Reminder of important clinical lesson

Brake fluid toxicity feigning brain death

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Summary

Brake fluid (glycol-based) toxicity is known to have a protean of central and peripheral nervous system manifestations. The principal component of this household poison is ethylene glycol. Toxic effect is generally attributed to peri-vascular deposition of calcium oxalate crystals in various tissues. However, clinical features resembling brain death have rarely been reported. We report a case of brake fluid toxicity simulating brain death in a 21-year-old healthy man who ingested it as a recreational agent.

BACKGROUND

Brake fluid constitutes ethylene glycol, the toxicity of which has been known for decades. But poor or inadequate history of its exposure may bewilder caring physicians, due to its diverse clinical presentation. A rule of thumb is the presence of high anion, high osmolar gap metabolic acidosis, that should prompt us to look for serum ethylene glycol exposure. In the literature, varied clinical manifestations have been mentioned. But a clinical state mimicking brain death has been reported only once.¹ Our patient progressively lost all brain stem reflexes and sensory motor nerve conduction. It was only through electroencephalographic studies that intact cortical function was revealed. This case is a rare scenario and emphasize the role of electroencephalogram in detecting brain function.

CASE PRESENTATION

A 21-year-old man presented with a 3-day history of recurrent vomiting, bilateral flank pain, headache, somnolence and decrease urine output. He was found to be in acute renal failure. Haemodialysis was started. However, no improvement was observed in renal function. Neurological examination showed that he was awake, alert, oriented, with good motor power, essentially no focal deficits. In subsequent days, he developed bilateral sensorineural deafness, facial palsy, fixed dilated pupils, with normal extraocular eye movements. It was on day 9 that the history of brake fluid ingestion was disclosed by the family members. He became areflexic by that time, but had good motor power. By day 10, he developed respiratory failure requiring transfer to the intensive care unit. He was intubated and started on mechanical ventilation. Examination on day 13 showed an absence of motor response to external stimuli, though he was not on any sedatives, and also an absence of all brainstem reflexes.

INVESTIGATIONS

At presentation his renal function was deranged. His blood urea was 35.5 mmol/l, creatinine 300 µmol/l, Na-131 mmol/l and K-3.8 mmol/l. His serum osmolality was 330 mosmol. His osmolar gap was 20 mOsm/kg and anion gap was

38 mEq/l. Arterial blood gas showed pH 7.15, PCO₂ 31 and HCO₃ 13.8. Although relevant, his urine was not examined for calcium oxalate crystal. His septic work-up was unremarkable. His CT brain was normal. Cerebrospinal fluid analysis on day 7 showed 47 white cells (lymphocytic predominance); protein was 2.27 g/l along with normal glucose. Nerve conduction study on day 8 showed an absence of motor or sensory response and this was reported as severe axonal sensorimotor polyneuropathy (figures 1 and 2). EEG on day 20 showed diffuse theta/delta slowing down with superimposed beta activity (figure 3). His MRI of the brain did not show any abnormality (figure 4).

DIFFERENTIAL DIAGNOSIS

A clinical scenario of acute renal failure with subacute onset cranial neuropathy, bulbar and respiratory muscle failure persuaded us to ponder about various clinical conditions. Guillain-Barré syndrome (GBS) was our top differential diagnosis. Although the patient had high protein in the cerebrospinal fluid, high white cell count was clearly contradicting the common entity of albumino-cytologic dissociation of GBS. Moreover, electrophysiological finding of no motor or sensory response was not quite befitting that diagnosis. Acute pandysautonomia was another differential, as the patient had evidence of cholinergic dysautonomia, that is, fixed dilated pupil, sensory neuropathy, absent corneal reflex, all of which are known to be associated with this condition. However, loss of other brainstem reflexes was not in accord with pandysautonomia. Botulism could have been considered; however, there was no history suggestive of exposure to botulinum toxin. Moreover, electrophysiological study was not in accord with botulism. Tick paralysis was a differential that needed to be considered. In this condition, one may get hypoactive to absent reflexes, bulbar paralysis and respiratory failure. In our case there was no history of exposure or visit to tick endemic areas. From the clinical stand point there was also no history of characteristic ascending paralysis of tick paralysis. We also thought of acute presentation of neuromuscular disease, that is, myasthenic crisis. However, electrophysiological study did not suggest

Figure 1 Sensory nerve conduction study of the right ulnar nerve shows an absence of response (NR).

