CASE REPORT

 γ -Benzene hexachloride poisoning leading to acute hepatorenal decompensation

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SUMMARY

γ -Benzene hexachloride is a commonly used insecticide of organochlorine group. Notable toxic effects include seizures, ataxia, confusion and other central nervous system dysfunction. A 30-year-old male farmer with suicidal ingestion of γ -benzene hexachloride poison developed acute intrinsic renal failure with azotaemia and diminished urine output along with features of acute fulminate liver failure. Renal function recovered with haemodialysis and liver parameters gradually normalised with time. As per our knowledge simultaneous acute hepatic and kidney injury associated with γ -benzene hexachloride poison is never reported in humans. The exact pathophysiology of this life-threatening complication is an enigma. However, oxidative stress has been postulated as a contributory factor as suggested in animal studies. Conservative management with successful outcome in our case stresses on the impact of watchful observation and aggressive management. He was discharged after 3 weeks at stable condition and was healthy at 3 months follow-up.

BACKGROUND

γ -Benzene hexachloride (GBH) or popularly known as gammaxene is a commonly used insecticide in rural as well as urban households and belongs to organochlorine compounds. Accidental ingestion is not uncommon among children and toxic manifestations comprise chiefly of central nervous system involvement. Acute liver or kidney injury in relation with GBH poisoning rarely reported but simultaneous failure of both these organs in acute GBH toxicity is never reported in literature. Anticipating this unusual yet life-threatening systemic toxicity in the background of GBH poisoning is required from healthcare providers and clinicians. As a corollary, in case of unknown poisoning with acutely precipitated hepatorenal decompensation, GBH poisoning should be ruled out. Moreover, absence of definite antidote and potential exacerbation of symptoms if treated with atropine, need to be addressed by primary care physicians. The triumph of conservative management in our case stresses on the impact of vigilant monitoring and aggressive management which can be lifesaving even in worse situations.

CASE PRESENTATION

A 30-year-old male farmer from rural India presented to the emergency department 6 h after ingestion of 50 mL of GBH as a suicidal attempt (Scabipid containing GBH 2% with cetrimide 0.5%

in emulsion base). There was no history of any other substance abuse. He had no known physical or psychiatric illness. There was no fever, altered sensorium, convulsion or any focal neurodeficit. The patient was given gastric lavage with activated charcoal. His vitals were stable at presentation. General as well as systemic survey including thorough neurological examination at the time of admission was unremarkable. Initial blood reports revealed a haemoglobin of 9 g/dL with total leucocyte count of 6800/mm³ (neutrophil 70%; lymphocyte 26%) and platelet count of 2.8 lac/mm³. Metabolic profile including electrolytes, renal and liver function parameters were within normal limits at the time of initial presentation.

INVESTIGATIONS

From the second day, the patient reported lethargy and nausea. Gradually his urine output started to decrease. It was 1200 mL/24 h on third day and by fifth day it was 200 mL/24 h. Mild dyspnoea started to appear along with worsening nausea. Elevated jugular venous pulse, bilateral basal crepitations in chest and diffuse abdominal tenderness jeopardised the patient. His sequential metabolic profile monitoring elucidated rapidly rising azotaemia, bilirubin and hepatic enzymes (table 1). P time was 33 s (control 11) with an international normalised ratio of 3.1. Ultrasonography of abdomen revealed raised cortical echogenicity of kidneys as well as mild ascites. Urine analysis revealed pus cells 8–10/high power field (HPF), red cells 10–14/HPF and protein 2+. There was no cast or crystals present. Arterial blood gas analysis revealed pH of 7.3 with bicarbonate 18 mEq/L (normal: 22–26 mEq/L). Viral markers of Hepatitis B, C and HIV infection were non-reactive. Immunological tests like complement levels (C3), antinuclear antibodies and antineutrophilic antibodies were unremarkable.

DIFFERENTIAL DIAGNOSIS

- Acute kidney injury
- Acute fulminant liver failure
- Leptospirosis
- Sepsis

TREATMENT

In the setting of acute kidney injury the patient was sent for immediate haemodialysis (HD). He underwent four sessions of HD over a week. Thereafter his urea and creatine gradually came down to normal within 3 weeks. Repeated liver function

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Table 1 Blood parameters in temporal profile

Parameter		Day 1	Day 3	Day 4	Day 5	Haemo-dialysis started	Day 8	Day 12	Day 15	Day 20
Kidney function tests	Urea (mg%)	40	67	80	106		NA	77	62	40
	Creat (mg%)	0.9	2.7	6.9	10.2		NA	3.6	2.1	1.3
	UO (mL)	1600	1200	800	200		1000	1200	1400	2000
Liver function tests	Bili (mg%)	0.8	1.6	2.6	4.1		6.0	4.3	NA	1.6
	AST (U)	31	128	339	467		400	140	NA	46
	ALT (U)	28	106	304	455		129	80	NA	56

ALT, alanine transaminase; AST, aspartate aminotransferase; Bili, bilirubin; Creat, creatinine; NA, not available; UO, urine output.

tests revealed gradual decrease of bilirubin and liver enzymes within hospital stay.

OUTCOME AND FOLLOW-UP

From the 10th day onwards, his urine output began to improve gradually and by day 15, it was 1400 mL/24 h. By 20th day, his urea and creatinine came as 48 and 1.3 mg/dL, respectively. His liver function also became normal. He had no more symptoms. He was finally discharged after 3 weeks at stable condition and at 3 months' follow-up, he did not develop any new complication.

DISCUSSION

GBH or popularly known as gammaxene is a commonly used insecticide in rural as well as urban households and belongs to organochlorine compounds. Usually, organochlorine compounds act as gamma amino butyric acid antagonists and block different voltage-gated ion channels in the central nervous system.¹ This causes release of excitatory neurotransmitters and agitation or seizures may be precipitated. Organochlorine compounds like GBH tend to be retained in lipid-rich tissues of the body and thus, blood levels often underestimate their body stores.¹ Common manifestations of toxicity of GBH include dermatitis, seizures, ataxia and confusion. Very rarely rhabdomyolysis, pancreatitis or disseminated intravascular coagulation (DIC) can occur.

Hepatic manifestations are rare and its exact mechanism has not been elucidated.¹ However, GBH is believed to interfere with hepatic oxidative metabolism and glutathione action. Also, there is alteration in Mg ATPase activity in liver tissues that may contribute to liver dysfunction.²

Cases of acute renal failure in GBH poisoning are also reported rarely in literature and mostly are related to rhabdomyolysis or DIC following GBH poisoning.³⁻⁴ Animal experiments have shown that GBH toxicity can cause lysosomal overload in renal proximal tubular cells with α -2 globulin chemical complex. It causes epithelial cell necrosis in cortical tubules due to oxidative damage.⁵ The clinical scenario in our patient

was also suggestive of acute intrinsic renal failure. Our patient did not develop myolysis or DIC.

There is no specific treatment of GBH poisoning. Symptomatic management, especially for seizures and myocardial depression is needed. Dopamine may be needed for hypotension. Cholestyramine has been used in some cases to prevent absorption of the toxin after oral ingestion.² In our case the patient was treated for renal failure with HD. Liver dysfunction improved spontaneously.

Learning points

- ▶ γ -Benzene hexachloride poisoning can present with acute hepatorenal failure.
- ▶ Potential exacerbation of symptoms if treated with atropine or pralidoxim.
- ▶ Watchful observation and aggressive management with haemodialysis can be lifesaving for the patient.

Contributors RP and RB were the treating physicians. GS reviewed literature. AT and RP wrote manuscript.

Competing interests None.

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