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Case Reports

Left Ventricular Noncompaction and Coronary Artery Disease: An Unexpected Combination

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Case Report

A 51-year-old man without cardiac risk factors was referred to the hospital after a complete left bundle block was discovered on an electrocardiogram; he had a 6-month history of fatigue and mild exertional dyspnea. Transthoracic echocardiogram showed a spongiform appearance of the left ventricle, with prominent hypertrabeculations in lateral and apical walls (Fig. 1A and B). The left ventricle was dilated and hypokinetic (diastolic diameter, 70 mm; ejection fraction, 25%).

Severe stenosis (90%) of both the middle tract of the left anterior descending artery and the first diagonal branch was detected by coronary angiography (Fig. 2). Lesions were treated with angioplasty and drug-eluting stent implantation. Cardiac magnetic resonance imaging confirmed the diagnosis of left ventricular noncompaction (LVNC) (Fig. 3A-D).

Discussion

Left ventricular noncompaction is a primary cardiomyopathy of genetic origin[1] that may cause congestive heart failure. Concomitant coronary artery disease (CAD) is uncommon, even more so in a patient without cardiac risk factors.

A theoretical explanation of this finding might be that the compaction of intertrabecular recesses in the left ventricular myocardium occurs simultaneously with the development of the coronary vasculature between 12 and 18 weeks' gestation.[2]

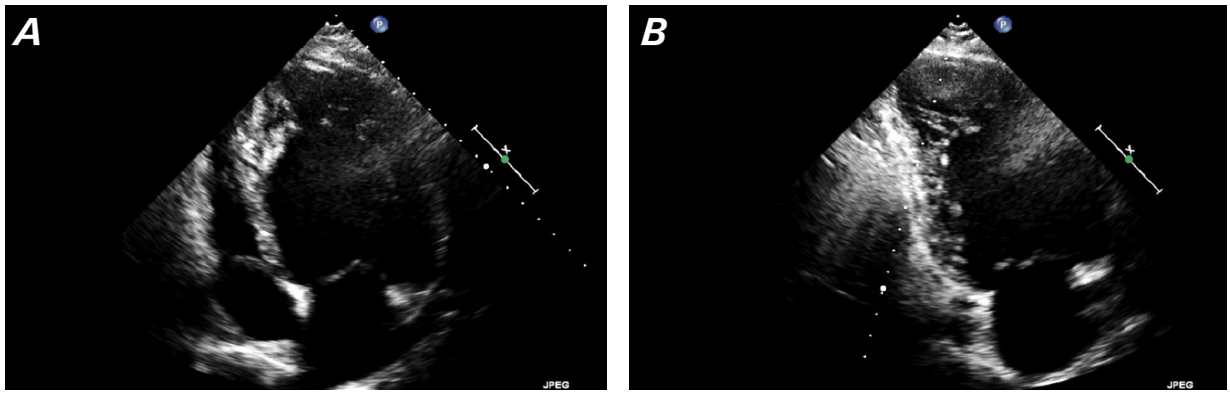

Fig. 1 Transthoracic echocardiography shows a dilated left ventricle with spongiform appearance and prominent hypertrabeculations in lateral and apical walls in **A)** a 4-chamber view and **B)** a 2-chamber view.

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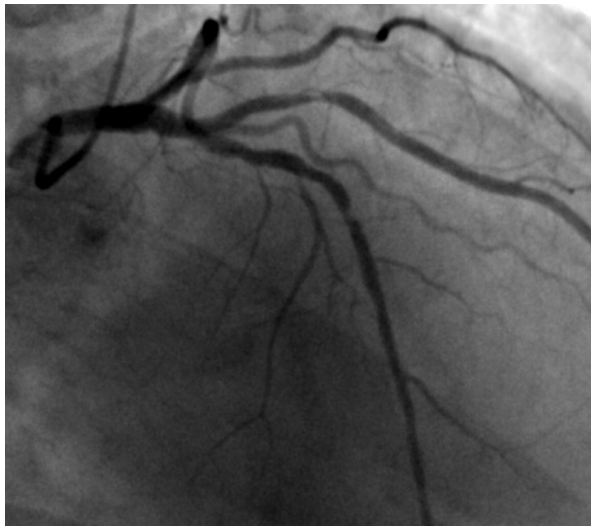

Fig. 2 Coronary angiography shows severe stenosis of both the middle tract of the anterior descending artery and the first diagonal branch.

Abbreviations and Acronyms

CAD	coronary artery disease
LVNC	left ventricular noncompaction

The anomalous origin of coronary arteries and coronary fistulae have previously been described together with LVNC in isolated case reports in the literature.

The co-occurrence of LVNC and CAD has been described only a few times before,[3-7] and prior literature on LVNC with congestive heart failure does not report any association with acquired CAD.

The presented case shows a peculiar association relevant for future reference in prospective studies to be done in cases of LVNC with cardiomyopathy.

