

Lactic Acidosis Secondary to Metformin Toxicity: A Case Report

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Background

Metformin is an oral antidiabetic drug in the biguanide class. It is the first-line drug of choice for the treatment of type 2 diabetes. The most common symptoms following overdose appear to include vomiting, diarrhea, abdominal pain, tachycardia, drowsiness, and, rarely, hypoglycemia. The major potentially life-threatening complication of Metformin overdose is metabolic acidosis. We report a case of fatal Metformin toxicity following an acute intentional Metformin intoxication

Cae Presentation

18 years 18-year-old female presented to the emergency department three hours after a suicide attempt with 30 grams of metformin, severely agitated, confused, with severe abdominal pain associated with persistent vomiting. Pulse: 120 / minute, BP: 80/60mmHg, Temperature was 37.0°C. and a respiratory rate of 28. The cardio-respiratory system was normal; her abdomen was soft and non-tender. Arterial blood gas analysis on admission showed a profound lactic acidosis: pH 6.8 (7.38 to 7.42); pCO₂:27 (35 45MmHg), pO₂:99, bicarbonate 4.2 (22-28 mmol/L) and lactate >15 (<1mmol/l)

Blood test results at presentation: HB: 11.2g, platelets 131 (150-450), Creatinine 1.8mg (0.74 to 1.35). Her family members took her to the toxicology emergency unit where gastric lavage was done, oral charcoal and intravenous fluids were given

During her ICU stay, Day 1: The patient had severe lactic acidosis, for which she received sodium bicarbonate (200 ml) infusion. She was

resuscitated by intravenous fluids (Ringer's acetate) and put on noradrenaline with no response, so adrenaline was added.

Day 2, Lactic acidosis was persistent and severe, so she received one hemodialysis session after a nephrology consultation.

Day 3, The patient was off adrenaline, but withdrawal of nor-adrenaline failed, so dobutamine infusion was added, supported by the data detected in echocardiography: EF 20%, akinetic whole septum, hypokinetic other whole segments, sparing apical segments (apical segments contract better than basal segments).

Day 4, the patient developed one attack of convulsions, generalized tonic seizures, followed by post-ictal confusion. Neurology consultation revealed an unremarkable MRI with diffusion and EEG. levetiracetam 500 was given twice daily.

Day 5: The patient was off nor-adrenaline, then off dobutamine with intact peripheral pulsations and normal blood pressure. Echocardiography was normal. EF 60%

Discussion

Metformin toxicity, though rare, can be life-threatening due to severe lactic acidosis. In this case, an 18-year-old female who ingested a large dose of metformin developed profound acidosis, shock, cardiac dysfunction, seizures, and gastrointestinal bleeding.

Prompt management including fluids, vasopressors, sodium bicarbonate, and hemodialysis, led to full recovery. The case highlights the need for early

recognition, supportive care, and timely dialysis in managing severe metformin overdose.

The rapid improvement in cardiac function and acid-base status following dialysis highlights the importance of extracorporeal toxin removal in cases of severe MALA. Although her initial presentation was critical, timely intensive care management resulted in a full recovery.

Conclusions

This case illustrates the potentially fatal nature of acute metformin overdose, particularly due to severe lactic acidosis.

Early recognition, hemodynamic support, correction of acidosis, and timely initiation of hemodialysis are key to improving outcomes.

Clinicians must remain vigilant for cardiac, neurologic, and gastrointestinal complications. Multidisciplinary management, including critical care, nephrology, and neurology teams, is often essential in guiding recovery.

Keywords

Lactic acidosis, Metformin-associated lactic acidosis (MALA), Hemodialysis, Critical care management, Extracorporeal toxin removal