Falls-associated elevation of troponin level in an older woman with normal coronary arteries

CASE REPORT

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ABSTRACT

A 75-year old woman with no history of ischaemic heart disease sustained a fall with scalp laceration. She had no chest pain. Troponin I was found to be high on admission and remained so for one day. Computed tomographic coronary angiogram was normal. She was discharged on low dose aspirin. The issue of non-ischaemic elevation of troponin is discussed.

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INTRODUCTION

Troponins are highly sensitive biomarkers of myocardial injury. They are useful in the diagnosis of acute myocardial infarction and risk stratification of acute coronary syndrome.[1] Nonetheless, troponin level may be raised in many conditions other than acute myocardial infarction, posing a diagnostic difficulty for clinicians.[2,3] We report an older woman who fell and sustained a scalp laceration. Troponin I was found to be high on admission; further investigation revealed no coronary artery disease.

CASE REPORT

In March 2015, a 75-year old woman was taken by ambulance to the emergency department with a head injury and scalp laceration secondary to a non-syncopal fall. She had normal cognitive function and no history of ischaemic heart disease, diabetes mellitus, or hypertension. She was a non-smoker and non-drinker, and was independent in walking and activities of daily living. She had been taking simvastatin 10 mg nocte for hyperlipidaemia for 3 years. The patient reported the fall to be accidental when she missed the curb of the street. She sustained a 5 cm long laceration on her left forehead. She had no loss of consciousness or any other symptoms or injury.

Computed tomography (CT) of the brain revealed no intracranial haemorrhage or skull fracture. In the ward, she was noted to be very nervous with a blood pressure of 200/100 mm Hg and a heart rate of 100 beats per minute. After a period of rest, her blood pressure settled to around 140/70 mm Hg and her heart rate dropped to 80 beats per minute.

Blood tests on admission revealed a raised troponin I level of 0.83 ng/ml (cut-off value for acute myocardial infarction, 0.5 ng/ml with 96% sensitivity and 94% specificity), and creatine phosphokinase (CPK) of 145 U/L (normal range, 40-160 U/L). Her renal and liver function tests were normal with a creatinine level of 53 umol/L. Electrocardiography demonstrated sinus rhythm and no Q wave or any ST or T changes. Because of the raised troponin I, she was started on low molecular weight heparin and aspirin 80 mg daily.

Subsequent blood tests showed that the troponin I level peaked at 2.22 ng/ml 6 hours after the first test and CPK remained static at 120 U/L. The troponin I level subsequently dropped to 1.9 ng/ml at 12 hours and 0.6 ng/ml at 18 hours.

She was seen by a cardiologist, and echocardiography showed normal contractility with ejection fraction of 60% and no valvular lesion. To exclude coronary heart disease, CT coronary angiogram was performed on day 2 and showed completely normal coronary arteries with an Agatstan

score of 0. This suggests an extremely low likelihood of any coronary artery disease in an asymptomatic individual.[4] Low molecular weight heparin was stopped after 3 days, but aspirin 80 mg daily was continued. She was then seen by a geriatrician who carried out a comprehensive geriatric assessment. She was advised to perform balance exercises such as Tai Chi and was scheduled for follow-up in 12 weeks. The patient was discharged on day 3 and remained well.

DISCUSSION

Falls and imbalance occur commonly in older people and are a major topic in geriatric medicine.[5,6] In Hong Kong, the prevalence of older people having at least one fall in the preceding 12 months ranges from 18% to 19.3%, with 75.2% sustaining an injury and 7.2% a serious injury.[7,8] Non-ischaemic cause of troponin elevation after falls has not been reported.

