Subclinical Hypothyroidism
A Reversible Cause of Complete Loss of Ventricular Lead Capture

Francesca Esposito, MD; Vincenzo Liguori, MD, PhD; Gennaro Maresca, MD; Annunziata Cerrone, BS; Ovidio De Filippo, BS; Bruno Trimarco, MD, PhD; Antonio Rapacciuolo, MD, PhD

Case Presentation
A 77-year-old male former smoker with hypertension, dyslipidemia, and diabetes mellitus was admitted to our institution for recurrent syncope. He had a history of previous non-Q-wave inferior myocardial infarction in 1980, which had never been investigated by coronary angiography. Echocardiogram showed left ventricular dilatation, inferior akinesia, and hypokinesia of the remaining segments with severe left ventricular dysfunction (left ventricular ejection fraction, 30%). Twenty-four–hour Holter ECG recording revealed 3 episodes of self-terminating sustained ventricular tachycardia. The patient underwent a coronary angiography that evidenced multivessel disease with chronic total occlusion of the proximal right coronary artery and of the mid left circumflex artery and diffuse disease of the left anterior descending artery without critical stenosis. Collateral flow ran from left anterior descending artery to left circumflex artery via diagonal branches (Rentrop 2); mid-distal right coronary artery was supplied by homo-contralateral vessel circulation via left circumflex artery collateral channels (Rentrop 3). No revascularization therapy was performed because both mid-distal right coronary artery and left circumflex artery were supplied by valid collateral flow and the patient was asymptomatic for both angina and dyspnea (New York Heart Association class I). Optimal medical therapy was established, including beta-blockers, loop diuretics, and mineralocorticoid receptor antagonists. Therefore, according to current guidelines, he underwent the implantation of a dual-chamber implantable cardioverter defibrillator (ICD). Through the standard left subclavian vein approach, we implanted a dual-coil passive fixation lead (Isoflex Optim, 1944/52 cm, St Jude Medical) at the right ventricular apex and a J-shaped passive lead (Endotak Reliance G 0286, Boston Scientific Corporation) in the right atrial appendage. The leads were connected to a dual-chamber defibrillator (Fortify ST DR, St Jude Medical) which was implanted prepectorally. Postimplantation parameters of both atrial and ventricular leads were normal (P-wave amplitude, 2.5 mV; atrial threshold, 0.75 V×0.5 ms; atrial lead impedance, 650 Ω; R-wave amplitude, 7.2 mV; ventricular threshold, 0.5 V×0.5 ms; ventricular pacing lead impedance, 490 Ω; shock impedance, 44 Ω). One month later, the patient was readmitted for persistent atrial fibrillation. Successful conversion to sinus rhythm with intravenous amiodarone was performed, and at discharge, oral amiodarone was continued. Predischarge thyroid function was normal. At 6-month follow-up ICD interrogation, intrinsic R wave was 8.1 mV, pacing impedance was 512 Ω, and shock impedance was 40 Ω, but analysis of stimulation threshold revealed complete lack of ventricular capture (maximum output tested, 7.5 V×1.0 ms; Figure 1). Atrial lead parameters were normal. Blood tests evidenced subclinical hypothyroidism: thyroid-stimulating hormone, 35.20 μU/mL (normal range, 0.3–4.2 μU/mL); free triiodothyronine, 3.7 pg/mL (normal range, 2.0–4.4 pg/mL); and free thyroxine, 0.87 ng/dL (normal range, 0.9–1.7 ng/dL). A standard posterior–anterior and lateral chest radiograph was performed. Chest radiograph showed ventricular lead to be in standard position. Because the patient was not pacing dependent and amiodarone is known to raise pacing threshold, this drug was discontinued and ICD interrogation was scheduled at 1 month. ICD interrogation 1 month after amiodarone withdrawal evidenced persistent lack of ventricular capture (maximum output tested, 7.5 V×1.0 ms). The patient was admitted to hospital for ventricular lead revision. Because of lack of radiological evidence of ventricular lead dislodgment and a subclinical hypothyroidism of new onset, we decided to postpone ventricular lead revision until after hypothyroidism had been adequately treated. Therefore, levothyroxine (50 mg/day) was started. On the third day, thyroid-stimulating hormone level was reduced to 24.57 μU/mL, and free thyroxine was 0.87 ng/dL. A standard posterior–anterior and lateral chest radiograph was performed. One month later, the patient was discharged on levothyroxine therapy. Three months after discharge, thyroid-stimulating hormone had returned within its reference range (3.3 μU/mL) and ventricular pacing threshold was 1.0 V×0.5 ms (Figure 3).
**Discussion**

Thyroid disorders are known to exert a multitude of adverse effects on myocardium and cardiovascular system, such as bradycardia, atrioventricular block, and prolonged QT interval. Hypothyroidism causing myxedema cardiomyopathy has been reported as a cause of increased ventricular pacing threshold and complete exit block. A previous report by Patton et al describes an isolated atrial lead dysfunction in a young woman with overt hypothyroidism attributable to Graves disease completely reversed with thyroid hormone replacement. Subclinical hypothyroidism is characterized by abnormal lipid metabolism, cardiac dysfunction, and diastolic hypertension. It is associated with a significant risk of atherosclerosis and ischemic heart disease and increased cardiovascular mortality at follow-up. The management of subclinical thyroid dysfunction is controversial, and there is no sufficient evidence that treatment is beneficial. A consensus panel recommends treatment with levothyroxine for patients with serum thyroid-stimulating hormone levels >10 μU/mL, especially when associated to decreasing free thyroxine levels, because the rate of progression to overt hypothyroidism is 5%. This is the first demonstration that a complete loss of capture can be attributable to subclinical hypothyroidism. Immediate ventricular pacing threshold restoration with levothyroxine administration strongly supports the hypothesis that subclinical hypothyroidism was the cause of exit block.

**Disclosures**

None.

**References**

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**Key Words:** amiodarone ◼ heart failure ◼ hypothyroidism ◼ thyroid gland ◼ thyroid hormones

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**Figure 1.** Twelve-lead ECG showing lack of ventricular capture, despite pacing artifact (circle).
Figure 2. ECG showing restoration of ventricular capture following levothyroxine administration.

<table>
<thead>
<tr>
<th>Time</th>
<th>Threshold (V x ms)</th>
<th>TSH levels (μU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-implantation</td>
<td>0.5 x 0.5</td>
<td>&lt;4.2</td>
</tr>
<tr>
<td>Six-month follow-up</td>
<td>&gt;7.5 x 1.0</td>
<td>35.20</td>
</tr>
<tr>
<td>Third day on levothyroxine</td>
<td>0.75 x 0.5</td>
<td>23.57</td>
</tr>
<tr>
<td>Three months after discharge</td>
<td>1.0 x 0.5</td>
<td>&lt;4.2</td>
</tr>
</tbody>
</table>

Figure 3. Ventricular pacing threshold vs time in relation to thyroid-stimulating hormone (TSH) levels. The figure highlights elevation of right ventricular threshold causing complete exit block, attributable to new onset subclinical hypothyroidism 6 months postimplantation. Short-term levothyroxine administration determines recovery of ventricular capture that persists 3 months after discharge, consistent with complete normalization of TSH levels.
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