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Troponine Ultra-sensible: Quelles Indications et Comment Interpréter les Résultats en Gériatrie Un cas d'Élévation de la Troponine chez une Octogénaire

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Résumé

La troponine T ultra-sensible (us), marqueur biologique spécifique du cœur, peut être élevée dans des conditions pathologiques autres que le syndrome coronarien aigu. Ces autres causes peuvent ou non être directement liées aux maladies cardiaques. Nous rapportons le cas d'une patiente de 85 ans présentant de multiples événements cardiovasculaires qui présentait une élévation de la troponine T us à 1088 pg/ml, probablement due à de multiples étiologies.

Mots-clés: Gériatrie ; syndrome coronarien aigu, troponine

Ultra-sensitive Troponin: What Indications and How to Interpret the Results in Geriatrics
A Case of Troponin Elevation in an Octogenarian

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Abstract

The ultra-sensitive troponin T (us), a specific biological marker of the heart, may be elevated in pathological conditions other than acute coronary syndrome. These other causes may or may not be directly related to heart disease. We report the case of an 85-year-old female patient with multiple cardiovascular events who presented an elevation of us troponin T to 1088 pg / ml, probably due to multiple etiologies.

Keywords: Geriatrics; acute coronary syndrome, troponin

Introduction

Troponins are the most sensitive and specific biomarkers of myocardial damage(Nallet et al., 2016).

The assay for ultra-sensitive troponins has recently been developed by several companies. It can detect concentrations 10 times smaller than previous techniques(Boukili M, 2012). However, elevations of hypersensitive troponins have been noted in patients without infarction(Chenevier-Gobeaux et al., 2013).

We present a case of troponin elevation in an 85-year-old female patient without chest pain and no ST-segment elevation.

Observation

This is an 85-year-old patient admitted to a comprehensive medicine unit in Saint Vallier sur Rhône (France) in February 2019, for right sciatic pain that occurred one week after falling down.

She has a rich cardiovascular history including high blood pressure (HTN); ischemic heart disease complicated by acute lung edema; a tight proximal interventricular artery stenosis with an active stent and an episode of paroxysmal atrial fibrillation. She also suffers from chronic kidney disease (CKD). She is on furosemide 500 MG: ½ tab in the morning, cordarone 200 mg: ½ tab in the morning, hydrochlorothiazide 25 MG P: ½ tab at noon 3 times a week; spironolactone 25 MG: 1 tab in the morning, RAMIPRIL 2.5 MG: 1 tab in the morning and ½ tab in the evening, Clopidogrel 75 MG: 1 at noon and Aspirin 75MG: at noon.

She complained of a mechanical lumbar pain radiating to the right lower limb not following a specific dermatomal pattern, associated with recent-onset dyspnea at rest.

On physical examination, the patient was slightly confused. Blood pressure (BP) was 90/64 mmHg, pulse 97 bpm; afebrile, the visual analogue scale (VAS) was rated at 4.

The Lasègue sign was positive when the right leg was elevated, with abolition of the patellar and Achilles osteotendinous reflexes on the right, and a positive Babinski reflex at the right side as well. There was no sensory disturbance. Cardiac auscultation found an arrhythmia with no added sounds. The remainder of the physical examination was unremarkable. A spinal x-ray revealed a T11 collapse confirmed by magnetic resonance imaging (MRI).

Biological assessment: Urea: 29.9 mmol/l; Plasma creatinine: 166 µmol/l; glomerular filtration rate (GFR) according to the mdrd method: 27ml/min; NT Pro bnp:12041 ng/l; Troponin T us: 1088 pg/ml (baseline: 124 pg/ml).

An electrocardiogram (ECG) performed showed atrial fibrillation (AF) with right bundle branch block (RBBB).

