Stress Cardiomyopathy due to Misuse of Transdermal Fentanyl Patches in an Elderly Patient

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Stress cardiomyopathy is characterized by transient systolic dysfunction of the apical and/or mid segment of the left ventricle. The main pathophysiology of stress cardiomyopathy is the excessive release of catecholamine. Opioid withdrawal can initiate a surge of catecholamine and an attack of stress cardiomyopathy. In this case, we report a case of stress cardiomyopathy due to iatrogenic withdrawal from transdermal fentanyl.

Key Words: Stress cardiomyopathy, Takotsubo cardiomyopathy, Fentanyl

INTRODUCTION

Stress cardiomyopathy, also called Takotsubo cardiomyopathy, is an increasingly reported syndrome generally characterized by transient systolic dysfunction of the apical and/or mid segment of the left ventricle and electrocardiographic changes that mimics acute myocardial infarction, with no evidence of obstruction in the epicardial coronary arteries1,2). The exact pathophysiology of this disorder is not well understood. However, the role of the excessive release of catecholamines has been demonstrated3).

Here, we report a case of stress cardiomyopathy secondary to acute fentanyl withdrawal.

CASE REPORT

A 65-year-old man presented at the Emergency Department in a comatose state with respiratory depression. His wife stated that, when she awoke in the morning, she found him lethargically lying on the bed. At the initial evaluation, the patient’s blood pressure was 120/80 mmHg, his heart rate was 100 bpm, and his body temperature was 36.4°C. He had miotic (pin-point) pupils and we detected 10 fentanyl transdermal patches (12 μg/dL) attached on his body. He had been prescribed fentanyl transdermal patches the day before due to knee pain. We assumed that the patient had mistaken fentanyl transdermal patches for simple pain relief patches and he had attached all the prescribed fentanyl patches to his body.

He was diagnosed with fentanyl intoxication. Therefore, all fentanyl transdermal patches were detached from his body. As the patient was in a comatose state with respiratory depression, a naloxone (known antidote of opiate overdose) infusion was started. His mental status improved after he received naloxone.

In 8 hours, he complained chest discomfort and dyspnea, and electrocardiography (ECG) showed a deep T-wave inversion and QT prolongation in the anterolateral leads compared to the initial ECG (Fig. 1). Laboratory investigations revealed elevated cardiac enzymes (creatinine kinase-myoglobin of 50.0 ng/mL, troponin I level of 2.10 ng/dL).

Furthermore, echocardiography revealed a decreased left ventricle (LV) systolic function (Ejection fraction (EF)=46%) with apical akinesia with ballooning of the LV and hyperkinesia of the mid-to-basal segment of LV (Fig. 2A). A diagnosis of
stress cardiomyopathy resulting from an acute fentanyl withdrawal reaction secondary to naloxone treatment was made. The patient was transferred to the cardiac care unit and received intensive therapy including diuretics, angiotensin converting enzyme inhibitors, and beta-blockers. He underwent a cardiac computed tomography (CT) to evaluate him for epicardial coronary artery stenosis. The cardiac CT showed nonsignificant coronary artery stenosis (Fig. 3). In seven days, his echocardiography normalized (Fig. 2B) and his ECG showed a residual anterior T-wave inversion. The patient’s condition stabilized and he was discharged after ten days in the hospital.

**DISCUSSION**

Stress cardiomyopathy is characterized by transient, regional systolic dysfunction of the left ventricle without obstructive epicardial coronary artery disease. It predominantly affects postmenopausal women and is frequently, though not always, triggered by an acute medical illness or an intense emotional or physical stress. The exact pathophysiology of stress cardiomyopathy is not well understood yet. The main hypothesis is that an abrupt increase in the level of serum catecholamines secondary to a stressful event may cause either microvascular coronary spasm or direct myocardial toxicity and initiate a reversible myocardial inflammation and dysfunction.

The sympathetic overstimulation can be caused by phaeochromocytoma, exogenous sympathomimetics, or the withdrawal of sympathetic antagonists, such as opioids. Fentanyl is potent, synthetic opioid analgesic, approximately 50–100

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**Fig. 1.** Electrocardiogram (ECG) revealed deep T inversion in lead V1-6 (A) compared to the initial ECG (B).

**Fig. 2.** (A) Echocardiography showed decreased left ventricle (LV) systolic function (EF=46%) with apical akinesia with ballooning of the LV and hyperkinesia of the mid-to-basal segment of the LV. (B) Follow-up echocardiography at seven days later revealed normal LV systolic function (EF=60%) without regional wall motion abnormality. EF, Ejection fraction.

**Fig. 3.** Cardiac computed tomography showed nonsignificant coronary artery stenosis.
times more potent than morphine on a dose-by-dose basis. Delivered via transdermal patches, fentanyl is the most widely used synthetic opioid in clinical practice.

Drug withdrawal can be associated with a hyperadrenergic state and central nervous system irritability, which may provide a pathophysiologic mechanism for withdrawal-associated stress cardiomyopathy.

In our case, the use of naloxone for the management of opioid intoxication from misuse of fentanyl patches triggered the opioid withdrawal.

Although the association between opioid withdrawal and stress-induced cardiomyopathy has already been reported \(^{11,12}\), to our knowledge, this is the first case of stress cardiomyopathy described after abrupt fentanyl transdermal patch withdrawal secondary to a drug-drug interaction.

With the ongoing increase in fentanyl transdermal patches prescription in old age, despite their potential benefits, inappropriate or incorrect use of fentanyl patches can lead to significant patient harm \(^{13}\).

Opioid withdrawal may trigger stress cardiomyopathy, therefore, this potential complication should be considered when managing opioid withdrawal patients.

Conflict of Interest Disclosures

The researchers claim no conflicts of interest.

REFERENCES


