Acute barium poisoning in a dog after ingestion of hand-held fireworks (party sparklers)

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Running title: Barium toxicity dog

Abbreviations

APTT – activated partial thromboplastin time

CK – creatinine kinase

ECG - electrocardiogram

ICP/MS - inductively coupled plasma/mass spectrometry

PCV – packed cell volume

PT – prothrombin time

SC - subcutaneously

TPP – total plasma protein

Abstract

Objective - To report a case of acute barium poisoning in a dog subsequent to ingestion of a common handheld pyrotechnic (sparkler).

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Case summary –A 5-year-old female neutered German shorthaired pointer presented with acute onset of generalized flaccid muscle paralysis and fasciculations, ptyalism and an irregular heart rhythm. Marked hypokalemia (1.9 mmol/L [mEq/L]; reference range [3.5-5.8 mmol/L [mEq/L]], acidemia (pH 7.20; reference range 7.38-7.44) and hypoventilation (PvCO₂ 55 mm Hg; reference range 40-50 mm Hg) were present on admission. Treatment consisted of fluid therapy, aggressive intravenous potassium chloride supplementation, gastric lavage and oral magnesium sulfate administration. Based on history and clinical presentation, barium intoxication after ingestion of combusted hand-held firework (sparklers) was suspected and a serum sample was submitted for barium analysis. The serum barium concentration determined by inductively coupled plasma/mass spectrometry was 2000 µg/L, a 3 orders of magnitude elevation above previously reported normal values in dogs. Within 18 hours of admission the clinical signs resolved and the blood potassium concentration normalized. The animal was discharged home 36 hours after admission. On follow up performed after 1 year and 5 years, no health issues were apparent.

New information provided – To the authors' knowledge, this is the first report of acute, life-threatening barium toxicosis characterized by flaccid paralysis, acidemia and severe hypokalemia occurring in a dog after ingestion of a popular pyrotechnic (sparkler) containing barium nitrate. Clinical signs may resolve within 24 hours with appropriate supportive care including aggressive potassium supplementation and chelation therapy.

Key words – Barium, intoxication, fireworks, hypokalemia, muscular weakness

Introduction

Barium is a highly toxic earth metal that does not exist in the elemental form in nature, but rather occurs in divalent cationic form and associated with other elements and with widely variable levels of water solubility.¹ Compounds with high water or acid solubility are of

greater toxicity after ingestion.² The insoluble barium sulfate is therefore safely used as contrast material in the field of medical imaging, while all other forms of barium may lead to intoxication after ingestion. In people, barium intoxication is often the result of occupational exposure to barium containing substances (eg, ceramics, mining, metal industry), occurs via accidental or intentional ingestion of household substances containing barium salts (eg, shaving powder, rodenticides [barium carbonate]), exposure to contaminated food or incorrectly formulated medical contrast solution, or consumption of barium containing objects, including fireworks (barium nitrate).³⁻¹³

Despite the almost ubiquitous presence of barium in our environment, the veterinary literature remains limited to brief reviews of the risks of barium toxicosis due to the toxicity of barium nitrate in fireworks, with only 1 previously reported case of barium intoxication in a dog.¹⁴⁻¹⁶ Characteristic clinicopathological features of barium poisoning are the acute onset and quick progression of flaccid paralysis coexisting with severe hypokalemia.¹⁷ Treatment consists of supportive therapy, aggressive potassium supplementation¹⁸ and chelation therapy.¹⁹ To the authors' knowledge this represents the first report of firework ingestion as the source of confirmed barium intoxication in the veterinary literature.

Case report

A 5-year-old, female neutered German shorthaired pointer (weight, 25 kg) presented for acute collapse, salivation and cold extremities. The owner, a veterinary general practitioner, reported the onset of clinical signs in the 8 hours prior to presentation, when the dog had commenced vomiting. Neurological signs in the form of ataxia, commenced 4 hours after the onset of gastrointestinal upset, with progression to acute collapse 8 hours after the onset of initial clinical signs. The owner was questioned about access to known toxins and recent medical history including medications and injuries however nothing relevant was

noted at that time. The dog was previously healthy, with unknown vaccination history and without tick prevention.

