

A Rare Case of Salmonella Myopericarditis in an Athlete: Diagnosis by Stool Polymerase Chain Reaction (PCR)

David McKinsey ^{1,*}

¹ Metro Infectious Disease Consultants, Kansas City, MO 64132, USA

* Corresponding author: dmckinsey@innovativeventures.com

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Introduction

Myocarditis, a potentially life-threatening disease characterized by inflammatory infiltration of the myocardium and/or degeneration of myocardial tissue, has many potential infectious and noninfectious etiologies [1]. The causative organism is often not elucidated.

Myocarditis most commonly affects individuals between the ages of 30 and 45 years, with 60% to 90% of cases occurring in males [1]. Prior to 2020, the incidence of myocarditis ranged from 4 to 14 per 100,000 people annually [1]. COVID-19 infection has been associated with an incidence of 59 to 64 per 100,000 per year. mRNA COVID-19 vaccines also have been linked to myocarditis (2.1 cases per 100,000 vaccine recipients per year), though much less frequently than infection. [1] The true incidence of myocarditis is likely higher, as many cases go unrecognized.

The broad range of etiologies of myocarditis includes infections, immune-mediated causes, and exposure to myocardial toxins. Viral infections are most frequently implicated, but various bacteria, fungi, and parasites also cause myocarditis [2]. The most common clinical manifestations are chest pain, which occurs in 82% to 95% of cases, followed by dyspnea (19% to 49%), syncope (5% to 7%), and palpitations [1].

Case Presentation

An 18-year-old white male high school senior and swimming champion presented after he experienced acute onset crushing substernal chest pressure, which radiated to his left shoulder and was associated with dyspnea, 5 days after returning from a family vacation at an all-inclusive resort near Cancun, Mexico. On the fourth day of his trip, he began experiencing recurrent episodes of liquid diarrhea accompanied by lower abdominal cramps. These symptoms persisted for 3 days. To control his diarrhea during his flight home, he took multiple doses of loperamide.

Upon arrival to the emergency department, his temperature was 37 °C, pulse 74 beats per minute, respiratory rate 18 breaths per minute, blood pressure 114/74 mm Hg, and oxygen saturation 96% while he was breathing room air. He appeared anxious but was in no acute distress. His physical examination was unremarkable. No cardiac murmurs, rubs, or gallops were noted, and the abdomen was nontender.

During his stay in Mexico, he consumed only bottled water and avoided undercooked meat, fish, and unpasteurized dairy products. He did, however, eat a large quantity of cut fruit. Notably, none of his family members reported similar symptoms during or after the trip. His family members had not consumed fruit.

White blood cell count was 9780 cells/mcL, with 66% polymorphonuclear cells, 22% lymphocytes, and 10% monocytes. Hemoglobin was 14.9 g/dL, and hematocrit was 43%. Serum glutamic-oxaloacetic transaminase was 61 U/L and glutamic-pyruvic transaminase 226 U/L. The highly sensitive troponin level was 46,737 ng/L. An electrocardiogram (EKG) revealed diffuse ST-segment elevation. Chest computed tomography scan with contrast was unremarkable.

Transthoracic echocardiogram demonstrated a left ventricular ejection fraction of 55% with no evidence of wall motion abnormalities. A subsequent cardiac catheterization showed normal coronary arteries.

Cardiac magnetic resonance imaging (MRI) revealed subendocardial late gadolinium enhancement of the basal, anterolateral, lateral, and inferolateral walls, involving less than 50% of myocardial wall thickness. Additionally, there was T2 hyperintensity, indicative of myocardial edema.

A stool gastrointestinal polymerase chain reaction (GI PCR) panel detected the *Salmonella* target, enabling the diagnosis of *Salmonella* myopericarditis complicating enteritis. Blood cultures were negative. The patient's chest pain resolved within a few hours. There was no evidence of arrhythmias or heart failure during his 3-day hospital stay and he was asymptomatic at the time of discharge.

The patient was treated with levofloxacin for 2 weeks for *Salmonella* infection.

His cardiologist prescribed colchicine for concomitant pericarditis and the patient's troponin level returned to normal within 2 days (Figure 1).

