

ECG changes after electroconvulsive therapy, cause or consequence?

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Electroconvulsive therapy (ECT) is a commonly used treatment in psychiatric disease for severe depression. It is considered to be a relatively safe procedure, although several case reports have described cardiac complications. Significant ECG changes may occur after this treatment [1–3] and echocardiographic abnormalities have been described [4–6]. Noninvasive evaluation of several cases after ECT failed to demonstrate underlying cardiac abnormalities [1, 7]. Despite the lack of invasive studies, a generally accepted viewpoint is that ECG changes after ECT are the result of increased sympathetic activity due to massive release from the hypothalamus [2]. However, in the following two cases, we demonstrate by using invasive evaluation that the relation between ECT and subsequent ECG changes may not always be explained by this theory.

Patient 1

A 67-year-old male was admitted to our cardiology department after ECT because of low oxygen saturation and persistent ECG abnormalities (Fig. 1). His medical history comprised hypertension, dilated abdominal aorta, diabetes and severe mental depression. MRI of his brain showed an old cerebral infarction. His ECG 6 months before ECT showed sinus rhythm with small biphasic T waves in leads V5-6.

After ECT he did not express any symptoms. Physical examination revealed no abnormalities. Blood pressure was 140/80 mmHg. His ECG showed a normal sinus rhythm of 67 beats/min with a left axis deviation. Negative T waves were seen in leads V1-5, which were new compared with previous ECGs. Laboratory investments showed maximal troponin-T level of 0.06 $\mu\text{g/l}$ (normal value $<0.05 \mu\text{g/l}$). All other parameters were normal except an increased d-dimer. Echocardiography showed a normal left ventricular function with mild concentric hypertrophy. Because of the combination of ECG changes, increased troponin level and low oxygen saturation at admission, coronary angiography was performed. In the proximal left anterior descending artery a significant stenosis was demonstrated at the division of the first diagonal branch. The other coronary arteries did not show stenoses. A few days later, the patient underwent elective PCI with stenting of the stenotic artery. The negative T waves on the ECG resolved. Afterwards he remained clinically stable.

Patient 2

A 78-year-old female patient, temporarily admitted to the recovery department after ECT, developed atrial fibrillation and chest pain. Her medical history showed hypertension, severe depression and paroxysmal atrial fibrillation. Her ECG 3 months before ECT was completely normal. Immediately after ECT her blood pressure was 230/140 mmHg, but later decreased to 110/45 mmHg. Examination of heart and lungs revealed no abnormalities. The ECG showed atrial fibrillation with a ventricular rate of 140 beats/min and new negative T waves in leads I, II, aVL and V2-6. The second ECG showed sinus rhythm and persisting negative T-waves (Fig. 2). Troponin T level was 0.02 $\mu\text{g/l}$ (normal) and no other laboratory abnormalities were found. A differential diagno-

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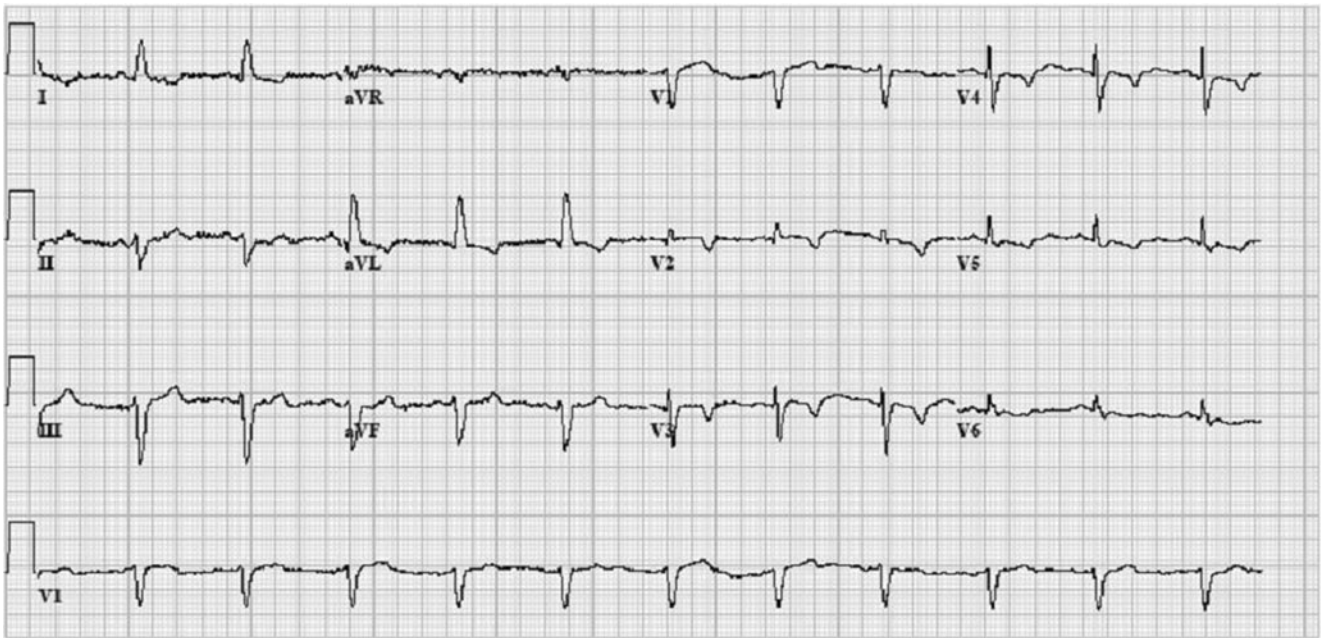