At admission, the dog was bright and alert but marked flaccid tetraparesis was evident. On cardiac examination, an arrhythmia with a heart rate of 60-80/min was auscultated. Although no diagnostic electrocardiogram (ECG) was performed, ventricular premature complexes were observed on the multiparameter monitors ECG^h. The remaining physical cardiovascular examination was normal and no respiratory abnormalities were identified. Mild hypothermia (37.3°C [99.14°F]) was detected at the time of admission. Reduced limb reflexes and low muscle tone affecting all extremities were present. Oral examination revealed an absent gag reflex and marked ptialism. Ocular findings included bilateral mydriasis, elevated nictitating membranes, reduced menace, intermittent nystagmus and reduced pupillary light reflex. Dark green/black feces was noted on rectal examination.

During the clinical examination (approximately 15 minutes post presentation), the clinical status of the dog further worsened; muscle twitching of facial muscles, including the tongue, panniculus muscle and eyelids, occurred and progression to flaccid paralysis occurred. Only minimal respiratory chest excursions were noted.

Initial diagnostic tests from blood collected at presentation included PCV, total plasma protein (TPP), serum biochemistry (including creatine kinase [CK]), venous blood gas analysis (Table 1). Serum biochemistry^a revealed increased albumin concentration, hypokalemia and an increased CK activity, increased packed cell volume and TPP (Table 1). The remaining values on the serum biochemical analysis were within normal limits. Initial venous blood gas analysis^b at 37°C (98.6°F) revealed changes commensurate with acute respiratory acidosis (Table 1) which was considered to result from acute respiratory muscle weakness. Repeated electrolyte analysis 30 minutes later confirmed the presence of a marked hypokalemia (Table 1).

Intoxication was considered as a likely differential diagnosis for the patient's presenting clinical signs suspected due to the history of indiscriminate eating, presence of muscle fasciculations, hypokalemia and associated generalised weakness, leading to treatment being instituted with IV administration of Hartmann's solution^c (5.6 mL/kg/h) with potassium chloride^d supplementation to achieve an administration rate of 0.36 mmol/kg/hr within 30 minutes of presentation. Hartmann's solution^c was selected due to the presence of marked acidemia in this case. Additional supportive care included flow-by oxygen supplementation (2-4 L/min), active warming^e, suctioning of the pharynx in order to keep the airway clear of pooled saliva, and nursing care practices specific for the care of the recumbent patient, the maintenance of sternal recumbency to limit the development of atelectasis, bladder care, turning of the animal every 4 hours and provision of padded bedding to prevent pressure sores. Maropitant^f (25 mg subcutaneously [SC]) and ranitidine^g (50 mg SC) were administered for their antiemetic, antacid and prokinetic effects. Monitoring during the initial treatment period consisted in repeat assessments of heart rate, respiratory rate, monitoring of ventilatory status via repeat venous blood gas analysis^b every 1-3 hours, oscillometric blood pressure^h and repeated rectal temperature every 15 minutes. In an attempt to reduce ptyalism in this patient and as therapeutic treatment trial to rule out cholinesterase inhibitor toxicosis, an initial dose of atropine sulfateⁱ (0.06 mg IV) was administered, followed by a second dose (0.24 mg IV) 30 minutes later, without any appreciable effect on the animal's condition. Despite commencement of potassium supplementation, the dog's respiratory rate slowed to 24/min and flaccid paralysis continued to be observed for the first 5 hours after presentation. Given the presence of marked respiratory acidosis in this patient, the possible need for mechanical ventilation was discussed with the owner, however was not initiated in this case.

Left and right lateral abdominal radiographs were taken approximately 2 hours after presentation and revealed numerous small metal-dense radio-opacities in the area of the stomach of the dog. A left lateral thoracic radiograph showed a radiographically normal chest.

Approximately 5 hours after presentation, the patient was internally transferred from the overnight emergency service to the internal medicine service for further care. At that time, the heart rate was 96 bpm, with cardiac auscultation revealing no murmur and a regular cardiac rhythm. The respiratory rate was 32/min and lung sounds were clear on auscultation. The animal continued to be recumbent, was found to have no gag reflex and had absent spinal reflexes in all limbs. Pain perception was normal. Abdominal palpation was unremarkable and revealed no pain. Dark brown semi-formed feces were noted on rectal examination. Rectal temperature was 37.0°C (98.6°F) despite active warming^e.