Troponins are regulatory proteins that control the calcium-mediated interaction of actin and myosin. The troponin complex consists of 3 subunits: troponin T, troponin I, and troponin C.[9] The skeletal and cardiac isoforms of troponin T and troponin I, but not troponin C, are distinct, and skeletal isoforms are not detected by the monoclonal antibody-based assays currently in use. Although troponin is sensitive and specific for detection of myocardial injury, it can be elevated in other conditions and is not specific for myocardial infarction.[10] Non-ischaemic causes of troponin elevation include sepsis, pulmonary embolism, renal failure, heart failure, pericarditis, cerebrovascular accident, tachycardia, Takotsubo cardiomyopathy, endurance exercise, angioplasty, cardiac contusion, and chemotherapy. The exact mechanism of non-ischaemic troponin elevation is complex and not well known. Troponins have a cytoplasmic pool and structural pool.[11] Myocardial injury first leads to release of the cytoplasmic pool resulting in a peak serum concentration at 24 to 36 hours after injury. The half-life of troponin in serum is around 2 hours, and the continuous elevation of troponin for a week or more depends on gradual degradation of the myofibrils and release of troponin from the structural pool as a result of myocardial injury. The mechanism for non-ischaemic troponin elevation is heterogeneous and multifactorial. In tachycardia, it is thought to be the result of shortening of diastole with sub-endocardial ischaemia.[12] Troponin raised after angioplasty might be the result of procedural complications such as side branch occlusion and thrombus formation.[13] In heart failure, apoptosis, myocyte damage from neurohumoral activation and inflammatory cytokines might lead to troponin increase.[14] Acute right ventricular strain secondary to increased pulmonary artery stenosis, and hypoxaemia and hypoperfusion secondary to low cardiac output are postulated causes of raised troponin.[15] In Takotsubo cardiomyopathy, physical or emotional stress leads to increased circulatory catecholamines and myocardial stunning and surge in troponin.[16] In pericarditis, there might be inflammatory involvement of the epicardium with consequent myocyte damage.[17] Inflammatory cytokines, hypotension, tachycardia, and increased catecholamines can act together and result in raised blood levels of troponin. After endurance exercise, a rise in troponin level may be due to the release of cytoplasmic troponin or increased permeability of myocyte cells under stress.[18] The reason for a rise in troponin in renal failure remains unknown.[19]

The exact mechanism of raised troponin in this patient was not known. There was no evidence of coronary artery disease on CT coronary angiogram, and her raised troponin subsided quickly in less than one day, probably owing to the release of cytoplasmic troponin with change of myocyte permeability as a result of catecholamine surge. She was quite nervous on the day of admission with high blood pressure and tachycardia but these subsided after resting. Probably because of the lack of myocardial cell injury, troponin I level dropped within a day. Other possible mechanisms of a transient rise in troponin include an imbalance of oxygen supply and demand secondary to sudden hypertension and tachycardia, shortening of diastole with sub-endocardial ischaemia associated with tachycardia, small vessel disease of the heart, and myocardial ischaemia secondary to coronary spasm. None of these conditions would be excluded by a normal CT coronary angiogram.

CPK is an enzyme found mainly in the heart, brain, and skeletal muscle. In the blood, CPK may be an indication of damage to CPK-rich tissue, such as in rhabdomyolysis, myocardial infarction, myositis, and myocarditis. In our patient, CPK was not elevated despite the raised troponin I level, indicating the absence of significant myocyte damage.

There are currently no data from studies evaluating the therapies aimed at reducing risk in patients with non-ischaemic troponin elevation. In most cases, aggressive antithrombotic and antiplatelet treatments are not indicated. Aspirin, unless it is contraindicated, may be given. Whether to check the troponin level in this patient was the clinical judgment of the admitting doctor. Atypical presentation of an older patient is common and well reported, often requiring comprehensive geriatric assessment supplemented by appropriate investigations to delineate the underlying problems.[20,21] Although the patient did not complain of any chest discomfort on admission, if the admitting doctor was concerned that a cardiac event might have occurred leading to the 'trip and fall', or that a heart event might have occurred after the fall, checking the troponin I level would be helpful to exclude such coronary event. Progressive troponin I rise in this patient with cardiovascular risk factors (age and hyperlipidaemia) mandated further tests such as CT coronary angiogram to exclude significant coronary artery disease. Although CT coronary angiogram has a small risk of contrast nephropathy and allergy, it is less invasive than conventional cardiac catheterisation.

CONCLUSION

Troponin may be elevated in the absence of acute myocardial infarction. Many conditions are associated with non-ischaemic troponin elevation, and their mechanisms are heterogeneous. There are currently no data from studies evaluating the therapies aimed at reducing risk in patients with non-ischaemic troponin elevation. Aspirin, unless contraindicated, can be given, as it appears to be relatively safe in most clinical circumstances.

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