female presented to our hospital with 5-days history of decreasing level of consciousness, during her admission at a public sector hospital. She was previously a known case of hypertension, and chronic kidney disease (not dependent on hemodialysis). On presentation she was drowsy with preserved orientation on arousal. Her blood pressure was 100/60 mmHg; pulse 110/minute, irregularly irregular; respiratory rate of 22/min and temperature 101°F. She had pedal edema, and raised JVP. Auscultation of the heart revealed a third heart sound. Lab investigations showed impaired renal function (urea 26.5 mmol/L; creat 193 mmol/L; normal Na and K); evidence of sepsis (TLC 47x103, Thrombocytopenia 50 x 103). PT, PTTK and D dimers were raised. The liver function tests were within normal limits. ECG showed atrial fibrillation with left bundle branch block. Chest Xray AP done in the intensive care unit showed calcification superimposed in the lower half of the cardiac shadow. A provisional diagnosis of sepsis leading to fluid overload and heart failure was made and the patient was prescribed broad spectrum antibiotics (Vancomycin, Flagyl, Meropenem). Multiple blood and urine samples were sent for culture. Echo revealed extensive mitral annulus calcification, calcification of the inferoseptum extending into the cardiac apex (see Figure-1).

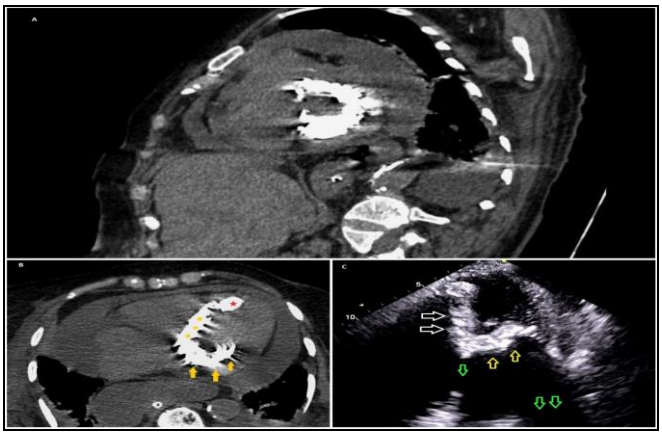

Figure-1: (A): CT scan; Modified coronal view showing the short axis of the heart with extensive C-shaped calcification seen of the mitral annulus, sparing only the anterior part. (B): CT; Horizontal long axis view showing extensive calcification of the mitral annulus (yellow arrows), the interventricular septum (yellow stars), and a large apical calcific chunk (red star). (C): Transthoracic echo showing modified Apical 4 chamber view showing extensive calcification seen in the inferoseptum (white arrows), & the posterior mitral annulus (yellow arrows), leading to extensive acoustic shadowing (green arrows)

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This calcification led to posterior acoustic shadowing and a limited echocardiographic study. In addition, there was Grade-3 diastolic dysfunction (restrictive) with E/A ratio of 3.5. Because of the akinesia of the calcified inferoseptum her left ventricular ejection fraction was reduced to 35%.

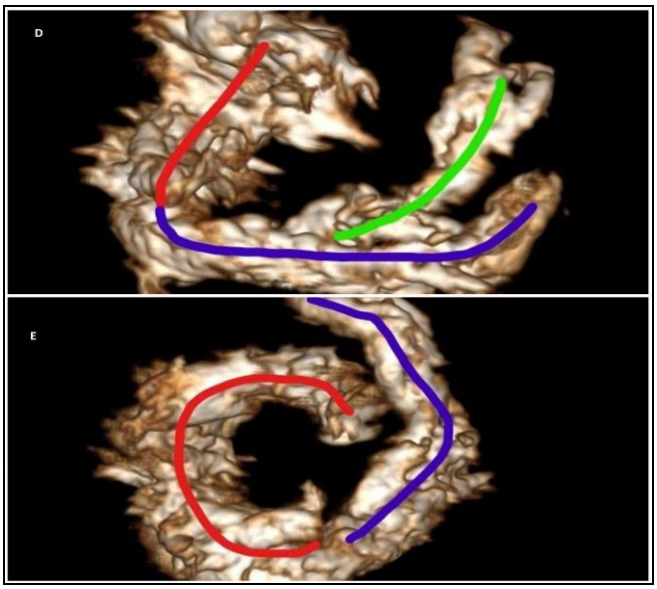

Figure-2: (D): 3D volume rendered Right anterior Oblique view of the calcium deposition "skeleton" in the heart showing the calcified mitral annulus (red line), and extension of this calcification into the inferoseptum (blue line), and the anteroseptum (green line). (E):3D volume rendered view from the left atrium of the calcium deposition "skeleton" in the heart showing the calcified mitral annulus (red line), and extension of this calcification into the inferoseptum (blue line)

