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METHADONE POISONING IN GEORGIA: NEUROTOXICITY AND CARDIOTOXICITY, CLINICAL FEATURES OF MULTIPLE ORGAN FAILURE

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Background. Methadone toxicity is dominated by respiratory depression, but cardiac, neurological and multiorgan injury are underrecognized, especially with stimulant co-use.

Case presentation. A 19-year-old man was found unconscious at home after reportedly using "crystalline methadone" and mephedrone (dose and timing unknown; no analytical confirmation). On arrival: GCS 3, miosis, respiratory rate 4–5/min, SpO2 80%, BP 85/55 mmHg, heart rate 126/min, temperature 35.6 °C. He was intubated; asystole occurred 9 minutes later, and circulation returned after 10 minutes of resuscitation. ECG showed sinus tachycardia (145/min). Tests showed pH 7.23, lactate 8.3 mmol/L, potassium 6.16 mmol/L, creatinine 208.7 μmol/L, creatine kinase 2250 U/L, AST 666 U/L and ALT 573 U/L. Echocardiography revealed diffuse hypokinesia with ejection fraction (EF) 18%; head CT was normal. Right cheek edema and left forearm hyperemia suggested prolonged-pressure injury.

Management and course. He required maximal doses of norepinephrine, phenylephrine and dobutamine, hydrocortisone, massive infusion therapy, potassium-binding therapy and acidosis correction. Vasopressors were weaned and he was extubated on day 3. Consciousness returned by day 2, with left-sided weakness and agitation, and emotional lability later. EF rose to 27% on day 2 and 55% on day 4; creatinine fell to 89.6 μmol/L by day 3. AST 3049 and ALT 4195 U/L on day 4 declined by day 6; platelets fell to 75×10^9/L before recovering. Dark urine and dense facial and left-arm swelling appeared by day 5. He was discharged on day 7 with residual facial swelling and limited left-arm joint movement.

Discussion. Coma, miosis and respiratory failure reflected opioid toxicity, while tachycardia (partly reflecting vasoactive support) may indicate a sympathomimetic (mephedrone) contribution. Arrest likely resulted from hypoxia, acidosis and hyperkalemia; rapidly reversible myocardial depression (EF 18% on day 1, 55% by day 4) fits toxic and hypoxic-ischemic cardiomyopathy. Organ dysfunction was multifactorial: muscle injury from prolonged

compression, shock and hypoxia likely explain the quickly resolving kidney injury, while the delayed transaminase peak suggests ischemic hepatitis with possible additional toxic injury. Transient left-sided weakness with normal head CT may reflect hypoxic-ischemic or compression injury.

Conclusion. Poisoning with methadone, particularly with stimulant co-use, can cause cardiac arrest, severe reversible myocardial dysfunction and multiorgan failure. Early airway control, serial echocardiography and monitoring of kidney, liver and muscle injury support good recovery.

Keywords: methadone; neurotoxicity; cardiotoxicity; cardiac arrest; multiple organ failure.