Fig. 1 ECG of patient 1 showing new negative T waves after ECT

sis was made and the following possible causes were taken into account: post ECT, post tachycardia due to atrial fibrillation, severe hypertension or myocardial ischaemia. Since the patient reported to have had previous anginal complaints, coronary angiography was performed. In the left anterior descending artery two significant stenoses were identified in the proximal and the mid section, respectively. Furthermore, a stenosis was found in the right coronary artery. Due to her psychiatric

condition, PCI was not performed and she was treated with anti-anginal medication and warfarin.

Discussion

External control of the heart by cerebral and neurohormonal factors is well established [8, 9]. Although ECT is a generally accepted, widely performed and safe therapy,

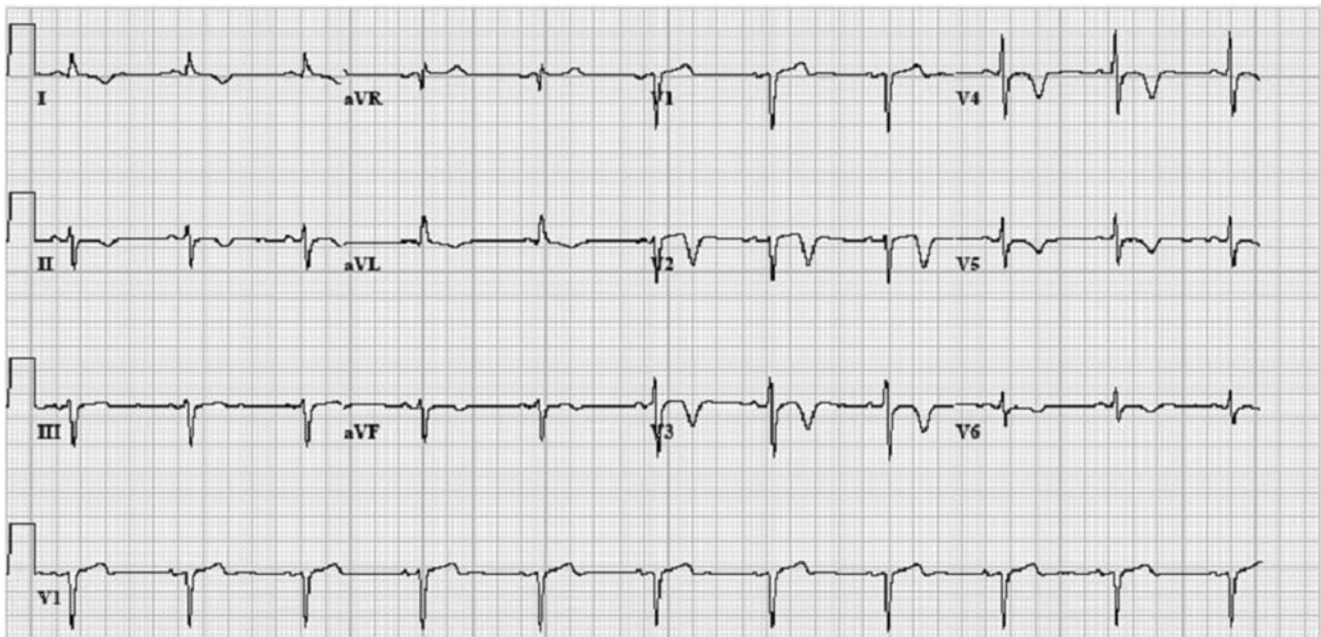


Fig. 2 Second ECG of patient 2, 8 h after ECT, showing persistent negative T waves

cardiac complications may occur. The most frequently observed are arrhythmias and T-wave inversion on the surface ECG, but also transient wall motion abnormalities have been reported [4–6]. Transient cardiac abnormalities have been explained by the concept of adrenergic excess [10]. In this concept, however, coronary artery disease was not considered to play an important role, because intense sympathetic activity was thought to be the exclusive explanation. One case report supported this concept by showing development of acute myocardial infarction during a course of ECT without underlying coronary artery disease [11].

In the present report, we describe two patients with an initially recognised ‘classic’ pattern of newly developed ECG abnormalities after ECT. However, after invasive evaluation, both patients were shown to have severe coronary artery disease. To our knowledge, this is the first study reporting two consecutive patients who were invasively evaluated after ECT, demonstrating that coronary artery disease may be the primary cause of ECG abnormalities. This finding is important because ECG abnormalities due to coronary artery stenosis have significant prognostic impact. As a consequence, we think that T-wave abnormalities after ECT cannot be used to differentiate between ECT-induced ECG changes and actual myocardial ischaemia. Useful clues to differentiate may be increased troponin levels and previous anginal complaints. Finally we suggest that shortly before ECT, a 12-lead ECG should be performed.

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