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Suspected Methadone-Gabapentin Co-Ingestion Associated with Rhabdomyolysis, Acute Kidney Injury, and Peripheral Neuropathy: A Case Report

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ABSTRACT

We report the case of a 27-year-old woman who presented with severe mixed respiratory and metabolic acidosis, rhabdomyolysis characterized by markedly elevated creatine kinase (CK), hyperkalemia, and acute kidney injury following an 18–20-hour period of unconsciousness. Upon admission, the patient reported ingesting an unknown quantity of methadone and gabapentin obtained from non-medical sources; however, no toxicological screening was performed to confirm this. Physical examination revealed hyperemic bullae on the right lower extremity and chest, consistent with prolonged immobilization. Despite aggressive intravenous fluid resuscitation, the patient developed anuria requiring renal replacement therapy. Her hospital course was further complicated by severe hemolytic anemia and urinary tract infection. As acute metabolic disturbances stabilized, a concurrent right sciatic neuropathy was identified, clinically suspected to be a compressive injury secondary to prolonged immobilization.

Keywords: Acute kidney injury; creatine kinase; gabapentin; hyperkalemia; methadone; rhabdomyolysis.

INTRODUCTION

The evaluation of patients presenting with prolonged unconsciousness requires a systematic approach to identify the life-threatening complications of immobilization. While exposure to central nervous system (CNS) depressants, such as methadone and gabapentin, is frequently suspected in these clinical scenarios, the absence of toxicological screening precludes a definitive etiological diagnosis. In this case, the clinical assessment was guided by the patient's own report of ingestion of methadone and gabapentin obtained from non-medical sources.

Rhabdomyolysis is a consequence of sustained mechanical pressure on muscle groups during prolonged immobilization. The resulting systemic release of intracellular creatine kinase (CK) and myoglobin can precipitate pigment-induced acute kidney injury (AKI), which may progress to anuria requiring renal replacement therapy.[1]

Simultaneously, localized pressure during the period of unconsciousness can result in significant compressive neuropathies: specifically, the sciatic nerve injury identified in this patient.

Other complications, such as anemia and urinary tract infections, can further worsen the clinical course, making management more challenging and potentially prolonging the hospital stay.

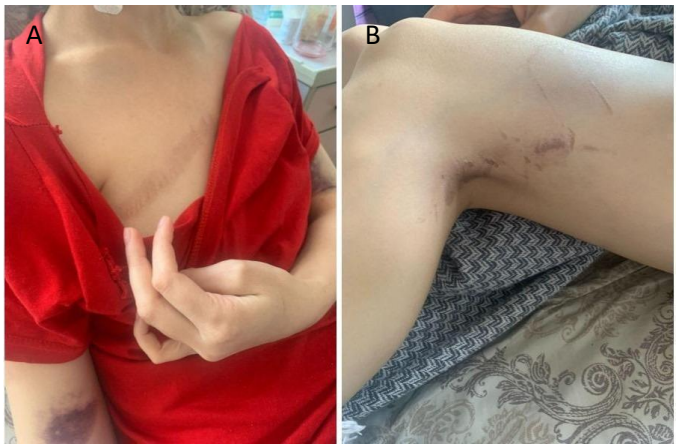
This case highlights the multi-organ complications of rhabdomyolysis and emphasizes the importance of a comprehensive clinical workup even in the absence of toxicological confirmation.

Case

A 27-year-old female was brought to the emergency department by ambulance after she was found unconscious at home. According to her family members, she had remained unresponsive for approximately 18-20 hours before discovery. Upon admission, the patient reported ingesting an unknown quantity of methadone and gabapentin tablets; however, no toxicological screening was performed to confirm this. At initial evaluation, she was hypotonic and somnolent, with sluggish responses to external stimuli and reported experiencing generalized weakness, dizziness, palpitations, dry mouth, and numbness in her lower extremities.

