Arrhythmogenic Right Ventricular Dysplasia - Value Of Fat Suppresion In MRI And Black Bold Spin Echo Images

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Introduction

ARVD is a poorly understood and often inadequately diagnosed form of cardiomyopathy. Cardiomyopathy leads to damage of the myocardial walls of the heart, causing them to become thickened and chambers to enlarge. Patients with ARVD often have arrhythmias, which can increase the risk of sudden cardiac arrest or death. MRI can be confirmatory by its unique ability to identify and characterize fatty tissue infiltration in right ventricular free wall. Other MR criteria are severe RV dilation, localized RV aneurysms, mild global dilatation and regional RV hyperkinesias. ECG findings are Epsilon wave just beyond the QRS complex in lead V1, wide QRS complex duration of greater than 110 msec in lead V1, inverted T waves in V1 V3, late potentials in LBBB. VES

Case report

A 39 year-old came to hospital with episodes of ventricular arrhythmias and also had a previous history of cardiac arrest. On admission ECG shows VT at a rate of 180/min with LAD with LBBB. Precordial concordance positive. Resting ECG showed T inversion in V1-V6. Holter showed frequent bigemini. No evidence of NSVT/VT or sinus pauses. Coronary study was normal with EF of 60%. Echocardiography did not reveal any abnormality. On these findings (Clinical/ECG), diagnosis of Right ventricular Dysplasia was made.

Cardiac MR was performed on 1.5 T unit using synergy coil and the following sequences. BFE axial sequence was obtained to to show the anatomic detail of cardiac chambers (Fig1). Axial T1-weighted black blood which showed evidence of fibro fatty replacement of myocardium of right ventricular free wall, mild RV dilatation, mild global dilatation and regional RV hypokinesia (Fig 2). RVOT was also dilated (Fig 4). Fat suppression sequence was also done, which confirmed fatty infiltration of right ventricular wall (Fig 3).

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abnormalities of the ventricular outflow tract and that structural abnormalities observed with MR imaging were often not detected with echocardiography. MR imaging can also be used to assess both systolic and diastolic function in great detail. Several studies have addressed the presence of right ventricular diastolic dysfunction as an early marker of disease; even systolic function is still preserved.

The typical criteria that can be demonstrated with MR imaging are fatty infiltration of the right ventricular myocardium with high signal intensity on T1 weighted images, fibro fatty replacement, which leads to diffuse thinning of the right ventricular myocardium, aneurysms of the right ventricle and right ventricular outflow tract. Dilatation of right ventricle and RVOT, regional contraction abnormalities and global systolic dysfunction. Castillo E et al suggested that intramyocardial fat detection in ARVD was better with fast SE MR imaging alone and combined with fat suppression than was gated SE MR imaging in vivo. Abbara S et al used fat suppressed in addition to non fat suppressed and observed that interobserver agreement and confidence in diagnosis and evaluation of intramyocardial fatty infiltration has increased. We have also used same pattern in presented case.

REFERENCES