Transient Left Ventricular Apical Ballooning

Magnetic Resonance Imaging Evaluation

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Summary: The magnetic resonance imaging characteristics of transient left ventricular apical ballooning are described in this report, and the features that distinguish it from acute myocardial infarction are emphasized.

Key Words: magnetic resonance imaging, cardiac imaging, transient left ventricular apical ballooning

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Transient left ventricular apical ballooning or "Takotsubo syndrome" is characterized by reversible left ventricular dysfunction affecting primarily the middle and apical regions with relative sparing of the base.[1–3] The syndrome was first reported 1990/1991 in the Japanese literature.[4,5] To date, there have been only a few cases reported.[2,3,6–11] Magnetic resonance imaging (MRI) findings in transient left ventricular apical ballooning have not previously been described.

Because patients frequently present with chest pain and dynamic electrocardiographic changes in addition to focal wall abnormalities, this syndrome is often mistaken for acute myocardial infarction.[2,3,7,9] Distinguishing features, however, include relatively mild cardiac enzyme elevation, the absence of significant coronary artery disease, and rapid improvement in left ventricular systolic function.[9]

CASE REPORT

A 42-year-old white woman presented with the acute onset of shortness of breath and chest pressure. Her medical history was significant for severe migraine headaches, and a particularly severe migraine had started several hours before the onset of chest pain. On presentation to the emergency department, her blood pressure was 81/61, with a heart rate of 110. Auscultation revealed bibasal rales. Her jugular venous pressure was 12 cm H_2O.

Electrocardiography showed sinus tachycardia with incomplete right bundle branch block (RBBB) and a prolonged QT interval, with a corrected QT interval of 491 milliseconds (normal range for women: <440 milliseconds). Cardiac enzymes were slightly elevated, with troponin I measured at 1.7 ng/mL (normal range: <0.06–0.50 ng/mL), creatine phosphokinase (CPK) measured at 189 IU/L (normal range: 24–170 IU/L), and creatine kinase–MB ([CK]–MB) measured at 24 UG/L (normal range: 0–7 UG/L).

The patient was referred for cardiac MRI for assessment of myocardial viability. Magnetic resonance imaging at 1.5 T (CV/I; General Electric Medical Systems, Waukesha, WI) showed an end-diastolic volume of 158 mL and an end-systolic volume of 124 mL, with an ejection fraction of 22%. On electrocardiographic-gated cine MRI (steady-state free precession pulse sequence), there was circumferential hypokinesis and akinesis in the middle and apical regions, respectively, with normal contraction of the base consistent with a pattern of left ventricular dysfunction, recently described as left ventricular apical ballooning (Figs. 1A, B). Perfusion MRI scans were obtained after intravenous administration of 0.1 mmol/kg gadodiamide contrast (Amersham Health, Princeton, NJ). Perfusion MRI scans showed no evidence of focal perfusion abnormalities corresponding to a specific vascular territory (see Fig. 1C). There was globally delayed perfusion of the myocardium, however, probably related to the reduced ejection fraction. Myocardial viability MRI approximately 15 minutes after an additional 0.1-mmol/kg intravenous dose of gadodiamide contrast (see Fig. 1D) indicated no evidence of myocardial enhancement that would have been otherwise typical for acute myocardial infarction.

The patient improved symptomatically with diuretics and beta-blocker therapy and was completely asymptomatic at the time of discharge. Left ventricular function was monitored by serial echocardiography. Although the ejection fraction was 10% at the time of admission, it had improved to 40% by the time of discharge on hospital day 7. A follow-up echocardiogram performed 1 week later showed normal left ventricular function, an ejection fraction of 60%, and no wall motion abnormalities.