Repeat blood gas and electrolyte analysis performed 7.5 hours after presentation revealed ongoing metabolic and respiratory acidosis, with marked hypokalemia and mild ionised hypercalcemia also present. A CBC^j was performed revealing a mild leukocytosis ($18.77 \times 10^9/L$; reference interval: $5.05-16.76 \times 10^9/L$, $0.75 \times 10^9/L$ lymphocytes; reference interval: 1.00-4.80), $1.74 \times 10^9/L$ monocytes; reference interval: 0.2 -1.50, $16.18 \times 10^9/L$ neutrophils; reference interval: 3-12, $0.08 \times 10^9/L$ eosinophils; reference interval: 0-0.8, $0.02 \times 10^9/L$ basophils; reference interval: 0-0.4, a hematocrit of 0.66 L/L [66.4%]; reference interval: 0.37-0.55 L/L [37-55%], a PCV of 70%, TPP of 70 g/dL and platelet count of $248 \times 10^9/L$; reference interval: $100-700 \times 10^9/L$. Prothrombin time (PT)^k and activated partial thromboplastin time (aPTT)^k were within reference interval (PT = 31 s; reference interval: 11-17 s, aPTT = 146 s; reference interval: 72-102 s), therefore, elapid snake envenomation, a common treatable differential for progressive lower motor neuropathy was excluded from the possible differential list.

Due to the progressive deterioration of the patient's neurological status several potential differential diagnoses including elapid snake envenomation, ixodes tick paralysis, polyradiculoneuritis, myasthenia gravis, botulism or peripheral neuropathy were considered. However, the patient's blood work derangements were not explained by these differential diagnoses more common in the local area, suggesting a more obscure cause of this dog's presenting symptoms such as barium intoxication. Further questioning of the owner revealed that in the days prior to the incident, sparklers, handheld pyrotechnics, were used by a child to create 'sparkler bombs' in the backyard. Two types of 'sparkler bombs' were built. For the first, bundles of sparklers were joined together, for the second, the combustible material was stripped off the wires and placed into individual aluminium foil packages. The dog was a known indiscriminate eater, and with remnants of the sparklers from the aforementioned experimentation located in the backyard, and metallic foreign material present in the stomach, sparkler ingestion with subsequent barium toxicosis was suspected. As there was only evidence of metal particles in the gastric lumen on assessment of abdominal radiographs, it would be more likely the animal had ingested the foil packet type "sparkler bomb." However, it is unknown whether the sparklers ingested in this case were unused or spent. The manufacturer provided details on the content of individual sparklers as containing 1.2g of particulate pyrotechnic mixture which consists of 54% barium nitrate, 30% iron, 7% resin ($C^{48}H^{42}O^7$) and 9% aluminium.^l Therefore, if the first type of sparkler bomb (aluminium foil packet) consisted of the particulate pyrotechnic mixture of only a single sparkler, it contained 648 mg of barium nitrate (25.9 mg/kg). The number of sparklers the animal ingested however, remains uncertain.

To confirm the presence of barium toxicosis, serum was collected approximately 7 hours after presentation and was submitted to an external laboratory^m that determined barium

concentrations by inductively coupled plasma/mass spectrometry (ICP/MS). The results were not available until 2 weeks later.

Additional potassium chloride was added to the Hartmann's solution to achieve a higher potassium infusion rate of 0.51 mmol/kg/hr. Continuous ECG monitoring with close monitoring of serum potassium concentrations was performed to assess for evidence of rebound hyperkalemia and adjustment of intravenous potassium supplementation as required. Approximately 8 hours after presentation, general anesthesia was induced with intravenous propofolⁿ titrated IV to effect to allow for endotracheal intubation, and maintained with isoflurane in oxygen. Although marked respiratory acidosis was observed in this patient, the case record did not indicate the use of mechanical ventilation during the procedure. Gastric lavage was performed; however, no recovery of toxins or foreign material was appreciated. Lavage was followed by administration of magnesium sulfate^o (6 g dissolved in water via orogastric tube, as per the oral pediatric dose of 250 mg/kg) for chelation of barium. Recovery from anesthesia was uneventful.

