Apical Hypertrophic Cardiomyopathy

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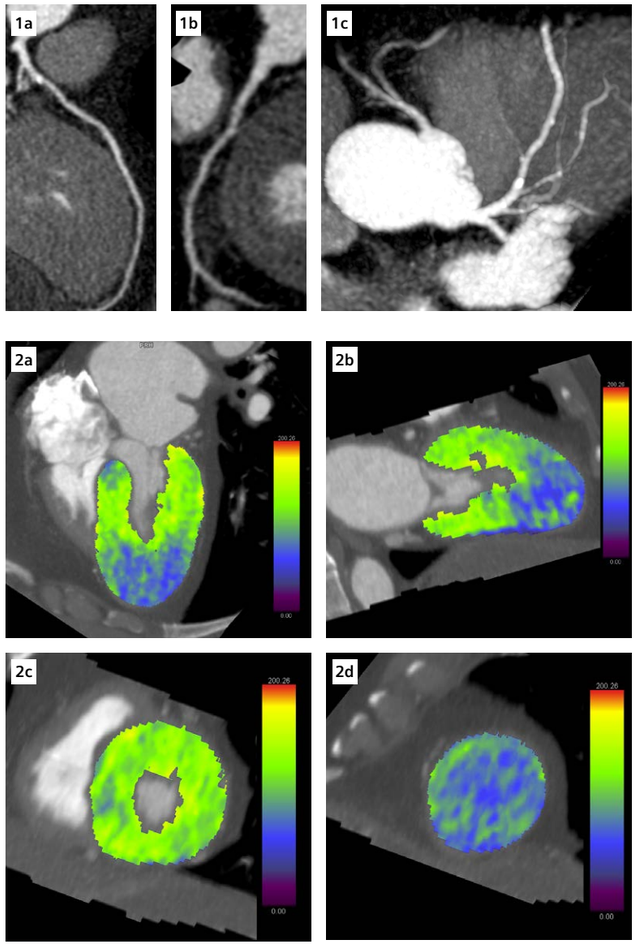


1 Curved MPR images (Fig. 1a, LAD; Fig. 1b, Cx) and a thin MIP image (Fig. 1c) of the coronary CTA show no significant stenoses.

2 Long (Figs. 2a, 2b) and short (Figs. 2c, 2d) axis views of the left ventricle show an extensive MBF reduction in the apical portion compared to the basal area.

History

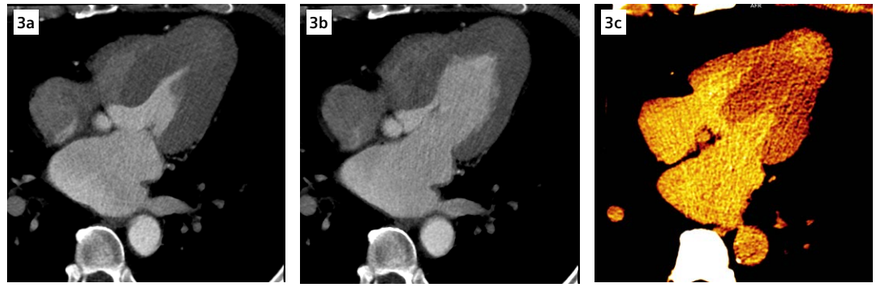
A 61-year-old male patient, complaining of progressive exertional chest tightness, relieved by rest, for the past 11 years, was referred to us with suspected coronary artery disease (CAD). Standard twelve lead electrocardiography (ECG) revealed ST-T segment changes on leads V2-V6 and T wave inversion. A coronary CT angiography (CTA) combined with ATP-stress myocardial CT perfusion was requested to investigate the coronary artery status and myocardial blood flow.

Diagnosis

Coronary CTA images showed mild stenoses in the left anterior descending artery (LAD), the left circumflex (LCX) and the right coronary artery (RCA), all caused by mixed plaques. The long-axis views of the left ventricle, in both systole and diastole, revealed a significantly thickened apical myocardium. Myocardial perfusion images showed an extensive reduction of myocardial blood flow (MBF) in the apical portion. No isolated perfusion defect was demonstrated. In the delayed scans, partial enhancement in the apical myocardium was shown, suggesting local fibrosis.

