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Case Report

Rare cause of ventricular tachycardia: Pheochromocytoma

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ABSTRACT

Pheochromocytoma is known from a wide range of clinical manifestations and can mimic other disorders which can lead to delay in diagnosis. We report a case of a young female presenting with chest pain, electrocardiographic changes, and episodes of ventricular tachycardia, finally diagnosed with this catecholamine-producing tumor.

<**Learning objective:** Pheochromocytoma is a rare catecholamine-producing tumor that can pose a diagnostic challenge due to its multiple manifestations mimicking various conditions, including cardiovascular disorders. Despite its infrequent occurrence, pheochromocytoma should be considered as a possible cause of life-threatening cardiac arrhythmias and electrocardiographic changes in patients with diagnostic difficulties and primarily suspected of having cardiovascular disease. Furthermore, the method of treatment is entirely different.>

Introduction

Pheochromocytoma is a rare and most often adrenally located catecholamine-producing tumor with diverse and vicious clinical manifestations and thus difficult to diagnose. Typically it expresses itself as a triad of abrupt episodes of headache, palpitations, and profuse sweating, accompanied by either sustained or paroxysmal hypertension or a labile blood pressure [1]. However, several case reports illustrate unexpected onset of the disease with failure of organs and systems, including multiple organ failure, as well as severe cardiovascular disorders, including life-threatening arrhythmias [2–9].

Case report

A 37-year-old healthy woman, smoker, with a family history of coronary artery disease (CAD), was admitted to our department with exercise-induced palpitations and chest discomfort. She complained about similar episodes increasing in frequency and severity over the previous year. Over the previous six months the episodes occurred several times a week, and lasted for 5–15 min. Three months prior to the admission, she was examined with echocardiography and an ambulatory Holter monitoring, and both tests were normal.

On admission the patient was stable with unremarkable objective examination. Electrocardiography (ECG) showed sinus rhythm 75/min and negative T waves in V1–V5 (Fig. 1A). Additionally, slightly elevated cardiac biomarkers with maximum troponin T (TNT) 26 ng/l (reference: <14 mg/l) and maximum creatinine kinase-MB (CK-MB) 6.6 mg/l (reference: <4.0 mg/l) led to subacute coronary angiography (CAG), which was normal. The remaining blood tests were normal and so were echocardiography, cardiac rhythm monitoring during hospitalization, and exercise test. ECG was normalized over the observation period (Fig. 1B). During the first admission she was normotensive (blood pressure ranged from 100/55 mmHg to 145/90 mmHg) and did not complain of symptoms characteristic for pheochromocytoma. Finally, her episodes were attributed to suspected paroxysmal supraventricular tachycardia, and she was discharged with a planned ambulatory ECG monitoring (R-test) in order to detect arrhythmia and a cardiac magnetic resonance imaging (MRI) to disclose possible ischemic changes.

Two months later, the patient was readmitted with similar symptoms, with exercise-induced palpitations and severe chest pain. On admission she was pale and hypotensive at 90/53 mmHg. Her pulse was 67/min, respiratory rate was 18/min, O_2Sat 100%, and temperature was 36.7 °C. The physical examination was normal. An arterial blood gas analysis showed respiratory alkalosis with a decrease in $PaCO_2$ due to hyperventilation, an increased plasma lactate 5.8 mmol/l (reference: 0.5–2.5 mmol/l), and plasma glucose 12.8 mmol/l (reference: 4.2–7.8 mmol/l). Renal, liver, and cholesterol blood samples were all normal. The initial ECG showed