ⁿTwelve hours after presentation, repeat blood gas and electrolyte analysis indicated normalisation of serum potassium concentration (5.2 mmol/L), with the patient's metabolic and respiratory acidosis resolving and the clinical abnormalities improving. Thereafter, the dog's serum electrolyte concentrations remained within reference interval. Twenty-four hours after presentation the animal was ambulatory. After resolution of the paralysis the patient was hospitalized for an additional 12 hours to allow for continued monitoring. The dog was discharged 36 hours after presentation without any clinically appreciable neuromuscular abnormalities and was eating and drinking well. Ten days after discharge, serum barium concentrations were reported by the laboratory (2000 µg/L), confirming the suspected barium intoxication. No consequences of the intoxication were apparent to the owner upon follow up 1 year after hospital discharge.

Discussion

This report highlights the potential for life-threatening barium toxicosis in dogs after ingestion of a handheld fireworks, or more specifically sparklers. The common sparkler, is a hand-held firework consisting of a rigid wire handle of which one end is coated with a particulate pyrotechnic mixture which generates sparks when ignited.²⁰ The main ingredients of the sparklers pyrotechnic mixture (part by weight) include, an oxidiser (25-50%), a fuel (3-27%), a metallic powder (3-14%), a combustion rate modifier (0-5%), a colourant (0-5%) and a binding agent (included with the fuel).^{15, 20} As there is no legal requirement for reporting the composition of sparklers on the package and the home-made 'sparkler bombs' being made by a minor, neither the type of barium salt nor the amount of barium per sparkler could be determined at the time of presentation and an estimation of the amount of barium salt ingested was not possible. When the major distributors of sparklers were later contacted, only the ingredient list for the sparklers was able to be supplied and only 1 manufacturing company supplied information on the percentage of barium contained in their product. Often sparklers contain barium nitrate as the oxidiser, with 2 main Australian suppliers^{p,q} of sparkler products confirming barium nitrate content in their products. One sparkler^p contained 51% barium nitrate, with the remaining constituents described as aluminium, sand and glue, and another^q containing 54% barium nitrate, aluminium, iron-Fe, potassium chlorate and phosphorus. This type of fireworks is widely available to the public and can be purchased without permit in most of the United States and in many countries.²¹⁻²³ Barium poisoning has been reported once in the veterinary literature, however in that case the source of barium was unknown.¹⁴ The authors believe that this report describes the first such occurrence in a dog after ingestion of fireworks, specifically sparklers containing barium nitrate. In people, a small number of case reports recount barium toxicosis subsequent to ingestion of unburned

sparkler or fireworks containing barium nitrate.^{12, 13, 24} Barium nitrate ($\text{Ba}[\text{NO}_3]_2$) is commonly included in sparklers and other fireworks as an oxidiser, but raises significant toxicological and environmental concerns.^{25, 26} $\text{Ba}(\text{NO}_3)_2$ and the compounds set free after burning of fireworks (e.g., BaO , $\text{Ba}(\text{OH})_2$, BaCl_2) are highly soluble and can therefore be readily absorbed if ingested. As such this report emphasises the unique risk that indiscriminately scavenging dogs are exposed to by chewing or ingesting materials in their environment that appear incredibly unpalatable, yet can be highly toxic.

Barium toxicosis results in the development of marked hypokalaemia which develops due to the barium ions interference with outward potassium transport.¹⁷ Potassium influx into the cell continues via Na^+/K^+ -ATPase, resulting in a net shift of potassium from the extracellular to the intracellular compartment and the marked hypokalemia associated with barium toxicosis.^{17, 27} Serum potassium levels as low as 1.7 mmol/L were identified in our case. From observations of two mass poisonings of people with barium via contaminated food, three distinct clinical stages of barium toxicosis emerged.^{28, 29} The first stage consisted in the development of gastrointestinal signs (e.g., vomiting and diarrhea) and mild paresthesia. The second stage was characterised by progressive lower motor neuropathy with the onset of muscular paralysis and loss of deep sensation, until in a final stage, generalised muscle paralysis including respiratory paralysis resulted.

