SHORT COMMUNICATION

Reversible pacemaker dysfunction in a patient with transient cardiac apical ballooning syndrome: a case report

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Transient cardiac apical ballooning syndrome (TCABS) is diagnosed by transthoracic echocardiography demonstrating apical akinesis with left ventricular (LV) apical ballooning and preserved mid-to-basal LV systolic function, and left heart catheterization showing the absence of significant obstructive epicardial coronary artery disease. Presenting symptoms are suggestive of an acute coronary syndrome and electrocardiogram findings mimic acute myocardial injury. Right ventricular involvement has been reported. We describe a case of acute pacemaker dysfunction caused by the TCABS, which responded to conservative therapy.

KEYWORDS
Transient cardiac apical ballooning syndrome; Pacemaker dysfunction

Case report
An 81-year-old female with a history of hypertension, hypothyroidism, and previously documented normal left ventricular (LV) systolic function was admitted to our institution with 5 days of progressive lower extremity oedema, dyspnoea, and substernal chest discomfort. One year prior to this admission, she had undergone implantation of a dual-chamber permanent pacemaker (Insignia I Plus DR, model 1298, Guidant Corp., St Paul, MN, USA) with subsequent concomitant radiofrequency catheter ablation of the atrioventricular node for the treatment of refractory atrial tachyarrhythmias.

Her cardiac rhythm at presentation showed atrial pacing with atrioventricular dissociation, complete heart block with escape of a junctional focus, and intermittent failure of ventricular capture. Her electrocardiogram (ECG) showed ST-segment elevation in leads V1, V2, I, and aVL and deeply inverted T-waves in leads II, III, aVF, and V3–V6 (Figure 1). Initial pacemaker interrogation revealed an acute increase in the right ventricular (RV) pacing threshold from a baseline value of 1.2 V at 0.5 ms to 2.7 V at 1.0 ms. In addition, the sensed R-wave amplitude was diminished from a baseline value of 8.9 to 5.2 mV, whereas the ventricular pacing lead impedance had decreased from a value of 1210 to 740 Ω.

Atrial lead characteristics were unchanged from baseline. The ventricular pacing output was subsequently increased to 6.5 V at 1.0 ms in order to observe an adequate safety margin. Troponin-T and creatine kinase MB levels peaked at 0.07 (normal, 0.03 ng/L) and 7.4 ng/L (normal 0–6.1 ng/L), respectively. A brain natriuretic peptide level was elevated at 921 pg/mL (normal, 160 pg/mL) in the setting of normal renal function. Serum electrolytes and thyroid stimulating hormone (TSH) were within normal limits, and no significant acid–base disturbance was present.

Transthoracic echocardiography demonstrated severe biventricular apical akinesis with LV apical ballooning and preserved mid-to-basal biventricular systolic function (Figure 2). Coronary angiography revealed the absence of significant obstructive epicardial coronary artery disease (no evidence of coronary artery spasm) and left ventriculography demonstrated apical ballooning with sparing of the mid-basal wall segments (Figure 3). All imaging studies demonstrated an intimate relationship between the apical placement of the RV pacemaker lead and the regional segments of biventricular dysfunction. Careful questioning of the patient revealed significant personal stress.

The patient was diagnosed with transient cardiac apical ballooning syndrome (TCABS) and treated aggressively with β-blocker, angiotensin-converting enzyme inhibitor, and diuretic therapy. Complete resolution of congestive heart failure symptoms occurred within 2 weeks. One month
following her admission, repeat transthoracic echocardiography demonstrated normalization of biventricular function. Repeat pacemaker interrogation at 10 days and 5 months following hospital discharge revealed improved RV pacing thresholds of 1.0 V at 1.0 ms and 1.5 V at 0.5 ms, respectively. The RV pacing lead impedances also returned to a baseline value of 1200 Ω.

### Discussion

We describe a case of reversible pacemaker dysfunction in a patient with TCABS. Transient cardiac apical ballooning syndrome is often characterized by ECG changes suggestive of an acute coronary syndrome. Although our patient’s initial ECG is consistent with those typically found in prior...
descriptions of TCABS, we cannot exclude the possibility of cardiac memory-induced T-wave changes secondary to acute loss of capture in the RV (Figure 1).

The majority of descriptions of TCABS have demonstrated transient LV apical ballooning/akinesis with sparing of the mid-to-basal LV wall segments, although other phenotypic manifestations, including reports of mid-chamber or basal LV ballooning have been reported. In fact, recent evidence of RV apical involvement with sparing of the base has been described.

In our patient, the abnormalities of pacemaker function were limited to the RV apical pacing lead, giving credence to the probability of RV apical involvement in this case of TCABS. Adjunctive imaging studies demonstrated an intimate relationship between the RV apical pacemaker lead and the apical wall segments affected. No reversible aetiologies were identified to explain the transient reversible nature of these ventricular pacing abnormalities, and the normalization of the ventricular pacing threshold values at the time of resolution of the transient biventricular apical dysfunction suggests a causal relationship.

In conclusion, we describe a patient with TCABS with biventricular involvement as a rare cause of transient reversible pacemaker dysfunction. Abnormalities of ventricular pacing can be expected to improve or normalize with the resolution of TCABS. Abnormalities of device function can typically be managed with changes in device programming and careful observation. This case suggests that the pathophysiology of the TCABS consists of dynamic reversible changes in the myocardial substrate, which are phenotypically expressed as both mechanical and electrophysiological abnormalities.

Supplementary Material

Supplementary material is available at Europace online.

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The authors state that they have seen and approved the paper and that the work has not been, and will not be, published elsewhere.

Conflict of interest

None declared.

References