



OPIOID INDUCED RHABDOMYOLYSIS PRESENTING WITH MYOPATHY

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ABSTRACT

Opioid is a psychoactive substance with high habit forming property. Opioid dependence and at times over dosing may cause acute muscle damage with elevated serum aspartate aminotransferase and creatine kinase activities, increased serum myoglobin concentrations, raised plasma creatinine concentrations, hypocalcaemia and hyperphosphataemia. These abnormalities may worsen if not treated and gets complicated due to the development of acute renal failure (requiring haemodialysis). Clinical evidence of myopathy is minimally reported but is known to occur in very few cases. The case described below presented to psychiatric OPD with muscle weakness and features of opioid poisoning.

KEYWORDS: Opioid, dependence, rhabdomyolysis, acute renal failure, hemodialysis, withdrawal.

INTRODUCTION

Opioids are highly addictive substances which when taken over prolonged period of time produces features of opioid dependence. The classical features of dependence include severe muscle cramps, profuse diarrhoea, abdominal cramps, rhinorrhoea, lacrimation, fever, yawning, piloerection, hypertension, pupillary dilatation, tachycardia and temperature dysregulation etc.^[1]

In very rare cases when these substances are taken in large doses in frequent span of time regularly may lead to condition called rhabdomyolysis.

Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of intracellular muscle constituents into the circulation. Creatine kinase (CK) levels are typically markedly elevated, and muscle pain and myoglobinuria may be present. The severity of illness ranges from asymptomatic elevations in serum muscle enzymes to life-threatening disease associated with extreme enzyme elevations, electrolyte imbalances, and acute kidney injury. In cases of severe dependence, opioid overdose, or other central nervous system (CNS) depressants leads to immobilization and ischemic compression of muscle.^[2]

Hypokalemia caused by potassium loss from exertion or excessive sweating after withdrawal that occurs^[3] can lead to notable complications. The role of potassium in the regulation of skeletal muscle blood flow appears to be important in the pathogenesis in this setting.^[4] During exercise, there is normally an appropriate increase in muscle perfusion to meet enhanced energy demands.

This hyperemic response is mediated in part by the release of potassium from skeletal muscle cells. The ensuing local elevation in the potassium concentration causes vasodilation, which enhances regional blood flow.^[5] However, the cellular release of potassium is impaired by potassium depletion. As a result, there is a lesser increase in blood flow, possibly resulting in cramps, ischemic necrosis, and rhabdomyolysis.^[6] Hypokalemia-induced impairment in muscle metabolism also may contribute to muscle dysfunction.^[4]

CASE REPORT

A 34 year old unemployed youth with family history of alcohol use in the father and depression in mother for last 5 years presented to the hospital with features of weakness of all four limbs, malaise, passage of darkened urine with background history of passing poor urine for last 2 months.

As patient was admitted in the hospital he manifested with symptoms of profuse sweating with body aches, nausea and vomiting after 48 hours of admission. He was noted to have elevated blood pressure with excessive yawning and was noted to constantly requesting for a discharge from hospital. As he was noted to be restless, dysphoric and exhibiting odd and aggressive behavior a high index of suspicion was seen to seek a psychiatric consult.

Detailed history revealed a long standing history of heroin and cocaine use since age of 14 years with peer group. He left school after his 6th class and would work as a car cleaner in various housing societies. He gave

positive history of sniffing petrol and diesel from fuel tank of vehicles which he would wash daily. To continue with his usage of daily intake of opioid he would resort to money lending from friends and doing overtime at his work. After 3 years he took to taking intravenous opioid use. He felt the high to be excessively more and he continued this route of access for next 13 years. He was asked to leave his house as he was noted to be abusive, violent and non trustworthy by his relatives. He continued use of IV morphine and heroin and would use codeine.

Individual would also consume country liquor, but whenever he had the money and chance, he would resort to heavy use of opioid. He started to peddle drugs for money after falling prey to people who would deal in such activities and managed to evade the law enforcement agency. In this period of time he exhibited weakness of muscles, exhaustion and when he was noted to be passing dark urine, a consult was sought and he brought by his friends on the advice of village elders.

- Detailed laboratory tests were done like Complete blood count (CBC), including hemoglobin, hematocrit, and platelets, Serum chemistries, Prothrombin time (PT), activated partial thromboplastin time (aPTT),
- Serum aldolase
- Lactate dehydrogenase (LDH)
- Cardiac troponin I
- Creatine kinase (most reliable and sensitive indicator of muscle injury)

Urine myoglobin and urine toxicology tests were done which were positive.

As he continued to show weakness of limbs tests like - Muscle biopsy, Immunoblotting, immunofluorescence, and genetic studies were done. CT Scan of brain was also done which revealed cortical atrophy.

Patient had evidence of hyponatremia, hypokalemia with positive tests for urine for myoglobin. CPK, LDH was elevated and serial blood urea and serum creatinine revealed findings of acute renal failure.

Patient was haemodialyzed and given restorative fluids with supportive therapy. Psychiatric management was done by use of Tab clonidine 0.1 mg TDS, Tab ibuprofen 400mg TDS, Tab Lorazepam 2mg TDS initially. After 48 hours tab Naltrexone 50 mg OD was initiated and he was given Tab Buprenorphine 10 mg per day. After his correction of fluid electrolyte abnormalities he was sent for counseling sessions on second week for motivation enhancement and psycho education.

His haematocrit and CPK, LDH levels gradually normalized. His urine toxicology tests were also done and was negative. The weakness in limbs recovered after 6 weeks of inpatient care and his renal function status showed complete recovery after another week.

Patient received 8 sessions of group therapy and was enrolled in Narcotic Anonymous group following his discharge after 10 weeks of inpatient care.

DISCUSSION

Opioid use has several complications besides being psychologically destructive, it renders a person to become a socially alienated and deviant person. Heavy intravenous use can cause increasing tolerance and craving for further use causing long term damage to the kidneys and musculo skeletal structure as was the case in the person mentioned in this case.

The presentation was with muscle weakness and darkened urine, but clinicians need to be aware that psychoactive substance use like opioids can cause massive destruction of muscle tissue and kidney architecture.^[7,8] Timely help and intervention both medical and psychiatric can prevent further damage and help in relapse prevention in affected individuals.

Advent of better medications and availability of anticraving drugs will be useful in follow up of such cases and it would be prudent to assess all such individuals for personality disorders and other psychiatric co morbidities like anxiety and depression.

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