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Hyperacute Phase Catecholamingergic Polymorphic Ventricular Tachycardia Captured on Computed Tomography

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A patient undergoes thermal ablation of an adrenal tumor, complicated by catecholaminergic polymorphic ventricular tachycardia. The constellation of radiological findings afforded by intraoperative computed tomography are presented.

Keywords Radiology; Interventional; Cardiology; Oncology; Ablation.

INTRODUCTION

Catecholamine excess is associated with a spectrum of functional and morphological changes of the heart. In acute excess, the syndromes of catecholaminergic polymorphic ventricular tachycardia (CPVT) and stress cardiomyopathy (SM) have been described. In chronic excess, structural changes have been described in patients with phaeochromocytoma and paraganglioma [1].

CASE REPORT

A 37-year-old female patient underwent thermal ablation of a right adrenal metastatic tumor. She had stage 4 lung adenocarcinoma maintained on targeted therapy. Two years prior, she underwent left adrenalectomy for another metastasis.

The procedure was performed by interventional radiologists under general anesthesia. The patient was placed in the left lateral position. Clonidine 1 μg/kg was administered before procedure for alpha blockade. Hydrodissection was performed to separate the right adrenal gland from the inferior vena cava for protection. Subsequently, a 15 g microwave applicator (Solero; Angiodynamics, Latham, NY, USA) was inserted into the tumor under direct computed tomography (CT) guidance and two 3-minute ablation cycles followed. Towards the end of the ablation, the patient was noted to have hypertension (230/185 mm Hg) and desaturation (93%). Cardiac monitoring showed polymorphic ventricular tachycardia. This was treated with magnesium and metoprolol and the haemodynamic changes resolved within a few minutes.

Immediately after the episode, non-gated arterial and venous phase CT of the chest and abdomen were obtained. There was patchy hyperenhancement of the subendocardial and mid-wall myocardium at the left ventricular apical segments, new from the pre-procedure arterial phase CT (Figs. 1 and 2). The proximal right coronary artery, left anterior descending artery and left circumflex artery were patent, excluding large vessel occlusion as etiology. There was new extensive left lung consolidation (the dependent side) in keeping with flash pulmonary edema.

The patient was mechanically ventilated post operatively with complete resolution of the pulmonary oedema. Serial echocardiogram showed resolving global left ventricular impairment with calculated ejection fraction troughing at 35% on day 0 and returning to 50% on day 3. No apical ballooning nor regional wall motion abnormality was seen. There was biochemical evidence of acute myocardial injury with Troponin I peaking to 3446 ng/L (0–17 ng/L) and NT-proBNP to 7230 ng/L (0–400 ng/L).

A diagnosis of acute polymorphic ventricular tachycardia secondary to catecholamine storm was made. SM was thought less likely given lack of morphological changes on echocardiogram.

The patient was discharged 4 days following procedure with no significant functional impairment. Three months later, she underwent another successful ablation procedure for a newly developed hepatic metastasis. Her cardiac changes were completely resolved on the intraoperative CT.

