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Case Presentation

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Myocardial Ischemia Demonstrated by 13N-Ammonia PET: Utility and Prognosis

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Abstract

Myocardial Perfusion Imaging (MPI) is considered an imaging study with high diagnostic and prognostic value in the approach of the patient with chest pain. Currently, Positron Emission Tomography (PET) is the modality of choice since, in addition to having increased availability and accessibility in the last decade, it has high sensitivity and specificity as well as better image resolution and low radiation exposure. The present case represents the typical clinical and imaging findings in patients with an ischemic process and this report highlights the usefulness of 13N-Ammonia MPI-PET in the early diagnosis, risk assessment and prognosis of patients with Coronary Artery Disease (CAD).

Keywords: Acute coronary syndromes; Myocardial perfusion imaging; Positron Emission Tomography; 13N-Ammonia; Chest pain

Introduction

Acute Coronary Syndromes (ACS), involving sudden heart blood flow reduction like ST-Segment Elevation Myocardial Infarction (STEMI) and non-ST-segment elevation ACS (NSTE-ACS),

are often associated with age, smoking, diabetes, high lipid levels, blood pressure, and body mass index. Primarily affecting older individuals, ACS typically presents as chest pain. Post-ACS, recurrent ischemic events are frequent, underscoring the importance of lifestyle changes such as adopting a mediterranean diet, exercising, and quitting smoking to enhance outcomes [1]. MPI, notably with PET/CT using 13N-ammonia and Fludeoxyglucose (18F), offers a noninvasive means for early CAD diagnosis, risk assessment, and prognostic evaluation, benefiting from its high speed, clarity, diagnostic precision, quantification capability, and low radiation exposure [2]. This review aims to showcase PET/CT's utility with 13N-ammonia in ACS patient prognosis and monitoring.

Case Presentation

A 75-year-old male presents with a history of oppressive precordial pain lasting over 30 minutes, occurring at rest without relieving factors. The patient has no signs of dyspnea or low output. Notably, he has a 40-year history of smoking quitting 23 years ago, in addition to a history of ACS, having experienced a NSTEMI approximately one month ago without reperfusion therapy. The biochemical profile shows total cholesterol of 254 mg/dL, LDL-Cholesterol of 180 mg/dL and HDL-Cholesterol of 56 mg/dL; resulting on a high atherogenic risk by a 6.5 Castelli Risk Index [3], suggesting obstructive pathology.

Resting Electrocardiogram (ECG) reveals inactive area in inferior wall **(Figure 1)**. MPI 13N-Ammonia-PET with pharmacological stress using adenosine was performed revealing non-transmural infarction of inferior wall with dorsal extension, as well as moderate ischemia in the territory of the Right Coronary Artery (RCA) **(Figure 2)**. Inferior and septal hypokinesia was demonstrated **(Figure 3 and 4)** in addition to decreased Myocardial Reserve Flow (MRF) in the RCA territory **(Figure 5)**. Left Ventricular (LV) systolic function is preserved with Left Ventricular Ejection Fraction (LVEF) of 53%.

