Transitory left ventricular non-apical ballooning syndrome: Diagnosis with multiple imaging modalities

Syndrome de ballonisation non apicale transitoire du ventricule gauche : diagnostic avec l’imagerie multimodalités

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A 57-year-old man was referred to our department with substernal chest pain. He was a smoker whose symptoms had started the day before, during an anxiety-associated conversation. At admission, the chest pain had almost disappeared. Serial electrocardiograms revealed a sinus rhythm associated with new inverted T waves, predominantly in the precordial leads (Fig. 1). Troponin T was raised to 0.36 ng/mL at admission and decreased to 0.22 ng/mL the following day. A third myocardial blood test was normal.

At admission, coronary angiography showed normal epicardial coronary arteries; the left ventriculogram demonstrated midventricular dilation and akinesis with a hypercontractile apex and base in favour of a transient LV non-apical ballooning syndrome (Fig. 2, Panels A and B; Video 1). One day later, on cardiovascular MRI, steady-state free-precession three-chamber cine sequences (Fig. 2, Panels C and D; Video 2) confirmed WMAs involving the middle portions of the left ventricle but not the LV apex. However, long-axis dark-blood T2-weighted sequences detected apical and midventricular oedema (Fig. 2, Panel E). No abnormality was found on first-pass perfusion or delayed enhancement MRI (Fig. 2, Panel F). Surprisingly, segmental 2D longitudinal strain of the basal anterior and anterolateral
Figure 1. ECG revealed a sinus rhythm associated with new inverted T waves, predominantly in the precordial leads.

segments was decreased significantly compared with the basal inferior and inferoseptal segments (Figs. 3 and 4). A bull’s eye map of peak systolic strain showed abnormal contraction in the apex. WMAs assessed using MRI and 2D speckle-strain analysis reversed rapidly, returning to the normal range at two months.

Figure 2. The left ventriculogram demonstrated midventricular dilation and akinesis with a hypercontractile apex and base in favour of a transient LV non-apical ballooning syndrome (Panels A and B). Cine MRI sequences confirmed WMAs involving the middle portions of the left ventricle but not the LV apex (Panels C and D). However, long-axis dark-blood T2-weighted sequences detected apical and midventricular oedema (Panel E). No abnormality was found on delayed enhancement MRI (Panel F).

Of particular interest, using cardiovascular MRI and 2D speckle-strain analysis, this case suggests that transient LV non-apical ballooning syndrome and classic LV apical ballooning syndrome may be different manifestations of a single syndrome, which presented in reality diffuse WMAs. Furthermore, it may be challenging to distinguish between
Figure 3. 2D strain segmental analysis in apical three-chamber view. Segmental 2D longitudinal strain of the basal anterior and anterolateral segments was decreased significantly compared with the basal inferior and inferoseptal segments.

Figure 4. Bull’s eye map of peak systolic strain showing abnormal contraction in the apex and in the anterior, anteroseptal and anterolateral walls.

patients with this suspected entity and other patients who have recanalized an acute thrombotic occlusion of a non-obstructive coronary artery plaque or have had an acute event as a result of thromboembolism. In such cases, striking differences, detected using multiple imaging modalities, probably help physicians to manage patients adequately and to decide on secondary prevention.

Conflict of interest
None.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2009.08.013.