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Case Report

Post-mortem finding of uncommon rheumatic heart disease with mural cardiac thrombi in a young individual: A case report

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Abstract

Sudden death in young individuals, though uncommon, is a profoundly distressing occurrence, with cardiovascular disorders being the leading causes. Cases of individuals brought dead often prompt inquiries into possible causes. We present the case of an 18-year-old male laborer with a background of chronic alcohol use and drug abuse. Autopsy revealed significant acute and chronic cardiac pathology, including mural thrombi and mitral valve stenosis, which was ultimately attributed as the cause of death due to heart failure. In forensic practice, careful evaluation for cardiac lesions—especially mitral stenosis and intracardiac thrombi—is essential in young adult deaths. Detailed gross and histopathological examination during autopsy is critical for establishing the definitive cause.

Keywords: Mitral valve stenosis; Thrombus; Rheumatic heart disease.

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1. Introduction

Sudden death in young individuals, though rare, is often linked to cardiovascular causes.[1] Fever and breathlessness, though common symptoms, are also seen in other conditions requiring exclusion before confirmation. We report the sudden death of an 18-year-old male with a history of drug abuse led to a medico-legal case and autopsy. Post-mortem examination revealed acute and chronic cardiac pathology. Gross and histological findings established congestive cardiac failure (CCF) due to mural thrombi and mitral valve stenosis in the context of rheumatic heart disease (RHD) as the cause of death.

2. Case Report

An 18-year-old male migrant laborer from Bihar, residing in Ahmedabad for three years, experienced fever and generalized weakness following fish and alcohol consumption on the night of 11/08/2020. A general practitioner administered intravenous fluids and prescribed oral medications including multivitamins, Rabeprazole, Cefixime, and Clonazepam. Symptoms persisted, and he developed worsening breathlessness at 2:00 AM on 13/08/2020, which intensified in the supine position and improved upon sitting. At 10:00 AM the same day, he became unresponsive and was taken to a private hospital, then referred to GCS Hospital at 10:55 AM in an unresponsive state. Cardiopulmonary resuscitation was unsuccessful, and he was declared dead. A history of alcohol consumption, cannabis use, and parenteral drug abuse was noted, but no significant past medical history. Due to unclear circumstances, a medico-legal case was registered, and autopsy was requested by police authorities. The post-mortem was conducted under safety protocols.

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Externally, the body was of average build (44.5 kg, 160 cm). A scar over the left medial malleolus and healing puncture abrasions on the right wrist, cubital fossa, and chest were noted. Internally, approximately 300 mL of yellowish turbid fluid was present in the pleural cavity, and frothy material was observed in the trachea. Lungs were edematous (right: 860 g; left: 460 g). The heart was enlarged (14.6 × 9.5 × 6.3 cm³; 510 g), with thickened left ventricular walls. A thrombus was found in the right atrium (5.3 × 4.2 × 2.7 cm³) and another in the left ventricle (4.2 × 3.7 × 2.2 cm³). The mitral valve showed stenosis (**Figure 1**) with thickened cusps and fused commissures; circumference was reduced to 5 cm (normal: 9–10 cm). The stomach contained 150 mL of semi-digested food without unusual or bad odor, indicating poisoning, infection, or decomposition. Toxicological screening for alcohol and other substances was negative. Viscera and selected organs were preserved for histopathology.

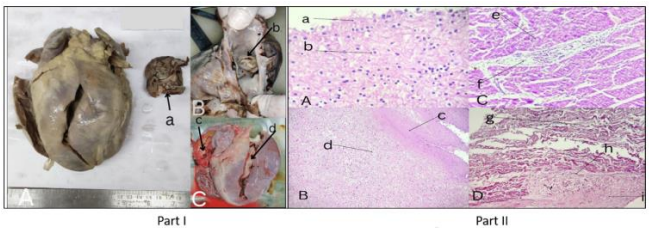

Figure 1: (Part I) A. Formalin-fixed heart with a. thrombus recovered from right atrium B. Formalin-fixed heart with b. mitral valve stenosis C. Dissected heart showing mural thrombi, dry, granular, firm, and adherent to the wall in c. right atrium, d. left ventricle, attached with inter-ventricular septum. **(Part II)** A. Section from a right atrial thrombus (40X) showing a. Fibrin + platelets (pink thread-like), b. Inflammatory cells (purple). B. Section from inter-ventricular septal thrombus (10X) showing c. Pink area - rich in fibrin, d. fibrin + RBCs + inflammatory cells. C. Section from left ventricle (10X) showing e. Myocardial muscles, f. Aschoff body in interstitial tissue of myocardium. D. Section from the right atrium (10X) showing g. Myocardium, h. Aschoff body (Fibrous – late phase, i. visceral pericardium (H and E stain)

Histological sections stained with hematoxylin and eosin revealed thrombi in the right atrium and left ventricle (**Figure 2**). Myocardial tissues showed congestion, pericardial fibrosis, and features of myocarditis in the left ventricle, including Aschoff bodies with “Anitschkow cells” characterized by caterpillar-shaped or "owl-eye" nuclei. The mitral valve demonstrated fibrosis and congestion without active inflammation. No evidence of infective endocarditis was seen. Pulmonary arteries, veins, and aortic segments were histologically unremarkable. Lung and liver sections exhibited chronic venous congestion; kidneys showed tubular cloudiness and congestion. Cerebral sections revealed edema and vascular congestion; the spleen showed congestion with focal fibrosis.