On admission, vital signs demonstrated tachycardia at 110 beats per minute (bpm), blood pressure of 130/80 mmHg, respiratory rate of 24 breaths per minute, and oxygen saturation of 94% on room air. Her body temperature was 36.60 °C. The pupils were equal, but miotic, with slow pupillary and corneal reflexes. Cardiac auscultation revealed muffled heart sounds, while the lungs exhibited clear vesicular breath sounds bilaterally. The abdomen was soft and non-tender, without rigidity or hepatosplenomegaly. Bullous, hyperemic lesions were noted on the anterior chest wall and both lower extremities, consistent with pressure-related skin injury secondary to prolonged immobilization (Fig.1A and 1B).

FIGURE 1. Skin findings on the twentieth day of hospitalization



An electrocardiogram obtained on admission revealed sinus tachycardia. Intravenous infusion therapy with normal saline was initiated. Subsequently, arterial blood gas analysis demonstrated severe mixed respiratory and metabolic acidosis, with a pH of 7.03, pCO_2 of 88.10 mmHg, bicarbonate of 15.3 mmol/L, lactate of 5.59 mmol/L, and an anion gap of 25.40 (Tab.1). Laboratory investigations revealed marked rhabdomyolysis, with a creatine kinase (CK) level of 8,340 U/L. Serial CK measurements and electrolyte trends were obtained to monitor the progression of muscle injury (Tab.2). Serum potassium peaked at 6.20 mmol/L. In addition to aggressive fluid resuscitation, she received supplemental oxygen and intravenous sodium bicarbonate. Calcium gluconate was administered to stabilize the cardiac cell membrane. The patient was transferred to the intensive care unit (ICU).

Urine dipstick analysis showed 2+ blood and 1+ protein. Microscopic examination demonstrated 0-2 red blood cells per high-power field and numerous muddy-brown casts, consistent with myoglobin-induced acute tubular necrosis.

TABLE 1. Arterial blood gas analysis on admission and during the clinical course

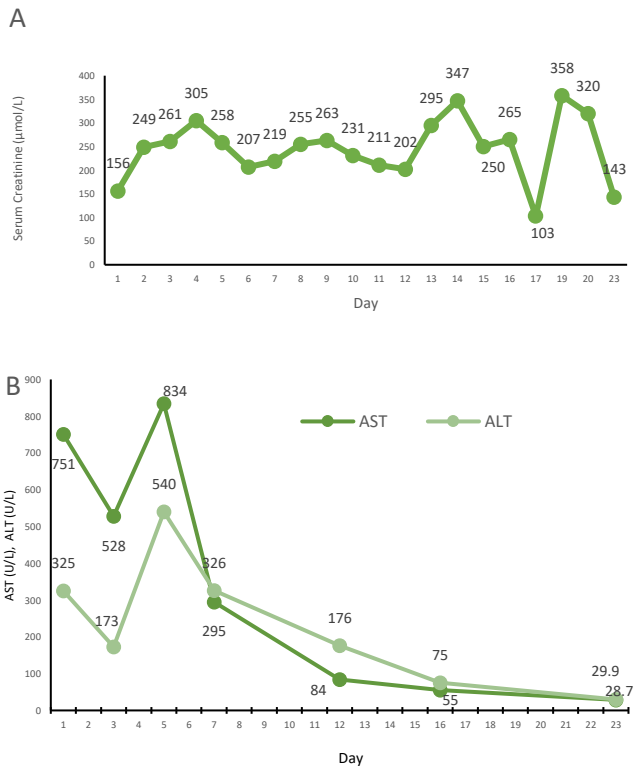
Day	pH	pCO_2 (mmHg)	HCO_3^- (mmol/L)	Lactate (mmol/L)
1	7.03	88.1	15.3	5.59
2	7.1	43.01	15.4	4.4
3	7.24	55.3	19.5	1.46
5	7.43	35.9	23.7	1.28
9	7.53	31.3	27.1	1.37
13	7.48	33.9	24.9	1.58
15	7.55	26.7	24.8	0.79
17	7.38	45.5	24.3	1.75
19	7.41	39.7	23.6	1.74
23	7.4	36.5	21.7	1.54

