Myocardial clefts are a recently described finding and are increasingly noted with the growing use of cardiac MRI. We describe a patient with multiple myocardial clefts on MRI.

Case
A 45-year-old man came for cardiac screening evaluation because of an abnormal ECG. He had undergone occupational health screening 8 years previously and had been noted to have T-wave inversion from V2 to V4. He had not pursued any further investigations. He was asymptomatic, with no history of syncope. Examination results were within normal limits. A 12-lead ECG again showed anterior T-wave inversion, with no associated Q waves (Figure 1).

A transthoracic echocardiogram showed mild asymmetrical interventricular septum thickening, measuring 14 mm in diastole (Movie I in the online-only Data Supplement). A treadmill exercise test showed horizontal ST depression in the absence of symptoms of chest pain. Because of the ECG and echocardiogram findings, a cardiac MRI was requested. This showed extensive crypt formation in the interventricular septum, with septal thickening up to 12 mm (Figure 2, Movies II and III in the online-only Data Supplement). Note was also made of an anomalous origin of the left circumflex coronary artery, and this was again seen on coronary angiography, although no obstructive plaque was detected. A diagnosis of multiple left ventricular clefts in the setting of early hypertrophic cardiomyopathy (HCM) was made, and genetic studies for structural cardiomyopathies have been requested.

Discussion
Case reports of myocardial clefts have previously been published in the literature; however, with the increasing use of cardiac MRI, it has been noted that such clefts can be seen in healthy controls also. Erol and colleagues define clefts as v-shaped fissures that extend >50% of the depth of the myocardium, which are not associated with any out-pouching of the myocardium, and that are associated with normal contractile function. These features differentiate left ventricular clefts from other structural changes such as diverticula and aneurysms.

Single or paired clefts have been observed in the interventricular septum and basal inferior wall of the left ventricle in healthy volunteers. In 1 study of 2093 patients undergoing coronary computed tomography evaluation, most for a coronary artery disease indication, 6.7% were found to have ventricular clefts; the majority of clefts were found in the interventricular septum, and only 0.7% of all patients had ≥3 clefts. The depth of clefts is also of interest, with a case–control study of HCM gene carriers and healthy controls (non–gene-carrying family members) showing that the gene carriers had deeper clefts, penetrating to 74% of the myocardium in comparison with 59% in controls (P<0.01). The presence of ≥3 clefts was highly specific for being a HCM gene carrier in this study.

Conclusion
Although isolated findings of single or paired left ventricular clefts may be a normal congenital variant, multiple clefts are rare and are more likely to be associated with a HCM phenotype. Follow-up of such findings may provide new insights into the pathogenesis of HCM changes.

Acknowledgements
No specific funding was received for this study. However the work of the Family Heart Screening Clinic is funded through charitable donations through the Mater Foundation’s Mater Heart Appeal.

Disclosures
None.

References


Figure 1. Twelve-lead ECG showing anterior T-wave inversion.

Figure 2. Cardiac MRI sequences showing multiple myocardial crypts in the interventricular septum.
Multiple Myocardial Clefts on Cardiac Magnetic Resonance Imaging
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Circulation. 2013;128:1388-1389
doi: 10.1161/CIRCULATIONAHA.112.000666

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