CORE

ided by Elsevie





LETTER / Cardiovascular imaging

Intramyocardial bridges: A real indication for cross-sectional cardiac imaging

S. Levesque^{a,*}, L. Cassagnes^b, J.R. Lusson^a, L. Boyer^b

^a Department of cardiology, Hôpital Gabriel Montpied, 58, rue Montalembert, 63003 Clermont-Ferrand, France

^b Department of radiology, Hôpital Gabriel Montpied, 58, rue Montalembert, 63003 Clermont-Ferrand, France

KEYWORDS Myocardial bridges; Coronary CTA; MRI Myocardial bridges (MB) are characterised by the intra-myocardial tunnelling of a large epicardial vessel, most often the left anterior descending artery. Often discovered by chance, the effect of their existence varies, with their possibly resulting in infarction or a fatal rhythm disorder. Coronary angiography, which is still the standard examination for diagnosis, poorly evaluates the ischaemic consequences on the myocardium unless combined with pressure measurements. Cardiac CT and MRI can help diagnosis by precisely evaluating the characteristics of bridges and their possible repercussions on the myocardium.

Clinical Case

We report here the case of a 51-year-old man with no cardiovascular risk factors, who consulted for mid-thoracic discomfort. The clinical examination was normal. The ECG had negative T waves in the apical region. Ultrasound showed a left ventricle of normal size and with normal kinetics and the maximum stress test was negative. A coronary CT scan was undertaken (General Electric 64-detector VCT HD scanner). Acquisition was synchronised, retrospective, at 120 Kv with a biphasic bolus injection of 75 mL of lomeron (iomeprol, Bracco, Milan, Italy) at $5 \,\text{mL/s}$, followed by a bolus of $40 \,\text{mL}$ of physiological saline at $4 \,\text{mL/s}$. Post-processing of the images highlighted two significant abnormalities. The first was a 2.7 cm long intramyocardial bridge of the middle part of the left anterior descending artery (LAD) with a depth of $3.2 \,\text{mm}$ (Fig. 1). The second was thinning of the tip of the left ventricle (Fig. 2a and b). This second image was confirmed by myocardial MRI (Siemens

* Corresponding author.

E-mail address: slevesque@chu-clermontferrand.fr (S. Levesque).

^{2211-5684/\$ —} see front matter © 2012 Éditions françaises de radiologie. Published by Elsevier Masson SAS. All rights reserved. doi:10.1016/j.diii.2012.03.007



Figure 1. Reconstruction of the LAD showing segment 2 of the LAD buried in the myocardium to a depth of 3.2 mm (a) over a length of 27 mm (b). It shows in section the path of the artery in the epicardial fat (c) and subsequently its intramyocardial route (d).

Avanto 1.5 T). The multislice cine sequences were acquired in the 4-chamber, long axis and short axis orientations. First-pass perfusion images were acquired in the same slice planes after injection of gadolinium (Dotarem[®]) at a dose of 0.2 mmoL/kg (and rate of 5 mL/s) and did not show any perfusion abnormality. Overall contractile function was normal (ejection fraction of 76%), as was myocardial mass (179 g, i.e., $94 g/m^2$). One image confirmed the existence of a microaneurysm of the tip, with no pericardial deformation. Delayed phase acquisition 10 minutes after a second injection of gadolinium showed enhancement of the thinned apical zone (Fig. 2d and e). Coronary angiography was performed which confirmed the existence of a middle LAD MB with almost complete closure of the lumen during systole (Fig. 3). Ventriculography showed stagnation of the contrast agent at the apex, without the latter being dyskinetic. Diagnosis was of a LAD MB responsible for apical ischaemic distress.

Discussion

This clinical case is worthy of note for the contribution made by cross-sectional imaging in evaluating the myocardial distress, which is seen perfectly using CT and MRI but is not evaluated by coronary angiography and ultrasound. These MBs have long been considered as benign variations as they are only 'physically' manifested during systole, while



Figure 2. Thinning of the apex of the LV visible on CT slices in 5-chamber view (a), vertical long axis (b) and in MRI VLA (c). Delayed enhancement of the same zone (arrows, d, e).

myocardial perfusion is maximal in diastole [1]. However, fractional flow reserve (FFR) and intravascular ultrasound (IVUS) functional studies have led to reconsideration of the phenomenon: there seems to be impaired relaxation in early diastole with considerable variation in diameters and intracoronary velocities [2]. Ishikawa et al. studied the anatomical peculiarities of intramyocardial segments in healthy hearts and infarcted myocardia in an autopsy series [3]. The myocardial thickness overlying the artery correlated with the presence of an infarction: $1005 \pm 703 \,\mu\text{m}$ in



Figure 3. Right anterior oblique coronary angiography showing systolic obliteration of the middle part of the LAD.

the infarction group vs. $797 \pm 526 \,\mu\text{m}$ in the healthy group (P < 0.05). The index thus calculated (thickness in $\mu\text{m} \chi$ length in cm) was significantly different in the infarction group (1997 vs 1294, P = 0.039). In our case, the MB index was calculated to be 8640.

Coronary CTA identified the tunnellised coronal portion and its morphological characteristics [4] (location, thickness and length) in a reliable and simple manner. Measurement of luminal reduction during systole is more difficult because of the usual motion artefacts associated with normal left ventricular contraction, but is probably more visible on phases from 30 to 35% of the RR interval, while the maximum diameter occursduring the 70 to 75% phase of the RR [5]. There have been no MRI studies of the repercussions of MBs.

The sensitivity of coronary CTA and MRI in detecting ischaemic coronary lesions is equivalent to that of coronary angiography [6], an imaging strategy that can also be used to characterise and assess the impact of MBs.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

- [1] Erbel R, Rupprecht HJ, Ge J, Gerber T, Görge G, Meyer J. Coronary artery shape and flow changes induced by myocardial bridging. Echocardiography 1993;1:71–7.
- [2] Ge J, Jeremias A, Rupp A, Abels M, Baumgart D, Liu F, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J 1999;20:1707–16.
- [3] Ishikawa Y, Akasaka Y, Suzuki K, Fujiwara M, Ogawa T, Yamazaki K, et al. Anatomic properties of myocardial bridge predisposing to myocardial infarction. Circulation 2009;120(5): 376-83.
- [4] Liu H, Huang MP, Liang CH, Zheng JH, Wu ZB. Detection and its clinical value of myocardial bridging with 64-slice spiral CT coronary angiography. Journal of Southern Medical University 2009;29(2):236-8.
- [5] Liu SH, Yang Q, Chen JH, Wang XM, Wang M, Liu C. Myocardial bridging on dual-source computed tomography: degree of systolic compression of mural coronary artery correlating with length and depth of the myocardial bridge. Clin Imaging 2010;34(2):83–8.
- [6] Donati OF, Scheffel H, Stolzmann P, Baumüller S, Plass A, Leschka S, et al. Combined cardiac CT and MRI for the comprehensive workup of hemodynamically relevant coronary stenoses. AJR Am J Roentgenol 2010;194(4):920-6.