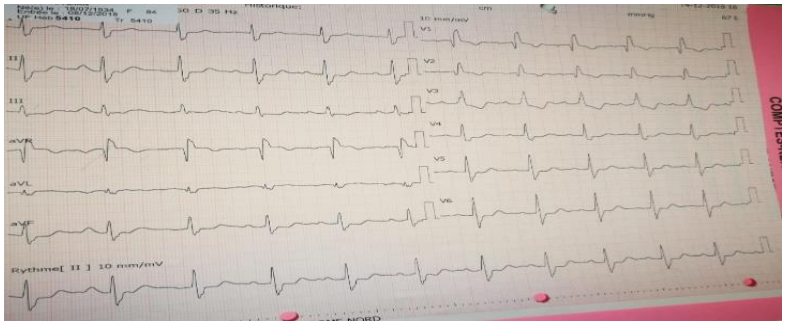

Figure 1. ECG december 2018

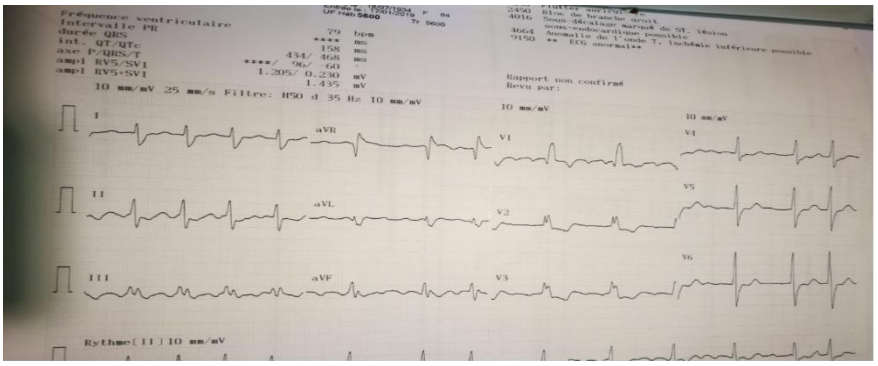

Figure 2. ECG january 2019

Faced with an elevated troponin level in a dyspneic patient without chest pain, a cardiology call was made urgently. The electrocardiogram showed an atrial fibrillation and a right bundle branch block and the transthoracic echocardiography a dilated right ventricle with a RV-RA gradient at 50mmHg; the cardiologist then suspected a pulmonary embolism and therefore recommended a chest CT angiography. However, since renal insufficiency contraindicates CT angiography, a lung scintigraphy was performed which was normal.

The nearest coronary angiography center was subsequently contacted to request a second cardiology opinion who had also concluded that the patient has an atrial fibrillation and a right bundle branch block, excluding the indication of coronary angiography.

On the other hand, Mrs. x presented since her admission a few episodes of drowsiness and sometimes confusion, so a brain CT scan was ordered which showed hypodense cortical subcortical areas in the left temporo-occipital and upper left frontal lobes leading to the conclusion of an ischemic vascular accident secondary to hypotension.

Management consisted of treatment with morphine, a reduction in the doses of RAMIPRIL and the discontinuation of HYDROCHLOROTIAZIDE

associated with an increase in the doses of CORDARONE and the introduction of ANTIVITAMIN K.

The clinical course was characterized by a disappearance of pain, dyspnea, confusion and a normalization of systolic BP to 110mmHg after one week.

Biologically, there was a regression of troponin with a drop to 850pg/ml after 24 hours and then to 177pg/ml.

Discussion

The assay for ultra-sensitive troponins has recently been developed by several companies. It can detect concentrations 10 times smaller than previous techniques. However, the improvement in this sensitivity is accompanied by a decrease in the specificity of identifying ACS (positive predictive value between 50 and 76%)(Boukili M, 2012).

Troponin is requested in cases of typical or sometimes atypical chest pain in the presence of risk factors. However, in geriatrics, the presentation of an ACS is more often atypical than in young people.

In an Indian study comparing the clinical presentation of ACS between a young population under 65 and an older population, it is noted that typical chest pain was the most common symptom in both age groups but was more frequent in young patients than in elderly patients (81.76% versus 50.47%; p <0.05). Atypical chest pain (28% versus 10.75%) or no chest pain (21.49% versus 7.53%) was observed more frequently in the elderly group compared to the younger group (p<0. 05). Dyspnea, palpitations, vertigo and syncope were reported more frequently by the elderly (Bhatia & Naik, 2013). In our patient, dyspnea, history of heart disease and comorbidities raised suspicion of an atypical presentation of ACS, without ST-segment elevation, thus justifying the dosage of troponin.

Indeed, in addition to age, comorbidities also influence the atypical presentation of ACS.

In their study, O manfrini et al (Manfrini et al., 2016) found that the risk of atypical presentation of ACS depends on the number of comorbidities. Thus, the risk of an atypical presentation of an ACS is 1.64 (95% CI: 1.42–1.90) in the event of a single comorbidity; 2.52 (95% CI: 2.05–3.10) for two comorbidities and 4.57 (95% CI: 3.39–6.17) for three or more comorbidities.

