International Journal of Research in Medical Sciences
Nazir I et al. Int J Res Med Sci. 2026 Mar;14(3):1167-1170
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20260639

Case Report

A case report on amphetamine-induced acute pancreatitis with concurrent rhabdomyolysis, acute renal failure and severe hyperkalemia requiring hemodialysis

Imran Nazir[1]*, Hanan M. M. Abdullah[2], Enas Abdulfattah I. Sindy[1], Ehdaa K. Boudal[1], Abdul Rahman A. Alwagdani[3], Ahmed Ramadan M. Elsaghir[3], Samaher H. S. Al Harbi[1], Haiqa Imran[4], Tareq Elsayed M. Abouelregal[3], Sayed S. Rahman[3]

[1]Department of Internal Medicine, SFHM, Makkah, KSA
[2]Department of Internal Medicine and ID, SFHM, Makkah, KSA
[3]Department of Nephrology, SFHM, Makkah, KSA
[4]Rahbar Medical College, Lahore, Pakistan

Received: 08 September 2025
Revised: 20 February 2026
Accepted: 21 February 2026

***Correspondence:**
Dr. Imran Nazir,
E-mail: imrannazir40@gmail.com

ABSTRACT

Amphetamine toxicity is increasingly linked to complications affecting multiple organ systems, such as rhabdomyolysis, acute kidney injury (AKI), and severe metabolic issues, such as hyperkalemia and high anion gap metabolic acidosis (HAGMA). Although acute pancreatitis due to amphetamines is rare, it should be considered in patients with abdominal symptoms and recent stimulant use. A 33-year-old healthy man presented with a week of epigastric pain, nausea, and vomiting. The patient presented with tachycardia, mild hypotension, and epigastric tenderness. Laboratory tests showed severe hyperkalemia (7.7 mmol/l), metabolic acidosis (pH 7.25, HCO_3^- 13.5 mmol/l), elevated creatinine (15.46 mg/dl), lactate (2.6 mmol/l), high CK (25,995 IU/l), and lipase (1,528 U/l). Amphetamines were detected by toxicology screening, and imaging showed interstitial edematous pancreatitis. Initial treatment failed to correct the metabolic issues; however, emergency dialysis led to rapid improvement. He underwent three dialysis sessions, recovered kidney function, and was discharged on day 7 with a follow-up appointment. This case highlights a rare but severe form of amphetamine toxicity, causing acute pancreatitis, rhabdomyolysis, and AKI with life-threatening hyperkalemia. Although amphetamines are not dialyzable, hemodialysis can effectively treat complications such as refractory hyperkalemia and acidosis. Clinicians should be alert to multisystem involvement and recognize that dialysis may be life-saving when standard treatments fail, even if toxin removal is not the main objective of treatment.

Keywords: Amphetamines, Acute pancreatitis, Rhabdomyolysis, Renal failure

INTRODUCTION

Amphetamine toxicity manifests as excessive stimulation of the sympathetic nervous system, resulting in vasoconstriction, elevated body temperature, and muscle damage.[1] There has been a growing number of reports on secondary issues, such as rhabdomyolysis and acute kidney injury (AKI).[2] High anion gap metabolic acidosis (HAGMA), particularly lactic acidosis, is common among critically ill patients and is associated with high mortality rates.[3] HAGMA occurs due to the buildup of unmeasured anions such as lactate, ketones, and certain toxins.[4] Severe hyperkalemia is an urgent, life-threatening condition due to its potential to trigger arrhythmias, requiring swift and standardized treatment protocols.[5] Hemodialysis (HD) becomes necessary when medical management fails or in

cases of kidney failure.[6] Amphetamine toxicity is characterized by a wide range of symptoms, including hyperthermia, agitation, vasoconstriction, and ischemia.[1] Although uncommon and primarily supported by limited case studies and animal research, amphetamine-type stimulants such as methamphetamine and MDMA (3,4-methylenedioxy-methamphetamine) have been associated with acute pancreatitis, potentially due to ischemia from severe vasoconstriction and catecholaminergic effects.[7-12] We report a case of simultaneous pancreatitis, HAGMA, and severe hyperkalemia following amphetamine use, which rapidly improved after HD.

