

Hyperkalemia during general anesthesia in two Greyhounds

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CASE DESCRIPTION

A 36-kg (79-lb) castrated male Greyhound (dog 1) and a 25-kg (55 lb) spayed female Greyhound (dog 2) underwent general anesthesia for dental care with similar perianesthetic protocols on multiple occasions from 2013 to 2016. Both dogs had periodontal disease but were otherwise deemed healthy. Both dogs developed clinically relevant hyperkalemia, with signs including loss of P waves on ECG tracings, during multiple anesthetic events.

CLINICAL FINDINGS

Dog 1 developed hyperkalemia during 2 of 2 anesthetic events, with ECG changes noted during the first event. Dog 2 developed hyperkalemia during 3 of 4 anesthetic events, with ECG changes identified during the second and third events. Serum potassium concentration for both dogs was within the reference range prior to and between anesthetic events. No underlying etiopathogenesis for hyperkalemia was identified for either dog.

TREATMENT AND OUTCOME

In each hyperkalemic event, the clinician stopped the dental procedure and continued to provide supportive care and monitoring while the dog recovered from anesthesia. The ECG changes resolved, and serum potassium concentration returned to the reference range rapidly after inhalant anesthetic administration was discontinued. The dogs were discharged from the hospital without further complications.

CLINICAL RELEVANCE

Hyperkalemia in anesthetized Greyhounds resulted in serious cardiac conduction abnormalities, which could be potentially fatal if not recognized and promptly treated. Further investigation into the etiopathogenesis, prevention and treatment strategies, and genetic or familial components of this condition is indicated. (*J Am Vet Med Assoc* 2019;254:1329–1334)

A 5-year-old 36-kg (79-lb) castrated male Greyhound (dog 1) was evaluated for dental care and biopsy of a cutaneous mass in May 2013. The dog was receiving a heartworm preventative^a and nutritional supplements (glucosamine and essential fatty acids). Abnormalities noted on physical examination included periodontal disease and a 0.5-cm-diameter, dark red, raised cutaneous mass on the sternum. Preanesthetic clinicopathologic analysis was performed at a local reference laboratory.^b Most preanesthetic clinicopathologic findings were within the respective reference ranges for the breed,¹ except for the presence of monocytosis ($1,184 \times 10^9$ monocytes/L; reference range, 0×10^9 monocytes/L to 840×10^9 monocytes/L) and 2+ urine protein concentration (with a urine specific gravity of 1.049 and inactive sediment). The serum potassium concentration was 4.1 mEq/L (reference range, 3.6 to 5.5 mEq/L). An

ECG performed prior to anesthetic induction revealed rare, intermittent atrial premature contractions.

The patient was premedicated with a combination of hydromorphone^c and midazolam^d (0.1 and 0.2 mg/kg [0.045 and 0.09 mg/lb], respectively, SC). A balanced electrolyte solution^e was administered through an IV catheter (7 to 10 mL/kg/h [3.2 to 4.5 mL/lb/h]) beginning just prior to and continuing throughout anesthesia. Following preanesthetic oxygen delivery and placement of monitoring equipment, anesthesia was induced with midazolam (0.15 mg/kg [0.07 mg/lb], IV) and propofol^f (4 mg/kg [1.8 mg/lb], IV), and an endotracheal tube was placed. Anesthesia was maintained with isoflurane^e in oxygen delivered through a standard small animal circle breathing circuit. The dog was insulated from the environment with towels and blankets, and thermal support was provided with a forced-air warming system^g and circulating warm water blanket. During anesthesia, heart rate and rhythm (ECG), SpO₂, PETCO₂ (capnography), indirect blood pressure (oscillometry), and rectal body temperature were monitored by use of a multiparameter monitor^h and recorded at 5-minute intervals. Monitoring parameters remained within acceptable ranges except as subsequently described.

