Acid Trip to the Hospital

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Introduction

- Illicit drug use can often be the cause of rhabdomyolysis, however lysergic acid diethylamide (LSD) ingestion as the cause of rhabdomyolysis has not been frequently documented.

- Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of intracellular muscle constituents into the circulation.

Case Presentation

Day 1:

- A 17-year-old male with a history of bipolar disorder on lithium was brought in from a college party with acute altered mental status.
- According to surrounding bystanders, following ingestion of an unknown substance he had "seizure-like" activity followed by increased combativeness.
- He arrived in the emergency department (ED) restrained to the gurney after being given intravenous lorazepam.

- ED Vitals: BP 127/79, HR 110, T 99.9F, RR 22, SpO2 90% on RA
- Intubated for airway protection and transferred to MICU
- Initial Labs

16.5 / 9.9 / 246 / 46.8	140 | 105 | 11 / 3.3 | 20 | 0.89	149

Lactate 10 mmol/L ⇑
Creatinine Kinase (CK) 401 U/L ⇑
Urine drug screen: +benzodiazepines

Acetaminophen, Salicylate, Lithium — Undetectable

CT Head: No acute intracranial abnormality

- Lactate normalized with IV hydration
- Patient self-extubated a few hours later and endorsed LSD ingestion.

Day 2:

- Transferred to the general medicine floor
- Morning routine labs:

141 | 108 | 20 / 3.6 | 24 | **2.72**		98

- Additional work up of AKI revealed

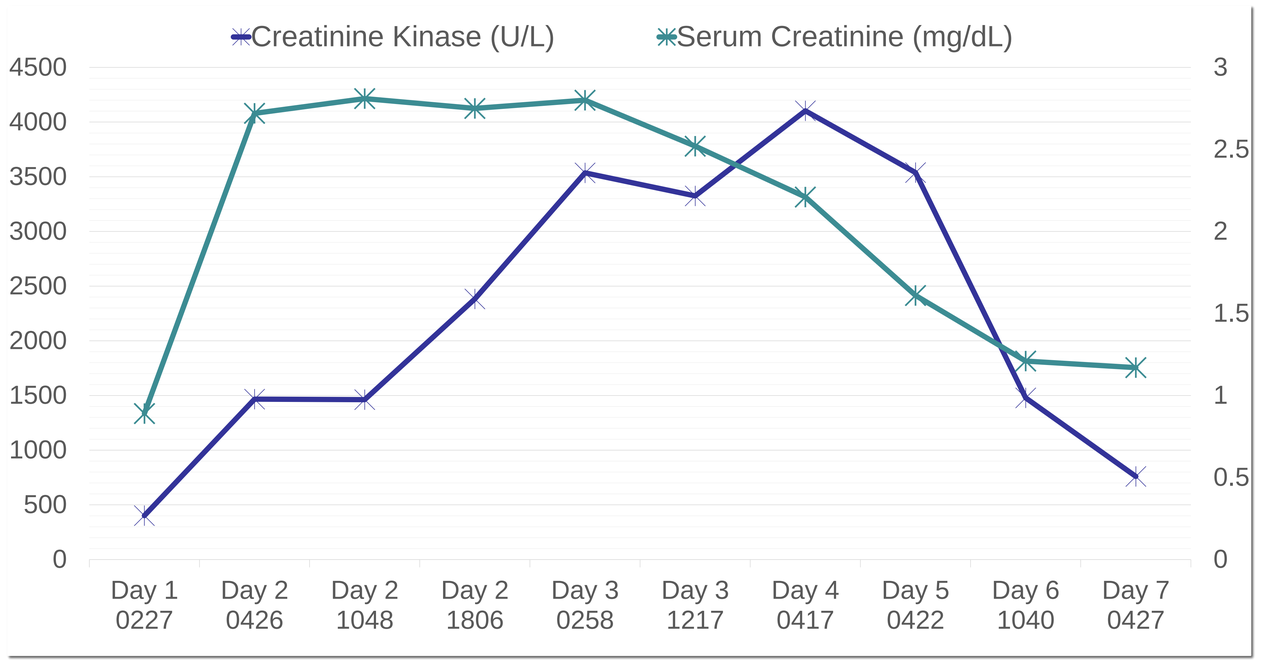
Creatinine Kinase 1467 U/L ⇑
Serum myoglobin 330 ng/mL ⇑
Urine myoglobin <1
Urine analysis: +blood (small)

AST 44 U/L
ALT 77 U/L
Uric acid 10 mg/dL
Phosphorous 3.9 mg/dL

- He denied any localizing musculoskeletal pain despite his CK elevation.
- CK peaked at 4103 U/L on Day 4
- Serum creatinine peaked at 2.81 mg/dL

Case Resolution:

- Diagnosis thought to be rhabdomyolysis as a consequence of LSD ingestion
- Resolved with aggressive IV hydration and discharged home.



Discussion

- LSD (street name: Acid) is a hallucinogenic substance that produces psychedelic effects including violent agitation that typically lasts 6 to 12 hours

- Rhabdomyolysis[1] is diagnosed in patients
  - With typical triad of muscular pain, weakness, and/or dark urine
  - plus a marked elevation in the serum CK (at least 5x the upper limit of normal)

- Myoglobin can also be found in the urine
  - Leads to darker colored urine
  - Not unusual for CK levels to remain elevated without myoglobinuria

- Serum CK begins to rise within 2 to 12 hours following muscle injury
  - Peaks within 24 to 72 hours
  - Decline usually within 3-5 days of cessation of muscle injury.
  - In patients whose CK does not decline as expected, continued muscle injury or the development of a compartment syndrome may be present

- Rhabdomyolysis due to illicit drug use has been described, this has not been well reported in patients with LSD ingestion, with only a few case reports in the literature[2,3].

- In one case series from the 1980s, LSD ingestion requiring the use of a straitjacket was implicated in the pathophysiology of rhabdomyolysis.
  - These aggressive movements lead to muscle damage and release of pigments that are toxic to the kidney.

- As LSD itself is not nephrotoxic, other etiologies of an AKI should also be investigated.

- It is important to monitor closely the renal function of patients who have suspected LSD ingestion, with a high clinical suspicion for rhabdomyolysis if renal dysfunction is present.

References

1. Giannoglou et al. The syndrome of rhabdomyolysis: Pathophysiology and diagnosis. European Journal of Internal Medicine 18 (2007) 90–100
2. Mercieca et al. Acute renal failure due to rhabdomyolysis associated with use of a straitjacket in lysergide intoxication. BMJ 288 (1984)
3. Berrens et al. Rhabdomyolysis after LSD ingestion. Psychosomatics. 2010 Jul-Aug;51(4):356-356.e3.