Amphetamine dose and patterns of use leading to multisystem toxicity

Amphetamine-related issues, as illustrated in this case, such as acute pancreatitis, rhabdomyolysis, acute kidney failure, and severe hyperkalemia, are affected by both the dosage and usage pattern. Acute toxicity generally arises from the consumption of high or excessive doses, whether for recreational purposes or due to overdose, resulting in intense sympathetic activation. Therapeutic amphetamine doses range from 5 to 60 mg/day (e.g., for ADHD treatment), but recreational users often ingest much larger amounts, sometimes surpassing 100–250 mg in a single session, especially with methamphetamine or MDMA.[13,14] Patterns of binge use can further increase systemic catecholamine levels, resulting in heightened physiological reactions within the body. Long-term amphetamine consumption leads to cumulative damage to blood vessels and organs due to sustained vasoconstriction, excess catecholamines, and oxidative stress.[15] In cases of acute high-dose exposure, severe vasoconstriction and hyperthermia can cause tissue ischemia and muscle toxicity, triggering rhabdomyolysis and subsequent acute kidney injury.[16] These downstream effects are often exacerbated by severe metabolic disturbances, such as high anion gap metabolic acidosis and potentially fatal hyperkalemia.[3,4] In uncommon instances, the acute use of amphetamines has been linked to pancreatitis, possibly due to ischemic or catecholaminergic damage to the small blood vessels in the pancreas; nonetheless, the precise pathophysiological process remains unclear.[7-12] The seriousness of these issues may require renal replacement therapy, especially when standard treatments are unable to resolve severe electrolyte imbalances and kidney dysfunction.[6,17]

CASE REPORT

A 33-year-old man with no previous medical history presented to the hospital with intense pain in the upper abdomen that extended to his back, along with nausea and frequent vomiting for the previous week. The patient reported no recent alcohol intake, no history of gallstones, and no new medication. The details of his medication history were unclear during the emergency department assessment, but he later accepted amphetamine abuse. Upon arrival, his vital signs were recorded as follows: blood pressure, 124/58 mmHg; heart rate, 118 bpm; respiratory rate, 22 breaths/min; oxygen saturation, 96% while breathing room air; and temperature, 37.0 °C. The patient was sweating and had tenderness in the epigastric region without any guarding. Found to have left thigh swelling and tenderness without skin changes (Figure 1).

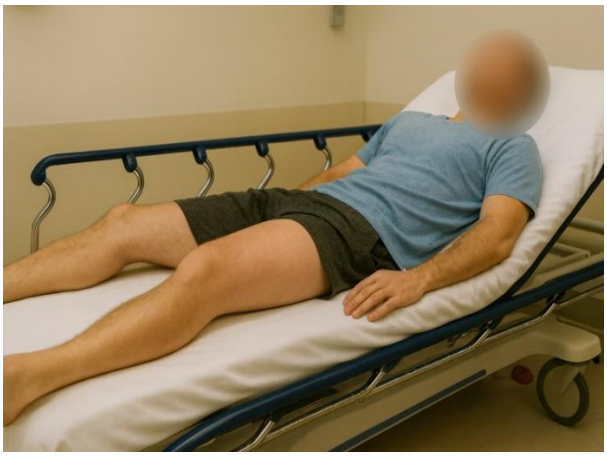

Figure 1: Left thigh swelling in a patient with amphetamine-induced rhabdomyolysis.