The same authors noted that the independent predictive variables (in descending hierarchical order) associated with an atypical presentation of acute coronary syndrome are stroke, renal failure, arteriopathy, chronic obstructive pulmonary disease (COPD), congestive heart failure, diabetes and age.

Thus, we note in our patient at least 4 factors (age, heart failure, renal failure and stroke) predictive of an atypical presentation of an ACS.

After the first concern which is to suspect an atypical presentation of an ACS in geriatrics and whether troponin dosage is necessary or not in this segment of the population, the second concern consists of the interpretation of a high troponin level.

Indeed, the interpretation of an elevated troponin is just as complicated as the detection of an atypical presentation of an ACS in this segment of the population, since several etiologies can be the cause of a troponin elevation without ACS.

In a Parisian study comparing 2 groups of patients aged below and above 70 years with a clearance below and above 60ml/min; it appears that the specificity of the ultra-sensitive troponin T threshold greater than 14 ng/l in the diagnosis of MI is 88% in those under 70 years old against 51% in patients over 70 years old ($p<0.001$). Similarly, the specificity of ultra-sensitive troponin T in the diagnosis of ACS, is 86% in patients with clearance >60 ml/min vs. 54% in patients with clearance <60% ($p<0.001$). This study also leads to similar conclusions for acute coronary syndrome without ST segment elevation(Chenevier-Gobeaux et al., 2013).

Renal failure is frequently associated with elevated troponin. This elevation is the consequence of progressive minor myocardial damage related to coronary artery disease, left ventricular hypertrophy and endothelial dysfunction(Bertinchant & Polge, 2004).

Our patient suffers from chronic kidney disease with a creatinine clearance of 27 ml/min. This renal insufficiency may therefore be one of the causes of troponin elevation in this particular case.

In the literature, several other diseases are found to be responsible for troponin elevation in the absence of coronary lesions. Among these diseases we find, heart failure; chronic obstructive pulmonary disease (COPD); tachycardia; stroke, certain digestive pathologies such as cirrhosis, certain infections, brady arrhythmia, syncope, GIT bleeding, myocarditis; hypertensive crisis, cardiac trauma, electrical cardioversion, pericarditis, infiltrative heart disease, pulmonary embolism and atrial fibrillation (Bardají et al., 2015; Lavoinne & Cauliez, 2004; Pruvot et al., 2006). Hence, in our patient, we note the presence of other probable causes of ultra-sensitive troponin elevation, in addition to the chronic kidney disease, namely stroke, atrial fibrillation and heart failure. The association of these conditions makes it difficult to interpret an isolated increased troponin levels without ST elevation.

Different mechanisms other than coronary syndrome may explain troponin elevation. Such as myocardial depression in sepsis and inflammatory syndrome; an imbalance of supply and demand in atrial

fibrillation, a subendocardial ischemia in left ventricular hypertrophy and an unsuitable response of autonomic nervous system in cerebral hemorrhage and stroke (Pruvot et al., 2006).

The elevation of troponin can vary from very significant up to 50 times in myocarditis or sepsis to a moderate elevation of 5 times in cirrhosis, gastrointestinal bleeding, hypertension, renal failure and respiratory failure(Lavoinne & Cauliez, 2004).

In our patient, the troponin was more than 50 times the normal threshold. This could be explained by the multiplicity of factors leading to an increase in troponin levels.

In this situation, where the probability of a heart attack is most often low or intermediate, two opposing attitudes should be avoided (Nallet et al., 2016):

- Adopting a pure cardiologic attitude leading to inappropriate hospitalizations in CICU/invasive explorations/antithrombotic treatments which can be useless and dangerous.
- Ignoring the result, since an elevated troponin has an important prognostic value and some ACS have a very atypical presentation.

In our context, cardiological opinions have ruled out an ACS. The management of the various comorbidities allowed a good clinical and paraclinical evolution with regression of troponin and a rapid return to the baseline.

Conclusion

Elevated troponin levels are common in the elderly. The imputation of this troponin elevation to an acute coronary syndrome is not always easy in geriatrics where atypical presentations are frequent as well as other causes that may be at the origin of a troponin elevation without ACS. A good analysis of the clinical situation and the benefit/risk evaluation is required for the best course of action.

Conflict of interest: Authors declaring that they have no conflict of interest

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