Elevated Cardiac Troponin T After an Epileptic Seizure: Is There a Concomitant AMI?

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Case Report

Abstract

Cardiac troponins are sensitive and specific indicators of myocardial injury and are traditionally used to support the diagnosis of myocardial infarction. However, raised cardiac troponins may also be found in a wide range of non-cardiac medical conditions. Examples include sepsis, neurological conditions, renal failure, burns and certain inflammatory and infiltrative diseases. Emergency physicians should consider these alternative diagnostic possibilities when faced with elevated cardiac troponins in the absence of other clinical and laboratory evidence to support a myocardial infarction. We report a case of a 29-year-old male who presented to the emergency department after an epileptic fit with a significantly elevated serum cardiac troponin. The association between seizures and raised cardiac troponins must be taken into account by emergency physicians to avoid misdiagnosing and over-investigating patients.

Keywords: Myocardial Injury; Myocardial Infarction; Troponin; Seizures

Introduction

Cardiac troponins are sensitive and specific indicators of myocardial injury and are traditionally used to support the diagnosis of myocardial infarction (MI) [1-3]. However, raised cardiac troponins may also be found in a wide range of non-cardiac medical conditions. Examples include sepsis, neurological conditions, renal failure, burns and certain inflammatory and infiltrative diseases [2-6].

This case reports a patient admitted after an epileptic fit with raised cardiac troponin T but with no other clinical and laboratory evidence to suggest an acute coronary syndrome. We discuss his evaluation, possible differential diagnosis and hypothesised pathophysiology for this phenomenon. There have been few similar cases reported and this case aims to highlight the possible association between seizures and raised serum cardiac troponins.

Case Report

A 29-year-old male with a past medical history of asthma and intravenous drug abuse presented to the emergency department with a witnessed seizure which lasted for 20 minutes and aborted spontaneously. His spouse described the jerking of his two upper limbs and associated eye-rolling and tongue biting. There was no loss of continence. The patient denied any pre-ictal aura, intercurrent illness, head trauma, any recent drug use or alcohol ingestion. Post-ictally, he was noted to be drowsy and confused and had one episode of non-bilious, non-bloody vomiting. He had a prior history of seizure in the past year, but defaulted neurology follow-up.

On arrival in the emergency department, the patient was cyanosed and hypoxic on room air with oxygen saturation of 85%. He was also tachycardic at 110 beats per minute. He was given supplemental oxygen and his oxygen saturation levels normalised thereafter. His cardiovascular, respiratory and abdominal examinations were unremarkable. The patient’s neurological examination was normal.

An electrocardiogram performed showed sinus tachycardia, T wave inversions in the inferior leads and Q waves in lead III. Serum cardiac troponin T was noted to be grossly elevated at 519 ng/L (normal <30 ng/L). A chest radiograph revealed bilateral lung infiltrates. Other haematological and biochemical blood tests were normal. The CT brain was done and it showed white matter changes with slight loss of volume, suggestive of leukodystrophy. A decision was then made to perform a computed tomography pulmonary angio-
gram (CTPA), to rule out a pulmonary embolism. However, in view of his previous drug abuse history, vascular access could not be achieved easily and hence the CTPA could not be performed. The radiologist on duty suggested a CT scan of the thorax, which subsequently ruled out the possibility of any significant pulmonary embolism. In view of the previous investigations and chest radiography findings of bilateral lung infiltrates, another diagnostic possibility considered was that of an acute coronary syndrome. The patient was hence admitted under the care of the Cardiovascular Medicine (CVM) department.

The patient was also referred to the Neurology department during his admission, where Magnetic Resonance Imaging (MRI) of his brain was performed. The MRI revealed leukoencephalopathy and unusual cystic changes possibly due to necrosis. The Neurologist made the diagnosis of epilepsy secondary to the underlying leukoencephalopathy. He tested negative for meningitis screening and there was no further progression of his symptoms. However, the patient ultimately refused to stay and was subsequently discharged against medical advice. Before his discharge, he was prescribed the anti-epileptic medication levetiracetam and follow-up appointments were arranged.

Discussion

Cardiac troponins consist of three different subunits (troponin C, troponin T and troponin I) and are located on the actin filament of skeletal and cardiac muscle. [3, 5] Troponin T and troponin I are the only two troponin isoforms which are expressed in cardiac muscles, hence the two are the more sensitive markers for myocardial injury. Raised cardiac troponins in the absence of typical electrocardiogram features like ST segment elevation are useful in identifying patients who are at high risk of suffering an acute coronary syndrome and serve to ensure timely referral for specialist intervention and management [5, 7].

Cardiac troponins may also be elevated in a number of situations which are not related to coronary plaque occlusion or rupture, which are important primary causes of ischaemic myocardial injury [1, 3]. There are also many secondary causes of ischaemic myocardial injury, which include cardiac arrhythmias, cardiomyopathies, procedures like coronary intervention and pulmonary embolism. Myocardial injury may also result from non-ischaemic causes and aetiologies for non-ischaemic myocardial injury may be traumatic, infective or inflammatory in nature or related to pre-existing comorbidities. These include disseminated sepsis, burns, renal failure, stroke, myopericarditis and receiving cardiotoxic medications during chemotherapy [2, 3, 6]. Other rarer aetiologies of raised cardiac troponins have been reported in literature and an example would be Takotsubo cardiomyopathy, which is stress cardiomyopathy related to apical ballooning [8].