ABBREVIATIONS

DAP	Diastolic arterial blood pressure
PETCO ₂	End-tidal partial pressure of carbon dioxide
MAP	Mean arterial blood pressure
SAP	Systolic arterial blood pressure
SpO ₂	Saturation of arterial hemoglobin as measured by pulse oximetry

Approximately 75 minutes after anesthetic induction, following partial completion of dental care and excisional biopsy of the skin mass, the dog was rotated to the contralateral recumbency. Fifty-five minutes later (130 minutes after anesthetic induction), a sudden decrease in heart rate (from 110 to 60 beats/min) was observed. Other monitored variables remained stable (body temperature, 37.6°C [99.7°F]; $PETCO_2$, 44 mm Hg; SpO_2 , 96%; and MAP, 70 mm Hg) and within the reference ranges except as subsequently described. Atropineⁱ (0.02 mg/kg [0.009 mg/lb], IV) was administered with little effect on the heart rate. Loss of P waves was noted on the ECG tracing, and a venous blood gas sample^j obtained from a lateral saphenous vein revealed a high plasma potassium concentration (7.7 mEq/L; reference range, 3.4 to 4.9 mEq/L). Hyperkalemia was presumed to be the cause of the sudden bradycardia and loss of P waves. A bolus of physiologic saline (0.9% NaCl) solution^k (10 mL/kg [4.5 mL/lb], IV) was administered, isoflurane delivery was discontinued, and dental care was rapidly completed. Patient monitoring was continued into the postanesthetic period. Forty minutes after discontinuation of isoflurane administration, the dog's plasma potassium concentration^l had decreased to 5.8 mEq/L and its heart rate had increased to a range of 150 to 160 beats/min; P waves were not visible on the ECG for an additional 20 minutes. One hundred minutes after isoflurane administration was discontinued, the dog's ECG tracing and acid-base status were considered normal and its plasma potassium concentration (3.6 mEq/L) was within the reference range. The patient was discharged from the hospital and reportedly did well overnight. The next day, the dog was reevaluated at our hospital; an ECG tracing was normal, and plasma potassium concentration (4.0 mEq/L) was within the reference range. A follow-up fecal examination for parasites was negative. The skin mass was diagnosed as a hemangioma on histologic analysis.

Three years later, dog 1 (then 8 years of age) was returned to the hospital for dental care. The only medication being given was heartworm preventative.^a Physical examination revealed periodontal disease and a transiently high heart rate consistent with stress associated with the veterinary hospital visit (200 beats/min) that decreased to a value considered normal for the breed in a hospital setting² (100 beats/min) after premedication and acclimatization. Preanesthetic clinicopathologic findings, including serum potassium concentration (4.0 mEq/L), were within the respective reference ranges.^b

Anesthetic drug protocol, monitoring, and supportive measures were the same as for the previous anesthetic episode, except that a non-potassium-containing crystalloid fluid^k (saline solution) was administered for IV fluid maintenance, ampicillin-sulbactam^l (22 mg/kg [10 mg/lb], IV) was administered after anesthetic induction, mechanical ventilation was instituted, and plasma potassium concentration^l

was monitored every 30 minutes beginning 1 hour after induction. The plasma potassium concentration progressively increased, reaching a peak value of 7.2 mEq/L 2.5 hours after anesthetic induction. Other monitored variables remained stable and within the reference ranges (body temperature, 38.0°C [100.4°F]; $PETCO_2$, 37 mm Hg; SpO_2 , 93%; and MAP, 72 mm Hg). Plasma concentrations of glucose and electrolytes remained within the reference ranges, and the dog's acid-base status was normal throughout the procedure. No ECG abnormalities were noted. The procedure was completed, and the patient was monitored closely throughout recovery; 2.5 hours after isoflurane administration was discontinued, the plasma potassium concentration was slightly low (3.1 mEq/L), and IV fluid therapy and monitoring were discontinued. The patient was discharged from the hospital. The dog's resting serum cortisol concentration was within the reference range (3.8 µg/dL; reference range, 1.0 to 5.0 µg/dL) during subsequent wellness screening in 2017.

