

ACUTE NON-RHEUMATIC MYOCARDITIS FOLLOWING GROUP G STREPTOCOCCAL PHARYNGITIS: A RARE MIMIC OF ACUTE MYOCARDIAL ISCHEMIA IN A YOUNG ADULT

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ABSTRACT

Background: Acute myocarditis is a rare but serious inflammatory condition of the myocardium, often triggered by infections or rheumatic heart disease. While group A *Streptococcus* is commonly associated with bacterial myocarditis, group G *Streptococcus* is rarely implicated. This case highlights a rare instance of non-rheumatic acute myocarditis also known as Streptococcal pharyngitis acute myocarditis caused by group G *Streptococcus* in a 19-year-old male without rheumatic fever.

Case report: A previously healthy 19-year-old male presented with chest pain, fever, and sore throat, prompting hospitalization. The diagnosis of acute myocarditis was based on elevated cardiac biomarkers, electrocardiographic abnormalities, and cardiac magnetic resonance imaging, with group G *Streptococcus* infection confirmed by throat polymerase chain reaction. The patient responded well to antibiotics and anti-inflammatory therapy.

Conclusions: This case highlights group G *Streptococcus*-induced pharyngitis acute myocarditis, stressing the need for prompt diagnosis and treatment in young adults presenting with recent streptococcal pharyngitis.

KEYWORDS

Streptococcal pharyngitis acute myocarditis (SPAM), group G streptococcus (GGS), pharyngitis, cardiac magnetic resonance, non-rheumatic myocarditis

LEARNING POINTS

- This case highlights a rare instance of non-rheumatic acute myocarditis also known as Streptococcal pharyngitis acute myocarditis, caused by group G streptococcus in a 19-year-old male without rheumatic fever.
- Streptococcal pharyngitis acute myocarditis may present with clinical features closely resembling an acute coronary syndrome; hence advance cardiac imaging is essential to confirm myocarditis and rule out true ischemic heart disease.
- Cardiac magnetic resonance imaging was used to diagnose Streptococcal pharyngitis acute myocarditis which showed subepicardial hyper-enhancement 4 weeks after discharge and resolution of symptoms.
- Prompt diagnosis was achieved, and the patient was treated with antibiotics and anti-inflammatory therapy in a timely fashion with good response.

INTRODUCTION

Pharyngitis, prevalent among children and young adults during colder seasons, typically stems from viral infections like adenovirus and rhinovirus. Yet, bacterial origins, notably group A *Streptococcus* (GAS) (*Streptococcus pyogenes*), are also common. Other bacterial causes include group C and group G *Streptococcus* (GGS), *Corynebacterium diphtheriae*, and *Mycoplasma pneumonia*. While often self-limiting, prompt antibiotic treatment is crucial if streptococcal infection is suspected, confirmed via rapid antigen detection tests and/or throat cultures, to reduce the risk of acute rheumatic fever (ARF). ARF, associated with complications affecting multiple organs including the heart, is thought to involve molecular mimicry between human cardiac myosin proteins and Streptococcal M protein^[1]. Cardiac sequelae of ARF encompass lesions of the mitral, aortic, and tricuspid valves, dilated cardiomyopathy, pericarditis, endocarditis, and myocarditis. Acute myocarditis, an inflammatory condition affecting the myocardium, primarily afflicts adults and can arise from various triggers including infection, autoimmune disorders, or rheumatic heart disease. While viral infections account for most cases of myocarditis, GAS is the primary bacterial culprit. Left untreated, GAS can provoke acute rheumatic fever, leading to a spectrum of cardiac complications necessitating immediate intervention. While GGS does not typically lead to rheumatic heart disease, instances of acute non-rheumatic streptococcal myocarditis, also known as Streptococcal pharyngitis acute myocarditis (SPAM) due to GGS have been reported^[2,3].