Possible sequelae reported include a marked flaccid paralysis with possible ventilatory failure, rhabdomyolysis and cardiac arrhythmias.^{17, 27} ECG irregularities, typically ventricular in origin are reported, with widening of the QRS complex, right bundle branch blocks, ST-segment depression and T wave flattening, ventricular premature complexes, ventricular couplets, triplets, runs of ventricular tachycardia, which can progress to ventricular fibrillation and ultimately death.^{14, 17, 27, 30} Ventricular premature contractions were reported by the initially treating veterinarian in this case.

Hypokalemia alone can lead to ECG abnormalities such as prolongation of the PR interval, increased amplitude and width of the P wave, flattened and inverted T wave, ST depression, prominent U waves, fusion of T and U waves resulting in apparent long QT interval when potassium is very low ($<2.7\text{mmol/L}$).³¹ With marked hypokalemia, frequent supraventricular and ventricular ectopic beats occur resulting in supraventricular tachyarrhythmias (atrial flutter and atrial tachycardia) and potentially life threatening ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation).³¹ While hypokalemia is often thought to be responsible for the majority of clinical signs of barium toxicosis, the severity of clinical signs was found to be more closely associated with barium concentration than the degree of hypokalemia in one human case report.³² Other findings, such as muscular fasciculations, vomiting and diarrhoea, acute kidney injury and other electrolyte abnormalities (eg, hypophosphatemia) may also be a direct effect of barium or the specific barium salt used, although this has not been examined in detail.^{7, 17, 30} Additionally, serum concentrations of sparkler compounds other than barium (eg, aluminium, zinc, iron, strontium) as well as electrolytes such as magnesium were not measured in this dog, but could potentially have complicated the clinico-toxicological picture.¹⁶ The dog in this case report presented with a history of acute vomiting and diarrhea, as seen in early stage intoxication in people, followed by quickly progressing muscle paralysis and fasciculations, observation of premature ventricular contractions and severe hypokalemia ($< 2.0\text{ mmol/L}$). Acute onset of flaccid paralysis and muscle fasciculations in combination with marked hypokalemia in our patient are suggestive of barium toxicosis. The case presented herein illustrates the acute toxic potential of barium compounds used in fireworks.

While a toxicologic etiology of the clinical signs observed was suspected, barium poisoning was not immediately considered at presentation. This is attributed to the rare occurrence of barium intoxication and the lack of a hint pointing towards the exposure of the

dog to barium containing material or any other toxin during the initial history taking. Thus, paralytic syndromes more commonly seen in the area were initially considered. These included tick paralysis (*Ixodes holocyclus*), acute polyradiculoneuritis, botulism and ingestion of other toxins including heavy metals such as lead and pesticides such as cholinesterase inhibitors. Upon identification of severe hypokalemia shortly after admission of the dog, barium intoxication was considered the likely cause of the clinical signs. The owner's statement regarding sparkler use coinciding with the occurrence of clinical signs corroborated that suspicion.

Measurement of serum barium concentration in dogs can be accomplished by utilization of human laboratory facilities and is typically conducted by ICP/MS. This methodology was used here and in a previous report of canine barium intoxication.¹⁴ Compared to that previous case, the barium concentration (2000 µg/L) in the dog reported herein was approximately 5-times higher, and more than 1000-times increased above reported normal values in dogs (0.7 to 1.9 µg/L).¹⁴ Reported serum barium concentrations in people with severe acute barium intoxications ranged from 107 µg/L to 20,200 µg/L.^{6, 10, 12, 13, 32-34} In 2 case reports where the source was firework ingestion, the barium values were 985 µg/L and 20,200 µg/L.^{12, 13} Alternatively, whole blood, gastric content and urine are suitable substrates for barium level determination.^{14, 35} While confirmatory, the turnaround time for serum barium levels limits the usefulness of this diagnostic test for acute patient management. In our case, the animal was already discharged by the time the test results became available. Treatment is therefore initiated on the basis of clinical suspicion from the patient's history, abnormalities on physical exam and further diagnostics. The presence of severe hypokalemia in conjunction with flaccid muscle weakness or paralysis provides the most specific clue to differentiate barium intoxication from other diseases.

