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A woman in her 70s with chest pain and elevated troponin T levels

EDUCATIONAL CASE REPORT

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Background

Troponin T is a biomarker of myocardial injury. In patients presenting with chest pain and elevated troponin T, acute coronary syndrome (ACS) is often suspected. However, alternative explanations must be considered.

Case presentation

A woman in her seventies was admitted to hospital with retrosternal chest pain and an initial elevated troponin T of 108 ng/L. Electrocardiogram and coronary angiography were normal. Further elevation to 263 ng/L raised suspicion of myopericarditis, even though cardiac MRI and echocardiography revealed no definitive pathology. Despite clinical improvement, troponin T remained persistently elevated at around 270 ng/L. Upon readmission 14 months later, troponin T levels were still elevated (89 ng/L) without corresponding symptoms. High-sensitivity troponin I was normal. Further biochemical testing revealed troponin T bound to IgG, consistent with macrotroponin and explaining the false elevation.

Interpretation

This case highlights the importance of considering biochemical factors when troponin results are inconsistent with clinical findings. Although macrotroponin was identified, the underlying cause of the initial elevated troponin T remains uncertain. The absence of cardiac findings suggests a non-cardiac origin, and the moderate, persistent elevation in creatine kinase raises the possibility of an underlying myopathic process. Diagnostic uncertainty can arise when biomarkers do not match the clinical picture.

Troponin T is a biomarker of myocardial injury. Patients presenting with chest pain and elevated troponin T in peripheral blood are normally investigated for acute

coronary heart disease. This case report illustrates that alternative explanations should also be considered when troponin T levels are elevated.

The patient was an active woman in her seventies who was still working part time. She had previously been found to have a mildly dilated aortic root, had hypertension, and was receiving treatment with an ACE inhibitor and a statin. Her general practitioner (GP) had referred her to the medical department following a two-day history of pressing retrosternal chest pain without radiation. The pain occurred at rest in the morning, increased during the day, and was resolved by the evening. It was exacerbated by walking uphill, and she stopped twice on the way to her GP due to worsening symptoms. At the time of admission, she still had chest pain. She was moderately hypertensive, with a blood pressure of 170/95 mmHg. The electrocardiogram (ECG) was normal, with no significant ST-segment or T-wave changes and no left bundle branch block. Initial blood tests showed an elevated troponin T level of 108 ng/L (reference range ≤ 14) and normal renal function (Table 1).

Chest pain and elevated troponin T levels raised suspicion of myocardial injury and non-ST-elevation myocardial infarction (NSTEMI). In line with the European Society of Cardiology's guidelines, and as recommended by the Norwegian Society of Cardiology, she was given a loading dose of 300 mg acetylsalicylic acid and referred for coronary angiography within 24 hours of admission.

Coronary angiography showed no significant stenoses, only wall irregularities in the left anterior descending artery and the right coronary artery.

The following day, troponin T remained elevated at 118 ng/L and the patient continued to experience chest discomfort. Aortic dissection was suspected as a possible cause, and a CT scan of the entire aorta was performed the same day.

CT of the aorta showed dilation of the aortic root measuring 48 mm, unchanged from three years earlier. Following a further rise in troponin T to 263 ng/L on the third day after admission, echocardiography was performed, demonstrating normal-sized, well-contracting left and right ventricles with no sign of myocardial infarction. Known dilatation of the ascending aorta, moderate aortic regurgitation and a minimal pericardial effusion were noted as incidental findings. Troponin T remained stable but elevated over the subsequent days (269–263–279 ng/L).

A persistent rise in troponin T is not typical of acute coronary heart disease, which is usually characterised by an initial rise followed by a gradual decline. The most likely cause was therefore considered to be myocarditis. The patient was consequently referred for cardiac MRI, which was performed five days after admission and seven days after symptom onset.

