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Troponin Elevation in Acute Cholangitis: A Case Report

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KEYWORDS

Acute cholangitis, elevated troponin, non-cardiac troponin, systemic inflammation

ABSTRACT:

The case report discusses an unusual presentation of elevated Troponin I in a 20-year-old female with a history of liver transplantation and recurrent cholangitis. Despite presenting with chest pain and ECG changes, subsequent cardiac investigations revealed no myocardial damage. The elevated troponin was attributed to systemic inflammation from acute cholangitis. This report highlights the need for careful interpretation of cardiac biomarkers in inflammatory conditions and offers the first known pediatric case linking cholangitis to transient troponin elevation

1. Introduction

Cardiac troponin T and I (cTnT and cTnI) are cardiac regulatory proteins that control the calcium mediated interaction between actin and myosin. Measurement of these cardiac enzymes is superior in terms of sensitivity and specificity in the identification of cardiac muscle damage [1,2]. However, previous reports have also suggested that elevated serum troponin level is not just confined to myocardial injury. Serum troponin levels have been reported to elevate in other conditions such as pulmonary embolism, heart failure, septicemia, renal failure, and post-chemotherapy. [3] In some of these conditions, such elevation has also been shown to be of prognostic significance. We present a patient with acute cholangitis, in whom elevated troponin was identified in the absence of any cardiac pathology. The purpose of this case report is to document a rare instance of troponin elevation in a pediatric patient with acute cholangitis, in the absence of cardiac pathology, and to propose systemic inflammation as a possible etiology.

2. Case Report:

A 20-year-old female with a history of congenital hepatic arterio-venous malformation for which she had undergone orthotopic liver transplant in the past, was admitted to the hospital with signs of ascending (acute) cholangitis. In addition, her history was complicated by poorly controlled type 1 diabetes mellitus and stage 2 chronic kidney disease. The patient had a history of recurrent cholangitis in the past and had a biliary drain in situ. Cardiology was consulted for an acute episode of chest pain during the hospitalization; that was unresponsive to opioid treatment. She remained hemodynamically stable and displayed no other concerning cardiac signs. An electrocardiogram was done which showed T wave abnormalities in the anterior precordial leads (Figure 1). There were no ST segment abnormalities. In comparison, her baseline ECG performed during a previous hospitalization was normal (Figure 2). Serum troponin-I level performed in view of the abnormal ECG, found elevated reading at 0.229 ng/mL (upper limit of normal = 0.03 ng/mL). An echocardiogram performed showed a structurally normal heart with normal biventricular systolic function without any focal wall motion abnormalities. Her chest pain subsided spontaneously in a few hours, and her troponin level normalized the next day. A follow-up electrocardiogram performed the next day showed normal sinus rhythm with resolution of prior T wave changes (Figure 3).

3. Discussion:

The case underscores the clinical importance of recognizing non-cardiac causes of troponin elevation, particularly in pediatric patients with complex hepatobiliary histories to avoid unnecessary cardiac interventions and misdiagnosis. Cardiac TnI is a sensitive marker for myocardial injury. Any elevation of troponin in a pediatric patient warrants an ECG and an echocardiogram, to evaluate for possible myocardial infarction or myocarditis. In some instances, diagnostic cardiac catheterization or advanced cardiac imaging may be warranted in addition. When the above cardiac workup is inconclusive, other potential etiologies should be sought after, including renal failure, hypothyroidism, pulmonary embolism, sepsis, or other inflammatory processes. Elevated troponin I in our patient was likely not a consequence of myocardial ischemia or myocarditis. Fox Et. al. have previously reported a case of a 51-year-old male with elevated troponin in association with acute cholecystitis. There have been no prior pediatric reports of similar elevation of troponin in association with gastrointestinal disorders. We hypothesize that the troponin elevation in our patient was secondary to her systemic inflammatory process related to ascending cholangitis. To the authors' knowledge,

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this is the first reported case in whom acute ascending cholangitis was associated with elevated troponin.

4. Conclusion:

The case highlights a rare association between acute ascending cholangitis and transient troponin elevation in the absence of cardiac pathology. Clinicians should be cautious in interpreting elevated troponin levels, especially in pediatric patients with underlying inflammatory conditions to prevent unnecessary cardiac interventions.

5. Figures:

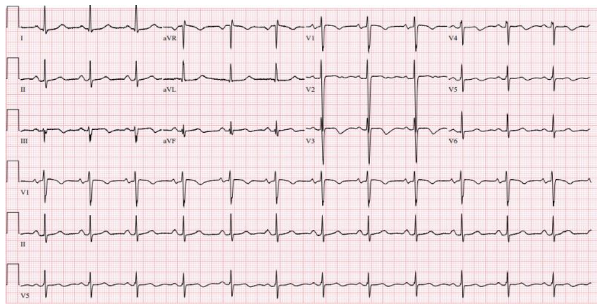

Figure 1: Electrocardiogram during the episode of chest pain

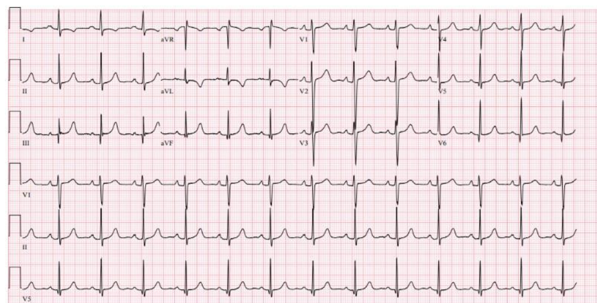

Figure 2: Electrocardiogram at baseline

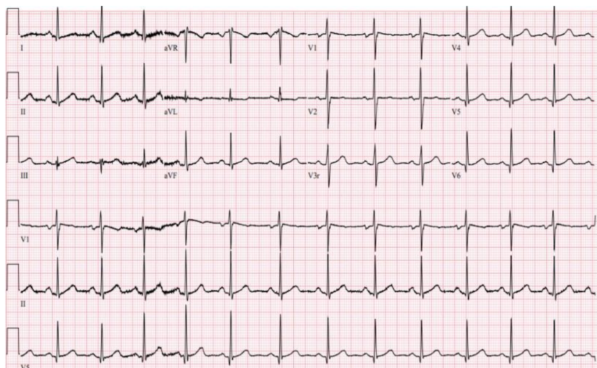

Figure 3: Electrocardiogram performed the next day of the chest pain and elevated troponin

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