TABLE 2. Serial trends of serum electrolytes and creatine kinase

Day	Potassium (mmol/L)	Calcium (mmol/L)	Phosphate (mmol/L)	Creatine Kinase (U/L)
1	6.02	1.15	2.2	8,340
2	4.69	1.05	2.4	5,125
5	3.74	0.88	1.45	4,355
8	3.7	1.07	1.4	2,410
13	4.54	1.02	1.3	931
15	3.85	1.12	1.02	435
17	4.89	1.13	1.23	283
20	4.46	1.14	1.3	140
23	4.1	1.24	1.3	135

Upon admission, the patient's serum creatinine was 156 µmol/L and continued to rise during hospitalization, reaching a peak of 358 µmol/L (Fig. 2A), with a concomitant increase in blood urea nitrogen to 22.58 mmol/L. These findings were consistent with acute kidney injury secondary to rhabdomyolysis. A urinary catheter was inserted for close monitoring of urine output.

FIGURE 2. Serial trends in serum creatinine (A), aspartate aminotransferase, and alanine aminotransferase (B)



Liver function tests revealed a progressive rise in transaminases, with aspartate aminotransferase (AST) and alanine aminotransferase (ALT) peaking at 751 U/L and 325 U/L, respectively (Fig.2B). In contrast, coagulation and cholestatic markers showed only minor fluctuations with

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admission values of prothrombin time (PT) 15.0 sec, activated partial thromboplastin time (aPTT) 27.8 sec, INR 1.17, total bilirubin 5.12 µmol/L, direct bilirubin 3.38 µmol/L, and γ-glutamyl transferase (GGT) 20.37 IU/L.

Blood count obtained at admission revealed leukocytosis with 11% band forms, suggestive of an ongoing systemic infectious process or systemic inflammatory response associated with rhabdomyolysis; therefore, empiric antibiotic therapy with ceftriaxone was started.

Hyperkalemia was successfully managed, with serum potassium decreasing to 4.69 mmol/L; however, ionized calcium levels reached a low of 0.88 mmol/L on the fifth day of hospitalization. Concurrently, laboratory analysis showed a peak hyperphosphatemia of 2.2 mmol/L (Table 2). These findings pointed towards the massive release of intracellular phosphate from necrotic myocytes, leading to subsequent calcium sequestration within damaged muscle tissue and the precipitation of calcium-phosphate complexes.

Despite aggressive intravenous hydration and trial of loop diuretics, the patient remained oliguric, producing approximately 150 mL of urine over 24 hours. Laboratory studies revealed persistent metabolic acidosis with a blood pH of 7.1 (Tab.1). Rising serum creatinine levels to 405 µmol/L indicated significant deterioration in renal function. Urine output dropped to 50 mL/24 hours, indicating anuria. In the setting of progressive azotemia, refractory acidosis, and anuria, renal replacement therapy was initiated. The patient underwent 10 hemodialysis sessions.

Following aggressive intravenous fluid resuscitation, serum creatine kinase levels began to decline within 24 hours. By the fifth day of hospitalization, the CK concentration had decreased by approximately 50%. Simultaneously, hemodialysis reduced phosphate levels, while calcium levels showed a reciprocal upward trend (Tab.2).

The clinical course was further complicated by hypoalbuminemia, with serum albumin decreasing to 2.94 g/dL, prompting administration of intravenous albumin. Concurrently, liver enzymes demonstrated a progressive increase, with AST at 834 U/L and ALT at 540 U/L (Fig.2B). The patient received reduced glutathione to reduce oxidative stress. Prophylactic anticoagulation with Fraxiparine 0.4 mg/0.4 mL and stress ulcer prophylaxis with 40 mg pantoprazole were also provided.

