paroxysmal atrial fibrillation when the diagnosis of Ebstein anomaly was made. At that stage tricuspid regurgitation was just mild-moderate in severity. Now, the clinical picture has significantly changed with worsening regurgitation and development of pulmonary hypertension. While the former complication is related to the syndrome the latter might not necessarily. It seems the development of left atrial myxoma was an additional cause for this patient’s pulmonary edema and pulmonary hypertension. Such combination has not been reported before, and it urges a critical echocardiographic examination in Ebstein patients who present with unexplained pulmonary hypertension.

Correspondence to:
Prof.Assoc.Gani Bajraktari, MD, PhD, FESC, FACC
Clinic of Cardiology, University Clinical Centre of Kosova
‘Rrethi i Spitalit’, p.n., 10000, Prishtina, Republic of Kosovo
Phone: + 377 45 800808
E-mail: gani.bajraktari@uni-pr.edu

References:

Myocardial Crypts: An incidental finding in a symptomatic patient with raised troponin

Ihab S Ramzy MD, PhD and Alina Hua MBBS, BSc

Department of Cardiology and Echocardiography Laboratory, Northwick Park Hospital, London, UK
DOI: 10.17987/icfj.v1i4.55

A 61 year old female of Asian origin presented with a two day history of intermittent sharp central chest pain radiating to the left shoulder, with each episode lasting a few seconds. Associated symptoms included exertional dyspnoea and limited exercise capacity to one flight of stairs. She denied any palpitations, pre-syncope or syncope. She had no orthopnoea, paroxysmal nocturnal dyspnoea or ankle swelling. She had stable pulmonary sarcoidosis, dual-chamber pacemaker insertion for symptomatic bradycardia 7 years ago, non-insulin dependent diabetes mellitus, hypertension, benign paroxysmal positional vertigo from the right ear, and hearing difficulties. Drug history included Metformin 500 mg, Ramipril 10 mg, Bendroflumethiazide 2.5 mg, Aspirin 75 mg and Simvastatin 20 mg. She had no known drug allergies.

Clinical examination revealed mild crepitations at the right lung base. She was haemodynamically stable and clinically euvoalamic. Twelve lead ECG showed paced rhythm. Chest x-ray showed satisfactory pacemaker wires position but was otherwise unremarkable. Blood tests were completely normal apart from a rise in Troponin I level to 0.10ug/L. A repeat Troponin level was 0.07ug/L (in house value <0.03 is considered normal).

In view of the patient’s risk factors for atherosclerosis and the rise in Troponin level, a decision was made to perform a coronary angiogram. This showed unobstructed arteries. Left ventriculogram showed a localised pouch at the inferior LV wall (Figure 1), which was confirmed on a transthoracic echocardiogram (Figure 2), and again with a cavity contrast echo for LV opacification (LVO) (Figure 3).

Figure 1: Left Ventriculogram showing a recess or pouch (arrow) in the inferior wall of the Left Ventricle.
Discussion: A myocardial crypt is a small recess within the LV myocardial wall communicating with cavity. This finding is occasionally seen on transthoracic echocardiograms, and is usually more apparent on cardiac computed tomographic angiography and cardiac magnetic resonance imaging (MRI). Brouwer et al.\textsuperscript{1} showed an increased incidence (70%) of myocardial crypts in patients with hypertrophic cardiomyopathy (HCM) mutation carriers compared to controls (12%). Similarly Maron et al.\textsuperscript{2} reported similar finding, with myocardial crypts present in 61% of the family members who were genotype positive and phenotype negative. These studies suggest that the myocardial crypts are usually located within the basal to mid inferoseptal wall and also the posterior wall. Although the frequency of myocardial crypts detected by TTE has been reported to be less than on CMR, their presence and locality should indicate the possibility that a patient may be a HCM mutation carrier. Thus, myocardial crypts may represent a novel imaging marker which alert for further clinical and family studies including genetic testing.

References:
