



Letter to the Editor

‘Stoned’ people can get stunned myocardium: A case of heroin withdrawal precipitating Tako-Tsubo cardiomyopathy

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ARTICLE INFO

Article history:

Received 6 July 2013

Accepted 13 July 2013

Available online 6 August 2013

Keywords:

Tako-Tsubo cardiomyopathy

Myocardial stunning

Broken heart syndrome

Heroin withdrawal

Apical ballooning syndrome

The patient is a 40 year old male with history of heavy inhaled heroin and tobacco use for >30 years who came to our detoxification unit with symptoms of nausea, vomiting and nervousness. He was started on hydroxyzine, zolpidem, nicotine patch and tapering doses of methadone. On the day following his admission he developed sudden onset palpitations. He denied chest pain, dyspnea, dizziness, abdominal pain or diaphoresis. Physical examination revealed a blood pressure of 121/78 mm Hg, heart rate of 114 bpm, respiratory rate of 20/min and oxygen saturation of 95% on room air. Cardiac auscultation disclosed normal heart sounds, no JVD, normal lung exam and no peripheral edema. EKG (*Fig. 1 bottom image*) showed a sinus tachycardia at 127 bpm, left axis deviation, T wave inversion in leads I, aVL, II, III, aVF and V3–V6 and Left Anterior Fascicular Block. These changes were new compared to the EKG performed on admission (*Fig. 1 top image*). Laboratory data showed CK 489 U/L, CKMB 11 ng/mL and an index of 2.4%, Troponin I 4.7 ng/mL; Na 131 mmol/L, K 4.3 mmol/L, Cl 91 mmol/L, BUN 28 mg/dL, Cr 0.9 mg/dL; AST 49 U/L, ALT 34 U/L, ALP 81 U/L, Total Bilirubin 1.67 mg/dL, Albumin 4.5 g/dL; WBC 9.55 K/ μ L, Hgb 13.5 g/dL, Hct 36.6%, Platelets 212,000 K/ μ L. Pro-BNP was markedly elevated at 28,638 pg/mL. Urine toxicology was positive for opiates, methadone and cannabinoids. Chest X-ray was unremarkable. He was immediately transferred to the Coronary Care Unit with a diagnosis

of NSTEMI and given Clopidogrel, Aspirin, Rosuvastatin and started on Heparin drip. A 2D-Echocardiogram showed severely reduced LVEF <25%, mid-infero-septal, apical septal, mid-inferolateral walls hypokinetic and mid-anterior wall akinetic (*Fig. 2*). He was transferred urgently for left cardiac catheterization which showed normal coronary arteries and severely reduced left ventricular systolic function. Findings were thus consistent with Tako-Tsubo cardiomyopathy. He was started on Metoprolol, Lisinopril and Rosuvastatin and Aspirin were continued. Troponin I trended down rapidly from 4.7 ng/mL to 0.74 ng/mL. TSH, Glucose and Iron levels were normal. His medical treatment was optimized and his symptoms improved. Repeat 2D-Echocardiogram 12 days later demonstrated normal left ventricular function >55% without any wall motion abnormalities (*Fig. 2*).

Tako-Tsubo cardiomyopathy (TCM) refers to transient acute heart failure with clinical presentation similar to acute myocardial infarction (AMI). It occurs in 1% to 2% of patients presenting with AMI [1]. Other terminologies used include broken heart syndrome, apical ballooning syndrome, stress cardiomyopathy and neurogenic stunning. It was first described by Sato et al. in 1991 [2]. Tako-Tsubo refers to the trap used by Japanese Fishermen to capture octopuses. Modified Mayo Clinic diagnostic criteria for TCM include (a) Transient hypokinesis, akinesis or dyskinesis in left ventricular mid-segments with or without apical involvement, (b) Regional wall motion abnormalities extending beyond a single epicardial vascular distribution; frequently associated with a stressful trigger, (c) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture, (d) New EKG abnormalities (ST segment elevation and/or T wave inversion) or modest elevation in cardiac troponin and (e) Absence of Pheochromocytoma and myocarditis. All 4 criteria must be met [3].