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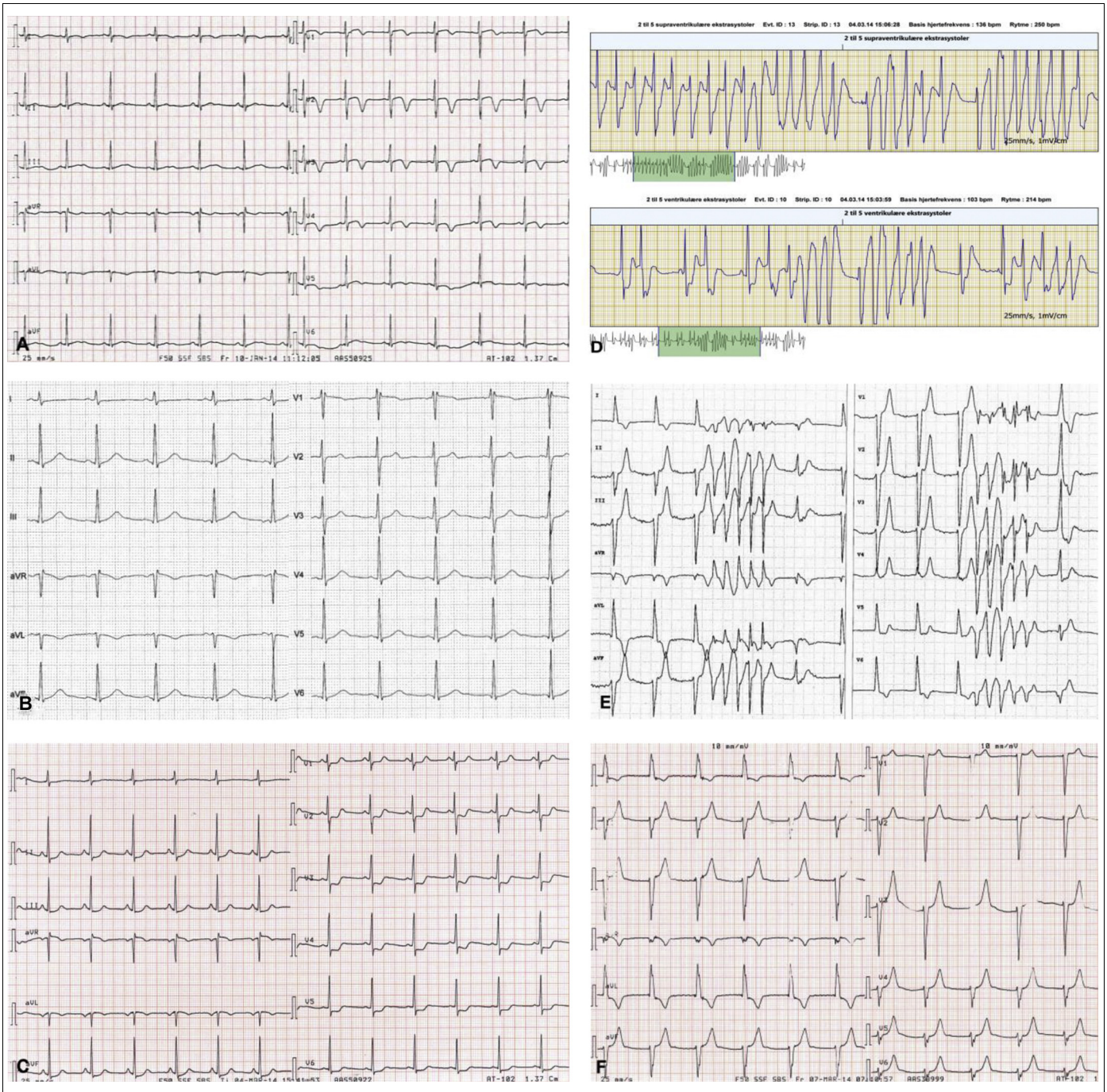

Fig. 1. Electrocardiogram (ECG) and R-test tracing. (A) ECG on the first admission mimicking evolved anterior wall myocardial infarction with negative T waves in V1–V5. (B) Prior observed ECG changes normalized during the observation period. (C) ECG on the second admission showing generalized ST-segment depressions of maximum 2 mm in V3–V4. (D) R-test tracing illustrating episodes of non-sustained polymorphic ventricular tachycardia. (E) ECG during an episode, showing nodal rhythm with non-sustained polymorphic ventricular tachycardia. (F) ECG during another episode, showing exclusively nodal rhythm.

sinus rhythm 77/min with generalized ST-segment depressions of maximum 2 mm in V3–V4 (Fig. 1C). An echocardiography revealed slightly reduced left ventricular ejection fraction (LVEF) with hypokinesia in the basal parts of the septum and the inferior wall. The chest X-ray was normal. The patient's symptoms, electrocardiographic and echocardiographic changes, as well as elevated cardiac biomarkers, maximum TNT 415 ng/l and maximum CK-MB 12.8 μg/l, led to the suspicion of non-ST-segment elevation myocardial infarction (non-STEMI) and the patient underwent subacute CAG. The examination again ruled out CAD and coronary vasoconstriction as possible causes of the symptoms. To our advantage, the day before the second admission, she had an ambulatory R-test that revealed frequent episodes of an aggressive non-sustained ventricular tachycardia (VT) 250/min, resembling torsades de pointes (Fig. 1D). A diagnostic electrophysiological study was performed without induction of the arrhythmia, despite aggressive stimulation protocols and even despite high doses of isoprenaline intravenously. Presence of an accessory pathway was ruled out. Subsequent cardiac MRI confirmed slightly reduced LVEF at 55% and a suspicion of regional ischemic lesion, corresponding to right coronary artery supply area. During the second hospitalization, we reported further symptomatic episodes of VT as well as