CASE REPORTS

# Troponin T elevation in lobar lung disease

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Two patients with a rise in cardiac troponin T (cTnT) concentrations during the course of lobar pneumonia, without any evidence of acute coronary syndrome or renal failure, are presented. The increase in cTnT concentration is considered a highly sensitive marker of cardiac injury, although it may also rise in other conditions and as a result of lobar pneumonia. Thus, this effect should be considered when the possibility of acute coronary syndrome in such patients is addressed.

Troponin T is an essential component of the contractile apparatus of striated muscle. During myocardial injury cardiac troponin T (cTnT) is released into the blood flow, thus creating a sensitive marker for such an injury.[1] Troponin T is used in everyday practice as a tool to diagnose myocardial damage. Although considered highly specific for myocardial injury,[1–3] accumulating data suggest its rise in other conditions. Raised concentrations of cTnT have been measured during the course of pulmonary embolism.[4 5] This rise of cTnT was attributed to temporary right ventricular dysfunction. Recently, levels of cTnT have also been shown to rise in various non-cardiac conditions, such as HIV, muscle dystrophies, and dyspepsia.[6–8] These accumulated data necessitate that practising physicians should be alert to other conditions that might cause a rise in troponin T and should be considered in the differential diagnosis. We present two cases of raised cTnT during the course of lobar pneumonia without apparent clear primary cardiac involvement.

## CASE REPORTS

### Case 1

A 73 year old woman presented with shortness of breath and cough of 10 days' duration and fever in the past few days. Her medical history included chronic obstructive lung disease. Examination on admission revealed a tachypnoeic woman with a respiratory rate of 24 breaths/min, blood pressure 140/90 mm Hg, pulse 95 beats/min, and temperature 38.1°C. On auscultation lung fields were clear except for fine crackles at the base of the right lung. Laboratory findings revealed a white blood cell count of $15.0 \times 10^9$/l with 90.3% granulocytes, plasma creatinine and urea were within the normal range, and cTnT was 0.1 ng/ml and rose to 0.15 ng/ml within few hours. The assay for troponin T was performed using Troponin T Stat (Cat No 2017423; Roche). An immunoassay for the in vitro determination of troponin T in human serum and plasma values found up to 0.037 ng/ml in healthy subjects. A cTnT concentration of 0.1 ng/ml is recommended as a clinical threshold. One month after discharge the patient's cTnT concentration was 0.04 and 0.02 ng/ml, creatine phosphokinase levels were normal, the electrocardiogram (ECG) showed no dynamic changes during hospitalisation, and the patient did not complain of chest pain. Chest radiography revealed right lower lobe consolidation.

### Case 2

An 80 year old man presented with weakness, shortness of breath, and purulent productive cough of a few days' duration. Six weeks earlier he had been hospitalised due to left upper lobe pneumonia and *Streptococcus pneumoniae* bacteraemia. He denied chest pain or discomfort. His past medical history was positive for chronic obstructive pulmonary disease related to heavy smoking. He had also had hypertrophic obstructive cardiomyopathy for the past 25 years, had undergone cardiac catheterisation with normal coronaries 10 years before, and had a permanent pacemaker. Physical examination on presentation revealed a temperature of 36°C, blood pressure 140/60 mm Hg, pulse 74 beats/min regular, room air pulse oximetry 85%, and a respiratory rate of 30 breaths/min. Heart examination revealed regular sounds with a 3/6 systolic murmur. No jugular distension was evident. On lung auscultation diffuse rales were heard with more prominent rales over the left upper and right lower fields. Abdominal examination was normal. There was no limb oedema. Laboratory data showed a white blood cell count of $12.8 \times 10^9$/l with 83.1% granulocytes and a blood creatinine concentration of 80 μmol/l. On admission troponin T was 0.05 ng/ml. An ECG revealed a pacemaker rhythm. Chest radiography revealed a new consolidation in the right lower lobe and a persistent consolidation in the left upper lobe; there was also a small left pleural effusion.

The patient was admitted to the medical ward for treatment with intravenous antibiotics. A computed tomogram (without contrast material) of the chest was performed which showed alveolar exudates in the left upper lobe and lingular and right lower lobe. Oronoscopy with lavage and cultures was uninformative. An ECG demonstrated left ventricular hypertrophy, good systolic function with mild mitral regurgitation, and pulmonary hypertension (70 mm Hg) attributed to chronic obstructive lung disease, without right ventricular distension or dysfunction. Repeat cTnT 48 hours after hospitalisation was 0.31 ng/ml, without any complaints of chest pain, ECG changes, or rise in creatine phosphokinase or lactic dehydrogenase. The patient received antibiotics and was discharged after one week of hospitalisation in a stable condition.

## DISCUSSION

We have shown in the present report that levels of cTnT may rise above the conventional cut off of 0.1 ng/ml without evidence of acute cardiac ischaemia or renal dysfunction in the presence of lobar pneumonia. It has been shown in the past that levels of cTnT may rise during pulmonary embolism.[4 5] This rise has been attributed to temporary right ventricular dysfunction or dilatation. We present two patients with raised troponin T and no evident coronary syndrome. We hypothesise that cTnT, being a highly sensitive marker of cardiac injury,[1] may rise to above normal levels as a result of a

**Abbreviations:** cTnT, cardiac troponin T; ECG, electrocardiogram

**Learning point**

Cardiac troponin T (cTnT) concentrations may rise as a result of lobar pneumonia, without evidence of acute cardiac ischaemia.

lobar pulmonary process, even without evident heart dysfunction. There is also evidence that higher baseline cTnT concentrations are found in congestive heart failure patients outside a hospital environment when compared with normal controls, and without evidence of an acute process.[9 10] It may therefore be prudent to allow for such an effect when considering the feasibility of an acute coronary syndrome in such patients.

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