CASE REPORT AND CLINICAL REVIEW

Methiocarb poisoning of a horse in Australia

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Snail bait poisoning is rare in horses. Cases have been reported, but clinical signs and subsequent prognostic indicators have been poorly documented and must be extrapolated from cases in companion animals. We describe in detail the poisoning of a horse that consumed a lethal dose of the carbamate, methiocarb. There are currently no guidelines for treating equine methiocarb toxicoses, but the principles of management are based on supportive therapy.

Keywords horses; methiocarb; poisoning; snail bait

Abbreviations
ACh, acetylcholine; LD₅₀, median lethal dose; PCV, packed cell volume; TP, total serum protein

Methiocarb and metaldehyde are the two predominant molluscicides used in Australia to control snail and slug populations in domestic and commercial gardens. Methiocarb (3,5 dimethyl-4-phenyl-N-methyl-carbamate) inhibits the activity of acetylcholinesterase and thereby increases the concentration of the neurotransmitter acetylcholine (ACh) within the synaptic cleft of cholinergic synapses (autonomic ganglia, parasympathetic neuroeffector synapses and the neuromuscular junction). It is usually prepared in a blue cereal-based pellet with a 2% w/v concentration (Baysol® Snail and Slug Bait, Bayer Australia, Pymble, NSW, Australia). Methiocarb poisoning is well documented in the companion animal literature, but cases of equine methiocarb poisoning have been documented twice previously, with one horse dying approximately 12 h after ingestion and the other horse surviving. To our knowledge, this is the first reported case of methiocarb poisoning of a horse in Australia.

Case report

A 10-year-old Stock Horse gelding presented to the Goulburn Valley Equine Hospital with a history of consuming approximately 1 kg of Baysol® Snail and Slug Bait (20 g/kg methiocarb; Figure 1). The horse was in good body condition and weighed approximately 400 kg. Clinical signs reported by the owners included profuse sweating, lateral recumbency, paralysis of the distal right hindlimb, generalised muscle tremors and hypersalivation. These clinical signs commenced within 2 h of ingestion. On presentation (approximately 6 h after ingestion), the horse was quiet but responsive. It was ambulatory but required a plantar splint extending from the heel to the hock to prevent ‘knuckling’ of the right hind fetlock. There was no history of a traumatic event. Muscle fasciculations of the hindquarters were initially observed, but resolved quickly. Abnormalities in the initial physical examination included moderate hypersalivation (Figure 2), tachycardia (56 beats/min) with no discernible murmurs and markedly reduced to absent gastrointestinal borborygmi in all four quadrants. The horse had signs of colic, evidenced by pawing at the ground and was also moderately pyrexic with a rectal temperature of 40.2°C.

Within 10 min of presentation, jugular blood samples were collected into EDTA and heparin tubes for routine haematology and clinical chemistry. Abnormalities detected were mild hyperbilirubinaemia (127 μmol/L; reference range 4–100 μmol/L), hyperproteinaemia (100 g/L; reference range 58–76 g/L), hyperalbuminaemia (46 g/L; reference range 28–38 g/L) and hyperglobulinaemia (54 g/L; reference range 22–40 g/L). Hepatobiliary enzymes were also increased, with a detected alkaline phosphatase of 271 U/L (reference range 50–250 U/L), aspartate transaminase of 5245 U/L (reference range 150–400 U/L), gamma-glutamyl transferase of 108 U/L (reference range 20–38 U/L) and glutamate dehydrogenase of 250 U/L (reference range <21 U/L). Serum creatinine (585 μmol/L; reference range 110–170 μmol/L) and urea (15.5 mmol/L; reference range 3.6–8.9 mmol/L) were also mildly elevated. The horse was mildly hypochloremic (81 mmol/L; reference range 98–110 mmol/L), had an elevated haemoglobin and increased haematocrit (65%; reference range 32–52%). These findings were consistent with mild to moderate dehydration. The total white cell count was within normal limits. There was a significant increase in creatine kinase (64,744 U/L; reference range 22–40 IU/L), which was consistent with muscle damage induced by the reported recumbency event and generalised muscle fasciculations.

Ultrasonographic evaluation of the abdomen was performed using a 3-MHz curvilinear transducer. The small intestine had generalised decreased motility, with mildly thickened intestinal walls. Several small bright hyperechoic foci were seen within the lumen of the small intestine (Figure 3).

