

## Case Report

# Positive Troponin T in a Chagasic Patient with Sustained Ventricular Tachycardia and No Obstructive Lesions on Coronary Angiography

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*The assessment and stratification of patients with chest pain in the emergency unit may indicate the appropriate therapy for each patient based on the probability of the presence of acute coronary artery disease and on the risk of its major cardiac events. That assessment is based on the triplet: clinical setting, electrocardiographic findings, and markers of myocardial lesion. We report the case of a 58-year-old male chagasic patient admitted to the emergency unit due to chest pain and palpitations, with an electrocardiogram showing sustained ventricular tachycardia and positive troponin measurement (0.99 ng/mL). The patient underwent coronary angiography, which evidenced no obstructive coronary artery disease.*

Troponin is an important marker of myocardial lesion with a fundamental role in risk stratification of patients with acute chest pain in the emergency unit. Although troponin has an excellent accuracy for identifying myocardial necrosis, it has been shown that its level can increase in numerous nonatherosclerotic cardiac diseases. We report the case of a chagasic male admitted to the emergency unit with chest pain and sustained ventricular tachycardia, positive troponin measurement, and no obstructive lesions on coronary angiography.

## Case Report

The patient is a white, 58-year-old male with chagasic dilated cardiomyopathy, who previously was in New York Heart Association functional class I, using a definitive ventricular pacemaker. He was admitted to the emergency unit complaining of oppressive chest pain, which had started 7 days before. The chest pain worsened on exertion, did not irradiate, was of small intensity, and had no other associated factors besides the sensation of palpitation and general malaise. On physical examination, the patient was conscious, oriented, and eupneic. His cardiac auscultation showed rhythmic cardiac sounds of normal intensity, and no audible heart murmurs.

His heart rate was 150 bpm, and his blood pressure was 130/80 mmHg. His lungs showed respiratory sounds in all fields and no rales. He had no signs of systemic congestion. The electrocardiogram on hospital admission showed sustained ventricular tachycardia and an approximate heart rate of 150 bpm (fig. 1).

After receiving intravenous amiodarone (300 mg), he regained his VVI pacemaker rhythm. Due to the history of chest pain, serum measurement of troponin T was taken 6 hours after admission, and the result was 0.999 ng/mL (reference value: 0.010 ng/mL). The serial measurements of CK-MB were normal, as were the hemogram, coagulogram, and urine I. On admission, creatinine was 1.5 mg/dL and potassium was 4.3 mEq/L. The hemodynamically stable patient, with a VVI pacemaker rhythm, was referred to the coronary unit, where he was maintained with intravenous nitroglycerin, heparin, amiodarone, and furosemide, in addition to oral beta-blockers, angiotensin-converting-enzyme inhibitors, acetylsalicylic acid, and clopidogrel. The echocardiogram showed a significant enlargement in the left ventricle, significant contractile dysfunction, no segmentary alterations in contractility, and moderate mitral insufficiency. On risk stratification for coronary heart disease, despite the difficulty in the electrocardiographic assessment because of the pacemaker rhythm, the patient was considered at high risk due to his history of chest pain associated with severe arrhythmia and ventricular dysfunction and positive troponin T measurement. The coronary angiography then performed showed no obstructive lesions (figures 2 and 3). No myocardial perfusion scintigraphy was performed.

While the patient waited for discharge from the coronary unit, he experienced sustained ventricular tachycardia with no pulse. He underwent electrical defibrillation, which was successful after the forth shock with a return of the pulse and hemodynamic instability. The patient continued to have episodes of nonsustained ventricular tachycardia with a pulse, which was reverted to the pacemaker rhythm by use of intravenous amiodarone. The patient was then referred for implantation of a cardioverter/defibrillator, which was successful. After this episode, the patient evolved with cardiogenic shock, acute renal dysfunction (peak creatinine – 6.4 mg/dL), and liver dysfunction with maximum values of ALT and AST of 5,011 UI/L and 12,560 UI/L, respectively. The serological research for hepatitis A, B, and C, cytomegalovirus, toxoplasmosis, and HIV was negative. The patient underwent hemodynamic monitoring with the Swan-Ganz catheter, which showed, in the initial hemodynamic measurements (with 10  $\mu$ g/kg/min of

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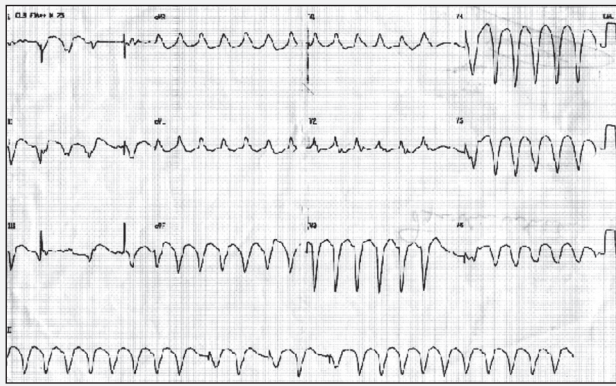


Fig. 1 – Electrocardiogram on admission.