All limb pulses were palpable. The other systemic review was unremarkable. Laboratory tests showed the following results: K^+ at 7.7 mmol/l (normal: 3.5–5.0 mmol/l), Na^+ at 121 mmol/l (normal: 135–145 mmol/l), Cl^- at 88 mmol/l (normal: 98–106 mmol/l), HCO_3^- at 13.5 mmol/l (normal: 22–28 mmol/l), anion gap at 19.5 mmol/l (normal: 8–12 mmol/l), venous pH at 7.25 (normal: 7.35–7.45), lactate at 2.6 mmol/l (normal: 0.5–2.2 mmol/l), creatinine at 15.46 mg/dl (normal: 0.6–1.3 mg/dl), CK at 25,995.33 IU/l (normal: 20–200 IU/l), AST at 134 U/l (normal: 10–40 U/l), ALT at 235 U/l (normal: 7–56 U/l), calcium at 1.87 mg/dl (normal total calcium: 8.5–10.5 mg/dl), negative for ethanol, normal serum osmol gap, and trace urine ketones. The serum lipase level was 1,528 U/l (normal range: 10–140 U/l). Upon admission, the urine drug test was positive for amphetamine. The ECG showed peaked T waves and a QRS duration of 120 ms. Doppler ultrasonography of left limb was unremarkable. Hepatobiliary ultrasonography did not reveal gallstones or biliary dilation. Computed tomography (CT) of the abdomen and pelvis revealed interstitial edematous pancreatitis without necrosis or pseudocyst development. The patient was diagnosed with acute pancreatitis according to the Atlanta criteria, which include typical pain, lipase levels exceeding three times the upper limit of normal, and imaging findings. Additionally, the patient had high anion gap metabolic acidosis, severe hyperkalemia, and acute kidney injury, likely due to a combination of hypoperfusion and rhabdomyolysis.

Hospital course and management

Immediate treatment for hyperkalemia was administered following established protocols, which included

intravenous calcium gluconate (to stabilize cardiac membranes), 10 units of regular insulin with dextrose (to promote intracellular potassium shift), high-dose nebulized salbutamol (a β2-agonist that also facilitates potassium uptake into cells), and intravenous sodium bicarbonate (used in cases of concurrent metabolic acidosis to further reduce serum potassium).[5,6] These temporizing measures are widely recommended in emergency settings to prevent life-threatening arrhythmias while definitive treatments are being arranged.

In cases of acute pancreatitis and rhabdomyolysis, aggressive intravenous fluid resuscitation with isotonic crystalloids is the standard practice to preserve renal perfusion, prevent tubular obstruction from myoglobin, and limit systemic complications.[2,16] Despite these interventions, the patient's potassium level initially decreased to 5.5 mmol/l but then increased to 5.7 mmol/l, accompanied by persistent high anion gap metabolic acidosis (HAGMA) and oliguria. These findings indicate inadequate renal clearance and intracellular potassium release due to ongoing muscle breakdown in the patient.

Given the refractory hyperkalemia, worsening acidosis, and evolving acute kidney injury (AKI), the nephrology team initiated intermittent hemodialysis (HD) using a temporary catheter on the day of admission. HD was chosen not for amphetamine clearance since amphetamines have a large volume of distribution and are poorly dialyzable, but to rapidly correct life-threatening electrolyte abnormalities, severe acidosis, and uremia, which are well-established indications for extracorporeal therapy.[6,14,15] The first HD session (4 h with a high-flux dialyzer) lowered the potassium level to 5.4 mmol/l and normalized the pH to 7.36. A second session, 36 h later, normalized the serum potassium to 4.4 mmol/l, and a third session on day four stabilized the electrolytes and supported diuresis. Supportive care, including analgesia, early enteral nutrition to maintain gut integrity and reduce infectious complications, and deep vein thrombosis (DVT) prophylaxis, was provided throughout hospitalization. No antibiotics were required because there was no evidence of infection at the time. By day six, CK levels decreased to 2,257 IU/l, and serum creatinine improved to 9.81 mg/dl. The patient was discharged on day seven in a stable condition with outpatient follow-up and referral for substance use counselling.

