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Interesting images

Cardiovascular [^{18}F]F-FDG PET/MRI in the diagnosis of acute cardiomyopathy inflammatory

PET/RM cardiovascular con [^{18}F]F-FDG en el diagnóstico de miocardiopatia aguda inflamatoria

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A 44-year-old woman, with slight cognitive delay, without cardiovascular risk factors, and history of multiple repetitive strokes in several cerebral territories, without showing relevant findings in arteriography in any of these acute episodes. MR angiography showed acute/subacute ischemic infarcts in multiple vascular territories, chronic microbleeds and superficial siderosis suggesting small vessel disease, including amyloid angiopathy among the aetiologies. The EKG showed sinus rhythm, with wide QRS and negative T in precordial derivations. The echocardiogram showed preserved systolic function (LVEF), without segmental alterations in contractility, with doubtful apical hypertrophy.

In the context of recurrent stroke, chronic anticoagulant treatment was initiated, presenting a new episode of left anterior ischemic stroke with normal arteriography. The study was completed with Holter-EKG showing sinus rhythm throughout the recording, with negative Ts and a cardiac MRI that showed cardiomyopathy with slightly depressed LVEF and Gadolinium retention with a mixed pattern: non-ischemic mesocardial foci with a tabby distribution and involvement mid-apical inferior transmural, as well as elevated T2 mapping values suggesting

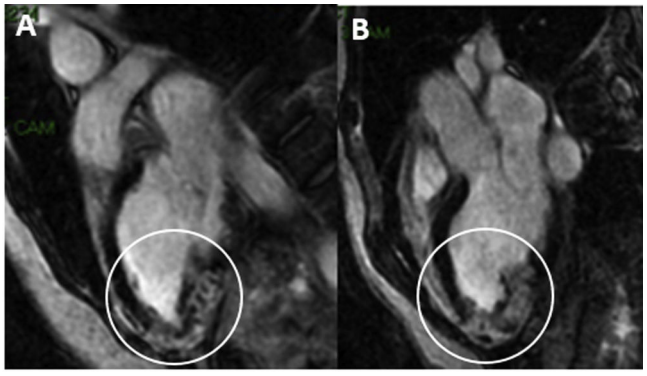

Figure 1. (A) and 3 chamber (B) end-diastole image of late Gadolinium enhancement sequence.
Hypertrophy is observed at the lower apical level and strict apex with the presence of late enhancement of transmural distribution in both segments. Very likely lamellar thrombus.
In the cine sequences (not attached due to their video format) a non-dilated left ventricle is evident with LVEF at the normal low limit value (LVEF 54%).

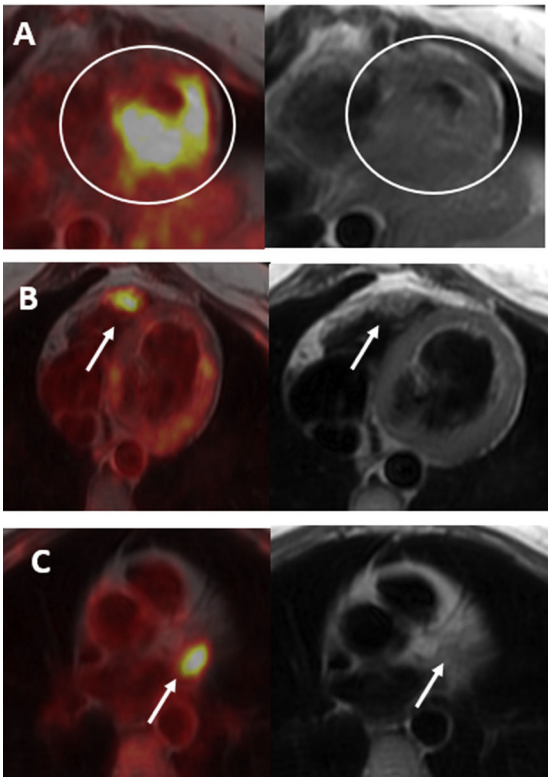

Figure 2. Axial PET/MR fusion images and axial CMR black blood sequences.
Hyperuptake of [^{18}F]F-FDG in lower mid-apical segments and apex of LV which is thickened on CMR (A), with active focus on the anterior wall of the right ventricle, with slight thickening on CMR (B) and hyperuptake of the tracer in the left appendage, without practical translation in CMR (C).
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oedema/inflammation.[1] The complete immunological and infectious study was negative and the coronary CT did not show lesions in coronary arteries.

The usefulness of [^{18}F]F-FDG PET has been described in differentiating between fibrosis and active inflammation in cases of myocarditis, as well as in evaluating response to treatment.[2] Given

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the mixed pattern evident on cardiac MR it was decided to perform a PET/MRI with [^{18}F]F-FDG, after completing a myocardial braking diet, which demonstrated hyperuptake of [^{18}F]F-FDG in the lower mid-apical and apex segments, confirming the inflammatory activity in the areas of fibrosis identified on MRI, as well as focal activity in the anterior wall of the right ventricle and in the left appendage, not identified solely by cardiac MRI (Figs. 1 and 2).

After confirmation of active inflammatory cardiomyopathy by the PET/MR study with [^{18}F]F-FDG, treatment with ACE inhibitors and beta-blockers was initiated. However, 2 months after a new ischemic stroke complicated by intracranial hemorrhagic transformation, the patient died. The autopsy showed the existence of a cardiomyopathy type Glucogenosis, allelic disease (GYG-1). This systemic disease is characterized by a late-onset myopathy, so in the biopsy study of inflammatory myopathies its genetic study is necessary.[3] In this case, PET/MRI with [^{18}F]F-FDG allowed us to confirm the inflammatory activity in left ventricle and its extension to the anterior wall of the right ventricle and the left appendage, not identified on MRI.

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