The evidence of significant coronary stenosis in combination with LVNC without documented dyslipidemia has led to the hypothesis of a common genetic involve-

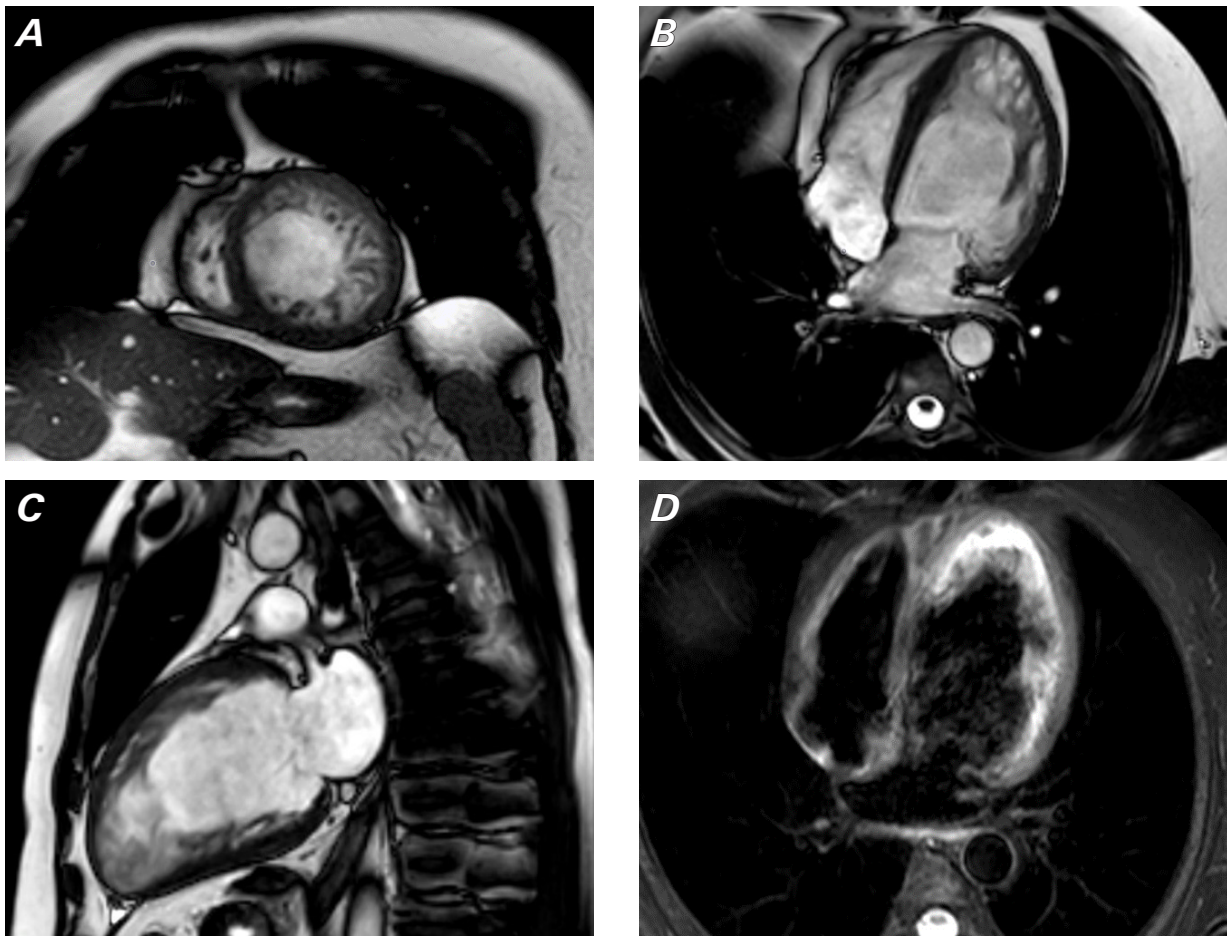

Fig. 3 Cardiac magnetic resonance imaging shows **A)** multiple hypertrabeculation as displayed in short-axis view. **B)** The end-diastolic ratio between noncompacted and compacted layers of more than 2.3 at the apex/midlateral areas in a 4-chamber view is diagnostic for left ventricular noncompaction. **C)** Spongiform appearance of the left ventricular apex in a 2-chamber view. **D)** Increased signal intensity caused by the blood pool of trabecular recesses on late gadolinium enhancement 4-chamber view.

ment for myocardial compaction and coronary endothelium development. This speculation is bolstered by the identification of a family with familial CAD and LVNC.[8]

In vitro and in the developing mouse heart, deletion of the Ino80 chromatin remodeler in vascular endothelial cells prevents ventricular compaction. This correlates with scarce coronary vascularization: Ino80 deletion results in a defect in coronary vessel formation, which means coronary arteries develop to become significantly smaller than normal.[9] Arbustini et al[10] provided an updated list of other genes that are associated with LVNC.

Therefore, LVNC is a morphogenetic defect that occurs at the site and time of embryologic development of the coronary arteries (intramyocardial section) that could potentially affect the biology of coronary arteries and increase the risk of early development of CAD.

In conclusion, whether the association of CAD with LVNC is coincidental is a question that needs further research. This report contributes to the case recording and opens the prospects for the possibility of some association in patients with LVNC cardiomyopathy.

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