DISCUSSION

This case highlights a rare but increasingly acknowledged pattern of multisystem toxicity caused by amphetamines, presenting as acute pancreatitis, rhabdomyolysis, acute kidney failure, high anion gap metabolic acidosis (HAGMA), and severe hyperkalemia, all of which necessitate hemodialysis. Although each of these complications has been individually linked to amphetamine use, their simultaneous occurrence is uncommon, emphasizing the significant systemic effects of sympathomimetic toxicity. The occurrence of HAGMA in patients is due to multiple factors. Elevated lactate levels (2.6 mmol/l) are likely indicative of tissue hypoperfusion resulting from vasoconstriction, agitation, and volume depletion, which are common in acute amphetamine toxicity.[1,3] Furthermore, acute kidney injury (AKI) resulting from rhabdomyolysis and hypovolemia leads to reduced acid clearance and worsens acidosis.[2,4] In cases where metabolic disturbances like severe acidosis (pH<7.2) or electrolyte imbalances do not respond to standard treatments, hemodialysis (HD) is employed not to detoxify amphetamines due to their high volume of distribution and poor dialyzability but as a critical measure to swiftly address acidosis and hyperkalemia.[6,14,15]

Severe hyperkalemia, a crucial element in this case, likely arose from a combination of rhabdomyolysis and acute kidney injury (AKI). Several factors, including hyperthermia, agitation, vasoconstriction, and direct toxic effects on the muscles, cause amphetamine-induced rhabdomyolysis.[5,16] The breakdown of skeletal muscle results in the release of significant amounts of intracellular potassium into the bloodstream. When kidney function is compromised, potassium excretion is hindered, leading to dangerously high serum potassium levels. Standard medical treatments, such as calcium gluconate, insulin–dextrose, beta-agonists, and bicarbonate, are typically the first line of defense.[5,6] However, this case illustrates the limitations of these treatments in severe toxicity cases. This underscores the importance of hemodialysis (HD) in effectively clearing potassium levels.

The patient was diagnosed with acute pancreatitis, a conclusion supported by the Atlanta criteria, which include typical abdominal pain, lipase levels more than three times the normal upper limit, and imaging findings indicative of pancreatitis. Notably, the patient had no history of alcohol use, gallstones, hypertriglyceridemia, or use of other medications commonly associated with medications. This points to a possible drug-induced cause, with amphetamine use being the most likely. Although rare, methamphetamine and MDMA have been linked to pancreatitis in case reports and small studies, potentially due to catecholamine-induced vasospasm, sphincter of Oddi dysfunction, or direct pancreatic ischemia.[7-12] The proposed mechanism involves significant vasoconstriction, leading to ischemic injury to the pancreatic tissue, which may explain the interstitial edematous pattern observed on imaging.

Additionally, while amphetamines are not effectively cleared by HD due to their high distribution volume and lipid solubility 10, HD remains vital for managing complications rather than directly addressing toxins.[6,14] This aligns with the growing evidence that extracorporeal therapy can quickly correct metabolic disturbances, such as severe acidosis and hyperkalaemia, thereby improving patient outcomes, even when the drug remains active in the body.[14,15] The nature and severity of the patient's complications likely suggest high-dose exposure or binge use of amphetamines, which can significantly elevate

circulating catecholamines and result in multiorgan dysfunction.[13,14] Long-term use may also increase the risk of vasculopathy and tissue damage, worsening the acute effects.[15] This case highlights the importance of early detection, aggressive supportive care, and maintaining a high level of suspicion for multisystem involvement in patients with known or suspected amphetamine toxicity.

CONCLUSION

This case highlights the dangerous combination of acute pancreatitis, acute kidney injury, hyperkalaemia, and severe metabolic acidosis that can occur after amphetamine use. When standard medical treatments fail to quickly control potassium levels or acidaemia, hemodialysis (HD) becomes essential. Prompt identification and a collaborative medical approach can expedite recovery.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Nazir I, Abdullah HMM, Sindy EAI, Boudal EK, Alwagdani ARA, Elsaghir ARA, et al. A case report on amphetamine-induced acute pancreatitis with concurrent rhabdomyolysis, acute renal failure and severe hyperkalemia requiring hemodialysis. Int J Res Med Sci 2026;14:1167-70.