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Title

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Permalink

<https://escholarship.org/uc/item/2fm0k4v3>

Journal

The international journal of cardiovascular imaging, 36(10)

ISSN

1569-5794

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Publication Date

2020-10-01

DOI

10.1007/s10554-020-01914-5

Peer reviewed

Myocardial Crypt, Diverticulum, or Aneurysm? CTA as an Adjudicator.

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Figure 1: Illustrates patient's coronary CT angiogram (coronary CTA). Imaging indicates a discrete cleft in myocardium of left ventricle (LV). The narrow, deep blood-filled invagination occupies more than 50% of the myocardial wall, and it is localized between the septum and inferior wall. By sensitively and accurately assessing this inferoseptal myocardial crypt (of 5mm origin), cardiac CTA offers a vital screening modality for potential hypertrophic cardiomyopathy (HCM) carriers.

Discussion

Myocardial crypts are congenital abnormalities that have been diagnosed in both healthy and hypertrophic patients [2]. Crypts (also known as clefts) within the LV myocardium may indicate an early pathological myocardial alteration in asymptomatic patients, and might predict future hypertrophic cardiomyopathy (HCM) [1]. Coupled with a family history of HCM, the crypt predicts gene carrier status [1].

Though myocardial crypt assessment has been traditionally accomplished via cardiovascular magnetic resonance (CMR), coronary CT angiogram (coronary CTA) offers increased spatial resolution with high-quality multiplanar and 3-D reconstructed imaging. Here, we present an image in cardiovascular applications to extend the CMR gold standard to CTA for accurate crypt diagnosis.

A 22-year-old female presented with chest pain and syncope. CT screening yielded a coronary artery calcium score of zero. CCTA confirmed absence of any CAD, but it identified a large inferoseptal myocardial crypt with 5mm origin (Figure 1). This narrow, deep blood-filled invagination penetrates more than 50% thickness of the adjoining compact myocardium in the long axis views. By definition, the “V” shaped fissure is therefore a myocardial crypt [2]. Because structural abnormalities like myocardial clefts can precede manifest hypertrophy, CTA offers a vital screening tool for patients who may carry HCM mutations.

For prognostic reasons, myocardial crypts or clefts must be clinically differentiated from diverticula and LV aneurysms [2]. Clearly illustrated via CTA, crypts are uniquely classified by fissure-like protrusions enclosed within the compacted myocardium. Congenital diverticula have narrow mouths and wide outpouchings that extend beyond the myocardial margin's confines: a clear morphological difference from crypts. LV aneurysms, in contrast, possess wide mouthed and thin walled characteristics [2]. Different still to myocardial crypts, pseudoaneurysms are a form of myocardial rupture that develop from trauma, infarction, or tuberculosis. They are contained entirely by the pericardium. Confirmed by CTA, our patient's abnormality is enclosed by a normally contracting well-perfused

myocardium. Therefore, our report supports a myocardial crypt: a congenital anomaly rather than an ischemic etiology.

In conclusion, this case image demonstrates CTA's well-suited modality to accurately assess myocardial crypts. CTA offers high spatial resolution, enabling the comprehensive assessment of crypt area, structure, and myocardial thickness. Coronary CT angiography clinically distinguishes clefts from other myocardial anomalies, in a noninvasive manner and with limited operator variability. Utilizing myocardial crypts as an imaging marker, CTA can identify patients with implications for diagnostic genetic testing before HCM development.

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