Although literature on myocarditis is abundant, non-rheumatic myocarditis triggered by streptococcal infections also known as SPAM due to GGS are relatively scarce. A 2022 systematic review on non-rheumatic myocarditis following Streptococcal pharyngitis/tonsillitis revealed common clinical features such as chest pain, elevated cardiac markers, and ST-segment elevations among the 25 cases that were reviewed^[4]. However, diagnosis and management varied, indicating a lack of standardized criteria. Here, we present a case of acute non-rheumatic myocarditis induced by group G *Streptococcus* in a 19-year-old patient, diagnosed based on initial symptoms and confirmed via extensive imaging.

CASE DESCRIPTION

A 19-year-old male with a history of gastroesophageal reflux disease and musculoskeletal chest pain presented with acute left-sided chest pain radiating to his back and molars. The pain, unassociated with exertion and relieved by acetaminophen, accompanied by fevers, cough, and sore throat, prompted a visit to an urgent care centre two days prior. Although symptoms initially improved, chest pain recurred, prompting hospitalization.

The patient had a family history of cardiovascular disease, including sudden cardiac death in his paternal grandfather and recent coronary artery bypass graft surgery in his father. He was previously evaluated for chest pain by a paediatric cardiologist in 2022 and 2023 with unremarkable results. In the emergency department, symptoms had subsided, but elevated troponin levels warranted admission. Respiratory viral panel was done to rule out viral infection. Suspected Streptococcal pharyngitis was detected and differentiated using Strep throat polymerase chain reaction assay as group C/G beta-haemolytic *Streptococcus* and the patient was found to be positive for GGS. Throughout hospitalization, troponin levels were monitored, peaking and then declining. A repeat electrocardiogram demonstrated sinus bradycardia and nonspecific ST and T wave abnormalities consistent with myocarditis (Fig. 1).

Due to the family history of coronary artery disease, the patient also underwent coronary computed tomography angiography, and results were negative for significant plaque or stenosis.

The patient was treated with ibuprofen, famotidine, colchicine and amoxicillin and subsequently cleared for discharge when his symptoms resolved with a plan for cardiac magnetic resonance (CMR) imaging as an outpatient. Discharge medications included colchicine and continued amoxicillin treatment.

CMR was done 4 weeks after discharge and showed subepicardial hyperenhancement consistent with myocarditis, supporting the diagnosis of acute non-rheumatic myocarditis induced by group G Streptococcal pharyngitis (Fig. 2). At 4-week follow up, the patient was chest pain free with no new symptoms.

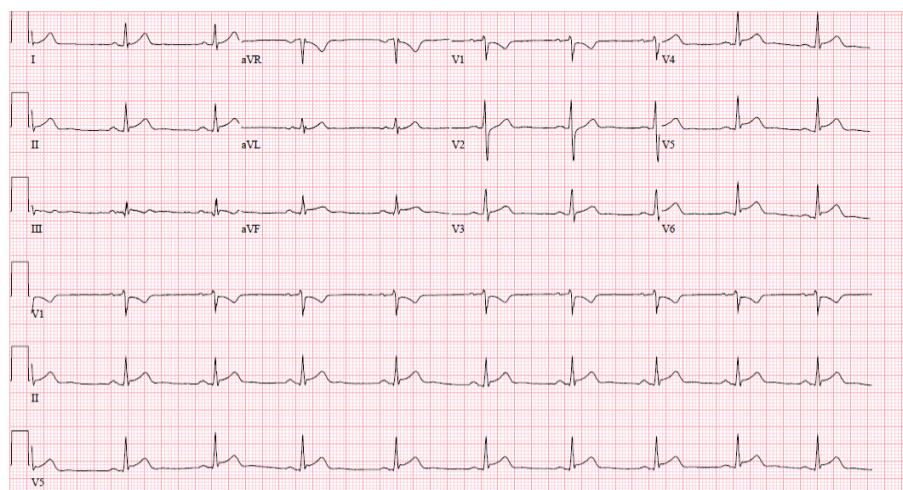


Figure 1. Electrocardiogram showing sinus bradycardia and nonspecific ST and T wave abnormalities consistent with myocarditis.

