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Case Report

Eosinophilic Myocarditis: A Rare Histopathological Finding and Diagnostic Challenge in Sudden Cardiac Death

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Abstract

Background: Eosinophilic myocarditis (EM) is a rare inflammatory myocardial disorder that may cause arrhythmia, heart failure, cardiogenic shock, or sudden cardiac death. **Objectives:** To describe the forensic and histopathological findings of a fatal case of EM and emphasize appropriate interpretation of pathological diagnosis, mechanism of death, and underlying etiology. **Methods:** A forensic autopsy and histopathological examination were performed in a 48-year-old man who died suddenly without documented preceding cardiac symptoms. **Results:** Autopsy revealed cardiomegaly, myocardial discoloration and necrosis, intracavitary mural thrombi, and no significant macroscopic coronary stenosis. Histopathology demonstrated myocyte degeneration and necrosis with dense eosinophil-rich inflammatory infiltration accompanied by lymphocytes, neutrophils, histiocytes, and hemorrhagic foci. These findings strongly supported EM as the principal pathological diagnosis. However, the etiology remained undetermined because complete toxicological, hematological, parasitological, autoimmune, and immunohistochemical investigations were not documented. A fatal arrhythmia was considered a plausible mechanism of sudden death but was not directly demonstrated. **Conclusions:** This case highlights the importance of integrating autopsy and histopathological findings while distinguishing pathological diagnosis, probable mechanism of death, and etiological classification in sudden death associated with EM.

Keywords: Eosinophilic myocarditis; Sudden cardiac death; Autopsy; Forensic pathology; Histopathology

Introduction

Eosinophilic myocarditis (EM) is an uncommon inflammatory disease of the myocardium characterized by prominent eosinophilic infiltration together with variable degrees of myocyte injury. Its clinical spectrum ranges from mild or nonspecific symptoms to fulminant heart failure, malignant ventricular arrhythmia, cardiogenic shock, and sudden cardiac death[1,2]. Myocarditis as a broader disease entity is increasingly recognized, but its true population burden remains difficult to estimate because asymptomatic and clinically unrecognized cases are common[3]. The rarity of histologically confirmed EM makes clinicopathological documentation especially important.

Clinical recognition of EM remains difficult because symptoms, electrocardiographic changes, cardiac biomarkers, and imaging findings may overlap with acute coronary syndromes and other forms of myocarditis. Contemporary consensus documents emphasize that myocarditis may arise from infectious, immune, toxic, and hypersensitivity related mechanisms, and that tissue diagnosis is particularly important when a specific inflammatory phenotype is suspected[4,5]. In forensic practice, the problem is amplified because the decedent may have had

little or no documented prodromal illness and the final diagnosis may depend on post mortem histopathology[6].

Sudden cardiac death is a time critical cardiovascular event whose immediate recognition is central to emergency response, while forensic investigation is required when death is unexpected and the cause is not clinically established[7] . EM is therefore relevant to both clinical and medicolegal practice. Published forensic series demonstrate that EM may be discovered only at autopsy and that secondary causes cannot be assumed absent merely because premortem clinical history is limited[6].

The main diagnostic gap in the present case is not whether the myocardium contains a prominent eosinophilic inflammatory process, but how confidently the pathological subtype, mechanism of death, and underlying etiology can be assigned. EM may be associated with drug hypersensitivity, parasitic infection, autoimmune disease, eosinophilic granulomatosis with polyangiitis, malignancy, and hypereosinophilic syndromes, while some cases remain without an identified cause[8,9]. Extensive necrosis and mural thrombosis can suggest a necrotizing or Löffler type phenotype, but classification requires correlation with systemic and laboratory findings.

This report asks whether eosinophilic myocarditis can be established as the principal pathological diagnosis in a sudden unexplained death on the basis of autopsy and histopathological findings while appropriately acknowledging the limits of etiological certainty. The objectives are to describe the gross and microscopic cardiac findings, present the forensic differential diagnosis, separate the pathological diagnosis from the suspected mechanism of death, and identify the additional investigations that would be required for a more definitive etiological classification.

