Journal of Renal and Hepatic Disorders

ORIGINAL RESEARCH

Automobile Paint Reducer Induced Acute Kidney Injury: A Case Series

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Abstract

The various aspects of the automobile industry also carry with it the risk for occupational health hazards with it. Toluene has also evolved as a commonly used drug by substance abusers. Accidental exposure or self-poisoning with these substances has been reported in literature. These substances can also cause distal renal tubular acidosis (RTA), acute tubular necrosis, glomerulonephritis and interstitial nephritis, rhabdomyolysis and myoglobinemia. In this series, we report about three patients who developed renal manifestations because of organic solvents. Two of the three patients had ingested the paint reducer substance and the third one was addicted to sniffing the toluene based paint reducer. All the patients had in taken these substances s with suicidal intent and developed acute kidney injury (AKI) and severe metabolic acidosis. One of the patients had features of rhabdomyolysis as well. The third patient was a substance abuser and had inhaled higher than usual dose and developed severe and refractory acidosis and mild kidney injury and required Renal Replacement Therapy (RRT) for acidosis. All the patients eventually recovered their kidney functions and were doing well during their follow-up. Toluene based organic solvents lead to acute neurological symptoms, accompanied by severe metabolic alterations, organ injury and dysfunction. An association of the development of hypokalemic paralysis and metabolic acidosis with toluene intoxication has been observed. The management of acute toluene toxicity is mainly conservative, consisting of electrolytes correction, acid-base and fluid abnormalities and renal replacement therapy in severe AKI. Organic solvent exposure may result in acute tubular necrosis, rhabdomyolysis, RTA and AKI irrespective of the intake route. Clinical suspicion of organ dysfunction and failure and timely induction of supportive care leads to a good outcome.

Keywords: Acute kidney injury; organic solvent; paint reducer; toluene

Received: 8 June 2021; *Accepted after Revision*: 19 July 2021; *Published*: 18 August 2021

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How to cite: Latief M, et al. Automobile Paint Reducer Induced Acute Kidney Injury: A Case Series. J Ren Hepat Disord. 2021;5(2): 10–13.

Doi: https://doi.org/10.15586/jrenhep.v5i2.116

Introduction

Organic solvents, commonly used in the automobile industry, have been reported to lead to kidney injury. These involve almost any compartment of the kidney i.e. glomerulus, tubules or vasculature. Moreover, these substances also aggravate the progression of underlying kidney diseases (1–5). Occupational health hazards are the known contributors to morbidity and mortality of workers in the industry. The factors contributing to this risk include the extent of exposure, ventilation maintenance, access to fluid intake, etc. A paint reducer, an organic solvent frequently used in the automobile manufacturing and repair industry,

is one such risk factor. Reducers are volatile solvents consisting of a mixture of organic compounds like aromatic hydrocarbons, halogenated hydrocarbons and naphtha, toluene, xylene, acetone, hexane, benzene and methyl isobutyl ketone etc. (6–8).

Toluene is a common constituent in many industrial solvents and has a known toxicity profile that shows environmental, accidental and intentional poisoning. Toluene finds its use in paints, paint reducers, polishing solvents, cleaning products, glues, adhesives, etc. (9). It is also a drug that substance abusers easily lay hands-on as an inhaled substance (glue sniffing). It leads to significant acute neurological effects such as euphoria followed by depression. The toxicity of toluene has been reported in the literature and impacts any organ system (10, 11). It is metabolised via cytochrome p-450 into hippuric acid and benzocaine acid and is excreted through the urine (11–13). It can also result in rhabdomyolysis, myoglobinemia, distal renal tubular acidosis (RTA) and acute tubular necrosis (14–18). The other clinical conditions that arise are a fulminant hepatic failure, lung haemorrhage, solvent encephalopathy, solvent psychosis, cerebral infarction, cerebellar dysfunction, cardiac arrhythmias and death (19–26). In this series, we report our experience with AKI because of toluene-based paint reducers.

Case Series

Case 1

A 40-year-old male working in an automobile repair workshop presented with multiple episodes of vomiting and altered sensorium after intentional consumption of around 200 mL of toluene-based paint reducer. The patient was poorly cooperative. His pulse rate was 124/min, blood pressure was 130/80 mmHg and oxygen saturation on ambient air was 94%. The patient was initiated on intravenous normal saline. The blood pressure remained stable, and over the next 4 days, his serum creatinine increased from 1.1 mg/dL to 6.3 mg/dL with anion gap metabolic acidosis. His haemoglobin was 14 g/dL. Serum creatine phosphokinase, lactate dehydrogenase (LDH), sodium, potassium, calcium and phosphorus levels were normal. His Liver functions were normal. Ultrasonography of the abdomen and bland sediment in urine was also examined. The patient received four sessions of dialysis during the hospital stay. On day 10, his urine output improved to 2 L per day with no metabolic acidosis, and serum creatinine was 2.1 mg/dL. The patient was discharged and was asked to follow up at 4 weeks. On day 24, his serum creatinine was 1.2 mg/dL and was suggested for continuing psychiatry follow-up.