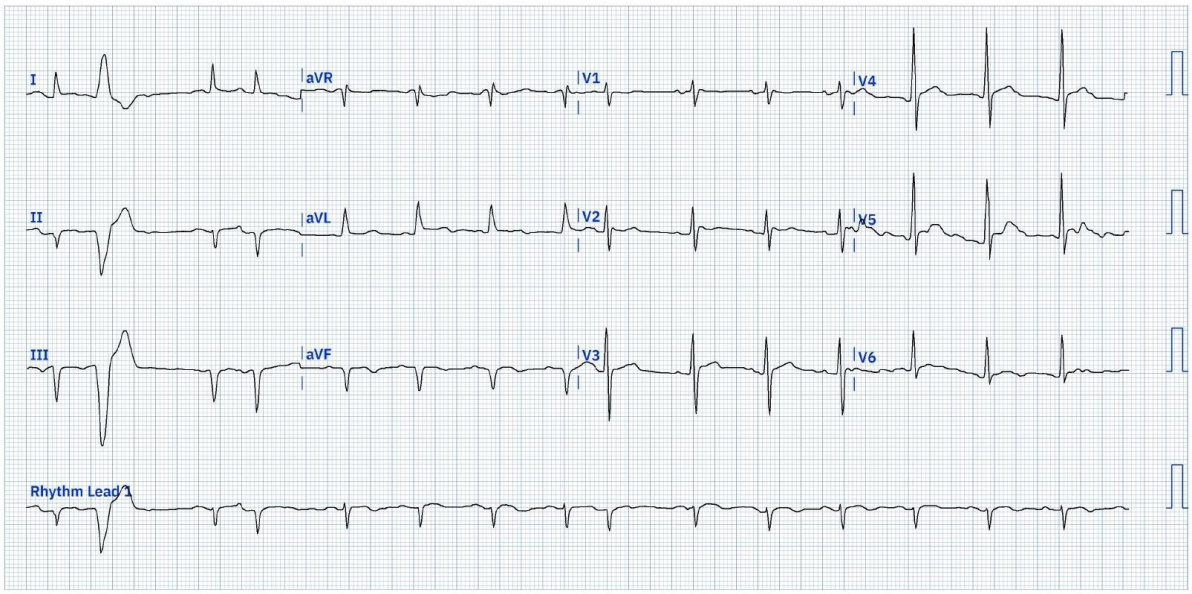

Figure 1: ECG with sinus rhythm and heart rate of 75 beats per minute, revealing inactive zone in inferior face with isolated ventricular extrasystoles.

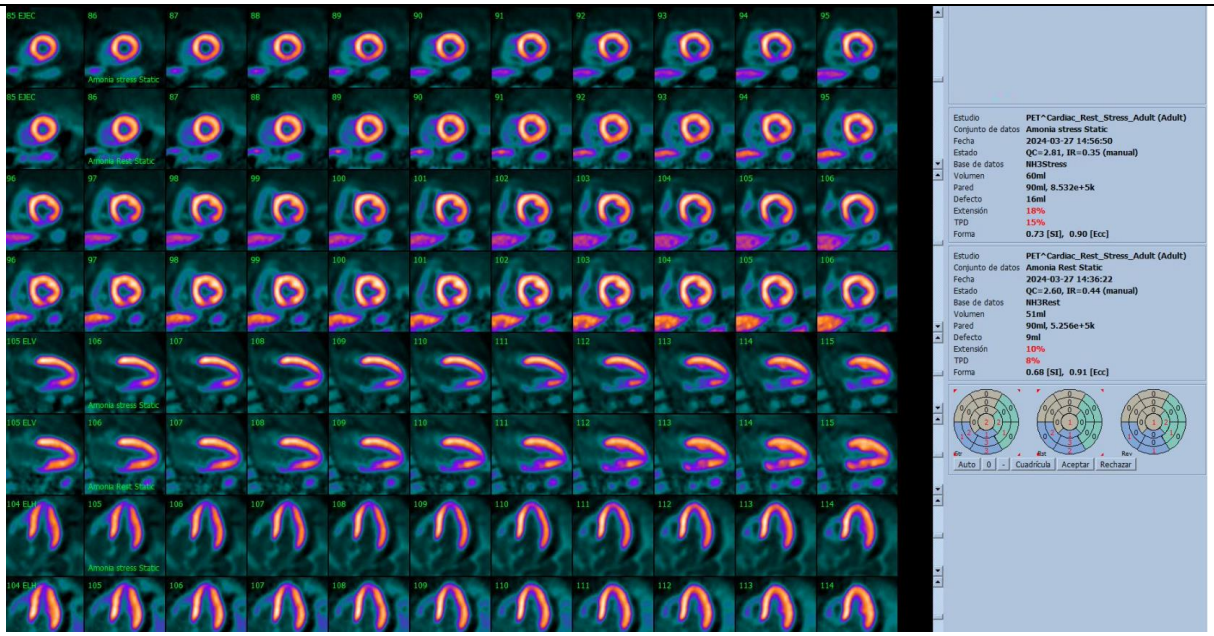

Figure 2: Non-transmural infarction of inferior location, mainly in its middle and basal thirds suggesting dorsal extension. Residual moderate ischemia in the apical and middle third and moderate ischemia in the basal third coinciding with right coronary artery territory.

