Letters to Editor

Survival after fatal pentobarbital ingestion

Sir,

Pentobarbital is a drug used in veterinary medicine for anaesthesia and euthanasia. Acute poisoning can be fatal due to cardiogenic shock, and respiratory arrest secondary to its potent action on the respiratory centre. The lethal dose for pentobarbital when ingested ranges from 2 to 10 g.[1] A case report of non-fatal pentobarbital overdose of 13 g is described.

A young male, working in the field of equine transport, presented to the hospital after being found unconscious, with empty vials of pentobarbital amounting to a total of 13 g. He had a respiratory arrest at scene and was intubated and transferred to hospital without the need of any sedation or muscle relaxant. On arrival, his core temperature was 32° C and his Glasgow coma score (GCS) remained 3/15 after administration of 1.6mg of naloxone. He had no pupillary or cough reflex present at that time. He had a blood pressure of 50/20 mmHg and had no other injuries. He was subsequently transferred to the intensive therapy unit where full invasive monitoring was established. He was slowly warmed up with warming blankets. Clinically, there were signs of aspiration, and a bronchoscopic lavage was performed prior to starting broad-spectrum antibiotics. The initial arterial blood gas (ABG) showed good oxygenation with lactic acidosis (pH 7.31, lactate 5). Urine toxicology screen was negative and serum paracetamol and salicylate levels were undetectable. Phenobarbital levels were undetectable, but local laboratories were not able to process pentobarbital levels. The nearest toxicology centre was consulted and activated charcoal lavage was started. They further advised a CT scan of the head if neurology did not improve within the next 24 hours. Over the next 12 hours, noradrenaline and dobutamine were commenced to target a mean arterial pressure of 65 mmHg. To enhance excretion, urinary alkalinisation by intravenous administration of 8.4% sodium bicarbonate was instituted and the urine output was targeted at >1 ml/kg/hour. This was stopped after 24 hours, as was activated charcoal lavage at 48 hours as advised by the toxicology unit. Nasogastric feed was subsequently started. GCS remained 3/15 despite no sedation. After the first 48 hours, the patient started having myoclonic jerks of his right upper limb, which was thought to be due to hypoxic brain injury. A single dose of lorazepam (4 mg) was administered and loading dose of phenytoin (1 g) was given. The CT scan of his head was normal. These movements continued despite treatment with phenytoin and lorazepam. At day 3, his cardiovascular function started improving and inotropes were successfully weaned off. However, his GCS remained 3/15 without sedation for 120 hours (5 days) post overdose, with the patient making no spontaneous respiratory efforts. An attempt was again made to process pentobarbital levels at various laboratories in the country, but the idea was given up due to cost and time restraints. On day 6 of admission, the patient started making spontaneous respiratory breaths and the myoclonic jerks stopped. He had not required any further inotropic support during this time, was absorbing the enteral feed, had normal renal function and his GCS had improved to 9/15. In view of this, his trachea was extubated. Post extubation, his GCS fluctuated between 7 and 13 and he found it difficult to clear his secretions. He became increasingly tachypnoeic as the day passed and had to be re-intubated. A second bronchoscopy was performed and secretions cleared. Overnight, he was commenced on a propofol and remifentanil infusion for tube tolerance. The next day after sedation hold, he was successfully extubated and later discharged to the ward.

Barbiturates are weak acids used as sedatives, hypnotics, induction agents and anticonvulsants.[2] Pentobarbital is a short acting barbiturate that is well absorbed, and its duration of action ranges from 4 to 8 hours with a half life of 15 to 50 hours.[3] It is rarely used in human beings in the UK. Overdose in human beings is therefore rare and should be suspected in patients who present as barbiturate overdose and work in the veterinary field. Pentobarbital overdose can lead to airway compromise, aspiration, cardiovascular collapse, sedation, coma, respiratory or cardiac arrest and death.[4] Treatment consists of airway protection, haemodynamic support, treating superadded infections and slow rewarming.[5] In early overdose, there is some role of gastric lavage with activated charcoal.[6] Forced alkaline diuresis and haemodialysis are of limited use as this drug is highly protein bound and lipid soluble.[7]

In this case, we were presented with a patient who had

taken a fatal barbiturate overdose. The unfamiliarity with the drug led us to treat it as phenobarbital overdose which was similar, but less effective. None of the local laboratories were able to process the pentobarbital levels due to the rarity. The patient had cardiac, respiratory and central nervous system failure from the overdose. The myocardial depressant effects of pentobarbital were marked, lasting more than 48 hours, but resolved first. Respiratory drive returned with spontaneous breathing on day 6. Central nervous system was the last to recover, which though delayed was complete. Although a suspicion of global hypoxic/hypoperfusion brain injury was raised earlier in the course of treatment, there was no functional deficit noticed on extubation. This was possibly also due to the neuroprotective effect of barbiturates.[8] The patient recovered without any neurological sequelae.

Vinodkumar Singh

Department of Anaesthesia and Intensive care, West Suffolk Hospital, NHS Foundation Trust, Bury St Edmunds, UK

Address for correspondence:
Dr. Vinodkumar Singh,
23, Airfield Road, Bury St. Edmunds, Suffolk, IP32 7PJ, UK.
E-mail: drvinbing@gmail.com

REFERENCES

1. Cravey RH, Reed D, Sedgwick PR, Turner JE. Toxicologic data from documented drug-induced or drug-related fatal cases. Clin Toxicol 1977;10:327-39.
2. Vickers MD, Stewart HC, Wood-Smith FG. Drugs in Anaesthetic Practice, 5th ed. London: Butterworth Heineman; 1978. p. 36-119.
3. Breimer DD. Clinical pharmacokinetics of hypnotics. Clin Pharmacokinet 1977;2:93-109.
4. Healy TE, Knight P, editors. Wylie and Churchill-Davidson's: A Practice of Anaesthesia. 7th ed. London: Arnold; 2003. p. 1255-65.
5. Hadden J, Johnson K, Smith S, Lawrence P, Giardina E. Acute Barbiturate Intoxication: Concepts of Management. JAMA 1969;209:893-900.
6. Sellers EM, Khouw V, Dolman L. Comparative drug adsorption by activated charcoal. J Pharm Sci 1977;66:1640-1.
7. Bloomer HA. Limited usefulness of alkaline diuresis and peritoneal dialysis in pentobarbital intoxication. N Engl J Med 1965;272:1309-13.
8. Kawaguchi M, Furuya H, Patel PM. Neuroprotective effects of anesthetic agents. J Anesth 2005;19:150-6.

Access this article online	
Quick response code	Website: www.ijaweb.org
	DOI: 10.4103/0019-5049.126838