that. Bare intoxication with diverse agents like ethanol, methanol was excluded by normal toxicology screen. However, as our lab's routine toxicology screen does not include ethylene glycol, it was not checked in this patient's blood upon admission. However, on day 9, when the history of exposure to ethylene glycol was revealed to us, we felt that it was too late to check its level, as the

patient by then had already undergone several sessions of haemodialysis. Hence, its level was not requested for. Severe metabolic encephalopathy from acute renal failure of unknown cause could not be fully attributed to the whole scenario. Finally, locked/pseudo-locked-in syndrome from varied aetiology was also kept in mind. As the patient had no neuroimaging evidence of brainstem

Figure 2 Motor nerve conduction study of right median nerve shows an absence of response.

Figure 3 EEG: a standard longitudinal bipolar montage showing diffuse theta and delta slowing with superimposed beta activity.

insult, true locked-in state was excluded. But pseudo-locked-in state from de-efferentation was well thought out.

TREATMENT

The patient presented with florid renal failure. Hence, he required regular haemodialysis to stabilise his renal function. Clinical finding of areflexia with initial nerve conduction studies showing severe axonal polyneuropathy led us to offer him intravenous immunoglobulin, five doses of which he received, thinking it may all be due to GBS. He did not show any response. As no history of illicit drug usage was obtained at the beginning of the illness, specific therapy for ethylene glycol intoxication was not offered.

Figure 4 Brain MRI axial cut fluid attenuated inversion recovery sequence shows normal appearance of white and grey matter and a normal ventricular system.

OUTCOME AND FOLLOW-UP

He began to regain his brainstem functions after about 2 months. At 9 months into his illness, he was discharged from the rehabilitation hospital, as he showed remarkable functional improvement. His latest examination revealed normal cognitive status, with bilateral sensorineural deafness, mild quadraparesis but he is able to ambulate independently. Neurological examination demonstrated a mildly dilated non-reactive pupil, with muscle power 4/5 and areflexia. His repeat nerve conduction studies showed low-amplitude compound motor action potential in median and ulnar nerves.

DISCUSSION

Brake fluid is a common agent used in industry and in automobiles for its ability to absorb water and to prevent overheating or freezing.² Ethylene glycol is the main constituent of brake fluid; reports of its potentially lethal effects if ingested first appeared soon after its widespread introduction in the 1930s. Widespread availability contributes to its popularity as a suicidal agent, and it is a common cause of accidental poisoning in children. The 1998 annual report of the Poison Control Centers Toxic Exposure Surveillance System reported 5376 exposures to ethylene glycol.³ It has little endogenous toxicity. Its half-life is about 7–10 h.⁴ Its toxic effect is actually derived from its metabolic products glycoaldehyde, glycolic and oxalic acids, which are highly toxic. These products stay in the body for many days. The lethal dose is estimated to be 100 ml.⁵ Oxalic acid binds with calcium to form calcium oxalate crystals that may deposit and thereby exert its toxic effect to different parts of the body including the brain, heart, kidneys and lungs. Oxalate crystals are commonly seen in urine examination of such affected patients. Once ingested, it simulates central nervous system toxicity of ethanol except that it does not exhibit the characteristic odour of ethanol.⁶ So usual initial

symptoms include confusion, lethargy, hallucination, slurred speech and ataxia.⁴ Rarely, seizures and tetany can occur. At the same time, acute renal failure with high anion and high osmolar gap metabolic acidosis sets in. So far published reports on central nervous system toxicity of ethylene glycol focus on the involvement of basal ganglia and brain-stem, with concordant brain imaging findings.⁷ Multiple cranial neuropathy occurring in ethylene glycol poisoning is reported in several case reports.^{8,9} Among them, facial diplegia and sensorineuronal deafness are frequently mentioned.⁸ But a state of clinical brain death with no associated imaging finding was found in only one case report.¹

Learning points

- ▶ Even without a history of intoxication, the presence of high anion, high osmolar gap metabolic acidosis should prompt one to search for ethylene glycol ingestion.
- ▶ In the absence of clinical brain stem reflexes, electroencephalograph may be the only indicator of brain function.
- ▶ De-efferentation is a recognised complication of ethylene glycol intoxication.
- ▶ Appropriate supportive care can allow near-complete functional recovery.

Competing interests None.

Patient consent Obtained.

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