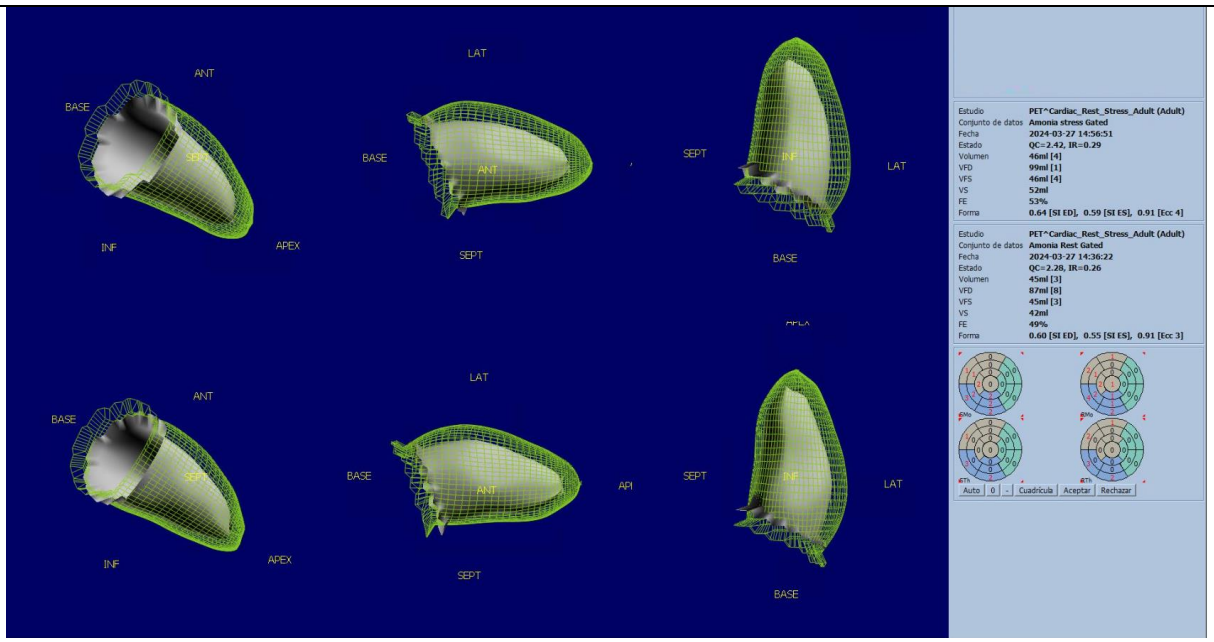

Figure 3: Preserved left ventricular systolic function with LVEF of 53%. Inferior and septal hypokinesia and decreased inferior systolic thickening.