Ninety percent of all cases have occurred in women 60–75 years of age. Most patients had a preceding physical or emotional trigger. Clinical features include acute sub-sternal chest pain (most common) followed by dyspnea, and less commonly syncope and palpitations. Common EKG findings include precordial ST elevation and T wave inversions. Inverted Tako-Tsubo has been described with hypokinesis involving base of the heart with apical sparing [4]. Fröhlich et al. demonstrated the value of NT-proBNP/Troponin T ratio in differentiating TCM from AMI [5]. Cardiac MRI may help to differentiate TCM from AMI by showing absence of delayed gadolinium hyper-enhancement in cases of TCM [6]. However cardiac catheterization remains the gold standard for diagnosis. Our patient's symptoms suggested Acute Coronary Syndrome but he had normal coronaries during cardiac catheterization. He was a heavy heroin user of >15–

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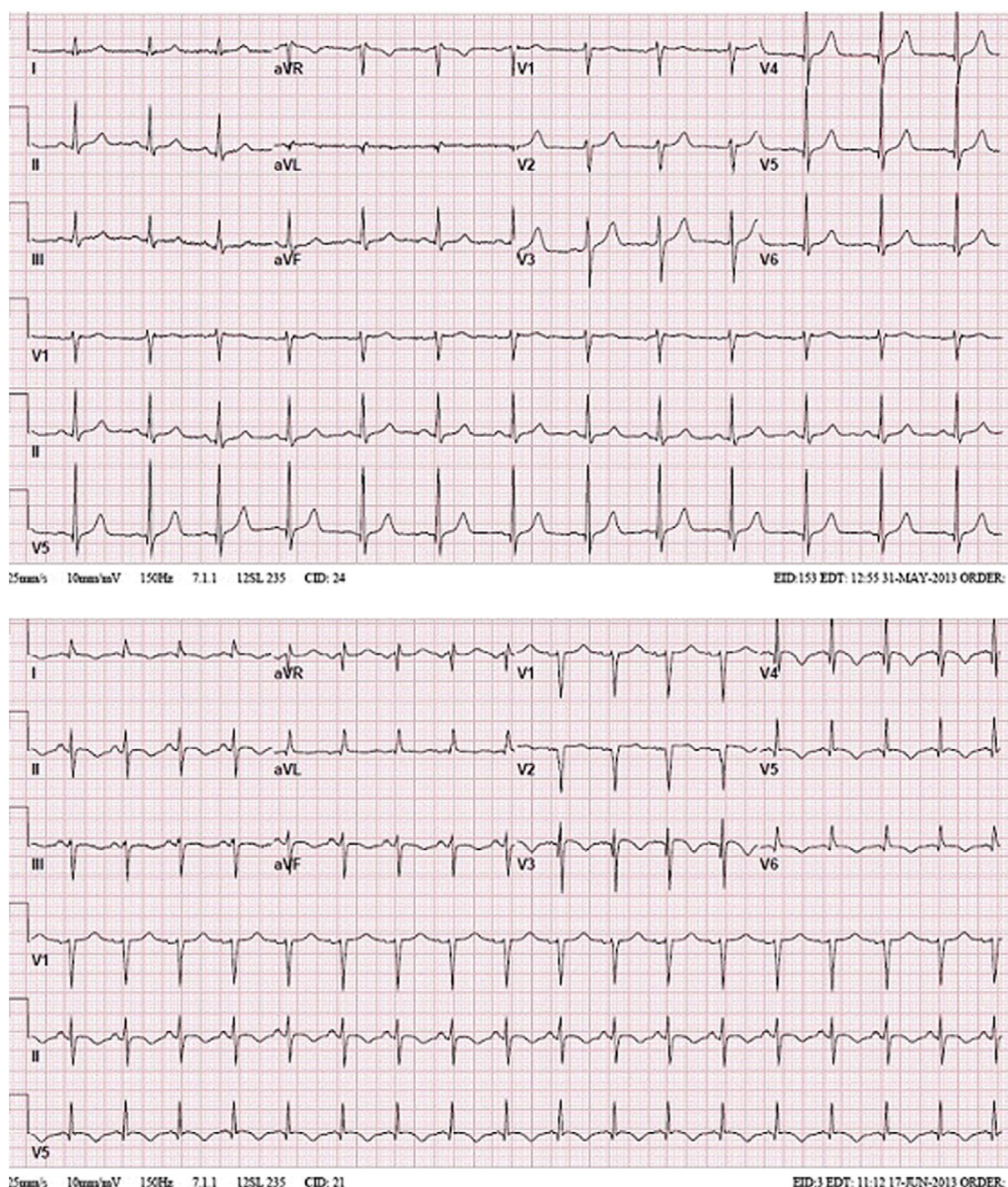


Fig. 1. 1. Normal ECG. 2. Sinus tachycardia at 127 bpm, left axis deviation and T wave inversion in leads I, aVL, II, III, aVF and V3–V6, Left Anterior Fascicular Block.