DISCUSSION

Streptococcal infections can manifest in various cardiac complications, with post-Streptococcal rheumatic fever being a recognized cause of pancarditis due to an autoimmune response to GAS infection. However, recent reports indicate a rising incidence of non-rheumatic Streptococcal myocarditis, which arises through distinct pathophysiological mechanisms^[4]. Given the concurrence of pharyngitis and myocarditis, it seems unlikely to be an immune-mediated phenomenon as is the case with acute rheumatic fever.

Our case presents a novel occurrence of acute non-rheumatic myocarditis triggered by GGS pharyngitis in a 19-year-old male. Acute non-rheumatic Streptococcal myocarditis, also known as SPAM, typically manifests within 5 days of the initial Streptococcal pharyngitis. It is characterized by cardiac symptoms, abnormal electrocardiography findings, and elevated cardiac biomarkers without obstructive coronary artery disease. SPAM represents an under-recognized yet treatable cause of ST elevation and chest pain in young adults with a history of pharyngitis^[5,6]. Timely recognition is crucial for appropriate management and optimal outcomes. In addition, ruling out myocarditis secondary to viral infection with nucleic acid amplification testing is also required to comprehensively assess the aetiology and guide treatment decisions effectively.

Distinguishing between rheumatic fever and non-rheumatic Streptococcal myocarditis is essential. While ARF typically exhibits a latency period of 2-3 weeks, SPAM presents more acutely. The revised Jones criteria aid in diagnosing ARF, as well as facilitating its exclusion. Streptococcal infection can be confirmed via typical symptoms alongside positive rapid antigen testing or throat culture. The gold standard for myocarditis diagnosis is CMR, which reveals focal subepicardial late gadolinium enhancement^[7]. CMR not only differentiates ischemic from non-ischemic heart disease but also enables monitoring of disease progression and resolution^[7]. However, its limited availability necessitates reliance on alternative diagnostic modalities, such as electrocardiography and echocardiography, which may reveal ST elevation, reciprocal changes, new wall motion abnormalities, or hypokinesis, alongside normal coronary angiography findings.

Treatment of SPAM centres on eradicating Streptococcal infection using antibiotics and controlling inflammation with anti-inflammatory agents. Differentiating between acute rheumatic fever and non-rheumatic myocarditis ensures appropriate management, emphasizing the importance of long-term antibiotic use in the former.

While GGS traditionally resides as commensal flora in the human upper respiratory tract, it may exchange genetic material with group A organisms, leading to a similar spectrum of illness. However, the precise underlying pathophysiological basis remains elusive, warranting further investigation. Despite advancements in understanding the systemic implications of Streptococcal pharyngitis, there



Figure 2. Cardiac magnetic resonance imaging 4 weeks after discharge. On delayed enhancement imaging, there is subepicardial hyperenhancement in multiple segments in a non-ischemic pattern. This pattern of increased signal on T2 weighted imaging and hyperenhancement is consistent with myocarditis.

remains a paucity of literature exploring the pathophysiology underlying non-rheumatic complications of GGS, beyond the shared M-protein homology with GAS. The limited availability of case reports and evidence-based guidelines for diagnosing and managing associated systemic sequelae support the pressing need for further rigorous scientific inquiry. The absence of a consensus on the mechanism of cardiac injury makes treatment of SPAM a challenge.

Further research efforts are essential for elucidating the intricate mechanisms driving SPAM, refining diagnostic strategies, and optimizing therapeutic approaches. Enhanced scientific understanding in this realm holds promise for improving patient care and clinical outcomes. Collaboration among clinicians and researchers is paramount to address the diagnostic challenges and to advance the treatment of SPAM.

CONCLUSIONS

This case highlights GGS-induced SPAM, emphasizing the importance of early recognition and treatment, particularly in young adults presenting with recent Streptococcal pharyngitis. Our case underscores the critical need to consider non-rheumatic Streptococcal myocarditis as a potential cause of chest pain and ST-segment elevation in this population. Given that SPAM is an under-recognized yet treatable condition, increasing awareness among healthcare providers is essential for timely diagnosis and management.

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