DISCUSSION

Transient left ventricular apical ballooning or Takotsubo syndrome was first reported in the Japanese population.[2–5] Recently, patients presenting with this syndrome have also been increasingly recognized in European and North American populations.[6,8,11]

The cause of transient left ventricular apical ballooning remains unknown. There are reports of transient left ventricular apical ballooning precipitated by physical or emotional stress.[7–9] In the case of our patient, it is likely that the physical stress of the severe migraine precipitated her presentation. The prognosis of transient left ventricular apical ballooning is favorable, with recovery of ventricular function within weeks.[8,9] This is certainly what was observed in our patient, with complete recovery of left ventricular function within 14 days.

Cardiac MRI provides morphologic and precise functional information about myocardial wall motion and

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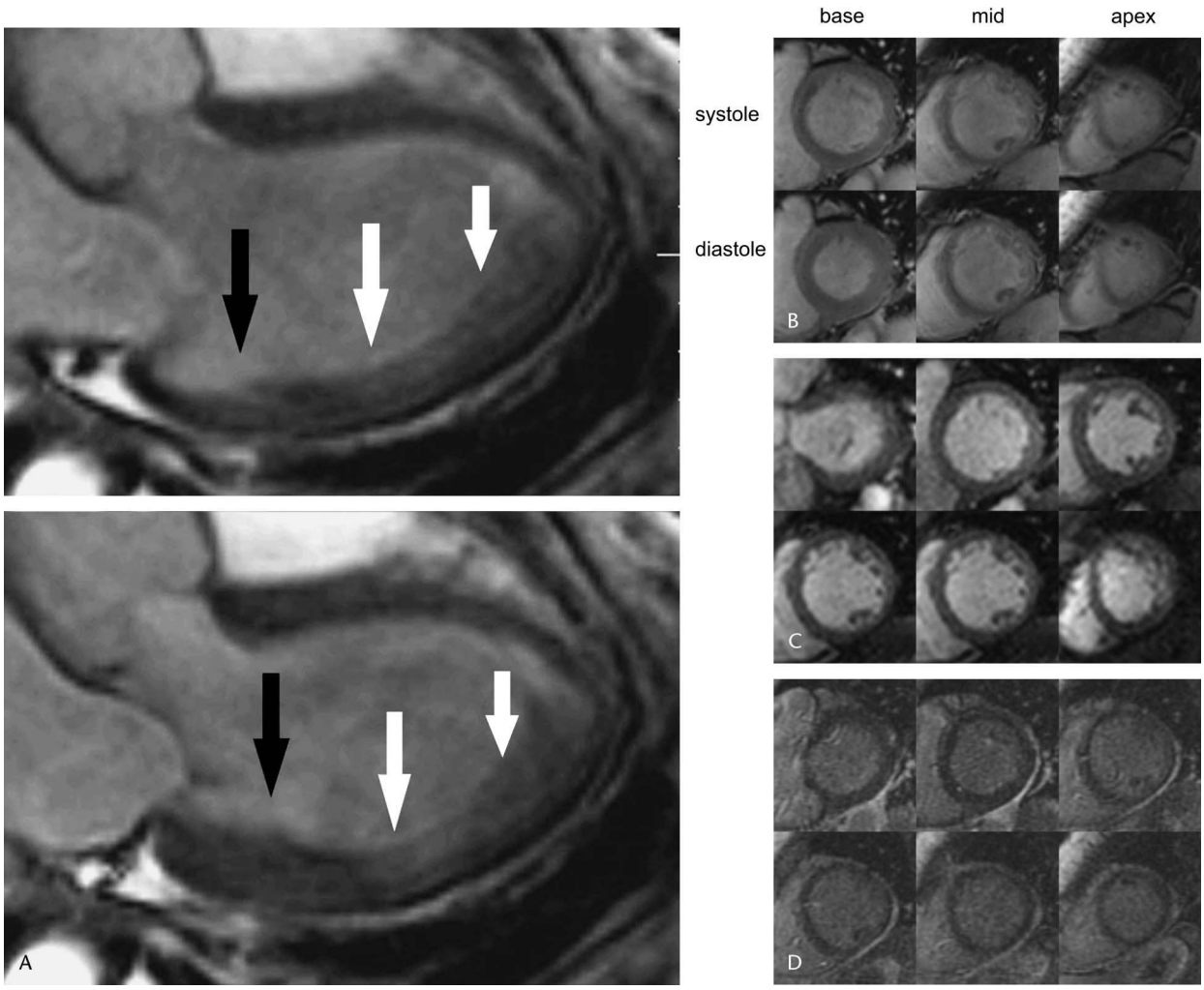