No specific antidote exists for barium toxicosis. Medical management is therefore limited to attenuation of further absorption and supportive care, foremost aggressive potassium supplementation and chelation with magnesium sulfate in combination with close monitoring of hyperkalemia. Induction of emesis was not a viable option in our case given the animal's neuromuscular impairment and the associated risk for aspiration of vomitus. Gastric lavage is labor intensive, but may be beneficial to remove large volume of barium containing material shortly after ingestion.³⁶ In this case, the gastric lavage occurred delayed and at least 12 hours after ingestion, which likely reduced the effectiveness of toxin removal significantly. However, the orogastric tube placement allowed the safe gastric administration of magnesium sulfate. Oral magnesium sulfate (Epsom salt) has been utilised as a chelation agent, which transforms highly soluble barium compounds into insoluble barium sulfate, thus attenuating further absorption of barium from the gastrointestinal tract.¹⁹ Administration of activated charcoal is ineffective, as it does not bind barium.⁶

The mainstay of curative therapy is reported as the intravenous administration of potassium.¹ In our case, intravenous infusions of potassium at doses up to the maximum recommended rate of 0.5 mEq/kg/hr (Kmax) were required for 12 hours until resolution of hypokalemia and could have been safely implemented earlier in the treatment plan. Our case remained hypokalemic until chelation therapy was commenced, it remains open whether this was a direct effect of the chelation therapy or a temporal coincidence with resolution of the hypokalemia. In refractory cases with cardiac instability or renal impairment, hemodialysis has been reported to be effective in reducing barium concentrations.^{30, 33} Respiratory paralysis, resulting in marked respiratory acidemia may require mechanical ventilation.²⁹

Despite the severity of the clinical signs and lack of mechanical ventilation in this case, the dog showed a rapid response to therapy and could be discharged to the owner 36 hours after admission and no long-term sequela were noted. This recovery is similar to the

only other reported case of barium intoxication in a dog, such that it seems reasonable to expect a favorable outcome with appropriate supportive care.¹⁴ In people, barium toxicoses has led to an overall mortality rate of 12% in published cases over the past 70 years, while two recent series of 44 cases in Brazil and 27 cases in Bangladesh led to the death of 21% and 44% of the affected individuals, respectively.^{11, 17, 29} Death from barium toxicosis may in part be due to delay of treatment or resource restriction in cases of mass poisonings with most death occurring due to respiratory failure, emphasizing the importance of quick access to intensive care for survival of severe cases of barium poisoning.

The amount of a toxin or chemical a victim consumes is related to the amount and concentration of the chemical ingested, frequency and duration of exposure and inversely related to both the body weight and total time of exposure of the affected individual. The risk of chemical ingestion can be determined by the resulting toxicoses that results in the event of exposure to the chemical. In the present case, barium toxicosis occurred as a result of exposure to barium nitrate in the form of a common hand-held pyrotechnic. Generally, the risk to animals to barium toxicoses due to fireworks ingestion is low due to limited exposure frequency and duration, with risk increasing during holiday periods and celebrations involving fireworks displays. No definitive proof exists that the barium intoxication reported here resulted from ingestion of sparklers as respective material could not be retrieved from the GI tract for analysis. However, given the history of the use of sparklers shortly before the clinical signs of acute intoxication, the ability of the animal to readily access the sparkler material, sparklers being a known source of highly water soluble barium salts (up to 54% barium nitrate), the radiographic presence of radiopaque material in the stomach likely representing metallic sparkler components (eg, iron, aluminium, barium) and the elevated serum barium levels, the most likely source of barium in this case cannot be disputed.

Sparklers can be purchased in supermarkets in many countries, including the United States, and thus must be considered as a possible source of barium intoxication in dogs.