Materials and Methods

Research Design

This manuscript is a descriptive forensic case report based on the available clinical history, gross autopsy findings, and myocardial histopathology from a single sudden death case. The report was structured to improve transparency in accordance with the principles of the CARE case reporting guidelines[10,11]. The design is descriptive and does not permit estimation of incidence, risk, treatment effect, or causal association. Diagnostic conclusions were therefore limited to findings directly supported by the available post mortem evidence.

Sample

The sample consisted of one deidentified deceased adult male, aged 48 years, who experienced sudden unexpected death and underwent forensic examination. The case was selected because cardiac gross examination and myocardial histopathology were available and demonstrated an eosinophil rich inflammatory pattern. No statistical sampling procedure or sample size calculation was applicable because the report concerns a single diagnostic case rather than an analytic population study. Potentially identifying details that were not necessary for interpretation, including ethnicity, the employer, and the specific workplace location, were omitted from the revised report.

Data Collection Techniques

Data were derived from the available case history, forensic autopsy record, gross cardiac examination, and histopathological examination of myocardial tissue. Gross assessment included heart size and weight, myocardial color and areas of apparent necrosis, intracavitary thrombus, and macroscopic inspection of the coronary arteries. Microscopic assessment focused on

myocyte degeneration and necrosis, the distribution and composition of the inflammatory infiltrate, and associated hemorrhagic change. Contemporary pathology guidance supports interpretation of myocarditis by integrating inflammatory infiltrates with evidence of myocyte injury and by correlating tissue findings with the clinical and pathological context[12,13].

The source record available for this revision did not document toxicology, premortem or post mortem eosinophil counts, parasitological testing, autoimmune investigations, immunohistochemistry, systematic histology of other organs, or molecular testing. Those investigations were therefore not assumed to have been performed. The available figure record also did not specify the histological stain or provide calibrated scale bars. Objective magnifications of 4 times and 10 times were retained because they were documented in the original figure caption; stain and scale details should be confirmed from the pathology laboratory record before publication.

Data Analysis Techniques

Analysis was descriptive and clinicopathological. Gross and microscopic findings were integrated to assess whether the features were compatible with eosinophilic myocarditis and to compare the pattern with major differential diagnoses, including hypersensitivity myocarditis, necrotizing eosinophilic myocarditis, Löffler type endomyocardial disease, giant cell myocarditis, and ischemic myocardial injury. No inferential statistical analysis was performed. The interpretation explicitly distinguished three levels of inference: the pathological diagnosis supported by tissue findings, the plausible mechanism of sudden death, and the underlying etiology, which requires a broader exclusion process than was documented in the available record.

Etical Consideration

The available manuscript files do not contain an ethics committee approval number, a formal ethics waiver, documentation of the legal authorization for the forensic autopsy, or documentation of consent for publication from the next of kin. Because this is a post mortem medicolegal case, ethical feasibility for publication depends on confirmation that the autopsy was performed under the applicable legal authority and that publication is covered by institutional approval, exemption, waiver, or next of kin consent as required by local regulations and journal policy. Confidentiality was strengthened in this revision by removing nonessential identifying details. Before publication, the authors should provide the exact approval or waiver statement and the consent status; no approval number or consent statement has been invented in this manuscript.

Case Description

A 48 year old man experienced sudden collapse and death at work. The available record did not document previous cardiac disease, routine medication use, drug exposure, allergy history, recent infection, chest pain, dyspnea, palpitations, syncope, or other prodromal symptoms. The absence of documentation should not be interpreted as evidence that these exposures or conditions were definitively absent. A forensic autopsy was performed to investigate the cause of the unexpected death.

