Poster Session -- Clinical case poster session 1

## P105

# A sustained VT during CMR scan. Physician does not fear much

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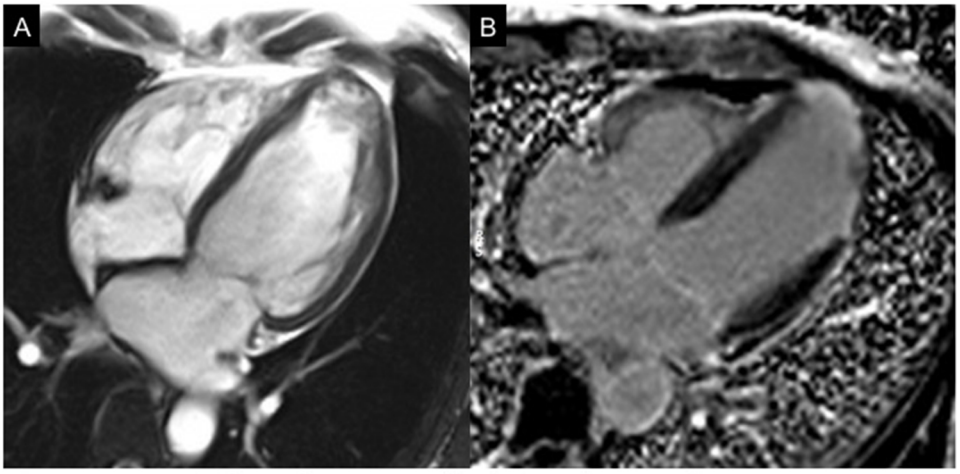
**Clinical Presentation:** a 51-years-old woman with history of palpitations and sporadic premature ventricular beats, presented to ED with chest pain. ECG revealed sustained polymorphic ventricular tachycardia (VT) with RBBB morphology. Amiodarone and lidocaine were partially effective while flecainide was initially able to maintain the sinus rhythm. The ECG showed a wide fragmented QRS (RBBB aspect) and negative T waves in lateral leads. Echocardiography disclosed dilated left ventricle (LV) with mild systolic dysfunction due to regional akinesia, prominent trabeculation in lateral wall. Troponin I and inflammatory markers were elevated as well. The patient underwent coronary angiography that ruled out any coronary disease and therefore, to identify the possible type of cardiomyopathy, an endomyocardial biopsy was performed but was inconclusive. Arrhythmia burden remained high with long episodes of VT despite antiarrhythmic therapy. Only esmolol seemed to reduce the episodes of VT. To better define the underlying disease, a cardiac magnetic resonance (CMR) during continuous esmolol injection was planned but, after functional sequences, the exam had to be stopped due to the occurrence of sustained VT. Amiodarone plus flecainide were needed to restore sinus rhythm beside esmolol. The few CMR data confirmed mild systolic biventricular dysfunction with LV non-compaction and dilated RV outflow tract. A second scan in a full body PET-CMR was scheduled showing an inflammatory cardiomyopathy with an enhanced uptake of FDG only in LV wall, Late Gadolinium Enhancement (LGE) was present transmurally in the apical and mid-segment of the anterolateral wall and as epicardial stria in inferolateral wall (Figure 1). The patient was discharged after 33 days on Flecainide and Metoprolol. Four months later a new CMR scan confirmed biventricular dilatation, a thinned and dyskinetic LV lateral wall with persistent areas of transmural LGE.

**Learning points:** the clinical data led us to think of an ischemic heart disease. Once we ruled it out, the diagnosis became challenging. Non-compaction could be due to a dilated cardiomyopathy and may be a consequence of LV remodelling rather than the cause, moreover troponin release is atypical in this setting. Cardiac sarcoidosis is commonly seen in basal segments, particularly of the septum and wall thinning is not the first manifestation. PET-CMR showed a LV inflammation with biventricular dysfunction. By Literature, the increasing use of CMR showed that in arrhythmogenic cardiomyopathy, the LV involvement is much more common than expected and a sizeable proportion of patients has a LV disease which parallels or exceeds the severity of right ventricular involvement, as in this case. With disease progression an epicardial scar can became transmural, causing thinning of myocardial wall over time. In our opinion, the most likely diagnosis was a hot phase of arrhythmogenic cardiomyopathy.

Abstract P105 Figure1.




Four chamber diastolic frame of cine sequence (first CMR scan) showing left venticle hypertrabeculation and lateral thinned wall (A). Post contrast image in the same long axis view (second CMR scan) showing myocardial fibrosis with epicardial/transmural pattern in lateral and apical segments.