20 bags a day. The abrupt interruption of this high dose of opiates (approx. 100 mg of heroin/bag) put the patient's myocardium in severe stress likely secondary to an adrenergic surge. Researchers have observed regional differences in β -adrenergic receptor density in the apex and base of the left ventricle which may account for the ballooning pattern observed in TCM [3].

Prognosis is generally good with spontaneous recovery in most patients within a few weeks. Complication rates are around 20% and include left ventricular failure, pulmonary edema, cardiogenic shock, mural thrombus, arrhythmias, ventricular rupture, and death

[6]. Beta-blockers and angiotensin-converting enzyme inhibitors are the drugs most commonly used to treat Tako-Tsubo cardiomyopathy. In-hospital mortality rate is <2%, with recurrence rate of 10% [6].

In summary, only three cases of opiate withdrawal associated TCM have been described previously. In our opinion this is the first case of heroin withdrawal (while on methadone maintenance) induced stress cardiomyopathy. The development of acute heart failure in an otherwise healthy young man with a history of opiate abuse should raise the suspicion of stress cardiomyopathy as part of the differential

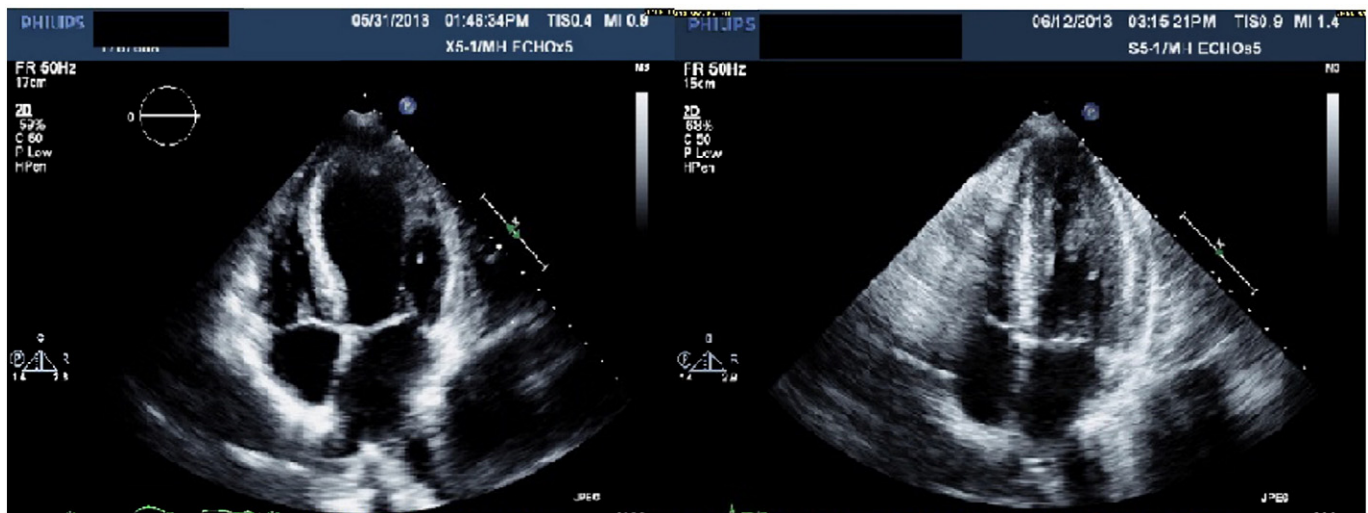


Fig. 2. 2D-Echocardiogram snapshots of a Four Chamber View during Systole demonstrating severely reduced LV function, an akinetic apex, hypokinesis of mid-infero lateral and mid-antero lateral walls (left) compared to a repeat study 12 days after demonstrating recovered and normal LV function without wall motion abnormalities (right).

diagnosis. Early diagnostic and therapeutic intervention with standard treatment for heart failure portends a good prognosis.

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