In this case, our patient did not report any typical symptoms suggestive of an acute coronary syndrome like chest pain, dyspnea or diaphoresis. Although the initial electrocardiogram suggested the possibility of a pulmonary embolism.

Subsequent Investigations were promptly carried out to ascertain the presence of pulmonary embolism. A CTPA would have been the most ideal investigation to rule out a pulmonary embolism but due to difficult venous access, a CT thorax was performed. The CT thorax ruled out the presence of any significant pulmonary emboli.

Our patient was a young healthy male with no history of malignancies or organ failure. On initial presentation, he was clinically non-toxic and his vitals were stable apart from tachycardia and hypotension which subsequently normalised on supplementary oxygen. His neurological function was grossly intact, which ruled out the possibility of stroke. The history obtained from his spouse ruled out the possibility of any traumatic injuries and this was corroborated during the physical examination.

Further assessment revealed that the patient had an underlying epileptic epilepsy disorder, as evidenced by the leukoencephalopathy and cystic changes detected on his MRI. Following extensive workup both in the emergency department and inpatient ward, the exact cause of the elevated cardiac troponin was still unknown.

Troponin release of unknown origin, in patients with different disorders, continues to remain controversial. Some of the hypothesis include [9-13]:

1. The ‘reversible’ ischaemia phenomenon, which is said to be due to the temporary reduction in coronary blood flow during certain situations, like seizures.

2. Autonomic involvement, resulting from alterations in the central nervous system, predominantly causing sympathetic overactivity.

3. Concomitant presence of vascular risk factors such as hypertension, hyperlipidaemia and diabetes. Hypertension has been noted to have the strongest association.

There have been previous similar case reports describing an increase in serum cardiac troponin levels after epileptic seizures, without any evidence of myocardial injury. Previous cases have identified patients with similar presenting complaints and raised serum cardiac troponins in the absence of myocardial injury in relevant investigations. A retrospective cohort study also identified a relationship between generalised convulsive fits and concomitant cardiac troponin release. However, these studies mainly identified the elevation of serum cardiac troponin I, while in our case, we report the elevation of cardiac troponin T [11-13].

A seizure happens because of uncontrolled electrical activity in the brain, which produces physical convulsions, sensory disturbances or syncope. During a seizure, especially in the case of our patient who had generalised tonic-clonic convulsions, the demand of myocardial oxygen consumption would be greatly increased due to tachycardia and increased cardiac contractility. Seizures also cause periods of apnea which reduce the oxygen supply to the myocardium. This mismatch in oxygen supply leads to myocardial injury and release of
cardiac troponins [9-12].

Another hypothesis for the increase in cardiac troponins following seizure has been attributed to the catecholamine surge resulting from autonomic nervous system dysfunction. The catecholamine surge results in increased myocardial contractility and myocyte damage due to the influx of calcium and other electrolytes that happens in response to the catecholamine surge. This sequence of events subsequently leads to the increase in serum cardiac troponins.

On the other hand, clinically defined MI is a rare complication of epilepsy. The factors for consideration, when assessing such patients, include:

1. Generalized tonic clonic seizures or status epilepticus can be considered as conditions which causes unphysiological cardiac stress

2. An epileptic may not be able to complain of symptoms such as chest pain or shortness of breath during seizure. Moreover, knowing that the ECG may not always be sufficiently diagnostic in all patients with MI, frontline clinicians have to bear this in mind when making decisions

3. The difficulty in obtaining an early ECG in a seizure patient, especially during the seizure episode

4. The phenomenon of sudden unexplained death in epilepsy does exceed that in the general population by some 24 times. Cardiac pathology indicative of cardiac injury, have been reported in these patients. These led to the consideration of suspected coronary artery disease in patients showing increased cardiac troponins following a seizure [14-19].

Conclusion

The 2012 Joint European Society of Cardiology, American College of Cardiology, American Heart Association and World Heart Federation Taskforce recommends that an elevated value of cardiac troponin, in the absence of clinical evidence of ischaemia, must prompt a search for other causes of myocardial necrosis [20].

There are many differential diagnoses for raised cardiac troponins. In most cases, raised cardiac troponins coupled with a typical clinical history supports the diagnosis of an acute coronary syndrome. In other cases, raised cardiac troponins may be an indicator of myocardial damage resulting from other pathologies. We advocate that Emergency Medicine physicians should be aware of the link between seizures and raised serum cardiac troponins. This would prevent physicians from misdiagnosing patients and ensures that patients receive the necessary investigations and treatments, in a timely fashion.

Conflict of Interest statement

We declare that we have no conflict of interest.
15. Lim W, Cook DJ, Griffith LE et al. Elevated cardiac tropo-

16. Van de Lende M, Surges R, Sander JW et al. Cardiac ar-
rhythmias during or after epileptic seizures. J neurol Neuro-
surg Psychiatry. 2015, 0: 1-6.

17. Lamberts RJ, Blom MT, Novy J et al. Increased prevalence
of ECG markers for sudden cardiac arrest in refractory epi-

18. Khattab AAA, Abd-Elnaby SA, Dwood AA-E et al. Cardiac
Troponin I level among children with epileptic seizures. The

19. Eskandarian R, Asghari N, Darban M et al. cardiac tro-
ponin levels after complicated and uncomplicated epileptic

20. Thygesen K, Alpert JS, White HD. Joint ESC/ ACC/AHA/
WHF taskforce for the re- definition of MI/ universal defini-