ESJ Natural/Life/Medical Sciences

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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Doi:10.19044/esj.2023.v19n12p131

Submitted: 23 December 2022
Accepted: 13 April 2023
Published: 30 April 2023

Cite As:
Seydou Moussa Y., Daou M., Andia A., Al-Azhari A., Brah S., Hamadou A. & Adehossi E. (2023). *Troponine Ultra-sensible: Quelles Indications et Comment Interpréter les Résultats en Gériatrie Un Cas d'Élévation de la Troponine chez une Octogénaire*European Scientific Journal, ESJ, 19 (12), 131. https://doi.org/10.19044/esj.2023.v19n12p131

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.

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

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, can be elevated due to causes 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 was recently developed by multiple companies. It can detect concentrations even 10 times smaller than previous dosing techniques(Boukili M, 2012). However, elevated ultra-sensitive troponins were detected in patients without infarction(Chenevier-Gobeaux et al., 2013).

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

We are going to present a case of elevated levels of troponins in an 85 years old female patient without chest pain or ST-segment elevation.

The case

The patient is an 85 years old female. She was admitted to a general medicine department in Saint Vallier sur Rhône (France) on February 2019, for right sciatica pain which started one week after she fell down.

She had a rich history of heart disease, including hypertension; ischemic heart disease which was complicated by acute lung edema; a tight proximal interventricular artery stenosis with an active stent, and an episode of paroxysmal atrial fibrillation. She suffered from chronic kidney disease (CKD) as well.

Her drug history included: furosemide 500 mg ½ tab in the morning, cordarone 200 mg: ½ tab in the morning, hydrochlorothiazide 25 mg: 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 75 mg at noon.

She complained of mechanical lumbar pain radiating to the right lower limb without following a specific dermatomal pattern, and a recent-onset dyspnea.

During the physical examination, the patient was slightly confused. The blood pressure was at 90/64 mmHg, the heart rate was at 97 bpm; afebrile, and the visual analog scale (VAS) was at 4 points.

The Lasègue test was positive when the right leg was elevated, both knee and Achilles deep tendon reflexes were diminished on the right side and a positive Babinski sign was detected on the right side as well. The sensory exam was normal. Arrhythmia was detected during cardiac examination but there was no murmur. The remainder of the physical examination was unremarkable.

A spinal x-ray had shown a collapsed T11 which was confirmed by magnetic resonance imaging (MRI).

Blood tests: urea: 29.9 mmol/l, serum creatinine: 166 µmol/l, glomerular filtration rate (GFR) using MDRD equation: 27ml/min, NT Pro bnp:12041 ng/l, ultra-sensitive troponin T: 1088 pg/ml (baseline: 124 pg/ml). An electrocardiogram (ECG) showed atrial fibrillation (AF) with a right bundle branch block (RBBB).