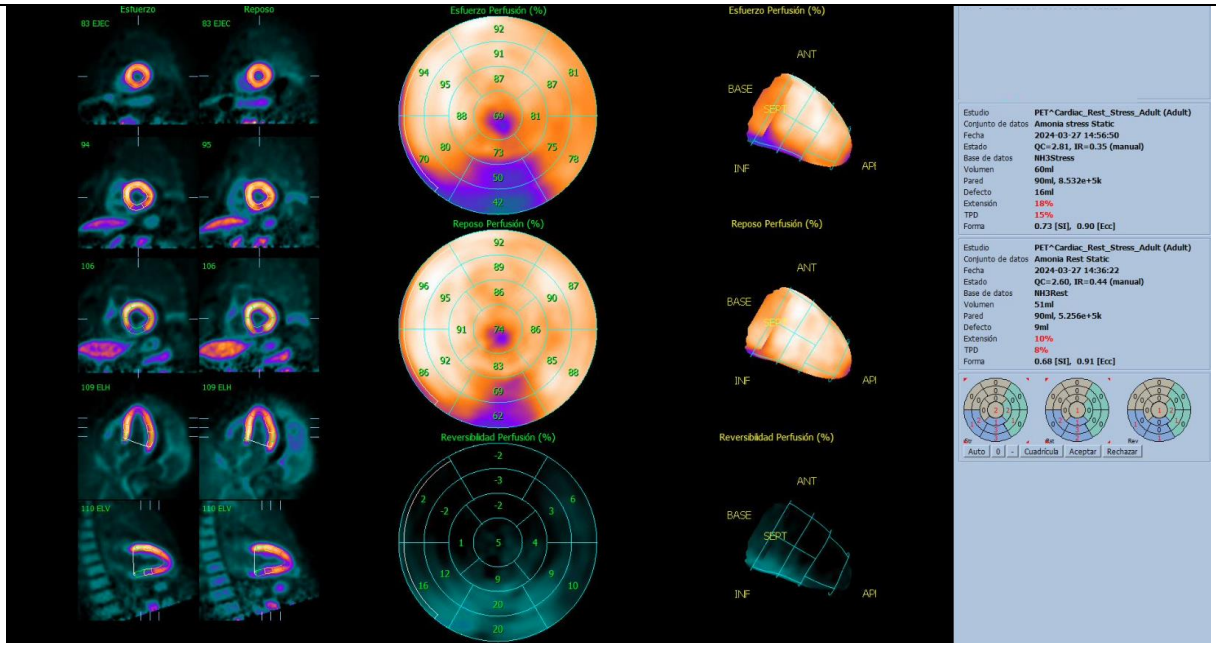

Figure 4: Segmental quantification revealing 9% ischemia.

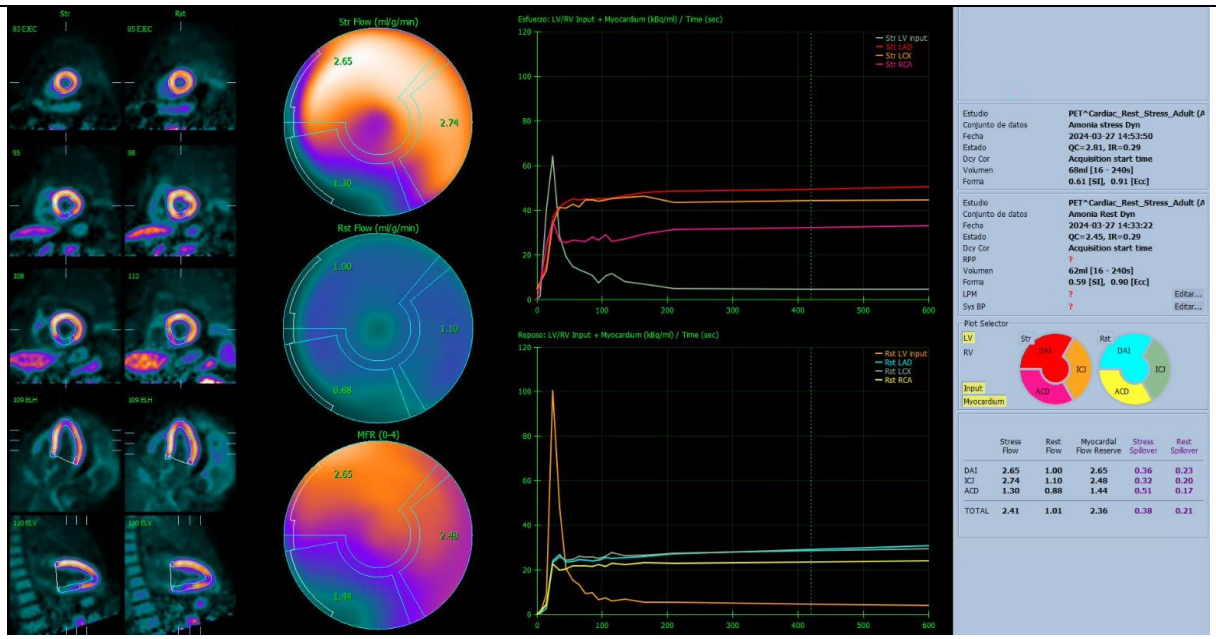

Figure 5: Decreased myocardial reserve flow in the right coronary artery territory.

Discussion

PET essential tool for patients with a history of Myocardial Infarction (MI) since it allows the evaluation of blood flow and MFR [4]. PET offers benefits such as reducing patient radiation exposure, identifying both local and global ischemia, detecting coronary stenosis, and measuring coronary blood flow dynamics and reserve during both stress and rest using a vasodilator like adenosine [5]. PET demonstrates sensitivity and specificity rates of around 90%, with an overall diagnostic accuracy of 80% for detecting CAD using 13N-ammonia PET-MPI [6,7]. The radiotracer 13N-ammonia has a half-life of about 10 minutes, with a typical dose of 10-20mCi for both rest and stress imaging [8]. MFR reflects microcirculatory function and disease of the epicardial coronary arteries and that patients with MFR less than 2 have a 70% higher probability of cardiac-related death [4]. Additionally, CAC greater than 400 carries a threefold increase in the risk of cardiovascular pathology. The combination of a perfusion agent such as nitrogen-13-ammonia or rubidium-82 and a metabolic agent like F-18 FDG is considered the gold standard for detecting myocardial viability [9]. The prognostic value of Coronary Artery Calcification (CAC) and MFR are key for predicting clinical perfusion. PET-CT allows for the acquisition of the Coronary Artery Calcium Score (CACS), which is an indicator of atherogenesis and prognosis to restage the cardiovascular risk mainly in patients with medium risk to high or low risk [4]. Used alongside the Framingham Risk Score (FRS) to assist in accurately predicting outcomes and identifying a higher prevalence of microvascular disease in patients with elevated CACS [7]. Research has shown that MFR reflects microcirculatory function and disease of the epicardial coronary arteries and that patients with MFR less than 2 have a 70% higher probability of cardiac-related death [4]. For patients without known Coronary Artery Disease (CAD), Myocardial Blood Flow Reserve (MBFR) offers significant clinical value. When combined with