Conclusion

Acute, life-threatening barium toxicosis characterized by flaccid paralysis and severe hypokalemia can occur in dogs after ingestion of a popular pyrotechnic (sparkler), but may resolve within 24 hours with appropriate supportive care. Acute barium toxicosis is very rarely reported but should be considered as a differential diagnosis for marked hypokalemia. As readily absorbable barium salts are present in several objects of every-day life, exposure is generally possible and careful history taking is essential.

Footnotes

^a Vetscan VS2, Abaxis, REM Systems, North Ryde, NSW, Australia

^b ABL77 Radiometer Pacific Pty Ltd, Mt Waverley, VIC, Australia.

^c Hartmanns Solution, Baxter Healthcare, Old Toongabbie, NSW, Australia

^d Sterile Potassium Chloride (Concentrate for Infusion) BP 10%W/V 10mL Ampoule, Pfizer Australia, West Ryde, NSW, Australia

^e Bair Hugger warming unit Model 505, Critical Assist, Mt Waverley, VIC, Australia.

^f Cerenia, Zoetis Inc, Silverwater, NSW, Australia

^g Zantac, GlaxoSmithKline, Australia Pty Ltd, Abbotsford, VIC, Australia.

^h SurgiVet[®] Advisor[®] Vital Signs monitor, Smiths Medical Australasia Pty, Ltd, Bella Vista, NSW, Australia.

ⁱ Ilium Atropine Sulphate, Troy Laboratories Pty Ltd (Discontinued product, now Ilium Atrosite 50mL injection (Atropine Sulphate 0.65mg/mL), Glendenning, NSW, Australia.

^j Vetscan HM5 Veterinary Hematology Analyzer, Abaxis Inc., REM Systems, North Ryde, NSW, Australia

^k IDEXX Coag Dx™ Analyzer, Coag Dx™ Citrate PT and Coag Dx™ Citrate aPTT cartridges, IDEXX Laboratories Pty Ltd, Rydalmere, NSW, Australia.

^l Lee, K, Liuyang Candour Fireworks Co., Ltd, 2017, personal communication, 6 April 2017

^m Queensland health Laboratories, Brisbane, QLD Australia submitted via QML Laboratories.

ⁿ Propofol, Sandoz Pty Ltd, Pyrmont, NSW Australia.

^o Faulding® Remedies Epsom Salts, Symbion Pty Ltd, Adelaide, SA, Australia.

^p Meteor Party Products Pty Ltd, 2017, personal communication, 5 April 2017

^q Gavin, M, Alpen Products Pty Ltd, 2017, personal communication, 5 April 2017

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Table 1: Point-of-care laboratory diagnostic test results and vital parameters at presentation and over the course of hospitalization of a dog with acute barium intoxication.

Time from presentation (hours)	PCV (L/L) [%]	TT P [g/L]	pH	PvCO ₂ [mm Hg]	BE [mmol /L]	HCO ₃ [mmol /L]	Na (mmol /L)	K (mmol /L)	iCa (mmol /L)	Cl (mmol /L)	HR (per min)	RR (per min)	T (°C)
0	0.81 [81]	92	7.20	55	-6.1	20.7	152	1.8	1.41	113	60-80	26	37.3
0.5			7.20	54	-6.1	20.7	152	1.9	1.41	113	60	28	37.2
1.5			7.21	44	-11.3	16.9	151	1.9	1.46	112	160	24	37.0
6.5	0.70 [70]	70	7.20	58	-5.0	21.8	149	1.7	1.47	114	96	32	37.0
9.5			7.24	50	-5.5	20.7	152	2.9	1.5	117			
10.5			7.28	45	-5.1	18.9	154	3.8	1.51	117			
12			7.33	32	-8.4	16.4	154	5.2	1.47	119			
15			7.35	34	-6.3	19.2	155	4.4	1.47	120			
28	0.46 [46]	60	7.44	36	0.4	24.1	152	4.7	1.44	113	88	20	38.3

TTP = total protein; PvCO₂, venous partial pressure of carbon dioxide; BE, base excess;

HCO₃, bicarbonate; Na, sodium; K, potassium; iCa, ionised calcium; Cl, chloride; HR, heart rate ; RR respiratory rate; T, rectal temperature

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