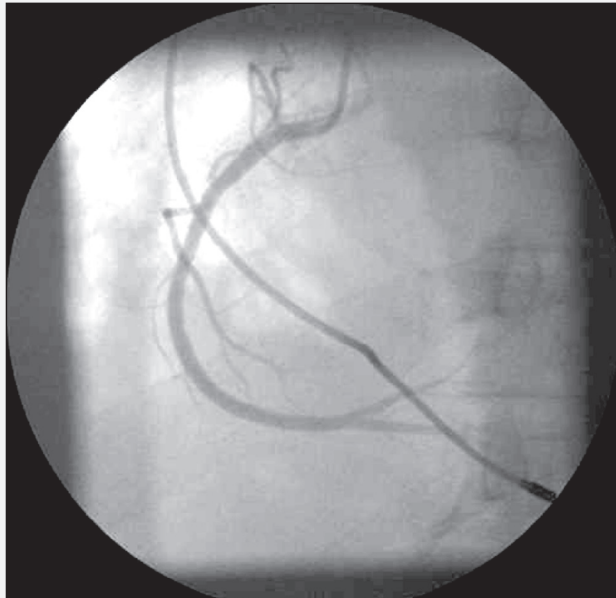


Fig. 2 – Right coronary artery.

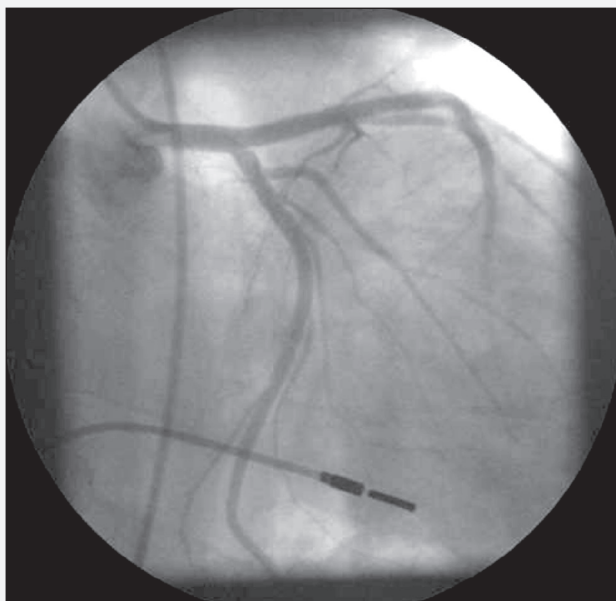


Fig. 3 – Left coronary artery.

dobutamine), a pulmonary capillary pressure of 30 mmHg, cardiac index of 2.4 L/min/m<sup>2</sup>, and peripheral vascular resistance index of 2052 D.s/cm<sup>5</sup>/m<sup>2</sup>. The respiratory measurements showed an oxygen supply of 450 mL/min/m<sup>2</sup>, consumption of 122 mL/min/

m<sup>2</sup>, and an oxygen extraction rate of 27%, with lactate measurement in the mixed venous blood of 1.8 mmol/L. The patient underwent a hemodialysis session with good clinical response, evolving to polyuria and gradual decrease in the levels of creatinine and hepatic enzymes. The patient was discharged from the coronary unit 12 days after admission.

## Discussion

Troponin is an important marker of myocardial lesion and plays a fundamental role in risk stratification of patients with acute chest pain in the emergency unit, due to its high sensitivity and specificity (100% and 83.3%, respectively, 12 hours after admission)<sup>1</sup> for identifying patients with unstable myocardial ischemic syndromes. It is better than the CK-MB measurement. Approximately 30% of patients who present with chest pain at rest, no elevation in the ST segment, and normal CK-MB levels are estimated to have positive troponin<sup>2</sup>. In addition, of the patients with unstable ischemic syndromes, no elevation in the ST segment, and normal CK-MB levels, elevation in troponin T or I identifies those at higher risk<sup>3</sup>, and a quantitative relation exists between the values of troponin T or I and the risk of death<sup>4</sup>. Despite this excellent accuracy in detecting myocardial lesions in acute coronary syndromes<sup>5</sup>, one cannot forget that troponins may be increased in several nonatherosclerotic cardiac diseases. Therefore, when interpreting the result of a troponin measurement, it is worth remembering that, although it is 100% specific for myocardial lesion, it is not 100% sensitive for myocardial infarction.