Cardiac MRI showed no sign of myocarditis. NT-proBNP was within normal limits. Based on the clinical presentation and the finding of minimal pericardial effusion on bedside echocardiography, myopericarditis was nevertheless considered the most likely diagnosis. The patient was discharged in good general condition after six days. Secondary prevention consisted of

dual antiplatelet therapy with acetylsalicylic acid 75 mg and clopidogrel 75 mg daily for three months, as an NSTEMI due to a spontaneously resolved thrombus could not be ruled out entirely. Colchicine 0.5 mg once daily for three months was also administered for myopericarditis.
At three-month follow-up, cardiac MRI showed no evidence of myocarditis, and myocardial scintigraphy showed no sign of myocardial ischaemia. Troponin T was 131 ng/L, creatine kinase (CK) 519 U/L (35–210), and CK-MB 9 µg/L (≤ 5). At follow-up nine months later, ECG showed sinus rhythm with frequent ventricular extrasystoles. Troponin T had further decreased to 109 ng/L, but due to persistently elevated levels, repeat CK testing was requested. This showed CK 222 U/L, CK-MB 5 µg/L and myoglobin 74 µg/L (≤ 65). As there was no clear explanation for the mildly elevated CK-MB and myoglobin, serum was sent for high-sensitivity troponin I analysis at another hospital trust.
The patient was admitted as an emergency two months later after a new episode of discomfort and pain in the left side of the chest. She reported that over the preceding year she had experienced regular episodes of discomfort/mild chest pain, usually in the morning and when stressed. The pain was reproducible on palpation along the parasternal area and chest wall. On admission, troponin T was 89 ng/L and myoglobin 70 µg/L. ECG showed sinus rhythm with frequent ventricular extrasystoles. Repeat echocardiography showed unchanged minimal pericardial effusion, and troponin T on the first day showed no dynamic change (89–94–86 ng/L).
Recurrence of myopericarditis was suspected. However, a review of the patient's medical record during the day-one ward round revealed the result of a high-sensitivity troponin I analysis from two months earlier, which was negative (< 4 ng/L), as well as a note to the GP questioning whether troponin T might have been falsely elevated. The current presentation was therefore interpreted as non-cardiac chest pain and possible costochondritis, an inflammation at the junction between cartilage and bone where the ribs meet the sternum.

On readmission, troponin T levels remained elevated. The chest pain was not related to physical activity or associated symptoms and was not consistent with classic symptoms of cardiac disease. As further investigations did not confirm myopericarditis, a biochemical explanation for the persistently elevated troponin T levels was considered. Through a local clinical biochemist, contact was established with the Department of Medical Biochemistry at Radiumhospitalet, which has specialist expertise in this type of problem. Here, serum proteins were separated into different fractions based on molecular size. This method requires experience, resources and special equipment but is particularly suitable for investigating suspected macro-troponin. Almost all troponin T in the sample was recovered in fractions containing proteins of 150–200 kDa, which is typical of complexes of IgG and troponin T. Given the marked differences in molecular weight between free troponin T (≈ 30 kDa), IgG–TnT (≈ 180 kDa) and IgM–TnT (≈ 1000 kDa), the persistent troponin T elevation was attributed to macro-troponin.

The patient was informed, and follow-up of the ascending aorta at the cardiology outpatient clinic was planned. She was referred for MRI of the thigh to exclude myositis. This examination showed no evidence of myositis.

Discussion

Troponin T and troponin I are not exclusively cardiac-specific proteins, but are present in all striated muscle (albeit with different amino acid structures in cardiac and skeletal muscle). In laboratory assays for troponin T and troponin I, antibodies against cardiac troponins are used, and these are regarded as cardiac-specific tests; however, in rare pathological conditions, skeletal muscle may express cardiac troponin isoforms. When there is a dynamic change in troponin levels, myocardial injury is normally suspected (1).