In May 2014, an 8-year-old 25-kg (55-lb) spayed female Greyhound (dog 2) was brought to the same facility for dental care. The dog was not receiving any medications, and abnormalities identified on physical examination were limited to severe periodontal disease and cataracts. Preanesthetic laboratory screening was performed in the hospital,^m and clinicopathologic findings, including serum potassium concentration (4.5 mEq/L; reference range, 3.5 to 5.8 mEq/L), were within the respective reference ranges for the breed. The dog received premedication with hydromorphone (0.1 mg/kg, SC), acepromazineⁿ (0.02 mg/kg, SC), and atropine (0.02 mg/kg, SC), and anesthesia was induced with midazolam (0.2 mg/kg, IV) and propofol (1.8 mg/kg [0.8 mg/lb], IV). Crystalloid fluid^c was administered at rates of 5 to 10 mL/kg/h (2.3 to 4.5 mL/lb/h) during the anesthetic period. Peri-anesthetic management and monitoring were otherwise the same as described for the first anesthetic episode for dog 1, except that serum potassium concentration was monitored^m every 30 minutes beginning 1 hour after anesthetic induction. Serum potassium concentration progressively increased to 7.5 mEq/L 3.5 hours after anesthetic induction. Other monitored variables remained stable and within the reference ranges (body temperature, 38.8°C [102.0°F]; $PETCO_2$, 36 mm Hg; SpO_2 , 99%; and MAP, 98 mm Hg). No clinical signs of hyperkalemia or ECG abnormalities were noted, but anesthesia was discontinued. Fluid administration and monitoring were continued into the postanesthetic period. Approximately 25 minutes after isoflurane administration was discontinued, the potassium concentration had decreased (6.6 mEq/L), and 70 minutes later it was within the reference range (3.5 mEq/L). Dog 2's condition remained stable, monitoring and fluid treatment were discontinued, and the dog was discharged from the hospital.

Seventeen months later, dog 2 (then 9 years of age) was returned to the hospital for dental care. The

dog was not receiving any medications, and physical examination abnormalities remained limited to periodontal disease and cataracts. Preanesthetic clinicopathologic screening was performed at a local reference laboratory.^b Clinicopathologic findings prior to anesthesia, including serum potassium concentration (4.0 mEq/L; reference range, 3.6 to 5.5 mEq/L), were within the respective reference ranges, except a 3+ urine protein concentration (with a urine specific gravity of 1.047 and inactive sediment); measurement of the urine protein-to-creatinine ratio was recommended but deferred by the client.

Anesthetic management was the same as previously described for this patient, except that saline solution (5 to 10 mL/kg/h, IV) was administered during the anesthetic period, ampicillin-sulbactam (22 mg/kg, IV) was administered after anesthetic induction, and mechanical ventilation was instituted. Plasma potassium concentration^l progressively increased from 3.7 mEq/L 1 hour after anesthetic induction to 6.5 mEq/L (reference range, 3.4 to 4.9 mEq/L) at the 3-hour time point. No ECG changes were noted; however, a decrease in MAP from 92 mm Hg (SAP and DAP, 125 and 75 mm Hg, respectively) to 62 mm Hg (SAP and DAP, 95 and 45 mm Hg, respectively) occurred with a small decrease in heart rate (from 127 to 120 beats/min). A fluid bolus (10 mL/kg, IV) was initiated. The blood pressure fluctuated but was stabilized within 15 minutes (changing from an MAP of 42 mm Hg [SAP, 50 mm Hg; DAP, 40 mm Hg] to 65 mm Hg [SAP, 85 mm Hg; DAP, 55 mm Hg]), at which time a loss of P waves was observed on the ECG tracing and a progressively increased plasma potassium concentration (7.0 mEq/L) was measured. Other physiologic monitoring variables were stable and within the reference ranges at this time (body temperature, 38.2°C [100.7°F]; PETCO₂, 41 mm Hg; SpO₂, 96%; MAP, 65 mm Hg; heart rate, 110 beats/min; and respiratory rate, 12 breaths/min). Isoflurane administration was discontinued, supportive care and monitoring continued, and recovery was uneventful. Thirty minutes later, the dog's plasma potassium concentration was lower (5.9 mEq/L) and the ECG tracing revealed no abnormalities. The dog remained stable, monitoring and IV fluid treatments were discontinued, and the dog was discharged from the hospital.