Results

Autopsy examination revealed an enlarged heart measuring approximately 16.2 × 10 cm and weighing about 600 g. The epicardial surface was pale brown with irregular dark reddish discoloration, most prominent over the left ventricular wall, as shown in Figure 1A and Figure 1B. These gross findings indicated significant myocardial abnormality but were not specific for a single etiology.

Sectioning of the left ventricle demonstrated a pale area interpreted grossly as necrotic, measuring approximately 3 cm in thickness, surrounded by a darker reddish zone of about 2.6 cm, as shown in Figure 1C. Mural thrombus was present within the cardiac chambers and was most prominent in the left ventricle. No significant macroscopic atherosclerotic stenosis was identified in the coronary arteries. This gross observation reduced the likelihood of severe obstructive coronary disease but did not by itself exclude ischemic myocardial injury.

Histopathological sections of myocardium showed degeneration of cardiac muscle fibers, wavy fiber change, loss of cohesion between adjacent myocytes, and focal to diffuse myocyte necrosis. A dense inflammatory infiltrate was present and was predominantly composed of eosinophils, with smaller numbers of lymphocytes, neutrophils, and histiocytes. Hemorrhagic foci were also identified within the damaged myocardium. The combination of eosinophil rich inflammation and associated myocyte injury was compatible with eosinophilic myocarditis.

Taken together, the gross and microscopic findings strongly support eosinophilic myocarditis as the principal pathological diagnosis. Extensive necrosis and mural thrombosis raise consideration of a necrotizing eosinophilic phenotype or Löffler type disease, but the available evidence is insufficient for definitive subtype classification. A fatal ventricular arrhythmia is a plausible mechanism of sudden death in this setting, although no rhythm was documented. The underlying etiology remains undetermined because a complete toxicological, hematological, infectious, parasitological, autoimmune, and systemic histopathological exclusion process was not documented.

Discussion

The principal finding in this case is a myocardial inflammatory process dominated by eosinophils and accompanied by substantial myocyte injury. This pattern is strongly compatible with eosinophilic myocarditis. The revised interpretation deliberately avoids equating the pathological diagnosis with a proven etiology. In a forensic setting, a complete etiological conclusion requires correlation with medication and exposure history, peripheral eosinophil data, toxicology, infectious and parasitic evaluation, autoimmune assessment, other organ findings, and, where appropriate, immunohistochemistry or molecular testing. None of those elements can be inferred from their absence in the available record.

Eosinophils can produce direct myocardial injury through release of cytotoxic granule proteins and inflammatory mediators. Major basic protein, eosinophil cationic protein, eosinophil peroxidase, and related mediators can damage cardiomyocytes and endocardium, producing edema, necrosis, hemorrhage, electrical instability, and thrombogenic endocardial injury[14,15]. Cardiac involvement in hypereosinophilic states may progress through inflammatory, thrombotic, and fibrotic phases, and the severity of cardiac injury is not always predicted by a single peripheral eosinophil measurement[16,17]. These mechanisms provide biological plausibility for the combination of necrosis, hemorrhage, and intracavitary thrombus seen in the present case.

The mural thrombi are an important part of the pathological pattern. In eosinophilic cardiac disease, endothelial and endocardial injury can create a prothrombotic surface, while ventricular dysfunction and blood stasis may further promote thrombosis. Hypereosinophilic syndromes are particularly associated with ventricular mural thrombi and later endomyocardial fibrosis[16]. However, the

presence of thrombus alone does not establish hypereosinophilic syndrome because a sustained blood eosinophil count and evidence of systemic eosinophil related disease were not documented in this case.