After the initial admission, myopericarditis was considered the most likely cause of the patient's chest pain and elevated troponin T. The condition is characterised by an inflammatory process primarily involving the pericardium, with only mild involvement of the myocardium, presenting with troponin release and/or ST-segment elevation on ECG (2). This is in contrast to perimyocarditis, in which myocardial involvement is more pronounced, resulting in reduced left ventricular function. The gold standard for diagnosis is endomyocardial biopsy, but this is only recommended when there are signs of heart failure. Cardiac MRI is used to detect myocardial inflammation, with a sensitivity of 73–88 % (3), as well as to exclude other diagnoses, while coronary angiography is performed to rule out coronary heart disease. Diagnostic criteria for myopericarditis include elevated troponin and pericarditis, together with at least two of the following: typical chest pain, pericardial friction rub, pericardial effusion and ECG changes. On review of the patient's symptoms and findings, the diagnosis appears to have been based solely on the elevated troponin T level, without other criteria being met.

Macro-troponin is an immune complex consisting of cardiac troponin (troponin T and/or troponin I) bound to endogenous anti-troponin antibodies. The complex will often, but not always, result in elevated measured levels of troponin T and/or troponin I (4–6). The prevalence varies between different populations and assay methods. For example, a large-scale Danish study of a healthy reference population showed a substantially higher proportion of macro-troponin I than macro-troponin T among participants with the highest values (7). Macro-troponin can have a molecular weight of ≥ 150 kDa and is too large to be filtered by the renal glomeruli. Due to reduced elimination, these immune complexes remain in the circulation and can lead to elevated troponin concentrations for a potentially prolonged period (weeks to months) in the absence of acute ongoing myocardial injury (8). The half-life of troponin T will increase from around two hours to weeks, corresponding to the half-life of IgG, thereby resulting in relatively stable and persistently elevated levels. This is consistent with the slow decline in troponin T from day 6 to 14 months in our patient.

Elevated troponin T is a marker of myocardial injury, and numerous investigations were performed, despite a low clinical suspicion of coronary heart disease. Both the elevated troponin T level on admission and the almost threefold increase by day 3 raised suspicion of unresolved myocardial injury or inflammation. The patient also showed no signs of heart failure, including Takotsubo syndrome (9), or arrhythmias that could explain the elevated troponin T. Troponin T levels can be elevated in severe renal failure (10), certain myopathies (11), severe rhabdomyolysis, severe sepsis and following strenuous physical exertion, none of which were suspected in our patient. We also found no definite cardiac source for the elevated troponin T during the two hospital stays.

A retrospective review of the patient's medical record revealed that the patient had experienced pain on palpation of the chest muscles since 2010. These myalgic chest pains are unlikely to explain the elevated and rising troponin levels at the time of the initial admission. In addition, the patient described progressive muscle weakness. This raised the question of whether skeletal muscle could be the source of a selective troponin T release (11). Inclusion body myositis can present with moderately elevated CK and disproportionate troponin T elevation (12). The patient was referred for MRI of the thigh, which showed moderate fatty infiltration without oedema. This does not support the diagnosis and is more likely related to ageing. We therefore found no definitive muscular source either.

In retrospect, CK, CK-MB and myoglobin could have been measured earlier, rather than only after three and twelve months, respectively. It was also unfortunate that the troponin I result sent to the GP was not identified at the time of the subsequent admission, prior to the ward round on day one. This result could have prevented an unnecessary repeat investigation, although it did not have clinical consequences in this case.

Clinicians must be alert to discrepancies between clinical findings and biomarkers in order to avoid patients undergoing repeated, costly and potentially hazardous investigations, such as invasive coronary angiography and contrast-enhanced CT scans. The laboratory should be contacted when troponin results do not fit the clinical picture. Appropriate measures may include repeat analysis of the sample, troubleshooting of the analysis instrument, assessment of possible interferences and use of complementary tests such as troponin I, CK, CK-MB, NT-proBNP and myoglobin in cases of elevated troponin T, as well as investigation for macro-troponin. Although macro-troponin was detected, we did not identify the cause of the troponin T release leading to macro-troponin formation in this patient. Nevertheless, the finding meant that no further investigations were undertaken and that the patient was reassured that she did not have significant cardiac disease.

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The article has been peer-reviewed.

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