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

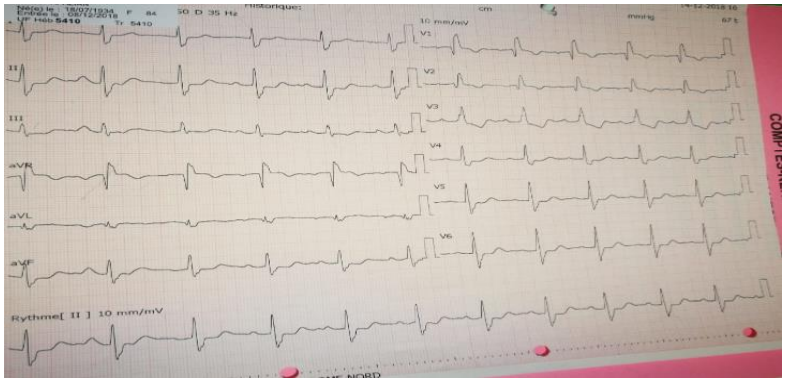

Figure 1. ECG December 2018

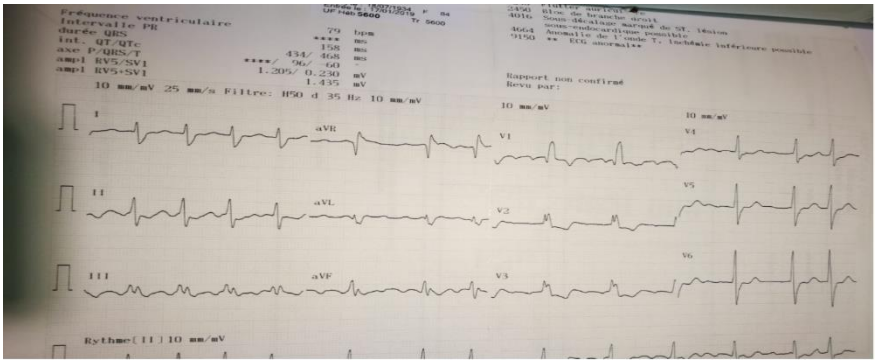

Figure 2. ECG january 2019

Since troponin levels were elevated and the patient was dyspneic, despite the absence of chest pain, a cardiologist's opinion was sought urgently. After an electrocardiogram and a transthoracic cardiac echography, the cardiologist had concluded that there was an atrial fibrillation, a right bundle branch block, and a dilated right ventricle with an RV-RA gradient at 50 mmHg, he also suspected a pulmonary embolism and recommended a chest CT angiography. However, the patient had a CKD which contraindicated the CT angiography, so lung scintigraphy was performed instead and it was normal.

The nearest coronagraphy center was contacted for a second opinion. The second cardiologist had also concluded that other than atrial fibrillation and a right bundle branch block there was nothing else, hence excluding any indication for coronagraphy.

Other than that, Mrs. X had presented, ever since her admission, some episodes of hypersomnia and sometimes even confusion, therefore a brain CT scan was performed which showed some cortical and subcortical hypodense areas in the left temporo-occipital and upper left frontal lobes. So, we concluded that it was an ischemic stroke secondary to hypotension.

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

The patient was managed with morphine and an antivitamin K, and we stopped the hydrochlorothiazide, decreased the dose of ramipril, and increased the dose of cordarone.

During follow-up the patient was no longer dyspneic nor confused, the pain had disappeared and the blood pressure had returned to normal at 110 mmHg one week after.

The troponin levels decreased to 850 pg/ml 24 hours later and then to 177 pg/ml.

Discussion

The assay for ultra-sensitive troponins was recently developed by multiple companies. It can detect concentrations even 10 times smaller than the previous dosing techniques. Nevertheless, despite the better sensitivity, there is a decrease in the specificity for Acute coronary syndrome (ACS) (predictive positive value between 50 and 76%)(Boukili M, 2012).

Troponins are ordered every time a patient has typical or atypical chest pain if he has other cardiovascular risk factors. Still, in geriatrics, ACS may have a more atypical presentation than the typical one seen in younger patients. In an Indian study comparing the clinical presentation of ACS between a population of patients younger than 65 and an older population, it was noted that typical chest pain was the most common symptom in both groups but was more common in younger patients than in the elderly (81.76% versus 50.47%; $p < 0.05$). Atypical chest pain (28% versus 10.75%) or no chest pain at all (21.49% versus 7.53%) were more frequently observed in the elderly group compared to the younger one ($p<0.05$). Dyspnea, palpitations, dizziness, and syncope were reported more by the older group (Bhatia & Naik, 2013).

In our case, dyspnea, history of heart disease, and other comorbidities made us suspect a non-ST-segment elevation ACS with an atypical presentation, justifying dosing the troponins.

In addition to old age, other comorbidities can lead to an also atypical ACS.

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

The same authors noted that the independent predictor variables (by descending order) associated with an atypical presentation of acute coronary syndrome are stroke, chronic kidney disease, arteriopathies, chronic obstructive pulmonary disease (COPD), congestive heart failure, diabetes, and old age.

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

Thus, we see that our patient had at least 4 predictive factors (old age, heart failure, chronic kidney disease, and stroke) for an atypical presentation of an ACS.