Eight months later, dog 2 (then 10 years of age) was brought to the facility a third time for dental care. The dog was not receiving medications, and the only abnormalities on physical examination were periodontal disease and cataracts. Preanesthetic clinicopathologic findings,^b including serum potassium concentration (3.9 mEq/L), were within the respective reference ranges, except for low serum phosphorus concentration (2.0 mg/dL; reference range, 2.5 to 6.0 mg/dL), 3+ urine protein concentration (with a urine specific gravity of 1.042 and inactive sediment), and urine protein-to-creatinine ratio (0.6; upper reference limit, < 0.5). There were no parasites detected on fecal screening.

Perianesthetic management remained the same as for the previous anesthetic episode. Plasma potassium concentration^l increased from 3.9 mEq/L 1 hour after induction of anesthesia to 5.3 mEq/L 30 minutes later. Other monitored physiologic variables remained stable until an acute decrease in MAP occurred 25 minutes later (approx 2 hours after induction of anesthesia), from 105 mm Hg (SAP and DAP, 130 and 90 mm Hg, respectively) to 62 mm Hg (SAP and DAP, 90 and 50 mm Hg, respectively). This change corresponded to a sudden loss of P waves and increased potassium concentration (7.3 mEq/L). The heart rate was relatively unchanged (from 130 to 125 beats/min) during this interval. Other monitored physiologic variables remained stable and within the reference ranges (body temperature, 38.4°C [101.1°F]; PETCO₂, 44 mm Hg; and SpO₂, 97%). Isoflurane administration was discontinued, supportive care continued, and the dog remained stable. Continued monitoring allowed identification of the return of visible P waves on the ECG tracing approximately 30 minutes after anesthetic delivery was discontinued; at this time, the plasma potassium concentration was still high (6.5 mEq/L). Seventy minutes after anesthetic administration was discontinued, the plasma potassium concentration was within the reference range (4.5 mEq/L). The dog's condition remained stable; monitoring and IV fluid treatments were discontinued, and the patient was discharged from the hospital.

Completion of dental care for dog 2 was planned 1 week later. The previously described anesthesia protocol was used, and the procedure was completed uneventfully. The dog's plasma potassium concentration^l was within the reference range (3.5 mEq/L) 45 minutes after induction of general anesthesia. A resting serum cortisol concentration was measured and found to be slightly high (7.0 µg/dL) during subsequent wellness screening in 2017.

Discussion

The present report highlighted a repeatable and potentially life-threatening occurrence of acute hyperkalemia with associated ECG changes during anesthesia in 2 Greyhounds. To the authors' knowledge, this has not been previously reported in the veterinary medical literature. Progressive increases in serum or plasma potassium concentrations and accompanying well-documented ECG changes associated with hyperkalemia³⁻⁵ were observed during these anesthetic episodes.

Many causes for hyperkalemia in veterinary patients have been described, including accidental ingestion or administration of potassium, acidemia, tissue damage (eg, injury associated with heat-induced illness, malignant hyperthermia, prolonged exercise, rhabdomyolysis, muscle hypoxia, or thrombosis), hyperkalemic periodic paralysis (an autosomal dominant genetic disease in horses⁶⁻¹² that was also reported to occur in 1 American Pit Bull Terrier¹³), parasite infestation (eg, whipworms), decreased renal excre-

tion (eg, with renal disease, hypoadrenocorticism, administration of diuretics or angiotensin-converting enzyme inhibitors, or other causes), inability to expel urine (eg, urinary obstruction or uroabdomen), and analytical causes (associated with hypernatremia, high platelet count, or high WBC count).³⁻⁵ None of these conditions were identified in either dog of this report.

Episodic hyperkalemia has been described in 3 pregnant Greyhounds,¹⁴ and as mentioned previously, it occurs with hyperkalemic periodic paralysis and malignant hyperthermia. Although malignant hyperthermia has been reported in Greyhounds,¹⁵⁻¹⁸ the breed may not have greater susceptibility, compared with mixed-breed dogs.¹⁹ Greyhounds can exhibit anxious responses in a hospital setting and may be predisposed to stress-induced hyperthermia, which can be compounded by perianesthetic shivering or pain.¹⁹ Importantly, both dogs of the present report remained normothermic during hyperkalemic episodes.

