

INTRODUCTION

- Opioid withdrawal has been known to cause major adverse cardiovascular events resulting in hemodynamic, vascular, and proarrhythmic consequences².
- However, opioid withdrawal has rarely been associated with stress-induced cardiomyopathy³.
- We discuss a case of a gentleman with a history of opioid misuse who presented with reversible acute myocardial dysfunction as well as stress-induced cardiomyopathy.

CASE PRESENTATION

- A middle-aged male with a medical history of intravenous fentanyl use presented to the emergency department (ED) from prison for shortness of breath and chest pain.
- Vital signs: afebrile, blood pressure of 168/110 mmHg, heart rate of 156 beats per minute, respiratory rate of 40 breaths per minute, oxygen saturation of 92% on room air
- Physical exam: ill-appearing, diaphoretic, tachycardic, tachypneic male in respiratory distress with diffuse coarse rhonchi noted
- Electrocardiogram (EKG) revealed diffuse ST-segment changes. This was discussed with cardiology, and it did not meet criteria for ST-elevation myocardial infarction (Figure 1A).
- Computed tomography angiography (CTA) demonstrated pulmonary edema without evidence of pulmonary embolism.
- Laboratory findings showed elevated troponins, pro-BNP, lactic acid, and white blood count.
- ED course was complicated by hypoxic respiratory failure requiring intubation after failed noninvasive positive pressure ventilation due to cardiogenic pulmonary edema.
- Due to diffusely abnormal EKG and unstable vital signs, interventional cardiology took patient for urgent cardiac catheterization. Coronary angiography showed no evidence of coronary artery disease but revealed severely depressed left ventricular (LV) function with an estimated ejection fraction of 5%. These findings were concerning for stress-induced cardiomyopathy (Figure 2).
- The patient was admitted to the Intensive Care Unit (ICU) for further management.
- While in the ICU central pulmonary artery catheter readings confirmed decreased cardiac index and output, and milrinone infusion was started for cardiogenic shock.
- Advanced heart failure team was consulted, and endocrinology ruled out thyroid-related causes for tachycardia-mediated cardiomyopathy.

Figure 1A: EKG on ED arrival

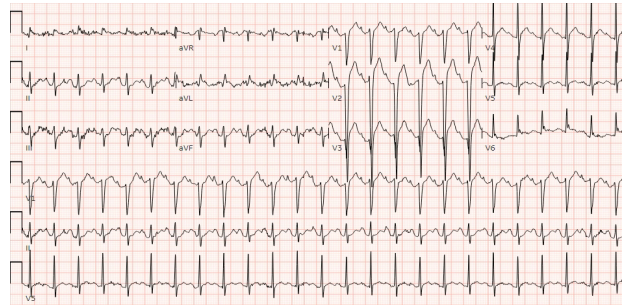


Figure 1B: EKG on outpatient follow up

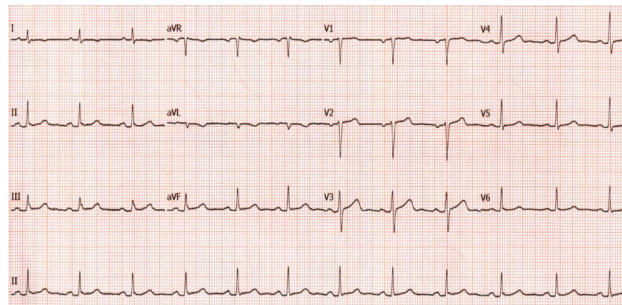
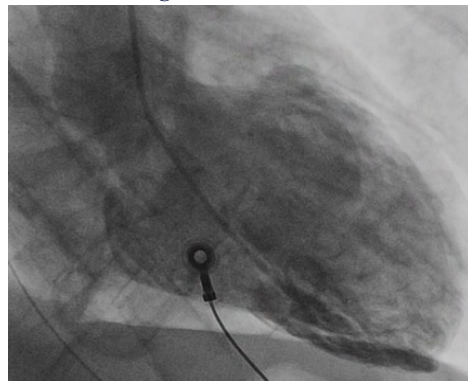


Figure 2: coronary angiography demonstrating left ventricular apical ballooning during systole also known as “takotsubo” configuration



CASE PRESENTATION (CONT.)

- Following aggressive diuresis and weaning of milrinone, the patient was successfully extubated and downgraded to hospital medicine service six days after admission.
- Patient tolerated guideline-directed medical therapy for heart failure with metoprolol, enalapril, and spironolactone.
- Patient was discharged back to his correctional facility with a wearable external cardioverter defibrillator.
- Once month later, patient followed up with outpatient comprehensive heart failure service with improved exertional dyspnea.
- Six months later a repeat echocardiogram showed normal LV function. A repeat EKG was also obtained as shown in Figure 1B.

DISCUSSION

- Increased catecholaminergic tone is a known trigger for stress-induced LV dysfunction.
- Per current literature, opioid withdrawal is known to cause a catecholamine surge, but this is rarely associated with stress-induced cardiomyopathy⁴.
- These patients are at high risk for ventricular arrhythmias, respiratory failure due to volume overload, and hemodynamic instability due to abrupt increase in myocardial oxygen consumption as exemplified in our case.

CONCLUSION

- Opioid misuse remains an epidemic with many challenges.
- This case highlights the importance of recognizing cardiovascular complications of opioid misuse including prompt screening and identification of stress-induced cardiomyopathy to facilitate emergent resuscitation and management^{1,2,4}.

REFERENCES

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