

IMAGE FOCUS

doi:10.1093/ehjci/jes168

Renal cell carcinoma presenting as a stress cardiomyopathy

Miguel Silva Vieira*, Nuno Antunes, Henrique Carvalho, and Severo Torres

Cardiology Department, Hospital Santo António, Centro Hospitalar do Porto, Largo Prof. Abel Salazar 4099-001, Porto, Portugal

* Corresponding author. Tel: +351 222 077 500; fax: +351 223 320 318. Email: zemiguelvieira@gmail.com

A 76-year-old woman was admitted to the emergency department with a squeezing severe chest pain starting at rest, 1 h before. Previous medical history was unremarkable. An electrocardiogram showed rapidly evolving ST-segment changes (Panels A–C). The rapid troponin I assay was positive (1.4 ng/mL). Unknown renal dysfunction was also noted (creatinine of 1.7 mg/dL). Apical akinesia was present on bedside echocardiogram (TEE). Urgent coronary angiography due to unremitting chest pain and hypotension excluded obstructive coronary disease. Ventriculography showed a striking pattern of apical akinesia (Panels D and E). The patient received the standard supportive care with gradual clinical improvement. The recovery of wall motion (Panels F and G) was seen in a cardiovascular magnetic resonance study, 2 weeks later, which also confirmed the absence of inflammation (Panel H), myocardial fibrosis, or infarction (Panel I) and found a right renal mass whose workup revealed a sporadic clear cell renal cell carcinoma (RCC) (Panels J–M). This phenotype—acute heart failure/acute coronary syndrome mimic, reversible LV dysfunction—suggests a stress cardiomyopathy. Recent research links catecholamine-mediated stunning to a protective anti-apoptotic transitory change in the β -adrenoceptor signalling pathway, resulting in a negative inotropic response. A higher density of β 2-adrenoceptors at the apex results in its increased sensitivity to circulating hormones. A transitory catecholamine-mediated myocardial apical stunning was likely triggered by the tumour, known to induce several types of paraneoplastic syndromes, frequently the initial presentation. Association with malignancies has been described, resulting from paraneoplastic phenomena with changes in adrenoceptor sensitivity/signalling, even lower psychic threshold for stress, but to the best of the authors' knowledge, this is the first report in a patient with RCC.

Figure 1. Rapidly evolving electrocardiographic ST-T segment changes on precordial leads (Panels A–C). Left ventriculography depicting typical apical akinesia and hypercontractile basal myocardium (Panels D and E). No apical ballooning on coherent gradient echo sequences of the left ventricle long-axis in systole and diastole (Panels F and G), oedema/inflammation (Panel H, short-tau inversion recovery sequence), fibrosis, or infarction (Panel I, late gadolinium enhancement sequence) seen on cardiovascular magnetic resonance. The recovery of wall motion is suggestive of Tako-Tsubo cardiomyopathy. Abdominal magnetic resonance showed a solid mass in the right kidney, disrupting the reniform shape, hypointense on T1-weighted images (Panel J), heterogeneously hyperintense on T2-weighted images with (not shown) and without fat suppression (Panel K), and enhancing following contrast administration (Panel L), findings suggestive of renal carcinoma. Nephrectomy histopathological examination confirmed the presence of typical clear cells (Panel M) of renal cell carcinoma, due to abundant lipid and glycogen in its cytoplasm.

Supplementary data are available at *European Heart Journal – Cardiovascular Imaging* online.

