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Case Report

Delayed cardiac conduction disease in a young adult following childhood Chagas infection

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Abstract

Presentation
A Bolivian man in his 30s presented to the ED with non-exertional chest pain and intermittent second-degree atrioventricular (2:1) block.

Diagnosis
Given a history of untreated childhood *Trypanosoma cruzi* infection and family history of Chagas heart disease, late cardiac manifestation was suspected. Structural and ischaemic causes were excluded.

Treatment
Antiparasitic therapy was initiated. Pacemaker implantation was considered but deferred following shared decision-making due to absence of symptoms.

Discussion
Chagas heart disease (CHD) may present decades after infection with conduction abnormalities. Recognition in the ED is essential to enable appropriate management and reduce the risk of progression to complete heart block and sudden cardiac death.

Introduction

Chagas disease, caused by *Trypanosoma cruzi* (*T. cruzi*), remains a leading cause of non-ischaemic cardiac disease and sudden cardiac death in endemic regions of Latin America. Cardiac involvement typically develops 20–30 years after infection and often manifests as conduction system disease, arrhythmias, or progressive myocardial dysfunction. Due to increasing global migration, emergency physicians in non-endemic countries are more frequently encountering

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patients with chronic Chagas heart disease (CHD), often without a prior diagnosis. Awareness of delayed cardiac manifestations is essential for timely identification and appropriate early management in the ED.

Case Report

A Bolivian man in his 30s presented to the ED with non-exertional chest pain. The pain partially improved with oral analgesia. He denied syncope, presyncope, palpitations, dyspnoea, or exertional symptoms. Electrocardiography demonstrated intermittent second-degree atrioventricular block with a 2:1 conduction pattern (figure 1). The patient reported untreated *T. cruzi* infection diagnosed at age eight. Both his mother and sister had documented Chagas-related heart disease. He was haemodynamically stable and examination was unremarkable. Serial cardiac biomarkers were normal. Point-of-care ultrasound demonstrated a structurally normal heart with preserved left ventricular systolic function and no regional wall motion abnormalities.

In the absence of ischaemia or structural abnormalities, intermittent 2:1 atrioventricular block in a young patient with untreated childhood *T. cruzi* infection raised concern for a late cardiac manifestation of Chagas disease. Given the recognised risk of progression to advanced conduction disease, he was admitted for cardiology evaluation and monitoring.

Cardiac MRI demonstrated no myocardial fibrosis, late gadolinium enhancement or structural abnormality. Exercise testing showed normal tolerance without inducible ischaemia, and coronary angiography demonstrated non-obstructive coronary artery disease. In the absence of an alternative aetiology, the conduction abnormality was attributed to chronic CHD. Pacemaker implantation was carefully considered but ultimately deferred. Given the absence of bradycardia-related symptoms and the intermittent nature of the conduction abnormality, immediate permanent pacing was not felt to outweigh the long-term complications of device therapy in a young patient. A shared decision was made to pursue medical management with close outpatient surveillance. Following multidisciplinary discussion with UK specialist services, antiparasitic therapy was approved through a World Health Organisation application process and subsequently initiated despite chronic infection, given the patient's young age and ongoing conduction disease. A loop recorder was implanted for longitudinal rhythm surveillance, with 3 monthly outpatient loop checks and outpatient clinic review scheduled at 6 monthly intervals. The patient remains under ongoing cardiology follow-up.

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CHD is associated with significant morbidity and mortality and encompasses a broad spectrum of clinical manifestations, including bradyarrhythmia, tachyarrhythmias, thromboembolic events, heart failure, and sudden cardiac death. CHD develops in approximately 30% of chronically infected individuals and frequently presents many years after the initial infection[1]. This case demonstrates delayed presentation of CHD as isolated conduction system disease more than two decades after childhood infection. Fibrosis and inflammatory damage to the atrioventricular node and His–Purkinje system are well-recognised pathological mechanisms and may precede overt myocardial dysfunction or heart failure[2].

In non-endemic settings, such presentations can be diagnostically challenging, as patients may lack structural heart disease and present with non-specific or incidental findings. Electrocardiographic abnormalities, including atrioventricular block and bundle branch disease, are among the most common cardiac manifestations of CHD and may represent the earliest clinical evidence of cardiac involvement[3]. Emergency clinicians therefore play a key role in recognising unexplained bradyarrhythmias in patients with relevant epidemiological risk factors, particularly given the potential for progression to advanced conduction disease or malignant arrhythmias.

Although antiparasitic therapy is most effective during the acute or early chronic phases, treatment may still be considered in selected patients with remote infection following specialist assessment[4]. In this case, treatment was supported following multidisciplinary specialist discussion due to the patients' age and evidence of cardiac involvement despite preserved structural function. Management of conduction abnormalities relies on surveillance and pacing strategies when indicated. In younger, asymptomatic patients with intermittent conduction disease, shared decision-making is essential when considering permanent pacemaker implantation, balancing the risk of progression to complete heart block against long-term implications of device therapy. Early recognition in the ED enables appropriate admission, specialist referral, and monitoring, thereby reducing the risk of sudden cardiac death in this population[5]. For emergency clinicians, maintaining a high index of suspicion in patients from endemic regions with unexplained conduction abnormalities is essential to avoid delayed diagnosis and optimise clinical outcomes.

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Declarations of Conflicts of Interest:
None declared.

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Figure 1. Electrocardiogram demonstrating intermittent second-degree atrioventricular block (2:1 conduction).