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Fulminant adrenergic myocarditis complicated by pulmonary edema, cardiogenic shock and cardiac arrest

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ABSTRACT

Adrenergic myocarditis is an uncommon presentation of pheochromocytoma and extremely rare cause of *de novo* acute heart failure (AHF). We present a case of a 31-year-old Caucasian woman with a history of hypertension and recurrent occipital headaches who was admitted to the emergency department due to severe *de novo* AHF presenting as pulmonary edema and cardiogenic shock. During the hospital admission the patient experienced asystolic cardiac arrest and was successfully resuscitated, intubated, and mechanically ventilated. Bedside transthoracic echocardiography revealed severe diffuse left ventricular hypokinesis with ejection fraction (LVEF) of 10%. Coronary angiography disclosed normal epicardial coronary arteries. The diagnosis of fulminant myocarditis was based on clinical, laboratory and imaging findings including cardiac magnetic resonance imaging (cMRI) Lake Louise criteria. STIR-cMRI sequences revealed myocardial edema in the lateral, inferior and posterior walls of the left ventricle, whereas T1-weighted early contrast-enhanced sequences showed myocardial hyperemia and capillary leak. An ultrasound and computed tomographic scan of the abdomen disclosed a solid, heterogeneous mass (3.6 × 3.2 × 2.8-cm) in the right suprarenal area. Urinary and plasma catecholamines and metanephrines were markedly elevated. A pheochromocytoma was suspected and laparoscopic resection of the tumor was performed after pharmacological preparation with phenoxybenzamine. The histopathological findings were consistent with pheochromocytoma. Follow-up cMRI showed complete reversal of myocardial edema and hyperemia. At 12-month follow-up, the patient has remained asymptomatic and normotensive with no recurrence of cardiovascular symptoms.

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1. Case report

A 31-year-old Caucasian woman with a history of hypertension and recurrent occipital headaches was admitted to the emergency department because of severe *de novo* acute heart failure (AHF) presenting as pulmonary edema and cardiogenic shock. During the hospital admission the patient experienced asystolic cardiac arrest and was successfully resuscitated, intubated, and mechanically ventilated. Her body temperature was 38.4 °C. When she was admitted to the intensive care unit, she was unconscious, pale, tachycardic (120–130 bpm) and hypotensive (70–85/60 mm Hg).

A 12-lead ECG showed a sinus tachycardia at 130 bpm with no signs of myocardial ischemia or prior infarct. Bedside transthoracic echocardiography (TTE) revealed severe diffuse left ventricular hypokinesis with ejection fraction (LVEF) of 10%.

Blood examinations showed the following: high-sensitivity troponin T, 3.46 (normal < 0.014) ng/mL; creatine kinase, 1406 (normal < 170) U/L; MB isoenzyme (CK-MB), 107 (normal < 24) U/L; white blood cells, 30,000/μL (normal, 4.5–10.0); C-reactive protein, 33.0 (normal < 3.0) mg/L. Serological tests for the most common cardiotropic viruses were negative. Multiple blood, urine and bronchial aspirate cultures were sterile. Anti-nuclear antibodies were negative.

Coronary angiography disclosed normal epicardial coronary arteries. Short tau inversion recovery (STIR) sequences of cardiac magnetic resonance imaging (cMRI) revealed high-signal intensity in the lateral, inferior and posterior walls of the left ventricle, consistent with myocardial edema (Fig. 1A). T1-weighted early contrast-enhanced sequences showed myocardial hyperemia and capillary leak. No late gadolinium enhancement was found. In addition, mild to moderate pericardial effusion was present (up to 13 mm) with no evidence of cardiac tamponade (Fig. 1B).

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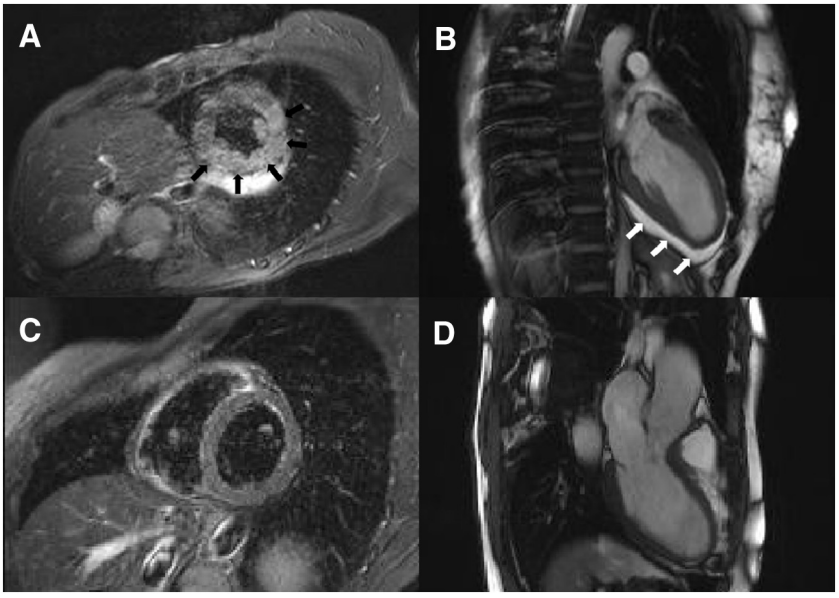

