Usefulness of Multimodality Imaging for Detection of Myocardial Infarction in Patients with Advanced Kidney Failure: Case Report

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Introduction

The third universal definition of acute myocardial infarction (AMI) is based on the elevation of troponin in association with ischemic symptoms, electrocardiographic changes and imaging findings.[1] In patients with chest pain, the diagnosis of AMI is performed by dosing serum markers of myocardial necrosis, particularly troponins, by means of changes in 12 - lead electrocardiogram (ECG) or by identifying changes in the contractile dynamics of the left ventricle to the transthoracic echocardiogram.[1]

In some cases, confounding factors may hamper the diagnosis, such as: (a) presence of previous changes in the baseline ECG, especially LBBB; (b) elevations of myocardial necrosis markers (MNM) resulting from situations other than AMI and; (c) old changes in contractility detected by transthoracic ECG.[2]

Serum cardiac troponin (Tn) is the most specific and most used MNM for the diagnosis of AMI.[1] Nevertheless, in some situations troponin elevation may not be due to an AMI, as in cases of acute pulmonary embolism, acute pericarditis, severe heart failure, myocarditis, and sepsis and kidney failure.[1] Patients with kidney failure have high probability of concurrent cardiovascular disease.[3] Furthermore, the cross-reacting proteins interfering with skeletal muscle, analytical imprecision and interactions with the dialysis membrane may cause elevation of troponin in 7% to 17% of patients with kidney failure.[3] When in doubt about diagnosis of AMI, ^{99m}Technetium pyrophosphate myocardial scintigraphy (^{99m}Tc-PYP) stands out as a noninvasive method capable of identifying areas of myocardial necrosis, thus helping in the diagnosis of AMI.[4]

^{99m}Tc-labeled phosphonate agents undergo chemical absorption with calcium. A large influx of calcium occurs during the evolutionary process of AMI. The calcium flows into the intracellular space and myocardial concentration of ^{99m}Tc-PYP follows such increase, with a maximum peak uptake about 48 to 72 hours after the acute event.[5]

The ^{99m}Tc-PYP is able to locate hydroxyapatite crystal structures found within the mitochondria of irreversibly damaged myocardial cells.[5] A study conducted in 52 patients undergoing ^{99m}Tc-PYP imaging, which underwent necropsy after death, showed sensitivity of 89% and specificity of 100% for ^{99m}Tc-PYP. The positive predictive value of the technique was 100%, and the negative predictive value was 72%.[6]

Keywords

Myocardial Infarction/scintigraphy; Diagnostic Imaging; Chronic Kidney Failure; Diphosphates.

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Case Report

Male patient, 70 years, with fever complaint and dyspnea on mild exertion for three days at first. He also mentioned intense epigastric pain during such period. He had a history of systemic arterial hypertension, chronic kidney failure (CKF) under conservative treatment, dyslipidemia, history of ischemic stroke, and chronic obstructive arterial disease (COAD) treated with percutaneous transluminal coronary angioplasty. His ECG at hospital admission showed third-degree left branch block, and his transthoracic echocardiography showed dyskinesia of the left ventricular apex, akinesia of 1/3 apical septum, and mild to moderate global systolic dysfunction of the left ventricle. His computed tomography (CT) of the chest showed perihilar pulmonary infiltrates on the right side, and bilateral pleural effusion. The patient was admitted and the treatment protocol for sepsis started. As he complained of epigastric pain and had gone from COAD, his troponin I levels were measured at his admission, that was equal to 11.9 ng/mL (normal value less than or equal to 0.05 ng/mL).[7]

A ^{99m}Tc-PYP myocardial scintigraphy was performed to confirm the diagnosis of AMI. Still chest images taken 4 hours following the administration of intravenous ^{99m}Tc-PYP showed anomalous accumulation of the radiotracer in the basal segment of the left ventricle lateral wall (Figures 1 and 2) and the scintigraphic tomographic images (or SPECT, single photon emission tomography) associated with the CT scan for attenuation correction and anatomic correlation confirmed the location of the necrotic area in that region (Figure 3). A myocardial perfusion study with ^{99m}Tc-sestamibi was also carried out, revealing uptake of the radiotracer in the apical and anterolateral left ventricular segments.

Discussion

Martins et al. described the importance of suspicion for other medical conditions in addition to AMI that can raise cardiac troponin levels,[8] namely: the evaluation of

chest pain with onset between 24 - 72 hours, LBBB, right ventricular infarction.[8]

The patient had CKF, third-degree LBBB at baseline ECG, pulmonary sepsis on admission, atypical chest pain with 72 - hours interval, an increase of Tn I and myocardial dysfunction new to the transthoracic echocardiogram, symptoms that provide accurate indications for carrying out the research on myocardial necrosis with ^{99m}Tc-PYP.[9]

When ischemia occurs, no change takes place in the phospholipid membrane, even in the absence of necrosis, leading to increased permeability of the membrane to calcium, related to the increased uptake of PYP.[5] The ^{99m}Tc-MIBI scintigraphy