normal perfusion, a high MBFR provides a robust negative predictive value, confirming the absence of CAD. It helps to verify the presence of CAD in cases of detected abnormalities and predicts the severity of the disease, such as in scenarios involving single-vessel abnormal perfusion or multiple vessels with abnormal MBFR. Moreover, it serves as a key indicator for identifying non-responders to treatment across all patient profiles.

In patients with established CAD, MBFR frequently shows abnormalities post-Coronary Artery Bypass Grafting (CABG), in those with a history of CAD, or following a myocardial infarction. While less beneficial in cardiomyopathy cases, a normal MBFR can effectively exclude the presence of CAD. Patients with renal failure typically exhibit abnormal MBFR. Post-Percutaneous Coronary Intervention (PCI), MBFR may also appear abnormal, yet its utility is enhanced when pre-PCI data is available. MBFR can confirm single-vessel disease, evident through both abnormal perfusion and MBFR in one vessel. Additionally, when perfusion is normal but MBFR is abnormal, it can identify balanced CAD and microvascular disease. Ultimately, MBFR is instrumental in recognizing non-responders to treatment in all patient categories [5]. Additionally, CAC greater than 400 carries a threefold increase in the risk of cardiovascular pathology. The combination of a perfusion agent such as nitrogen-13-ammonia or rubidium-82 and a metabolic agent like F-18 FDG is considered the gold standard for detecting myocardial viability [9]. Normal perfusion or ischemic segments suggest viable myocardium, while reduced perfusion with increased FDG uptake at rest indicates hibernating yet viable myocardium. Poor uptake of both agents signifies scarred myocardium. Identifying hibernating myocardium of necrotic tissue, It is vital to determine the appropriate therapy to follow and assess risk benefit for revascularization [9]. Reports indicate that up to 25% of MI patients develop Heart Failure (HF) within one year, and 75% within five years. Despite advances in therapies and devices, HF remains a leading cause of morbidity and mortality in MI and coronary disease patients [10]. Necrotic tissue is replaced by a non-contractile fibrotic scar, which impairs ventricular function. The size and extent of the scar are proportional to post-MI mortality. Additionally, the reduction in ventricular contractility, with the consequent decrease in cardiac output, triggers a neurohormonal cascade as a compensatory response, ultimately leading to Heart Failure (HF) in the patient [10]. It is known that for every 5% increase in the scar size, there is a 19% increase in one-year mortality and a 20% increase in hospitalizations [10]. PET is advantageous because it allows for the evaluation of Left Ventricular Ejection Fraction (LVEF) immediately after peak stress due to the short half-life of PET radiotracers, unlike SPECT, which measures post-stress LVEF 35–40 minutes later. This makes PET more effective in targeting ischemic heart disease [11]. This particular case exemplifies the typical clinical and imaging findings in a patient with suggestive data of an ischemic process, such as history of CAD, typical chest pain and a suggestive ECG. This case report underscores the use of PET as the study of choice for adequate diagnosis, risk stratification and prognosis evaluation in the approach of CAD.

Conclusion

Ischemic heart disease represents the main cause of morbidity and mortality in Mexico and worldwide, which is why the use of nuclear medicine

has revolutionized the approach to these patients. It allows for proper stratification and provides clinicians with relevant information for appropriate therapeutic decision-making. The application of radiotracers with high specificity and the reduction of adverse effects on patients make PET/CT a key method for myocardial viability assessment, leading to proper therapeutic management decisions. Currently, there is greater availability of this type of equipment. However, it is necessary to have professionals for interpretation.

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