Following discharge, the patient was advised by Cardiology to refrain from any exercise for 2 months. A follow-up cardiac MRI scan 10 weeks after diagnosis of myocarditis demonstrated minimal late gadolinium enhancement suggestive of scar formation with no signs of ongoing inflammation. A repeat echocardiogram showed an improved ejection fraction of 60%. Prolonged event monitoring, which included a period of intense physical activity, was negative. Consequently, the patient was cleared by his cardiologist to resume competitive swimming, with the precaution of using a waterproof cardiac monitor during the first 2 weeks of activity. No arrhythmias were detected during this period. He resumed competitive swimming at the collegiate level and has remained asymptomatic.

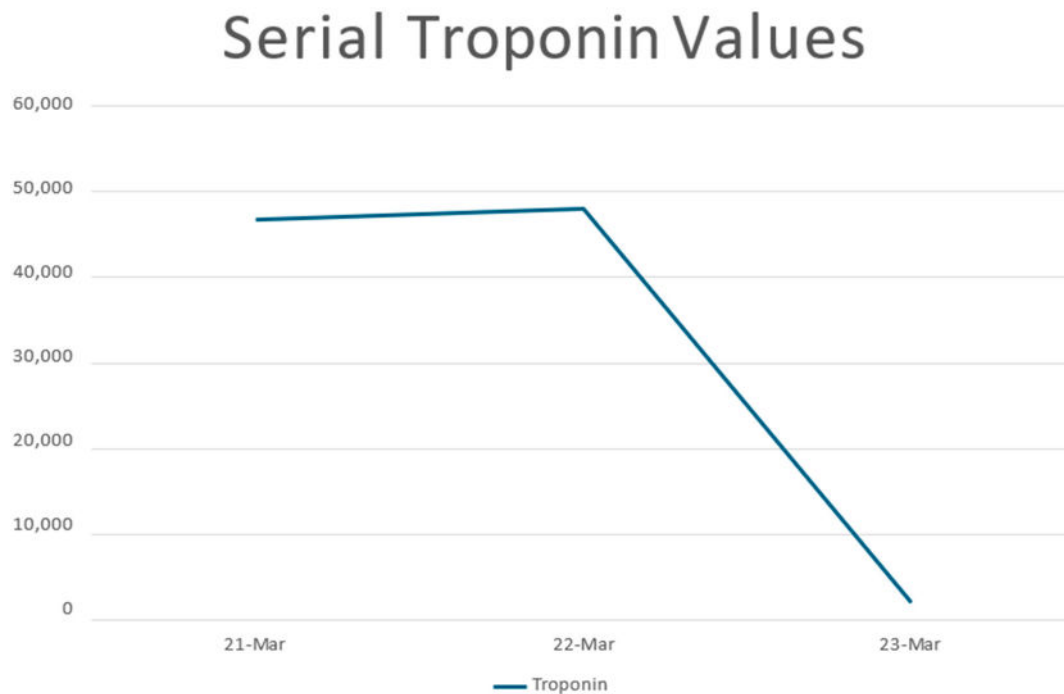


Figure 1: Serial Troponin Values.

Discussion

The inflammatory infiltration of myocardium, in the absence of coronary artery stenosis or preexisting cardiac disease, is classified as either fulminant, acute nonfulminant, chronic active, or chronic persistent myocarditis [1]. Fulminant myocarditis is characterized by severe cardiovascular dysfunction and Class IV congestive heart failure, whereas the acute non-fulminant form presents with moderate cardiac dysfunction, which, in some cases, may progress to heart failure. Over time, the inflammatory infiltrates in acute nonfulminant myocarditis tend to resolve [1].

Infections, particularly those caused by the cardiotropic viruses Coxsackie group B and other enteroviruses, remain the most common etiologies of acute myocarditis. Advanced HIV infection may cause chronic active myocarditis leading to dilated cardiomyopathy. Other infectious causes include rickettsiae and *Trypanosoma cruzi*, the parasite that causes Chagas disease. SARS-CoV-2 is the most recent addition to the list of known viral causes.

The diagnostic evaluation of myocarditis includes laboratory studies (troponin, complete blood count, and inflammatory markers), EKG, echocardiography, and cardiac MRI, which can demonstrate characteristic late gadolinium enhancement. Cardiac positron emission tomography scan can be used to diagnose myocarditis caused by sarcoidosis. Endomyocardial biopsy, which is rarely performed, can establish a histologic diagnosis in some cases. Screening laboratory studies for autoimmune disorders may be diagnostic. Notably, viral serologies are not routinely recommended as part of the diagnostic workup [1].