A nasogastric tube was passed and 8 L of reflux was obtained. The contents were turquoise in colour and although pellets were not visualised, the colour was consistent with the blue dye used in the Baysol® Snail and Slug pellets. A diagnosis of methiocarb toxicity was made, based on the history, clinical signs and refluxed contents.

The horse was placed on prophylactic antibiotics: procaine penicillin (22,000 IU/kg IM twice daily (Bomacillin, Bomac Animal Health, Hornsby, NSW, Australia)) and gentamicin sulfate (6.6 mg/kg IV once daily (Gentam 100, Troy Laboratories, Smithfield, NSW, Australia)). Flunixin meglumine (1.1 mg/kg IV twice daily (Flunixin, Troy Laboratories)) was administered as an antiinflammatory and the horse was maintained nil per os and refluxed as required. Within hours of presentation, the horse began to deteriorate...
noticeably. Its mucous membranes were initially injected, but then became muddy. The capillary refill time was more than 2 s and the heart rate increased to and remained consistently between 60 and 68 beats/min. The respiratory effort and rate increased to 32 breaths/min. Gastrointestinal borborygmi were absent on auscultation and the horse demonstrated increasing signs of colic, which included increased pawing at the ground and restlessness.

A 14-gauge catheter (MILA International Inc., KY, USA) was placed in the right external jugular vein and intravenous fluids were commenced. An initial bolus of 20 L of Lactated Ringer’s Solution (Baxter, Old Toongabbie, NSW, Australia) was followed by a constant rate infusion of 3 L/h. A lignocaine constant rate infusion was also commenced as a prokinetic for the treatment of ileus. A loading dosage of 1.3 mg/kg over 15 min was followed by a constant rate infusion of 0.05 mg/kg/min.

Within hours of commencing the intravenous fluids, flunixin meglumine and lignocaine therapy, the horse became more comfortable, with a heart rate varying between 46 and 56 beats/min, resolution of the pyrexia and a decrease in colic signs. However, the ileus and associated reflux continued and by 12 h post-presentation, 24 L net reflux had been obtained via nasogastric intubation.

By 36-h post-admission, the horse was stable but continued to show signs of poisoning. The heart rate continued to vary between 46 and 56 beats/min, mucous membranes were pink with a mild hyperaemic margin and capillary refill time was >2 s. Nasogastric reflux continued at a rate of approximately 7–15 L every 6 h (total reflux volume by 36 h after presentation was 55 L), with the refluxed material returning to a more normal colour and consistency. The horse remained quiet and appeared to have normal mentation. It was seen to urinate several times but not defecate. There was still an absence of gastrointestinal borborygmi on auscultation. Packed cell volume (PCV) and total serum protein (TP) at this stage were 52% and 72 g/L, respectively, indicating mild haemoconcentration.

At 48 h after admission, the horse began to deteriorate again, becoming increasingly dull and lethargic, but with intermittent periods of restlessness, hyperaesthesia and hyper-responsiveness to auditory stimuli. The horse’s mucous membranes became injected, with capillary refill time >3 s. The PCV and TP both increased to 68% and 8.6 g/L, respectively, despite the ongoing intravenous fluid therapy. Because of its poor progression and poor response to therapy, euthanasia was advised and agreed to by the owners.

The horse was euthanased by intravenous injection of barbiturate. A necropsy was performed. The stomach was intact and the contents consistent with the later nasogastric reflux material obtained. The small intestines were distended with mildly thickened walls, consistent with the initial ultrasonographic findings. The small intestinal contents were of normal colour and consistency, but in some areas were tinged with blue. Pellets were not visible. The heart, lung, liver, spleen and kidneys all appeared grossly normal. No tissue samples were submitted for toxicological analysis, as rapid oxidation of methiocarb would have rendered tissue residue levels undetectable.5

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Figure 1. The box of ‘Baysol Snail and Slug Bait’ consumed by the horse.

Figure 2. Hypersalivation at the time of presentation.

