Georgian Scientists
ქართველი მეცნიერები
Vol. 8 Issue 4(1), 2026
https://doi.org/10.52340/gs.2026.08.04(1).11



TOXIC CARDIOMYOPATHY AND THE SPECTRUM OF CARDIOTOXICITY IN SEVERE POISONING: A CASE STUDY

Eka Kurdadze[1,2], Ninia Intskirveli[2]

[1]Anesthesiology, Reanimatology and Toxicology Department, TSMU, Tbilisi, Georgia.
[2]First University Clinic, TSMU, Tbilisi, Georgia

Background. Poisoning can injure the heart through direct myocyte toxicity, rhythm and conduction disturbances, vasodilation and hypoxic-ischemic damage. Troponin elevation with ventricular dysfunction is easily mistaken for acute coronary syndrome.

Case presentation. A 19-year-old man was transferred from another hospital, intubated and on inotropic support for shock, after loss of consciousness following use of an unknown narcotic; a urine immunoassay there was positive for methadone. Troponin I had risen from 0.94 to 1.6 ng/mL there and was 6.49 ng/mL (normal ≤0.03) in our laboratory. He was referred for coronary angiography. On arrival, BP was 122/79 mmHg on norepinephrine (0.3 μg/kg/min), heart rate 53/min with sinus bradycardia and no acute focal ECG changes, pupils were miotic, potassium 3.08 mmol/L, lactate 1.36 mmol/L and CRP <5 mg/L. Chest radiograph showed perihilar infiltrates and coronary angiography showed intact arteries. First echocardiography showed ejection fraction (EF) 35%, diffuse hypokinesia, impaired relaxation and mild pulmonary hypertension (PASP 35 mmHg).

Management and course. Mechanical ventilation, infusion therapy and potassium correction continued; norepinephrine was tapered and stopped, and he was extubated on day 3. A second echocardiography 16 hours after the first showed EF 55% (58% on day 3), and troponin I was 0.03 ng/mL by day 6. Antibacterial therapy was added for rising inflammatory markers (CRP 167 mg/L on day 4, 9 mg/L on day 8). A repeat cardiac marker was negative during chest pain and anxiety on day 4. He was discharged on day 8, hemodynamically stable.

Discussion. Intact coronary arteries and absent ST changes excluded acute coronary syndrome; troponin elevation with rapidly reversible left ventricular depression indicated toxic cardiomyopathy. Likely mechanisms are direct methadone effects on myocardial ion channels, hypoxia from respiratory depression, and catecholamine or inotrope exposure; recovery of EF within 16 hours suggests myocardial stunning rather than necrosis, although changing loading

conditions and inter-study variability may contribute. Methadone cardiotoxicity spans bradycardia, QT prolongation and arrhythmia, reversible cardiomyopathy and cardiac arrest, whereas calcium channel blocker poisoning typically causes vasodilatory shock with preserved EF; echocardiography helps separate these patterns.

Conclusion. In poisoning with troponin elevation and new ventricular dysfunction, early coronary angiography and serial echocardiography distinguish toxic cardiomyopathy from acute coronary syndrome and guide supportive care, which allows rapid recovery of ventricular function.

Keywords: toxic cardiomyopathy; methadone; cardiotoxicity; troponin; poisoning.