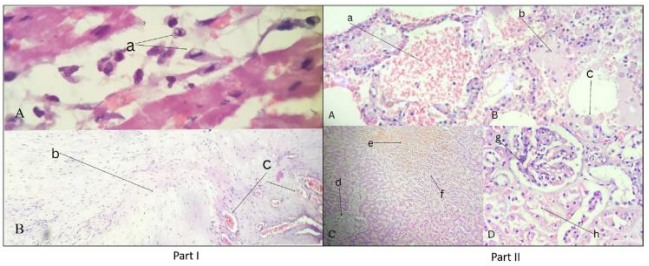

Figure 2: (Part I) A. Section from left ventricle (100X) showing a. Anitschkow cells with caterpillar nuclei with mild chronic inflammatory cells infiltrate in the background. B. Section from mitral valve (40X) showing b. Fibrosis, c. Congestion. (H and E stain); **(Part II):** A. Section from right lung (40X) showing a. Intra alveolar hemorrhage. B. Section from left lung (40X) showing b. Oedema c. Hemosiderin-laden macrophages. C. Section from the liver (10X) showing d. Portal triad, e. Congested sinusoids, f. Centrilobular necrosis. D. Sections from both kidneys (40X) showing, g. Congested glomerulus, h. Renal tubules – cloudy changes. (H and E stain)

These findings collectively indicated acute and chronic cardiac pathology consistent with RHD, and the cause of death was determined to be heart failure due to mural thrombi and mitral stenosis.

3. Discussion

Cardiovascular diseases are among the leading causes of sudden death in young adults. Although the presence of fever and breathlessness are common symptoms, the orthopnea and history of drug abuse indicated an alternative etiology. These features, along with clinical signs of CCF, pointed toward myocarditis, a serious manifestation of rheumatic fever. When myocarditis coexists with valvular disease, a rheumatic origin is likely.

Globally, RF remains a major cause of acquired heart disease in the young.[2] It is an autoimmune response to group A β-hemolytic streptococcal infection, usually presenting as pharyngitis. However, only a small proportion (3–5%) of affected individuals progress to RF.[3] While the acute illness resolves over time, valvular damage persists and leads to chronic RHD, often manifesting as mitral valve (MV) stenosis.[4] The mitral and aortic valves are most frequently affected, with isolated MV involvement in two-thirds of cases and aortic involvement in about 25%.[3,5] Though more common in females, this case involved a young male patient.[3]

Waller BF et al. confirmed that MV stenosis is a hallmark of RHD, and in this case, Aschoff bodies—indicative of rheumatic carditis—were noted.[6] These nodules develop in three stages: early (fibrinoid degeneration), intermediate (granulomatous with Anitschkow cells and histiocyte palisading), and late (fibrotic healing).[7] Histology in this case revealed fibrosis with minimal Anitschkow cells, lymphocytes, and plasma cells, suggesting a late-stage lesion,

which corresponded with exacerbated dyspnea even in the supine position.

A notable finding in this case was the presence of two large intracardiac thrombi. Intracardiac thrombus formation, particularly in association with MV stenosis, is well-documented in RHD.[6] Rohila AK et al. reported a rare case of right atrial thrombus in RHD with severe mitral regurgitation, while Waller BF et al. found thrombi in about 25% of cases with MV stenosis.[8]

Thrombi in ventricular cavities are common whenever there is coexisting coronary artery disease, which was absent in this case.[6] Rodriguez JB et al. linked left ventricular thrombi to anterior myocardial infarction and reduced ejection fraction, both absent here.[9] Chronic methamphetamine use has been associated with left ventricular thrombus and embolism,[10] but there was no histological evidence of drug-related endocarditis or IV drug use complications. Right-sided infective endocarditis and related thrombus, usually seen in IV drug users, were also ruled out by negative microbial stains.[5,11]

Mansueto G et al. evaluated thrombus age histologically and through immunohistochemistry (IHC), categorizing thrombi as early (≤1 h), recent (>1–24 h), recent medium (>24–48 h), medium (>48–72 h), or old (>72 h).[12] Platelet aggregation, lymphocytic infiltration, and fibroblast appearance helped in dating. Though IHC was not performed due to resource constraints, histology revealed fibrin, platelets, and lymphocytes without fibroblasts, ruling out post-mortem clots and suggesting a thrombus age of 1–24 hours—correlating with the brief history of respiratory symptoms and collapse.

4. Conclusion

RHD is a heart disease mainly affecting the MV, causing stenosis. It primarily impacts young to middle-aged individuals, especially females. While global incidence and mortality have declined, RHD remains a public health issue in low-income areas. A thorough examination of cardiac pathology, particularly MV stenosis and thrombi, is crucial in sudden young deaths. Autopsy findings, including histopathological thrombus dating, are essential for determining cause and time of death.

5. Source of Funding

None.

6. Conflict of Interest

None declared.

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Nil.

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