Case 2

This patient was a 20-year male working in the automobile repair unit who presented with complaints of nausea, myalgias and decreased urine output after having consumed around 150 mL of paint reducer. There was no history of loss of consciousness, seizures, drug abuse, trauma, vomiting, fever or loose stools. His blood pressure was 140/88 mmHg, with a pulse rate of 110 bpm. His clinical examination revealed mild tenderness of limbs. Investigations revealed normal blood counts: serum creatinine, 5.4 mg/dL; serum potassium, 6.2 meq/dL; sodium, 141 meq/dL. Threefold elevation of liver enzymes, normal bilirubin, serum calcium and phosphorus were normal. Normal anion gap metabolic acidosis with serum bicarbonate of 15 meq/dL with a positive urine anion gap was observed. The patient's Creatine phsophokinase (CPK) was 1040 U/L, and LDH was 560 U/L. Urine for myoglobin was negative. Rhabdomyolysis and AKI with RTA were induced by the suspected organic solvent. The patient was initiated with intravenous normal saline, received three sessions of haemodialysis and was given oral bicarbonate 0.5 meq/kg. Over the next 2 weeks, the patient's clinical condition improved. At discharge, the lab analysis revealed a serum potassium level of 3.9 meq/dL, serum bicarbonate of 24.2 meq/dL and creatinine of 1.4 mg/dL. The patient was seen by a psychiatrist, received behavioural therapy,continues to follow the treatment and is doing well.

Case 3

A thirty-two-year patient was missing from his home for 2 days and was found by his parents. The patient was a driver by profession and had previously undergone rehabilitation 2 years before for drug abuse behaviour. But patient had relapsed and had started sniffing toluene-based organic solvent. The patient had locked himself up in his car and consumed more than his usual dose and developed altered sensorium before being found by his family. On presentation, the patient was not oriented and was talking irrelevantly. His blood pressure was 110/90 mmHg with a pulse rate of 134/bpm. The echocardiography showed sinus tachycardia. Lab analysis revealed serum creatinine levels of 2.9 mg/dL, sodium of 150 meq/dL, potassium of 5.1 meq/dL, normal calcium and phosphorus (bland urine sediment). His CPK, LDH liver functions were normal. The patient had a urine output of 0.6 mL/kg/h and severe metabolic acidosis (normal anion gap) and received two sessions of haemodialysis, intravenous saline, oral bicarbonate replacement and dramatically recovered. On day 7 of his hospital stay, all parameters had normalised. Hence the patient was discharged and attached to a rehabilitation centre.

Discussion

Organic solvents, particularly toluene-based, have become a common occupational hazard and a frequent abused

substance. These cause acute neurological symptoms, severe metabolic abnormalities and associated organ injury. One of the predominantly observed side effects of toluene intoxication is metabolic acidosis. Organic solvents can also result in liver injury and rhabdomyolysis. Some risk factors that suggest poor outcomes include altered mental status, renal failure and severe acidemia. Although our patients had both organ impairment and altered mental status, they managed to do well (11).

Toluene causes glomerular and tubular damage, Fanconi syndrome and hematuria. Proteinuria is common, although it seldom is in the nephrotic-range levels. Occupational exposure of toluene in patients with established glomerulonephritis is a known risk factor for progression to end-stage renal disease. Direct induction of acute tubular necrosis and acute oliguric renal failure are also associated with toluene intoxication (5, 15, 16, 27–29). Rhabdomyolysis results either because of direct toluene muscular injury or prolonged immobility. They are usually seen with ingestion of toluene, but is reported in patients with thinner intoxication by inhalation (5, 30–32). Moreover, electrolyte abnormalities like hypokalemia and hypophosphatemia can also result in rhabdomyolysis and hence AKI.

The muscular phosphate depletion from severe hypophosphatemia impairs ATP production, which leads to muscle injury by altering the oxygen-haemoglobin dissociation curve (33, 34). Type 1 distal RTA which is characterized by an inability to lower urine pH, despite acidemia results, in hyperchloremic metabolic acidosis with hypokalemia. The degree of hypokalemia is also an important factor and can lead to rhythm abnormality and or hypokalemic paralysis. The primary suggested mechanism in toluene-induced type 1 distal RTA is an inability of the distal tubule to excrete hydrogen ions and an increased production of hippuric acid by toluene metabolism (16, 35). Management of acute toluene-based solvent toxicity is largely conservative, and the measures taken are influenced by the severity of organ involvement. Broadly it involves correction of the electrolytes, acid-base and fluid abnormalities. The management focuses on aggressive potassium replacement, maintaining hydration and close monitoring of the cardiac rhythm. Large doses of bicarbonate and potassium replacement are often needed. Patients should be closely observed for the development of organ dysfunction. Rhabdomyolysis should be ruled out in patients particularly, with impaired renal parameters. A timely introduction of renal replacement therapy is needed. Mild transaminitis, which is reversible, has been previously reported in the literature (10, 32).

Conclusion

Organic solvent exposure may result in acute tubular necrosis, rhabdomyolysis, RTA and AKI irrespective of the route intake. Clinical suspicion of organ dysfunction and failure and timely induction of supportive care leads to good outcomes.

Conflict of Interest

No potential conflict of interest was reported by the authors.

Funding and Support

The authors did not receive any funding for this study.

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