Figure 3. Abdominal ultrasound showing hyperechoic foci within the small intestine.
Acetylcholine is a neurotransmitter in both the autonomic (ganglionic transmission and parasympathetic post-ganglionic transmission) and somatic (neuromuscular junction) nervous systems. Thus, methiocarb poisoning, with inhibition of the activity of acetylcholinesterase, can have wide reaching consequences for the animal and it may be difficult to predict accurately the clinical presentation and progression of equine methiocarb toxicity. In this case, the horse demonstrated diverse clinical signs that could be attributed to involvement of parasympathetic, sympathetic and somatic nervous systems. In contrast to the companion animal literature concerning organophosphate and carbamate toxicity, the horse did not exhibit any forms of increased gastrointestinal activity. Instead, no faeces were seen to be passed throughout the treatment period. Neurological deficits were diverse, varying from localised palsy to generalised muscle fasciculations and altered mentation.

Discussion

In contrast to the organophosphates, methiocarb binds reversibly to acetylcholinesterase. Thus, it is possible for animals to recover spontaneously. The accumulation of ACh causes prolonged depolarisation of nicotinic receptors (in sympathetic and parasympathetic ganglia and at neuromuscular junctions) and of muscarinic receptors in the parasympathetic nervous system. Clinical signs of carbamate toxicity in the dog are thus varied and pertain to the degree that nicotinic and muscarinic receptors are affected. Initially, parasympathetic signs are seen because of the overstimulation of muscarinic receptors located primarily in the heart, eye, gastrointestinal tract and smooth muscle. Associated clinical signs may include hypersalivation, lacrimation, polyuria, increased gastrointestinal activity leading to defecation and diarrhoea, emesis, increased bronchial secretions, bradycardia and miosis. Clinical signs associated with the increased stimulation of sympathetic nicotinic cholinergic receptors may include tachycardia, tachypnoea, mydriasis, peripheral vasosconstriction and gastrointestinal relaxation. Clinical signs associated with the excessive stimulation of nicotinic somatic neuromuscular junctions may include muscle stiffness, fasciculations, tremors, ataxia and paralysis, whereas signs associated with the nicotinic receptors within the central nervous system include restlessness, anxiety, depression and seizures. It is of note that diarrhoea and increased gastrointestinal activity in companion animals is well documented, but has never been documented in cases of equine carbamate toxicity. The signs observed in the horse presented here could be explained by stimulation of both the autonomic and neuromuscular cholinergic receptors.

The ongoing and worsening clinical signs can be explained by the continued absorption of methiocarb. Studies of dogs undergoing gastric lavage on presentation have failed to demonstrate a substantial rate of recovery of ingested poison, especially if the lavage was delayed for more than 60 min. The clinical effectiveness of gastric lavage in horses after the ingestion of a toxic substance has not been assessed. However, it is likely to be beneficial only if performed shortly after ingestion. In this case, gastric lavage was performed approximately 6 h post-ingestion.

Atropine sulfate is a parasympatholytic. It is a competitive antagonist of ACh, binding to muscarinic receptors and thus decreasing parasympathetic stimulation. Nicotinic receptors are not affected by atropine sulfate. It is therefore unlikely that the presumptive sympathetic-induced ileus would have benefited from the administration of atropine. Gastrointestinal stasis can also cause prolonged retention and, thus absorption, of toxic material. Atropine sulfate has been used to reduce parasympathetic signs of bradycardia, hypersalivation, excessive airway excretions and profuse diarrhoea in companion animals with methiocarb poisoning. Administration was considered in the present case, even though the literature suggests that the dose rate and time of administration are not as important compared with that of the companion animal literature. However, atropine sulfate was not administered because of the absence of bradycardia and the previously resolved hypersalivation. It was also deemed contraindicated in this case by the lack of gastrointestinal borborygmi and the presence of nasogastric reflux. It is of interest that in both of the previous reports in horses atropine sulfate was used. Alexander administered 9 mg of atropine intramuscularly approximately 8 h after ingestion. The horse survived, but it can be argued that atropine was administered to an already stabilising animal. Edwards administered repeated doses of atropine sulfate intravenously, with 26 mg administered within 6 h of methiocarb ingestion (20 mg within the first 70 min). A unique clinical feature of this present case compared with the previous reports is the presence of gastrointestinal reflux. Reflux is a common indicator of gastrointestinal disease in the horse. It is hypothesised that the reflux occurred because of local irritation to the stomach resulting in gastritis and/or a more generalised ileus secondary to the overstimulation of sympathetic nicotinic cholinergic receptors. It is unknown why previous cases demonstrated no gastrointestinal involvement, but it may be related to the dose of toxin. Furthermore, it is also possible that there is an interspecies variation in the muscarinic and nicotinic receptors of the equine and canine gastrointestinal tracts, because diarrhoea as a clinical sign has not been reported in any of the equine cases.