By hospital day 14, the patient developed severe anemia, with hemoglobin dropping to 5.1 g/dL. Laboratory studies showed an MCV of 87.40 fL and a platelet count of $174x10^9$/L, both within normal limits. Coagulation studies, including aPTT (27.05 sec), PT (13.7), and INR (1.06), were unremarkable. No active or occult hemorrhage was identified. The presence of normocytic normochromic anemia, alongside elevated biochemical markers, specifically lactate dehydrogenase (LDH) of 1,243 U/L and indirect bilirubin of 2.1 mg/dL, pointed toward hemolytic anemia possibly caused by mechanical trauma (shear stress) from a kink in the dialysis circuit. Hemodialysis was temporarily halted, and the patient required a transfusion of three units of packed red blood cells to stabilize hemoglobin levels.

The following day, laboratory findings were notable for elevated neutrophil counts and worsening renal function, with creatinine reaching 374 µmol/L. Urinalysis revealed proteinuria (100 mg/dL), marked hematuria, and moderate leukocyturia. Microscopic examination showed 25-30 white blood cells per high-power field and numerous intact red blood cells. Additionally, moderate uric acid crystals were present, along with a significant number of fungi and bacteria. Blood cultures were negative for bacteremia. These findings were suggestive of a complicated urinary tract infection. An infectious disease specialist was consulted, and antimicrobial therapy was changed from ceftriaxone to piperacillin-tazobactam to provide broader coverage against gram-negative rods.

Following the correction of metabolic and infectious complications, a neurology consultation was requested to address the persistent numbness in the right lower extremity. Physical examination revealed swelling in the right hip and gluteal region. Functional assessment demonstrated impaired right hip flexion, reduced muscle strength, and diminished Achilles and patellar reflexes. Notably, there was a complete absence of active movement in ankle dorsiflexion and plantarflexion, resulting in a clinically significant foot drop. While these findings were highly suggestive of right sciatic nerve injury, no electrodiagnostic studies or advanced neuroimaging were performed during the hospitalization. A duplex scan of lower extremity vessels confirmed normal arterial and venous flow, ruling out primary vascular pathology. The deficit was managed as compression neuropathy. Therapeutic intervention was initiated with a combination of cytidine monophosphate, uridine monophosphate, and vitamin B complex to support nerve recovery.

The patient was discharged in a stable condition on hospital day 23. Outpatient physical therapy and electrical stimulation were recommended for ongoing rehabilitation of right lower limb neuropathy.

In a follow-up interview, the patient reported that her recent serum creatinine levels have returned to normal and she has achieved a full recovery from acute kidney injury without progression to chronic kidney disease. Outpatient nerve conduction studies confirmed sciatic axonal neuropathy. The patient is currently undergoing electrical stimulation with significant symptomatic improvement, though she has not yet reached a full neurologic recovery.

DISCUSSION

This case highlights a complex presentation of rhabdomyolysis-induced acute kidney injury following prolonged immobilization, further requiring multiple hemodialysis sessions, secondary hemolytic anemia, and compression neuropathy.

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Rhabdomyolysis can be triggered by trauma, infection, extreme exercise, and various pharmacologic agents.[2] It is characterized by dissolution of skeletal muscle and release of intracellular contents, including creatine kinase, myoglobin, and aminotransferases. The pathogenesis involves dysregulated calcium influx due to sarcoplasmic injury or ATP depletion, leading to protease activation and myocyte disruption.[3]

Following the destruction of myocytes, several life-threatening electrolyte imbalances, such as hyperkalemia, hyperphosphatemia, and hypocalcemia, may emerge.[4] Rapid release of intracellular potassium into systemic circulation, combined with hypocalcemia driven by precipitation of calcium-phosphate complexes, poses an immediate risk of cardiac arrhythmias.[5]

Common clinical manifestations of rhabdomyolysis include reddish-brown urine due to myoglobinuria, muscle weakness, fatigue, pain, and cramps.[6] Urine dipstick testing may yield a false-positive result for blood in the absence of red blood cells, reflecting the presence of myoglobin.

