



Troponin Elevation in Acute Cholangitis: A Case Report

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Keywords: Acute cholangitis; Elevated troponin.

Abstract

Background: Cardiac troponins T and I are highly sensitive and specific markers of myocardial injury. Although typically associated with cardiac pathology, elevated troponin levels have been described in several non-cardiac conditions, including sepsis, renal dysfunction, pulmonary embolism, and certain gastrointestinal diseases. However, reports in pediatric patients are rare.

Case presentation: A 20-year-old female with a history of congenital hepatic arteriovenous malformation status post orthotopic liver transplant, poorly controlled type 1 diabetes mellitus, chronic kidney disease stage 2, and recurrent cholangitis presented with acute ascending cholangitis. During hospitalization, she developed acute chest pain with new T-wave abnormalities on ECG. Serum troponin-I was elevated at 0.229 ng/mL (normal < 0.03 ng/mL). Despite these findings, she remained hemodynamically stable, and echocardiography demonstrated normal biventricular function without wall-motion abnormalities. Her chest pain resolved spontaneously, troponin normalized within 24 hours, and repeat ECG returned to baseline.

Discussion: Comprehensive cardiac evaluation did not support myocardial ischemia or myocarditis. Given the rapid normalization of troponin and absence of structural or functional abnormalities, the elevation was most consistent with a systemic inflammatory response to cholangitis. Although non-cardiac causes of troponin elevation are recognized, no pediatric cases associated with gastrointestinal pathology have been reported.

Conclusion: This case describes the first known pediatric presentation of transient troponin elevation in the setting of acute ascending cholangitis, emphasizing the need to consider systemic inflammatory causes when cardiac evaluation is unrevealing.



Background

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 are also reportedly elevated 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.

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. 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, not relieved with opioids. She was hemodynamically stable without any other concerning cardiac signs or symptoms. 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 by the next day. A repeat electrocardiogram performed the next day showed normal sinus rhythm without any T wave abnormalities (Figure 3).

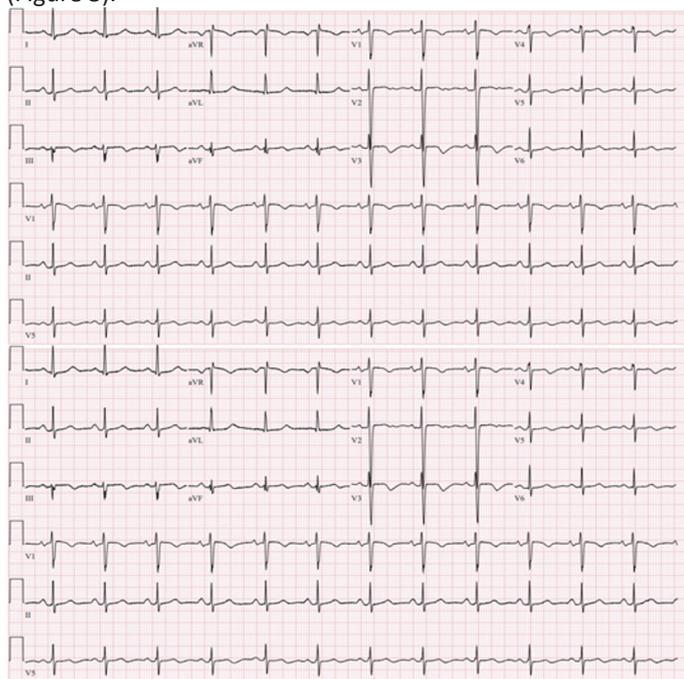


Figure 1: Electrocardiogram during the episode of chest pain.

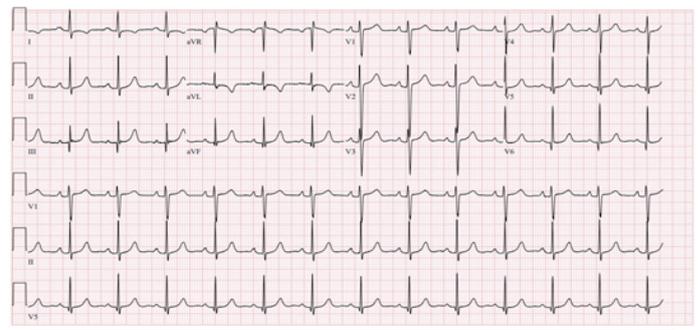


Figure 2: Electrocardiogram at baseline.

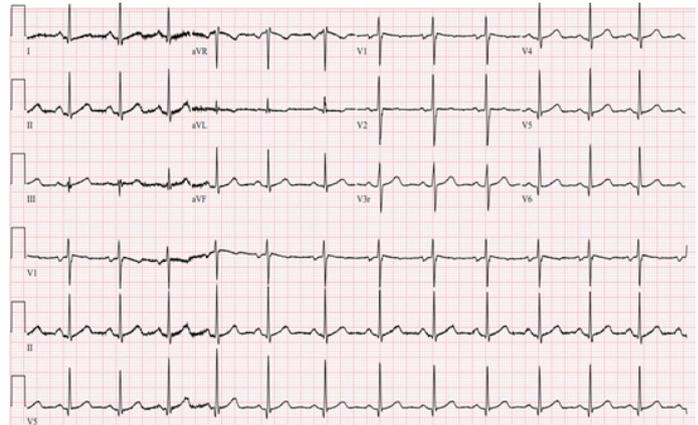


Figure 3: Electrocardiogram performed the next day of the chest pain and elevated troponin.

Discussion

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, and sepsis or other inflammatory processes. Elevated troponin I in our patient was likely not a consequence of myocardial ischemia or myocarditis. Fox et. al. has previously reported a case of a 51-year-old male with elevated troponin in association with acute cholecystitis. There have 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 this is the first reported case in whom acute ascending cholangitis was associated with elevated troponin.

Author declarations

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Conflict of interest

All the authors have no conflicts of interest to disclose.

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