Activated charcoal is a safe and commonly used adsorbent for a wide variety of ingested toxins, but was not administered to this horse because of the significant amount of nasogastric reflux. In addition, its efficacy in adsorbing poorly water-soluble compounds, such as methiocarb and metaldehyde, is debatable. The exact amount of methiocarb ingested is unknown. Although the owner reported that the box was previously unused and unopened, the horse was unlikely to have consumed the entire 1 kg of pellets. The box was considerably damaged and contained no contents on presentation. The owner stated there were very few residual pellets in the yard where the horse was found and therefore it is assumed that the horse consumed the majority of the box’s contents.

In previous case reports, an outcome was determined within 12 h of methiocarb ingestion. The affected horse either died 12 h after consumption or recovered uneventfully. The doses of ingested methiocarb varied. In the fatal case, 5 g methiocarb (125 g of 4% w/v) was consumed, whereas the horse that survived had consumed 4 g methiocarb (100 g of 4% w/v). The horse in this case report ingested 20 g of methiocarb (1 kg of 2% w/v), but did not demonstrate the reported severe clinical signs nor was there resolution of the toxicity within 12 h. There is currently no equine median lethal dose (LD₅₀) for methiocarb toxicity. An LD₅₀ in dogs of 18 mg/kg of methiocarb has been reported, so extrapolating from this, the LD₅₀ in a 500-kg horse would be 9 g of methiocarb. The horse in this case report consumed more than...
double this amount, whereas the dose in the previous reported cases was less than this. Although it would be advantageous to have an approximated LD₅₀ for horses, species differences in gastrointestinal function and pharmacokinetics make extrapolation difficult.

Elevation of hepatobiliary enzymes has not been previously documented in the horse. We speculate that the mild elevation could be secondary to detoxification.

The presenting signs of a methiocarb-affected animal vary considerably. Some possible factors that may contribute to the clinical signs at presentation and the clinical course are: time of presentation after methiocarb consumption, dose, the relative involvement of muscarinic versus nicotinic receptors and the degree of spontaneous reversion of the carbamate–acetylcholinesterase bond.

This is the first report of methiocarb poisoning of a horse in Australia. Because of the low incidence in horses, and the varying clinical signs and inconsistent prognoses, little is understood about this rare poisoning. Treatment protocols and LD₅₀ have been extrapolated from human and small animal toxicology and pharmacology. Equine clinicians should be aware of this potentially fatal source of poisoning in horses and further research is needed to successfully predict prognostic factors in future equine cases.

References


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BOOK REVIEW


This is the first edition of Low stress handling, restraint and behavior modification of dogs and cats: techniques for developing patients who love their visits. Its primary aim is to be a reference book for veterinarians and nurses to better manage and handle dogs and cats in a clinical setting. It is presented in a simple, easy-to-read format with an accompanying DVD and provides a practical outline of dog and cat behaviour for the small animal clinician.

The first section provides a guide to behaviour patterns and the interpretation of these patterns, while the latter sections cover animal handling, counterconditioning and preventative behaviour modification. Each technique described includes a photo guide demonstrating the techniques.

Low stress handling, restraint and behavior modification of dogs and cats presents, in very simple terms, how to understand interpret patient behaviours and how to deal with various common situations encountered in veterinary practice. There are examples of classic handling techniques we have all adopted over the years along with new ideas that may be of benefit to experienced practitioners and good advice to pass on to owners for training. This book has a place in all small animal veterinary practices that hire new graduates or junior nursing staff. It contains easy and quick reference diagrams to allow inexperienced staff to manage dogs and cats in the clinical setting. The table of contents allows you to easily identify the area you need, e.g. canine restraint for jugular venipuncture, and the book presents the handling techniques in a summarised form with easy to follow pictures.

The accompanying DVD provided complements the book nicely. It demonstrates the techniques with clear explanations that allow inexperienced handlers to better understand what is described, how animals behave and misbehave, dealing with difficult dogs and preventing problems. Low stress handling, restraint and behaviour modification of dogs and cats is interesting for both new and experienced veterinarians. It demonstrates new approaches to common problems in patient examination and treatment and is a great reference text for practices that hire new graduates. It would also be a great reference manual for practice nurses and technicians. I have not seen another resource book that explains dog and cat behaviours and handling in such a simple fashion.

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