Necrotizing eosinophilic myocarditis deserves specific consideration because the reported myocardium showed extensive necrosis together with a dense eosinophilic infiltrate. Necrotizing eosinophilic myocarditis is an aggressive histological phenotype associated with rapidly progressive cardiac failure and sudden death[18]. Comparable biopsy proven cases have demonstrated extensive eosinophilic inflammation, necrosis, cardiogenic shock, and malignant ventricular arrhythmia[19]. The present morphology is compatible with this spectrum, but definitive classification should remain cautious because the original material did not document the distribution of necrosis throughout the heart or a complete exclusion of secondary causes. 1) Hypersensitivity myocarditis remains another important differential diagnosis. It may occur in association with prescription drugs, over the counter medication, herbal products, vaccines, or other exposures and can be clinically silent before sudden deterioration. Eosinophilic myocarditis associated with allergic or hypersensitivity conditions has also been reported with intracardiac thrombi[20]. In the present case, the available history did not identify a medication or allergy exposure, but missing documentation cannot exclude such exposure. Therefore, the revised manuscript does not classify the case as idiopathic. 2) Löffler type endomyocardial disease and hypereosinophilic syndrome are also relevant because both can be associated with endocardial injury, mural thrombosis, restrictive physiology, and later fibrosis. Contemporary reviews emphasize that cardiac involvement in hypereosinophilic syndrome may evolve from an early inflammatory stage to thrombotic and fibrotic stages[21]. Severe eosinophilic myocarditis can also occur as part of systemic hypereosinophilic disease and may ultimately require advanced heart failure therapy[22]. In this case, the absence of documented persistent peripheral eosinophilia, systemic organ involvement, and endomyocardial fibrosis prevents confident assignment to this category. 3) Giant cell myocarditis is a major histopathological differential because it can cause fulminant myocarditis, ventricular arrhythmias, and sudden death. Its diagnosis requires identification of multinucleated giant cells in an appropriate inflammatory background, which was not reported in the examined myocardial sections. Other inflammatory cardiomyopathies and systemic vasculitic disorders should also be considered when the clinical and pathological context warrants. The absence of these features in the available myocardial sections makes them less favored, but limited tissue sampling can never exclude every focal process with complete certainty[2,12]. 4) Ischemic myocardial injury also requires careful consideration. Wavy fibers, myocyte necrosis, hemorrhage, and ventricular thrombosis are not unique to myocarditis. The absence of significant macroscopic coronary stenosis lowers the probability of severe fixed obstructive coronary disease but does not exclude coronary spasm, transient thrombosis, embolism, microvascular ischemia, or an early ischemic event. The dense eosinophil rich inflammatory infiltrate associated with myocyte injury supports myocarditis, yet the revised interpretation recognizes that ischemic injury should be excluded through the full forensic and histopathological context rather than by gross coronary inspection alone[2,5].

Histopathology is central to the diagnosis in cases such as this because the disease was not suspected before death. Consensus pathology recommendations emphasize appropriate

myocardial sampling and correlation of inflammatory infiltrates with myocyte injury, clinical information, and ancillary studies[12]. Endomyocardial biopsy guidance similarly highlights that tissue diagnosis is most useful when the result may identify a specific myocarditis phenotype or guide etiological investigation[13]. In the post mortem setting, extensive myocardial sampling can provide an opportunity to characterize distribution and severity more completely than a limited biopsy, but the diagnostic value still depends on careful documentation and ancillary testing.

Cardiac magnetic resonance has become a major noninvasive tool for suspected myocarditis through assessment of edema, myocardial injury, and late gadolinium enhancement patterns[23]. Contemporary reviews place magnetic resonance, biomarkers, electrocardiography, echocardiography, and selective endomyocardial biopsy within an integrated diagnostic pathway[24,25]. Nevertheless, histopathology remains important when a specific inflammatory phenotype is suspected, and classic reviews continue to recognize tissue examination as a reference method for defining myocarditis subtypes[26]. In this case, premortem cardiac imaging was not available, so post mortem tissue examination carried the main diagnostic weight.