Fig. 1. Cardiac magnetic resonance imaging (cMRI). A. STIR short axis plane - increased intensity of the lateral, inferior and posterior wall of the left ventricle (black arrows); B. 2-dimensional (2D) TOF sagittal images showing mild to moderate pericardial effusion (white arrows); C–D. Follow-up cMRI sequences showing complete regression of myocardial lesions.

An abdominal ultrasound disclosed a solid, heterogeneous mass (3.6 × 3.2 × 2.8-cm) in the right suprarenal area (Fig. 2). Computed tomography (CT) scan of the abdomen confirmed the presence of the oval, well-defined right adrenal mass with attenuation values of 30–40 Hounsfield units (Fig. 3).

During hospitalization, the patient required temporary pacing because of symptomatic paroxysmal third-degree atrioventricular block detected on 24-hour Holter monitoring (Fig. 4). In the following days, the clinical symptoms and laboratory parameters significantly improved. TTE performed at discharge showed a recovery of left ventricular contractility (LVEF 55%).

Based on markedly elevated plasma and urine catecholamines and metanephrines, a pheochromocytoma was suspected and the patient was transferred to the endocrinology department for further investigation. The laparoscopic resection of the tumor was conducted after pharmacological preparation with phenoxybenzamine. The histopathological findings were consistent with pheochromocytoma. Follow-up cMRI showed complete reversal of myocardial edema and hyperemia (Fig. 1C–D). At 12-month follow-up, the patient has remained asymptomatic and normotensive with no recurrence of cardiovascular symptoms.

2. Discussion

This case illustrates a very rare presentation of adrenal pheochromocytoma as fulminant adrenergic myocarditis complicated by pulmonary edema, cardiogenic shock and cardiac arrest.

Pheochromocytoma is an uncommon catecholamine-producing tumor arising from chromaffin cells of the adrenal medulla (85–90%) or extra-adrenal paraganglia with an estimated annual incidence of 2–8 per million population [1,2]. The majority of adrenal pheochromocytomas may secrete both norepinephrine and epinephrine due to the activity of phenylethanolamine *N*-methyltransferase (PNMT) – an enzyme that converts norepinephrine to epinephrine [2]. Rarely,

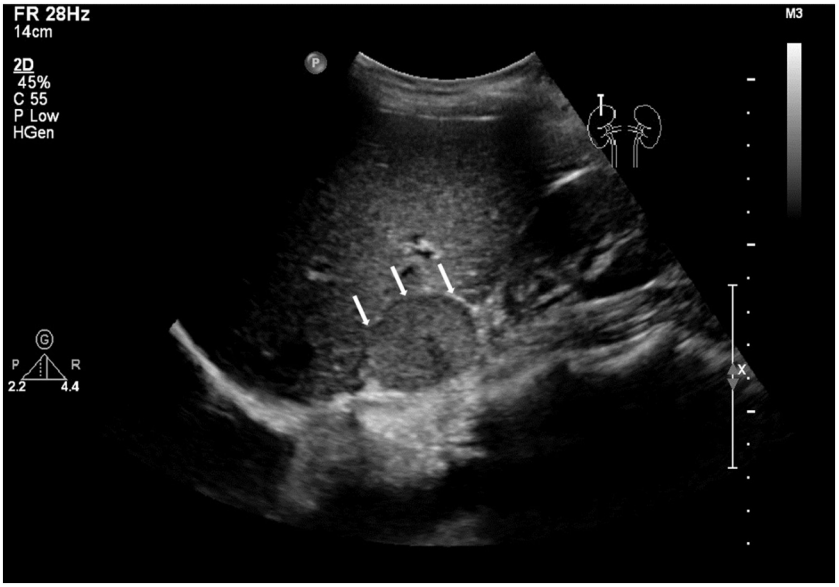

Fig. 2. Abdominal ultrasound revealed a solid, heterogeneous mass (3.6 × 3.2 × 2.8-cm) in the right suprarenal area (white arrows).

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