The cornerstones of treatment of myocarditis include management of heart failure and arrhythmias. Colchicine and nonsteroidal anti-inflammatory drugs may be used for concomitant pericarditis. Immunosuppressive therapy is reserved for eosinophilic or sarcoid myocarditis. Targeted therapy for autoimmune diseases may also be employed. Exercise restriction is recommended. The use of intravenous immunoglobulin has not shown a clear benefit in the treatment of myocarditis.

The role of antimicrobial therapy for myocarditis has not been assessed in detail because the causative organism is often not identified, and randomized controlled clinical trials of anti-infectives have not been conducted [3]. However, emerging research in the field of molecular biology has suggested that antiviral agents may reduce viral replication within cardiomyocytes in vitro. Skin cells may be converted into pluripotent stem cells by adding Yamanaka factors. Stem cells are engineered into cardiomyocytes [4]. Once such engineered cells have been infected with SARS-CoV-2, the addition of remdesivir decreases viral replication. Cellular models of cardiomyocytes infected with other microorganisms in vitro and then treated with antimicrobials could be useful in guiding future clinical trials.

Salmonella myocarditis has been rarely reported in the literature; only 9 cases have been described, including 5 cases of *Salmonella typhimurium* and single reports of infection caused by *S. enteritidis*, *S. choleraesuis*, and *S. heidelberg*. Mortality was 77.8%. In addition, 6 cases of myopericarditis have been reported, caused by *S. enteritidis*, *S. typhimurium*, and *S. agona*, with an overall mortality rate of 33.3%. [5] Clinical trial data for treatment of *Salmonella* myocarditis are unavailable. Experts suggest treatment with ciprofloxacin, levofloxacin, or ceftriaxone. Potential alternative regimens include azithromycin, cefixime, or trimethoprim-sulfamethoxazole.

Competitive athletes with a history of myocarditis require thorough cardiac assessments 3 to 6 months after illness. This evaluation includes resting and exercise EKGs, a 24-hour Holter monitor, and an echocardiogram. Criteria for clearance of athletes to return to competitive sports include lack of symptoms, normal exercise echocardiogram, absence of arrhythmias, and normal left ventricular systolic function [6].

This patient did not consume undercooked food or unpasteurized dairy products. It is plausible that some of the fruit he consumed in Mexico was cut with knives that had been rinsed in contaminated tap water. The Centers for Disease Control and Prevention offered the following guidance regarding travelers' diarrhea: "When you travel to areas of risk, remember to 'boil it, cook it, peel it, or forget it'" [7]. Caution is necessary when consuming food items that may not have been handled or prepared under strict sanitary conditions.

This case highlights the importance of considering non-viral infectious causes of myocarditis, including salmonellosis, when evaluating patients with antecedent gastrointestinal symptoms.

Salmonella-associated myocarditis and myopericarditis remain rare but have been associated with substantial mortality, particularly in cases with fulminant presentations [7]. The true incidence of *Salmonella* myocarditis is likely much higher than reported, as many cases likely go undiagnosed. This patient's favorable outcome, which included full recovery and return to competitive swimming, emphasizes the importance of early recognition and treatment of bacterial myocarditis.

Additionally, the case highlights the potential utility of GI PCR testing for establishing the microbial etiology of myocarditis. The sensitivity and specificity of GI PCR in myocarditis cases have not been established, and prospective studies of the role of this test in diagnosing infectious causes of myocarditis have not been carried out. Data from such studies could be utilized to clarify the relative frequencies of the various causes of myocarditis in different populations and to aid in devising empiric treatment strategies.

Conclusions

Myocarditis has dozens of microbial etiologies. *Salmonella* myocarditis has been rarely reported but likely has been underdiagnosed. Myocarditis can be life-threatening, particularly in fulminant cases, but the prognosis could potentially be improved with prompt diagnosis and antimicrobial treatment. This case highlights the importance of considering *Salmonella* as a potential cause of myocarditis, especially when cardiac manifestations are preceded by gastrointestinal symptoms. Competitive athletes who develop myocarditis can safely resume their activities following the resolution of their illnesses and clearance by a cardiologist. Gastrointestinal PCR testing holds promise as a tool

for identifying some of the microbial causes of myocarditis and for improving empiric and targeted treatment regimens.

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