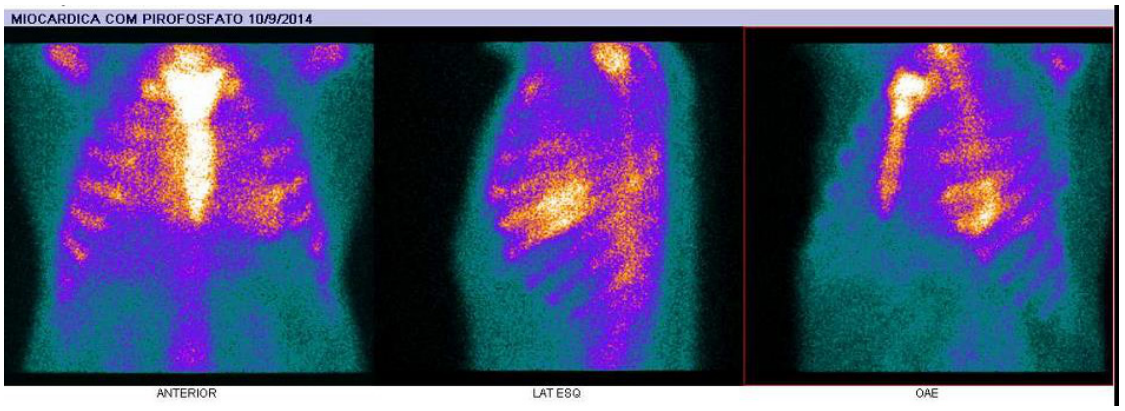

Figure 1 – *^{99m}Tc-pyrophosphate myocardial scintigraphy: planar images of the chest showing high uptake in cardiac basal lateral segment, suggestive of acute myocardial infarction in healing stage.*

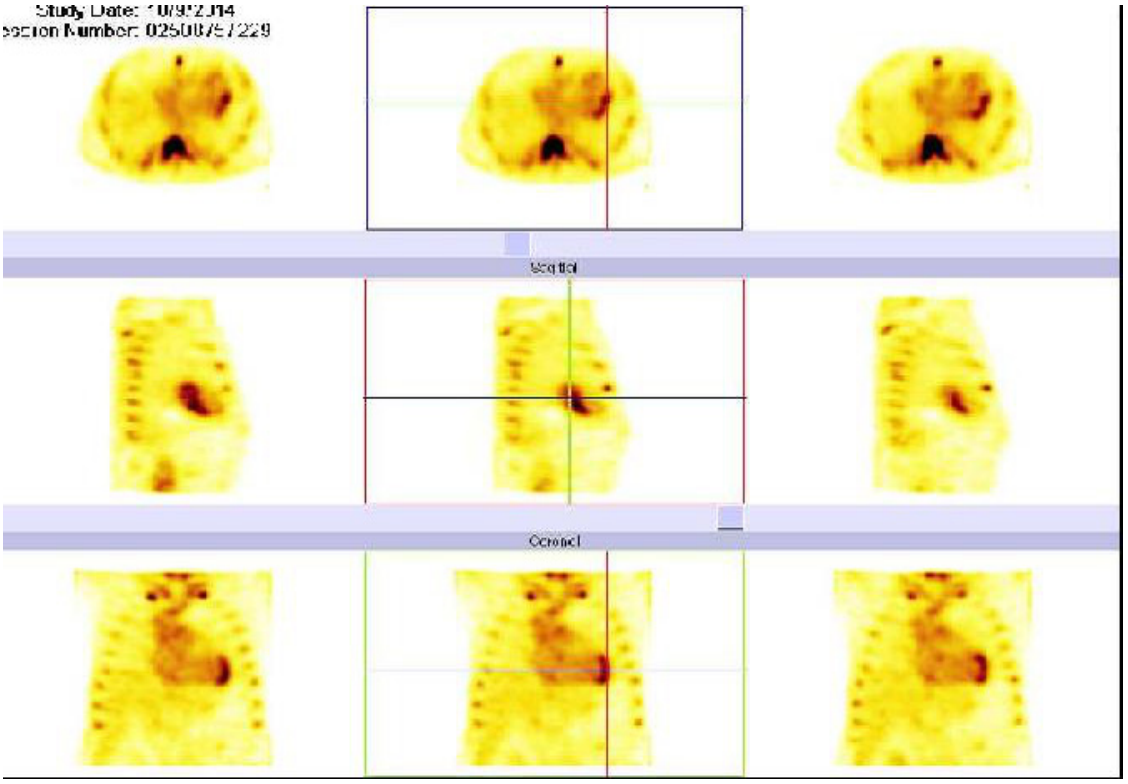

Figure 2 – *4-hours SPECT of chest showing high uptake of 99mTc-pyrophosphate in cardiac basal lateral segment, suggestive of acute myocardial infarction in healing stage.*