We asked for a high-resolution CT scan chest, which revealed extensive C-shaped calcification of the mitral annulus sparing the anterior part of the annulus (Figure-2). This calcification was extending into the inferoseptum connected to a large apical nodule of calcium, as well as tentacles of calcium extending into the anteroseptum. These changes explained the extensive immobility of the interventricular septum. Blooming artefacts were noted due to the excessive calcification. Despite all measures the patient continued to deteriorate and passed away 72 hours later. Her blood cultures revealed growth of Klebsiella pneumoniae post-mortem.

DISCUSSION

Dystrophic calcification is more common than metastatic calcification, most of it being accounted for by post myocardial infarction (MI) related myocardial necrosis.[1] Post MI dystrophic calcification has been documented at 8% in one study in MIs more than 6 years old.[2] Myocardial calcification may occur as an extension of mitral annular calcification,[3] or calcific pericarditis. Metastatic calcification usually occurs as a consequence of systemic calcium homeostasis (hypercalcemia) in a healthy or diseased tissue. It is seen in hyperparathyroidism (primary, secondary, or tertiary) with vitamin D disorders and in oxaluria.[4] It is most commonly reported in patients on maintenance hemodialysis, however in these cases the calcification is most commonly found in the mitral annulus and the aortic valve. Acute metastatic calcification has been seen in cases of acute sepsis.[5,6] Post heart transplant patients have been reported to have myocardial calcification as well.[7] Myocardial calcification has significant functional consequences. The calcification of the mitral annulus affects it's descent significantly during the contraction of the left ventricle, which negatively affects the suction pump action of the left ventricle on the left atrium. It thus leads to diastolic dysfunction and even a restrictive physiology in extreme cases. It can also cause focal wall motion abnormalities,[8] and life-threatening arrhythmias if there is calcification in the conduction system.[9] In our patient left ventricular systolic and diastolic dysfunction with restrictive physiology were noted. The global cardiac calcium score was applied in a cohort of patients. It was shown that there was almost a linear relation between the heart calcification score and total mortality and risk of ischemic stroke.[10] Evaluation of myocardial calcification begins with its identification usually on an imaging study followed by an investigation of its cause and effects. The chest X-ray film may show areas of calcification in the heart as was seen in our case. An echocardiogram usually reveals the extensive areas of calcification as highly echogenic foci with posterior acoustic shadowing. In severe cases it may limit the echo study. Both these features were seen in our case. The gold standard modality for identifying cardiac calcification is CT scan of the heart. Plain Cardiac CT reliably shows the location and extent of the calcification and hyperdense areas within the myocardium and other cardiac structures. There may be blooming artefacts related to this as seen in our case. the strength of cardiac CT in this case is the ability to create multiplane reconstructions (MPR) and view the calcification in its full extent in 3D. The other strength of cardiac CT is the volume rendered images which help to recreate a complete "skeleton" of the myocardial calcification while excluding the non-calcified part of the heart. We demonstrated the "prawn" shaped calcification in our patient using this

technique. In our patient the extensive myocardial calcification was multifactorial; due to the chronic kidney disease and resulting disturbed calcium homeostasis. Although it may be difficult to prove but we cannot rule out the possibility of superadded sepsis associated calcification.

CONCLUSION

Myocardial calcification is an under diagnosed entity that leads to considerable morbidity and mortality. Modern imaging methods allow a prompt diagnosis but a high index of suspicion has to be maintained for accurate detection of medical condition.

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Conflict of Interest: None.

Author's Contribution

Following authors have made substantial contributions to the manuscript as under:

RY: Manuscript writing, drafting, concept and editing

NB: Image processing, editing, Manuscript writing & review

SH: Manuscript writing, review and image processing

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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