Several clinical conditions that result in cardiac lesion could increase serum troponin levels. If the troponin levels are elevated and the clinical findings are not consistent with myocardial infarction, other causes of myocardial lesion should be considered. The possibility of a false-positive result should be considered last. The following numerous conditions other than atherosclerosis could result in myocardial lesion: myocarditis, cardiomyopathy, congestive heart failure, sepsis, pulmonary embolism, rhabdomyolysis, thoracic contusion (trauma), coronary embolism caused by endocarditis, mural thrombus, prosthetic valves, neoplasias, inflammatory processes, including viral infection by coxsackie B, radiation-induced coronary stenosis, congenital anomalies of the coronary arteries, use of cocaine and other numerous conditions, such as Hurler's syndrome, homocysteinuria, rheumatoid arthritis, and systemic lupus erythematosus<sup>6</sup>.

Khan et al<sup>6</sup> studied 102 consecutive patients who had positive troponin I, 35 of whom did not have a final diagnosis of unstable myocardial ischemic syndrome, and only 3 complained of chest pain. The following diseases were found: nonischemic dilated cardiomyopathy, muscular diseases, central nervous system diseases, HIV, chronic renal failure, sepsis, and pulmonary and endocrine diseases. The mean value of troponin in patients with no atherosclerotic coronary arterial disease, however, was significantly lower than that in patients with acute ischemic syndromes (2.0±1.9 vs. 27.4±28.2 ng/mL; P<0.0001)<sup>6</sup>.

Basquiera et al<sup>7</sup> studied 39 chagasic patients with troponin T measurements and found only one positive result in the group with more severe heart disease. Those authors concluded that troponin levels are not associated with the earliest stages of the myocardial lesion in Chagas' disease. In addition, Ianni et al<sup>8</sup>,



studying the evolution of cardiovascular events in chagasic patients with the undetermined form of the disease, reported 1.2% of symptoms of coronary arterial disease in a follow-up of up to 177 months. An autopsy study<sup>9</sup> comparing 35 chagasic patients with 54 nonchagasic patients reported that the frequency of myocardial infarction and coronary atherosclerosis was similar in both groups, although the incidence of infarction without coronary artery disease or with minimum lesions was greater in chagasic patients as compared with that in nonchagasic patients.

Another important and controversial issue is related to troponin measurement in patients with renal dysfunction. Detectable levels of troponin T may be found in several patients with end-stage renal disease without acute heart disease; however, data in some studies showing false-positive results of troponin T in patients undergoing hemodialysis have been erroneously extrapolated to troponin I measurements. Although the precise mechanism of troponin I clearance is not known, no current evidence exists showing false-positive results in end-stage chronic renal patients; therefore, troponin I is believed not to be dialyzable<sup>10,11</sup>. On the other hand, in patients diagnosed with unstable myocardial ischemic syndrome and renal dysfunction, troponin T can predict the short-term prognosis independent of creatinine clearance, as reported by Aviles et al<sup>12</sup> in a study comprising more than 7,000 patients.

Finally, in a study with 889 consecutive patients, Bakshi et al<sup>13</sup> found 21 patients with positive troponin I and normal coronary

angiography or with mild coronary artery lesions. Of those, 6 were diagnosed as secondary to cardiac arrhythmias, 4 being supraventricular and 2 associated with ventricular tachycardia. Of those 2 patients with ventricular tachycardia, one had mild lesions on cine coronary angiography, and the other had normal coronary arteries.

Troponin measurement in the emergency unit revolutionized the medical care of patients with unstable myocardial ischemic syndromes and their prognostic assessment. However, sometimes we are faced with patients whose troponin is positive and coronary angiography shows no obstructive lesions. In such circumstances, interpreting the result as false positive may be a comfortable, passive attitude.

We report the case of a patient with previously compensated chagasic dilated cardiomyopathy and discrete alteration in the renal function on admission, who complained of chest pain and palpitations. His electrocardiogram showed sustained ventricular tachycardia, reverted with intravenous amiodarone. Due to the presence of chest pain and severe ventricular arrhythmia, the troponin measurement was performed aiming at risk stratification for coronary artery disease. As the troponin result was undoubtedly positive, cine coronary angiography was performed, and no atherosclerotic obstructive coronary artery disease was observed. Both Chagas' disease and sustained tachyarrhythmia could cause an elevation in troponin, although the possibility of a false-positive result could not be definitively ruled out.

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