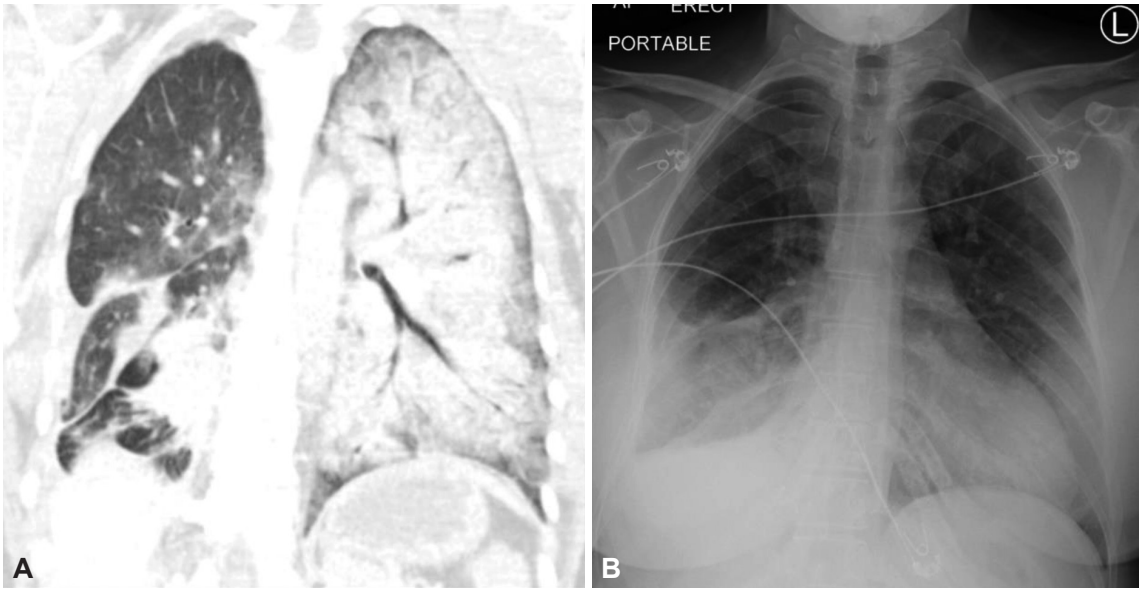

Fig. 1. Flash pulmonary edema. A: Coronal reformatted intraprocedural CT image demonstrating extensive left lung consolidation with subpleural sparing, reflecting flash edema in the dependent lung secondary to cardiac dysfunction. Right lower lobe opacity is due to a previously treated lung tumor. B: Erect frontal chest radiograph taken day 3 post ictus demonstrating complete resolution of the left lung consolidation and chronic right lung changes. CT, computed tomography.