FIGURE 1. A, Diastolic (upper image) and systolic (lower image) left ventricular long-axis cine magnetic resonance imaging (MRI) scans (electrocardiographic-gated, steady-state, free precession pulse sequence; repetition time (TR)/echo time (TE) = 3.3/0.9) demonstrating circumferential akinesis of the middle and apical regions (white arrows) with normal contraction at the base (black arrow). B, Diastolic and systolic short-axis cine MRI scans (electrocardiographic-gated, steady-state, free precession pulse sequence; TR/TE = 3.3/0.9) demonstrating circumferential akinesis of the middle and apical regions with normal contraction at the base. C, Perfusion MRI scans (notched interleaved echo planar gradient echo sequence, TR/TE = 6.8/1.5) from the heart base (left upper image) to the apex (right lower image) showing no evidence of focal perfusion abnormalities corresponding to a specific vascular territory. Left ventricular dilatation is present at the middle and apical levels. D, Delayed enhancement viability MRI scan (inversion recovery–prepared, fast gradient, echo blood suppression sequence; TR/TE = 87/38) from the heart base (left upper image) to the apex (right lower image) indicating no evidence of myocardial enhancement that would have been otherwise typical for acute myocardial infarction. Dilatation with thinning of the left ventricle is evident in the lower row at the middle and apical levels.

contractility. First-pass perfusion imaging and delayed enhancement imaging after administration of gadolinium contrast enable the distinction between reversible and irreversible myocardial ischemic injury, regardless of the extent of wall motion or the age of the infarct.[12,13] Myocardial infarction is characterized on MRI by signal enhancement of the infarct area 10 to 20 minutes after gadolinium contrast administration using an inversion recovery–prepared, gradient-recalled, echo pulse sequence.

The differential diagnosis for this patient included myocardial infarction or less common entities such as myocarditis or sarcoidosis. Patients with each of these conditions may present with acute left ventricular dysfunction; however, patients with both acute and chronic myocardial infarction show typical enhancement on MRI viability studies that corresponds to the involved coronary artery territories. Patients with myocarditis or sarcoidosis also show enhancement on MRI, but the pattern is typically focal without a typical coronary artery distribution.[8] In the patient in this report, the area of myocardial dilatation and severe dysfunction was localized to the apical portions of the left ventricle. The areas of dysfunction showed no delayed enhancement, suggesting that the myocardial injury was reversible.

The mechanism of pathogenesis of transient left ventricular apical ballooning remains unknown. Pathologic findings from endomyocardial biopsy include polymorphonuclear and mononuclear cell infiltration with focal myocyte injury and no evidence of acute myocarditis, and macroscopic necropsy examination in a patient with transient left ventricular apical ballooning did not show evidence of recent myocardial infarction or scarring.[2,3,7] Magnetic resonance imaging findings in our patient were consistent with these reported findings.

In summary, transient left ventricular apical ballooning is an unusual entity that typically resolves within weeks if appropriate supportive care is given. Magnetic resonance imaging findings of left ventricular dilatation and decreased function that spares the heart base, combined with the lack of gadolinium enhancement, may be characteristic in the correct clinical setting. Given the many overlapping clinical features of transient left ventricular apical ballooning and other causes of acute myocardial dysfunction, MRI may be a useful modality in helping to diagnosis left ventricular apical ballooning.

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