

Epileptic Seizure vs. Myocardial Infarction: the Significance of Cardiac Troponin Levels

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Elevated cardiac troponin level is considered to be highly specific for myocardial injury, and if correlated either electrocardiographically or clinically, establishes the diagnosis of acute myocardial infarction. We report a patient with ischemic changes on ECG and increased troponin levels following a generalized tonic-clonic seizure but with no evidence of cardiac injury.

Patient Description

A 53 year old woman with a history of epileptic seizures treated with lamotrigine was admitted to the emergency room after a 15 minute episode of a GTC seizure that was terminated after intramuscular injection of 15 mg midazolam. On admission to the emergency room, physical examination revealed general symmetric weakness that gradually resolved within 24 hours. Her lungs were clear on auscultation and cardiovascular examination was normal, with regular rhythm and no gallops or murmurs. The ECG chart showed sinus tachycardia with 1 mm ST segment elevations at lead I and aVL. The patient's blood tests were within normal range with the exception of mildly elevated levels of creatine phosphokinase (255 U/L, normal < 170 U/L) and cardiac troponin I (4.8 ng/ml, normal < 0.5 ng/ml), which was consistent in a subsequent test the following day. Echocardiography at admission revealed normal global and regional left ventricular function with an ejection fraction of 60%. Exercise ECG and exercise thallium SPECT performed on the fourth day of her admission were normal, showing no perfusion defects. During the entire hospitalization the patient denied any type

of pain and her physical examination was consistently normal. After her discharge from hospital, the patient ruled out any clinical complaints and returned to full physical activity within 2 weeks. Routine blood tests including CPK were all within normal range.

Six years prior to the current admission, a screening test revealed that the patient was diabetic. Since then, she lost 70 kg (body mass index drop from 42 to 22) with a drastic change in lifestyle that for the past year has included four weekly training sessions in a triathletic program. During the last 3 years, the patient's HbA1C was consistently lower than 5 g/dl without any need for anti-diabetic drugs. Low density lipoprotein levels ranged between 100 and 120 mg/dl and high density lipoprotein levels were 60–70 mg/dl due to treatment with simvastatin (40 mg daily). Blood pressure values were also within the normal range. An annual ophthalmological examination revealed no evidence of diabetic retinopathy.

Comments

The present report describes a patient admitted for GTC seizure with suspected cardiac involvement manifested by increased cardiac troponin I and transient ST elevation on the ECG. Although cardiac troponin I elevation is considered a specific marker for myocardial damage, its interpretation in the setting of GTC seizure is more controversial.

Patients who developed myocardial infarction with increased cardiac troponin I level after GTC seizures have been described [1]. Importantly, these patients had

typical clinical complaints and abnormal echocardiographic and cardiac perfusion scans. Similar to our case, another report describes a patient with an increase in troponin level (> 5 ng/ml) following GTC seizures, yet the patient was asymptomatic with normal echocardiography and coronary angiography [2]. These findings raise questions regarding the mechanism and significance of troponin elevations following GTC seizures.

GTC seizure, like the one reported here, can be considered as a physiological cardiac stress test that increases heart rate, blood pressure and myocardial contractility. Tonic muscle contraction augments cardiac afterload and further increases cardiac oxygen demand. Therefore, a prolonged and isolated seizure can impose a perfusion/demand mismatch that may be severe enough to produce sub-endocardial ischemia in the absence of coronary flow impairment.

Although the cardiac troponin I immunoassay is considered to lack any cross-reactivity with troponin I found in human skeletal muscle, 17% of patients admitted to the emergency room with rhabdomyolysis (defined as CPK > 1000 U/L) but without any cardiac symptoms or findings had elevated levels of cardiac troponin I [3]. Our patient, despite her intensive physical exercise regimen, does not seem to fall into the latter category since her CPK level was 255 U/L with an abnormal ECG chart. Another condition that might cause seizures and elevated troponin levels is acute stroke [4]. However, even in stroke patients, elevated cardiac troponin I levels are thought to arise from neurally mediated myocyte damage. It has been suggested that an imbalance of the auto-

GTC = generalized tonic-clonic

CPK = creatine phosphokinase

onomic nervous system with resulting excess of sympathetic activity and increased catecholamine effect on the myocardial cells promotes this damage. Other causes found to increase cardiac troponin I in the absence of coronary artery disease are advanced renal failure, pericarditis, myocarditis, sepsis, hypotension, rapid atrial fibrillation, pulmonary embolism and coronary vasospasm. Elevated rheumatoid factor and heterophile antibodies were also reported as a false positive cause mimicking cardiac troponin I elevation [5].

In our patient we found no neurological deficit, permanent or temporary, and no radiological evidence of hemorrhagic or ischemic stroke on brain computed tomography. Clinical, imaging and laboratory tests ruled out other suggested etiologies such as those described above. Follow-up examination in our patient revealed normal rheumatoid factor levels. Finally,

we believe that the mechanism of cardiac troponin I elevation in epileptic seizures is most likely explained either by transient and reversible supply-demand mismatch at the myocardial level or, alternatively, neurally mediated transient myocyte damage as described in patients with stroke and cerebral hemorrhage. Current data on this issue are still lacking and a well-established single mechanism has not yet been elucidated.

We conclude that cardiac troponin elevation and ECG changes without clinical evidence – by non-invasive or invasive tests – of myocardial injury may represent a neuro-hormonal stress or excessive myocardial wall tension imposed by a prolonged GTC seizure, which therefore reduces specificity for acute coronary syndrome. Clinicians should be aware of this suggested mechanism when assessing patients with elevated cardiac troponin I following generalized tonic-clonic seizure.

References

1. Chin PS, Branch KR, Becker KJ. Myocardial infarction following brief convulsive seizures. *Neurology* 2004;63:2453–4.
2. Brobbey A, Ravakhah K. Elevated serum cardiac troponin I level in a patient after a grand mal seizure and with no evidence of cardiac disease. *Am J Med Sci* 2004;328:189–91.
3. Li SF, Zapata J, Tillem E. The prevalence of false-positive cardiac troponin I in ED patients with rhabdomyolysis. *Am J Emerg Med* 2005;23:860–3.
4. Dixit S, Castle M, Velu RP, Swisher L, Hodge C, Jaffe AS. Cardiac involvement in patients with acute neurologic disease: confirmation with cardiac troponin I. *Arch Intern Med* 2000;160:3153–8.
5. Makaryus AN, Makaryus MN, Hassid B. Falsely elevated cardiac troponin I levels. *Clin Cardiol* 2007;30:92–4.

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