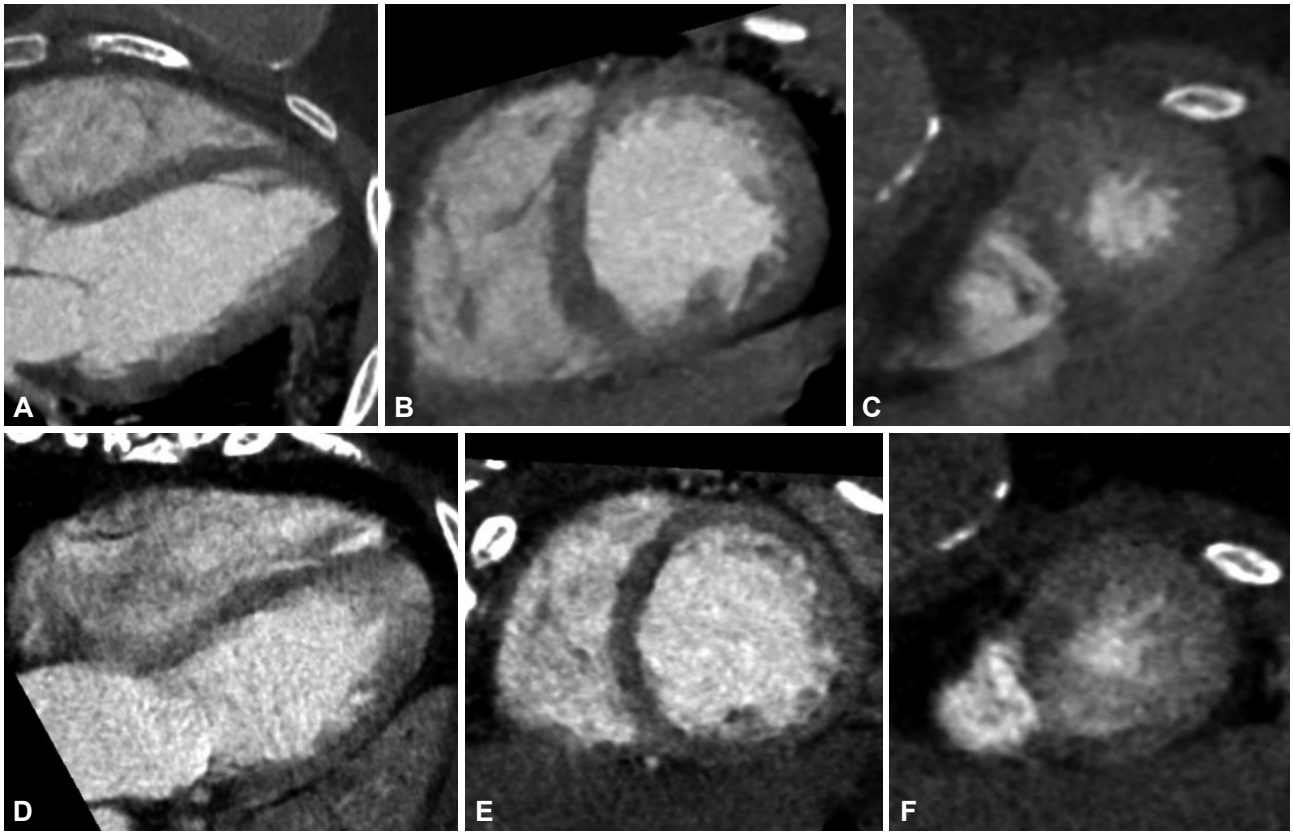

Fig. 2. Immediately pre-ablation arterial phase CT image reformatted to vertical long axis view (A) and short axis view at mid (B) and apical levels (C). Normal appearances of the myocardium within limitations of non-dedicated, non-gated study. Immediately post-ablation arterial phase CT image again reformatted to vertical long axis view (D) and short axis view at mid (E) and apical levels (F). There is hyperenhancement of the AHA segments 13, 14, 16, and 17 (apical, apical lateral, apical posterior, and apical inferior), with relative sparing of the apical septal segment. The basal and mid segments are not involved. CT, computed tomography; AHA, American Heart Association.

DISCUSSION

The imaging features of SM are well described on echocardiogram and magnetic resonance imaging (MRI). In the acute phase, there is myocardial edema (hyperintensity on T2-weighted triple inversion recovery sequences, prolonged T2 and T1 on parametric mapping) as well as kinetic and morphological abnormalities (hypokinesia and apical ballooning on echocardiogram and cine MRI). In the chronic phase, the changes are either completely reversible or they may be variable degrees of myocardial scarring (delayed gadolinium enhancement on MRI) [2].

In CPVT, the focus in the literature is often on the genetic basis or electrophysiological manifestation, with imaging features not well described. A small study analyzing MRI taken in the interictal phase of 20 patients found either normal findings or occasional non specific features such as ventricular dilation and myocardial non-compaction [3].

The proposed pathophysiology of catecholamine associated cardiomyopathy includes direct toxicity and myocardial stunning secondary to microvascular spasm [4]. Our case offers a rare glimpse into the pathophysiology as we managed to image the patient during the hyperacute phase. Although CT is not typically used to image cardiomyopathy due to limited soft tissue and temporal resolutions, it is perhaps the only modality capable of capturing these hyperacute changes.

Three hypotheses can be offered for the present CT findings of myocardial hyperenhancement: First, there may be loss of autoregulation of the distal vasculature, with increased blood and contrast flow to the damaged myocardium, giving "luxury perfusion" as seen in the case of brain infarcts. Second, there may be transient increase in membrane permeability of the stunned myocardium, leading to pooling of contrast in the extracellular space. Third, there may be venous congestion leading to retention of contrast. Determining the correct hypotheses would require further study.

Ethics Statement

Informed consent has been obtained for this publication.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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None

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