The most serious complication of rhabdomyolysis is acute kidney injury, primarily mediated by myoglobin-induced tubular toxicity, vasoconstriction, and ischemia. Nephrotoxic effects of myoglobin are intensified in the presence of an acidic urine pH caused by metabolic acidosis and hypovolemia.[7] Monitoring urine output and initiating early and aggressive fluid resuscitation with crystalloid solutions are essential for preventing and treating AKI. These measures restore renal perfusion and increase urine flow rate. Additionally, urinary alkalinization with sodium bicarbonate may be considered to prevent precipitation of myoglobin and uric acid in the distal convoluted tubule, correct metabolic acidosis, and reduce the risk of life-threatening hyperkalemia.[8]

When supportive measures fail to manage hyperkalemia, hyperazotemia, metabolic acidosis, or persistent anuria, intermittent hemodialysis is required. Current research indicates that rhabdomyolysis-associated AKI is linked to increased in-hospital mortality. Approximately 70-80% of survivors successfully discontinue dialysis within 90 days.[9] While about 70% of patients eventually regain baseline kidney function, it is important to note that in 27% of cases, the condition may progress to chronic kidney disease.[10]

Beyond renal complications, prolonged immobilization often results in compression neuropathy. Sustained external pressure on nerves can compromise the vasa nervorum and cause axonal damage. Additionally, the dissolution of skeletal muscle typically leads to significant inflammatory edema and increased intracompartmental pressure, further exacerbating axonal injury.[11] The physiological timeline for axonal regeneration could take up to 6-12 months for a definitive clinical outcome to be apparent.

Apart from neurological considerations, maintaining hematologic integrity is another important concern during critical illness. Anemia in patients undergoing dialysis during prolonged hospitalization is often multifactorial. Potential contributors include hemodilution secondary to aggressive fluid resuscitation, iatrogenic blood loss from frequent phlebotomy, and the suppressive effects of systemic inflammation on erythropoiesis.[12] The absence of coagulopathy or identifiable gastrointestinal and internal hemorrhage, a rapid decline in hemoglobin, should prompt investigation into intravascular hemolysis. In the setting of renal replacement therapy, high shear stress and turbulence generated when blood is forced through stenotic catheters, thrombi at dialyzer ports, or kinked tubing result in erythrocyte fragmentation.[13] Severe anemia requires prompt blood transfusion and the temporary discontinuation of dialysis to prevent further erythrocyte destruction, while the circuit integrity is evaluated.

The reported co-ingestion of methadone and gabapentin provides a plausible explanation for the initial clinical presentation of unconsciousness and miosis. While rhabdomyolysis is a recognized complication of opioid exposure, combined opioid-gabapentinoid toxicity remains poorly described.[14,15] In this case, an 18-20-hour period of immobility, possibly precipitated by the synergistic depressive effects of these substances, could have resulted in sustained external pressure and subsequent compression neuropathy and the necrosis of muscle fibers. However, as toxicological confirmation was not obtained, the attribution of these clinical findings to specific agents remains presumptive, based primarily on the patient's history and the observed toxidrome.

CONCLUSIONS

This case demonstrates the severe multi-organ consequences of prolonged immobilization in the setting of suspected CNS depressant co-ingestion. Rhabdomyolysis triggered a cascade of life-threatening complications, including mixed acid-base disturbance, acute kidney injury requiring renal replacement therapy, hemolytic anemia, urinary tract infection, and compressive sciatic neuropathy. These findings reinforce the need for a multidisciplinary approach when evaluating patients with prolonged unconsciousness and add to the limited literature describing the combined toxicity of opioids and gabapentinoids as a precipitant of immobilization-related injury, even in the absence of toxicological confirmation.

INFORMED CONSENT

Written informed consent for publication was obtained from the patient, and all identifying details were removed or modified to protect patient privacy.

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