Case Report

Left ventricular non-compaction in pregnancy

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Abstract

Left-ventricular non-compaction (LVNC) represents an arrest in the normal process of myocardial compaction, resulting in multiple, prominent, persistant trabeculations and deep inter-trabecular recesses communicating with the ventricular cavity. LVNC is a rarely encountered cardiomyopathy and few cases have been reported in pregnancy. In this case report we present a patient who referred to our clinic with symptoms of heart failure during pregnancy and whose echocardiographic examination revealed prominent trabeculations in the left ventricle.

Keywords: pregnancy, non-compaction, heart failure

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Left ventricular non-compaction (LVNC), also known as 'spongy myocardium', represents an arrest in the normal process of myocardial compaction, resulting in the persistence of multiple prominent trabeculations and deep inter-trabecular recesses communicating with the ventricular cavity.[1,2] The presentation may follow a spectrum from no symptoms to poorly functioning dilated ventricles, a high incidence of ventricular arrhythmias, and systemic emboli.[3]

The diagnosis is usually made using echocardiography and, increasingly, magnetic resonance imaging (MRI), despite lacking universally accepted criteria.[4] Multi-detector computed tomography (CT) may also help detect the abnormal architecture of the left ventricle.[5]

Left ventricular non-compaction is a rare cardiomyopathy that appears more often in children than adults, with only a few cases reported in pregnancy.[6,7] In this report we present a patient who was referred to our clinic with symptoms of heart failure during pregnancy, and whose echocardiographic examination revealed prominent trabeculations in the left ventricle.

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Case report

A 19-year-old postpartum woman with dyspnoea was referred to our clinic for further evaluation. Although she had not experienced any cardiac symptoms during her pregnancy, she felt progressive dyspnoea and began coughing three weeks before her delivery. She had presented to an institution where she was hospitalised for a few days, before she was referred to our clinic.

Her medical history was unremarkable and she had no family history of heart failure. On physical examination, her heart rate was 98 beats/min and her blood pressure was 100/70 mmHg. She had jugular venous distention, diffuse crepitation rales in both lungs, and an apical 2–3/6 pansystolic murmur.

An electrocardiogram showed non-specific T-wave changes. Echocardiography revealed a dilated left ventricle (end-systolic diameter 49 mm) with an ejection fraction of 20%, severe mitral and tricuspid regurgitation, and mild aortic regurgitation. Pulmonary artery systolic pressure was estimated at 57 mmHg from tricuspid regurgitation. Two-dimensional echocardiography demonstrated prominent trabeculations of the left ventricle, with colour Doppler examination showing penetration of blood flow into the sinusoidal recesses formed by these trabeculations (Fig. 1).

Since there is no specific therapy for the condition, the patient was treated with beta-blockers, angiotensin converting enzyme inhibitors, diuretics and acetyl salicylic acid. Neither prophylactic anti-arrhythmic therapy nor anticoagulant therapy was given. At her three-month follow-up examination, her ejection fraction and functional capacity had improved.

Discussion

Left ventricular non-compaction is a rare congenital cardiomyopathy caused by an arrest in the normal process of endomyocardial morphogenesis. This disorder is characterised by multiple prominent trabeculations and deep inter-trabecular recesses communicating with the ventricular cavity.[1,2]

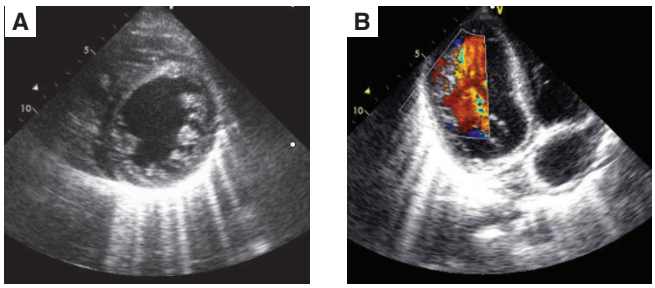

Fig. 1. A: increased left ventricular trabeculation B: colour Doppler showing blood flow between the trabeculations.