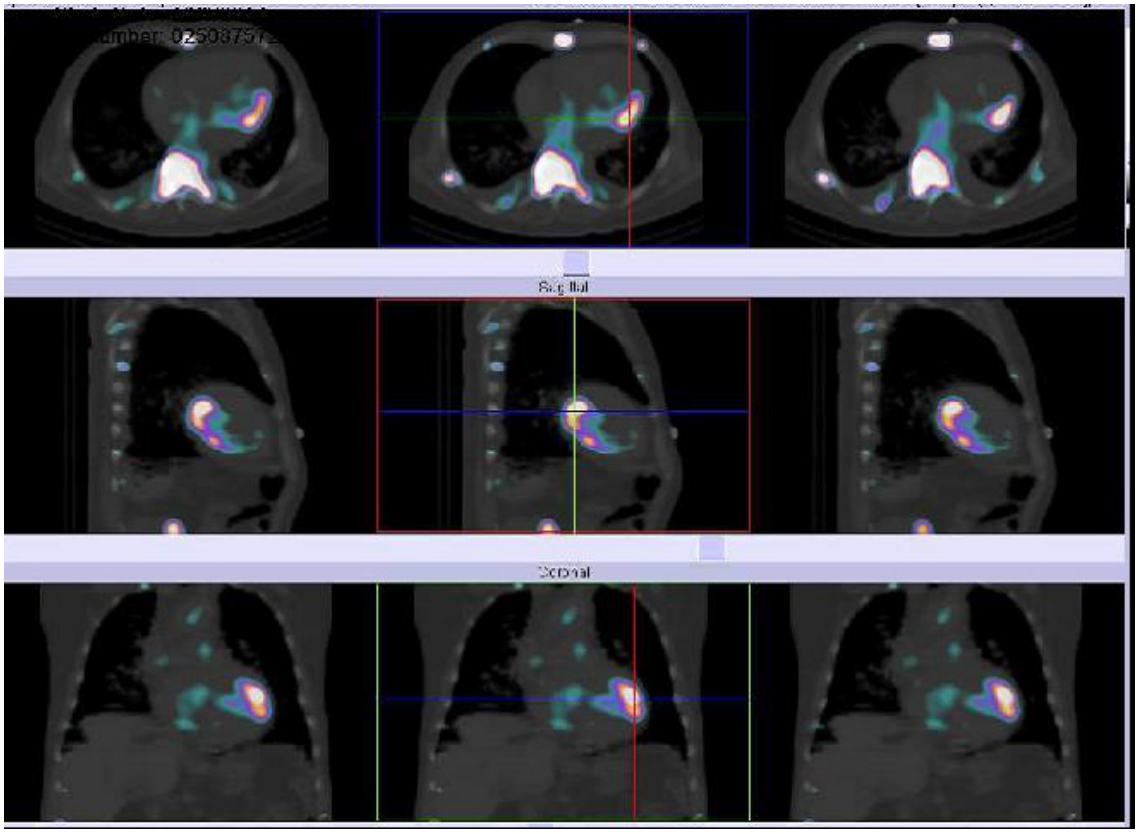

Figure 3 – *4 - hours SPECT/CT of chest showing hyperfixation of ^{99m}Tc-pyrophosphate radiotracer in cardiac basal lateral segment, suggestive of acute myocardial infarction in healing stage.*

portrays myocardial perfusion by high-affinity attachment to the mitochondria of muscle cells, and is one of the markers of choice for the detection of perfusion defects, showing ischemic tissue injury.[2]

Particularly in this case, myocardial perfusion imaging with MIBI scintigraphy and myocardial ^{99m}Tc-PYP scintigraphy showed a high uptake of the ^{99m}Tc-PYP radiotracer in the basal lateral segment, and a compatible area of low uptake for the ^{99m}Tc-MIBI radiotracer in rest images, typical of functional impairment. The comparison between images from different radiotracers strongly suggests recent transmural myocardial infarction.

Cardiovascular magnetic resonance (CMR) scan is a good diagnostic test to view infarction areas. This method uses a specific sequence performed 10 to 20 minutes after intravenous administration of gadolinium-based contrast – a paramagnetic metal that increases the signal intensity of structures in images.[10] However, in patients with CKF, the gadolinium-based contrast constitutes a relative contraindication for the examination in order to prevent exposing the patient to the potential risk of developing systemic nephrogenic fibrosis.[10]

In case of comorbidities capable of increasing serum troponin I, additional scintigraphic examination was critical to define the diagnosis and therapy.

Authors' contributions

Research creation and design: Veras MF, Azevedo JC, Mesquita CT; data collection: Veras MF, Mesquita CT; data analysis and interpretation: Veras MF, Azevedo JC, Mesquita CT; manuscript drafting: Veras MF, Azevedo JC, Mesquita CT; critical revision of the manuscript for important intellectual content: Veras MF, Mesquita CT; exam recommendation and monitoring of clinical evolution, as well as definition of the patient's therapeutic approach: Gamarski M, Mesquita ET; Literature review: Mesquita ET, Alves JG.

Potential Conflicts of Interest

No relevant potential conflicts of interest.

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Academic Association

This study is not associated with any graduate program.

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