Comments

Hypertrophic cardiomyopathy (HCM) is characterized by left ventricular (LV) hypertrophy with various phenotypes, of which apical HCM accounts for approximately 2–15%.[1, 2] Clinically, patients with apical HCM may present with exertional angina or dyspnea and may have electrocardiographic findings similar to those with CAD or in acute coronary syndromes.[1, 3] Some of these patients may be admitted for ICA due to suspected CAD. Therefore, a pre-interventional differential diagnosis is important. Cardiac MR imaging has its advantages,[1, 4, 5] is however limited to its availability and by contraindications. A coronary CTA combined with ATP-stress myocardial CT perfusion can be performed to investigate the coronary arteries, the myocardium thickness and the MBF. In this case, mild coronary stenoses with unmatched extensive reduction of MBF in the significantly thickened apical portion are revealed. This is highly suggestive of an apical HCM.[1–3] These results assist the physicians in making a prompt diagnosis and appropriate treatment planning, as well as to assess the patient's prognosis. ●



3 Long axis views in the systole (Fig. 3a) and diastole (Fig. 3b) show a thickened apical myocardium, partially enhanced in the delayed phase (Fig. 3c), suggesting local fibrosis.

References
[1] Baxi AJ, Restrepo CS, Vargas D, Marmol-Velez A, Ocazionez D, Murillo H(2016). Hypertrophic Cardiomyopathy from A to Z: Genetics, Pathophysiology, Imaging, and Management. Radiographics;36(2): 335-354.
[2] Maron BJ, Maron MS(2013). Hypertrophic cardiomyopathy. Lancet;381(9862): 242-255.
[3] Ho CY, Lopez B, Coelho-Filho OR, et al.(2010). Myocardial fibrosis as an early manifestation of hypertrophic cardiomyopathy. N Engl J Med;363(6):552-563.
[4] Brouwer WP, Baars EN, Germans T, et al. (2014). In-vivo T1 cardiovascular magnetic resonance study of diffuse myocardial fibrosis in hypertrophic cardiomyopathy. J Cardiovasc Magn Reson;16:28.
[5] Maron MS(2012). Clinical utility of cardiovascular magnetic resonance in hypertrophic cardiomyopathy. J Cardiovasc Magn Reson;14:13.

Examination Protocol

Scanner	SOMATOM Force		
Scan area	Heart	Heart	Heart
Scan mode	ECG Triggered Sequential scan	Stress Myocardial Perfusion	Dynamic 4D Spiral
Scan length	107 mm	105 mm	105 mm
Scan direction	Cranial-caudal	Shuttle	Shuttle
Scan time	3.8 s	34 s	6 s
Tube voltage	90 kV	70 kV	80 kV
Effective mAs	398 mAs/rot.	275 mAs/rot.	307 mAs/rot.
CTDIvol	20.2 mGy	39.73 mGy	24.17 mGy
DLP	218.1 mGy cm	419.59 mGy cm	255.2 mGy cm
Rotation time	0.25 s	0.25 s	0.25 s
Slice collimation	144 × 0.6 mm	192 × 0.6 mm	192 × 0.6 mm
Slice width	0.75 mm	1.5 mm	1.0 mm
Reconstruction increment	0.5 mm	0.7 mm	1.0 mm
Reconstruction kernel	Bv40 (ADMIRE 3)	Qr36	Qr36
Heart rate	74 – 85 bpm	72 – 85 bpm	78 – 83 bpm
Contrast	370 mg/mL	370 mg/mL	–
Volume	45 mL + 45 mL saline	44 mL + 44 mL saline	–
Flow rate	5.0 mL/s	5.5 mL/s	–
Start delay	Bolus tracking at the ascending aorta with 100 HU + 5s	5 s	7 minutes after CTA

The outcomes by Siemens Healthineers customers described herein are based on results that were achieved in the customer's unique setting. Since there is no "typical" hospital and many variables exist (e.g., hospital size, case mix, level of IT adoption), there can be no guarantee that other customers will achieve the same results.

In clinical practice, the use of ADMIRE may reduce CT patient dose depending on the clinical task, patient size, anatomical location, and clinical practice. A consultation with a radiologist and a physicist should be made to determine the appropriate dose to obtain diagnostic image quality for the particular clinical task.