Acute cocaine myocarditis: a word of caution

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An otherwise healthy 25-year-old male presented to the Emergency Department with new onset severe retrosternal chest pain. Electrocardiogram demonstrated lateral ST-segment elevations in leads I and AVL with inferior ST-segment depression (Panel A). Initial troponin I level was 1.8 ng/mL quickly rising to >200 ng/mL within first 8 h. Coronary angiography revealed normal coronary arteries (see Supplementary material online, Video S1). Echocardiogram showed regional wall motion abnormalities involving inferolateral segment with moderately reduced left ventricular ejection fraction (LVEF) (see Supplementary material online, Video S2). He had episodes of non-sustained ventricular tachycardia on telemetry. Urine toxicology screen was positive for cocaine and marijuana.

Cardiac MRI (CMR) study obtained within 24 h of presentation revealed moderately reduced systolic function (LVEF = 42%) with regional wall motion abnormalities and diffuse circumferential increased T2-signal in the midwall and epicardial regions corresponding to the areas of acute oedema/inflammation seen on pre-contrast T2 mapping (Panel B). Late gadolinium-enhancement (LGE) images revealed similar circumferential midwall and epicardial ring enhancement suggesting acute myocarditis with substantial myocardial necrosis (Panel C). Native heart biopsy was declined by the patient and family. The patient was treated for cocaine-induced myocarditis with ACE-inhibitors, beta-blockers, and statin therapy with resolution of symptoms, arrhythmias, and normalization of biomarkers. A 30-day follow-up CMR study demonstrated less prominent circumferential LGE suggesting remarkable transformation of necrosis to fibrosis (Panel D), with interval adverse LV remodelling and persistent LV dysfunction (Panel E) (Supplementary material online, Video S3). Of note, despite resolution of the ST changes, residual QRS fragmentation was observed corresponding to the transformation of myocardial necrosis into fibrosis as demonstrated in the 30-day follow-up CMR study. (Panel F).

Temporal changes associated with acute cocaine cardiotoxicity are not well delineated in the literature. In a series of 40 autopsies with cocaine-associated deaths, Virmani et al. (1988) demonstrated that myocarditis with mononuclear infiltrate was nearly 10 times more common than acute thrombotic coronary occlusion. This case illustrates such presentation of acute cocaine-induced myocarditis with aggressive myocardial necrosis and unfavourable cardiac remodelling clearly demonstrated by serial CMR studies. In the absence of coronary artery disease, the CMR study should be considered in symptomatic patients following acute cocaine use and positive biomarkers to assess the extent of myocardial injury that could be prognostically important.

Supplementary material is available at European Heart Journal online.