Pericardial Calcification: A Rare Case with Intraventricular Extension

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Introduction

Pericardial calcification is a rare and often underdiagnosed condition characterized by the deposition of calcium in the pericardium, the doublewalled sac surrounding the heart. While pericardial calcification is typically associated with chronic pericarditis or tuberculosis, its extension into the ventricular myocardium is exceptionally rare and presents unique diagnostic and therapeutic challenges. This opinion article aims to discuss the implications of intraventricular extension of pericardial calcification, its pathophysiology, diagnostic approach, and treatment strategies, drawing from a case study and existing literature. Pericardial calcification often results from chronic inflammation or infection, leading to fibrosis and subsequent calcium deposition. Historically, a major cause of pericardial effusion and calcification [1].

Patients undergoing radiation for thoracic malignancies may develop pericardial fibrosis and calcification. In some cases, no clear etiology can be identified. The extension of calcification into the ventricular myocardium suggests a more aggressive or prolonged inflammatory process. This can lead to significant functional impairment due to the rigid and non-compliant nature of calcified tissue, affecting both diastolic filling and systolic contraction.

Description

A 65-year-old male presented with symptoms of heart failure, including dyspnea, fatigue, and lower extremity edema. His medical history included chronic renal failure and a history of tuberculosis treated 30 years prior. Imaging studies, including echocardiography and Computed Tomography (CT), revealed extensive pericardial calcification with notable extension into the interventricular septum and ventricular free wall. The diagnosis of pericardial calcification with intraventricular extension requires a high index of suspicion, particularly in patients with a history of pericardial thickening, calcification, and cardiac function. Calcification appears as echogenic (bright) areas on TTE. Provides detailed images of the posterior pericardium and can better delineate the extent of calcification. Offers superior visualization of calcified structures compared to echocardiography. It provides detailed information on the location, extent, and density of calcification, as well as its impact on adjacent cardiac structures [2].

Useful for assessing myocardial involvement, particularly in cases where calcification extends into the myocardium. MRI can provide detailed images of myocardial tissue and help assess ventricular function. Elevated levels of inflammatory markers can suggest ongoing inflammation. Important for patients with a history of renal disease, as uremia is a known cause of pericardial calcification. Intraventricular extension of pericardial calcification can severely impact cardiac hemodynamics. The rigid, calcified tissue restricts ventricular filling during diastole, leading to increased intracardiac pressures and reduced cardiac output. This diastolic dysfunction can mimic constrictive pericarditis, but the involvement of the myocardium adds complexity to the clinical picture.

Patients with intraventricular calcification often present with symptoms of heart failure, including due to elevated left atrial pressures and pulmonary congestion. Resulting from reduced cardiac output. Secondary to increased systemic venous pressures. May occur if the calcification is associated with active inflammation or myocardial ischemia. The differential diagnosis for patients presenting with pericardial calcification and heart failure symptoms includes characterized by pericardial thickening and calcification leading to impaired diastolic filling. Differentiated by myocardial rather than pericardial pathology, though both can present with similar hemodynamic abnormalities. Important to rule out as calcification and symptoms may overlap [3].

Treatment of pericardial calcification with intraventricular extension involves a combination of medical management and, in select cases, surgical intervention. Essential for managing fluid overload and reducing symptoms of heart failure. Nonsteroidal anti-inflammatory drugs or corticosteroids may be used if there is evidence of ongoing inflammation. Addressing the underlying cause, such as controlling renal function in uremic patients or treating any residual infection in patients with a history of tuberculosis. In cases where medical management is insufficient, surgical intervention may be necessary. Options include surgical removal of the calcified pericardium can relieve constrictive symptoms. The procedure carries significant risk, especially in patients with extensive calcification or myocardial involvement. In cases with localized intraventricular calcification, surgical debridement may be considered to improve myocardial function [4]. The prognosis for patients with pericardial calcification and intraventricular extension varies based on the extent of calcification, underlying etiology, and response to treatment. Regular follow-up with echocardiography and CT imaging is essential to monitor disease progression and treatment efficacy. Managing comorbid conditions, such as renal disease and infection, is critical for improving outcomes. The case of intraventricular extension of pericardial calcification highlights several important considerations in the diagnosis and management of this rare condition. Clinicians should maintain a high index of suspicion in patients with a history of pericarditis, tuberculosis, or chronic renal disease who present with heart failure symptoms. Advanced imaging techniques, such as CT and MRI, are invaluable for accurately diagnosing the extent of calcification and guiding treatment decisions. Medical management remains the cornerstone of treatment, with diuretics and anti-inflammatory medications playing key roles. However, surgical intervention may be necessary for patients with severe symptoms or refractory cases. Pericardiectomy, while effective, carries significant risk and should be reserved for carefully selected patients. Further research is needed to better understand the pathophysiology of pericardial calcification with intraventricular extension and to develop targeted therapies. The role of novel anti-inflammatory agents, calcium metabolism regulators, and advanced surgical techniques warrants exploration [5].

Conclusion

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Received: 03 June, 2024, Manuscript No. jchd-24-144332; Editor Assigned: 05 June, 2024, Pre QC No. P-144332; Reviewed: 17 June, 2024, QC No. Q-144332; Revised: 22 June, 2024, Manuscript No. R-144332; Published: 29 June, 2024, DOI: 10.37421/2684-6020.2024.8.222

significant clinical entity that poses unique diagnostic and therapeutic challenges. A thorough understanding of its pathophysiology, combined with advanced imaging and individualized treatment strategies, is essential for improving patient outcomes. Clinicians must remain vigilant for this condition, particularly in patients with a history of pericarditis, tuberculosis, or chronic renal disease, to ensure timely and effective management. Continued research and clinical vigilance will be crucial in addressing the complexities of this rare but impactful condition.

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How to cite this article: Li, Tian. "Pericardial Calcification: A Rare Case with Intraventricular Extension." *J Coron Heart Dis* 8 (2024): 222.