After the primary concern, which is suspecting an atypical ACS in a geriatric patient and whether or not to order a troponin assay, the secondary concern consists of how to interpret an elevated troponin level in this particular population of patients. Indeed, the interpretation of an elevated troponin level in such a case is as complicated as detecting an atypical ACS, since many other etiologies can cause its elevation even without an ACS.

A Parisian study compared two groups of patients ages younger and older than 70 years with a glomerular filtration rate lower and higher than 60ml/min, and they noticed that the specificity of an ultra-sensitive troponin T threshold > 14 ng/l for myocardial infarction diagnosis is 88% in the younger group compared to 51% in the group older than 70 years old ($p<0.001$). Also, the specificity of ultra-sensitive troponin T for myocardial infarction diagnosis was 86% in patients with GFR > 60 ml/min versus 54% in patients with GFR < 60% ($p<0.001$). Therefore, this study had the same conclusions concerning acute coronary syndrome without ST elevation(Chenevier-Gobeaux et al., 2013).

Chronic kidney disease is frequently associated with elevated troponins. This elevation is a consequence of minor myocardial damage due to coronary artery disease, left ventricular hypertrophy, and endothelial dysfunction(Bertinchant & Polge, 2004).

Our patient had chronic kidney disease with a GFR of 27 ml/min, so this can be a contributing factor to the elevated troponins seen in her case.

In the literature, we find that many other diseases can lead to troponin elevation in the absence of coronary lesions. These diseases include heart failure; chronic obstructive pulmonary disease (COPD); tachycardia, stroke, other digestive diseases such as liver cirrhosis, some infections, bradyarrhythmia, syncope, gastrointestinal bleeding, myocarditis, hypertensive crisis, cardiac trauma, electrical cardioversion, pericarditis, infiltrative cardiomyopathies, pulmonary embolism, and atrial fibrillation (Bardají et al., 2015; Lavoinne & Cauliez, 2004; Pruvot et al., 2006). Thus, we notice that our patient had multiple causes, other than chronic kidney disease, that could have been responsible for elevated ultra-sensitive troponin levels which were: stroke, atrial fibrillation, and heart failure. Having all these diseases at the same time makes the interpretation of isolated increased troponin levels without ST elevation difficult.

Different mechanisms may lead to troponin elevation without the coronary syndrome. It could be due to myocardial depression in sepsis and inflammation, an inadaptation of supply and demand in atrial fibrillation, subendocardial ischemia in left ventricular hypertrophy and an inadaptation of

 ISSN: 1857-7881 (Print) e - ISSN 1857-7431

the autonomic nervous system in cerebral hemorrhage and stroke (Pruvot et al., 2006).

Troponin elevation can vary depending on the cause. It can be 50 times more elevated than the normal threshold in the case of myocarditis or sepsis and only moderately elevated, no more than 5 times the normal value, in liver cirrhosis, gastrointestinal bleeding, hypertension, chronic kidney disease, and respiratory failure(Lavoinne & Cauliez, 2004).

In our case, troponin levels were elevated 50 times more than the normal value, which can be explained by the presence of multiple factors contributing to this elevation.

In this kind of situation, where the probability of a myocardial infarction is mostly weak or intermediate, we should avoid two opposite attitudes (Nallet et al., 2016):

- Adopting a pure cardiology vision, leading to hospitalizations in departments inappropriate for the patient's needs such as CICU, to invasive investigations and antithrombotic treatments which can be, not just useless, but also dangerous.
- Neglecting the results, since a troponin elevation could reveal an atypical ACS and has a prognostic significance.

In our case, two cardiologists' opinions had eliminated an ACS. The management of different comorbidities led to a good clinical result as well as a decrease in troponin levels until it returned to the baseline.

Conclusion

Troponin elevation is common in elderly patients. Linking this elevation directly to the acute coronary syndrome isn't as simple in geriatric patients where the atypical presentation of ACS is as frequent as other non-ACS-related causes of troponin elevation. An adequate analysis of the clinical situation as well as the benefit/risk ratio is of the utmost importance for better management.

Conflict of interest: authors declare that they have no conflict of interest

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