From a forensic perspective, the mechanism of sudden death should be distinguished from the pathological diagnosis. EM can produce electrical instability through inflammatory injury, necrosis, edema, and ventricular dysfunction, making fatal ventricular arrhythmia biologically plausible[1,6]. However, an arrhythmia was not recorded in the available case materials. The most defensible formulation is therefore that eosinophilic myocarditis was the principal pathological abnormality and the most likely cardiac cause underlying the sudden death, with fatal arrhythmia considered a plausible mechanism rather than a directly demonstrated event.

The broader cardiovascular context also supports attention to thrombosis and rapid recognition of cardiac emergencies. Literature in Healthy Tadulako Journal discusses cardiovascular risk pathways related to hyperhomocysteinemia and thrombosis, although those mechanisms should not be interpreted as direct evidence for eosinophilic myocarditis[27]. Another HTJ study illustrates the diagnostic value of integrating clinical information with histopathological examination in a different disease context, tuberculosis, reinforcing the general principle that difficult diagnoses require clinicopathological correlation[28]. These references are used for their scientific context rather than as evidence of a direct causal pathway in the present case.

The main strength of this report is the combination of gross cardiac findings and histopathological evidence of prominent eosinophilic inflammation with myocyte injury in a sudden death case. The principal limitations are equally important: incomplete premortem history, no documented toxicology, no documented peripheral eosinophil count, no parasitological or autoimmune work up, no reported immunohistochemistry, no documented systematic examination of other organs for eosinophilic disease, and no documented ethics or publication consent information in the source file. These limitations restrict etiological classification and require cautious cause of death wording. Future forensic reports of similar cases should document the full exclusion process, pathology methods, stain and scale information, and the legal and ethical basis for publication.

Figure 1. Gross Macroscopic Findings of the Heart. (A) External cardiac surface with pale brown appearance and dark reddish discoloration. (B) Additional gross view of the heart. (C) Ventricular section showing a pale myocardial area surrounded by darker reddish tissue. Mural thrombus was identified on gross examination.

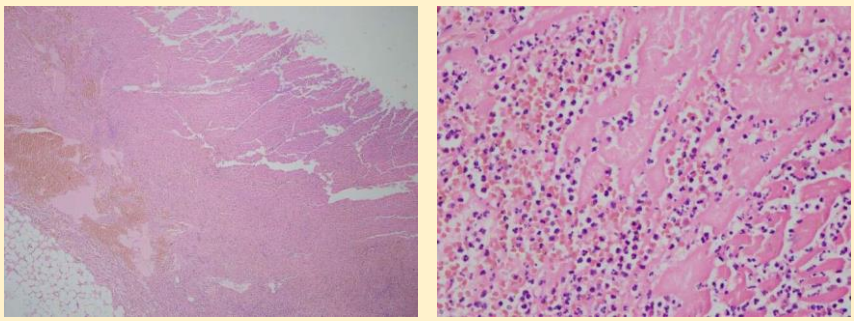

Figure 2. Microscopic Findings of the Heart. (A) Objective magnification 4 times showing myocardial degeneration and wavy fiber change. (B) Objective magnification 10 times showing dense eosinophil rich inflammatory infiltration with myocyte injury. The histological stain and calibrated scale bars were not documented in the available case record and should be confirmed from the pathology laboratory record before publication.

Conclusion

The autopsy and myocardial histopathological findings in this sudden death case strongly support eosinophilic myocarditis as the principal pathological diagnosis. Extensive myocardial necrosis and mural thrombosis raise consideration of a necrotizing eosinophilic or Löffler type phenotype, but the available evidence does not permit definitive subtype assignment. A fatal ventricular arrhythmia is a plausible mechanism of sudden death, although it was not directly documented. The etiology should remain undetermined because the source record does not document a complete evaluation for drug or hypersensitivity exposure, parasitic disease, hypereosinophilic syndrome, autoimmune disease, toxicological causes, or other secondary conditions. Careful separation of pathological diagnosis, mechanism of death, and etiological classification improves the forensic interpretation of rare inflammatory cardiac deaths. Future reports should include complete ancillary testing, pathology methods, and explicit ethics and publication consent documentation.

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