Interestingly, serum potassium concentrations have been shown to increase during and immediately after racing in some Greyhounds.²⁰ However, the few articles that describe exercise-associated hyperkalemia also report proportional increases in sodium and chloride concentrations, suggesting this change is likely the result of intravascular fluid translocation or dehydration.²⁰⁻²⁵ A recent study²⁶ by one of the authors found that Greyhounds have significantly lower basal aldosterone concentrations than non-Greyhound dogs, despite having similar plasma renin- and angiotensin-converting enzyme activities. It is possible that this contributes to the development of hyperkalemia or the inability of Greyhounds to rapidly or effectively respond to increases in serum potassium concentrations when they occur.

Perianesthetic hyperkalemia has been reported in large nondomesticated cats during anesthesia.²⁷ Eleven captive felids (3 lions and 8 tigers) underwent general anesthesia with midazolam, medetomidine, ketamine, and isoflurane and developed progressive increases in serum potassium concentration, with 1 tiger becoming hyperkalemic (serum potassium concentration, 6.5 mEq/L; reference range, 2.8 to 4.8 mEq/L); the authors measured circulating aldosterone concentrations and found that the concentrations increased over time (3.5 hours), suggesting a response to increased serum potassium concentration but not a cause for it.²⁷ Progressive increases in plasma potassium and glucose concentrations (and initial hypoinsulinemia, which resolved over time for the group) were also noted and hypothesized to be induced by the inhibitory effects of the α_2 -adrenergic receptor agonist (medetomidine) on insulin production.²⁷ In the hyperkalemic tiger, concurrent hyperglycemia was greater than, and insulin concentration lower than, the group's mean concentration at the same time point.²⁷ Both Greyhounds of the present report remained euglycemic throughout anesthesia, and neither dog received an α_2 -adrenergic receptor agonist.

Intra-anesthetic hyperkalemia has been reported in human patients with propofol infusion syndrome, which is characterized by rhabdomyolysis, severe lactic acidosis, hyperkalemia, acute renal failure, cardiac failure, and death associated with constant rate infusion of propofol.^{28,29} Initially hypothesized to be dose dependent and duration dependent, propofol infusion syndrome has been reported with intra-anesthetic hyperkalemia as the only clinical manifestation and with short-term constant rate infusions of a typical propofol dose in people.²⁸ Propofol was administered to the dogs of this report as a single induction dose (not constant rate infusions), and the doses used were at the low end of the recommended range.³⁰

We were unable to find any published peer-reviewed reports pertaining to anesthesia-induced hyperkalemia in Greyhounds; however, other veterinarians anecdotally indicated by personal communication⁰⁻⁵ that they had observed this phenomenon. Additionally, 1 report³¹ of hyperkalemia-related perianesthetic death was found in an online library of articles related to Greyhounds.³¹ A genetic component for the anesthesia-related hyperkalemia described in these 2 dogs remains unknown. The 2 dogs described in this report did share a common ancestor; however, popular studs are likely overrepresented in contemporary racing dog pedigrees. Recently, the authors documented the development of hyperkalemia in 36 of 95 anesthetic events in Greyhounds evaluated between 2013 and 2017; 7 hyperkalemic episodes occurred within the first 1.5 hours of general anesthesia, and 29 occurred after ≥ 2 hours of general anesthesia.¹ Prospective genetic and pedigree studies could shed light on whether this phenomenon is familial in nature. Additionally, redistribution of potassium associated with channelopathies, large muscle mass, occult myopathy and rhabdomyolysis, endocrinopathy, adverse drug effects, and other potential causes warrant investigation.

Owing to the seriousness of the consequences associated with hyperkalemia and the rapidity with which it may escalate, vigilant monitoring and supportive care are indicated for Greyhounds undergoing general anesthesia. Interventional or emergency treatments that are well documented in the literature³⁻⁵ are suggested if the circulating potassium concentration is increasing to concentrations greater than the upper limit for the equipment used and the procedure cannot be staged or halted, even if there is no evidence of ECG changes or other physiologic anomalies. Strategies to decrease circulating potassium concentration may include IV fluid administration (through dilution and promotion of excretion as a result of increased renal perfusion) and treatment with dextrose, insulin, and sodium bicarbonate (through intracellular movement of potassium).³⁻⁵ Historically, administration of physiologic saline solution was recommended for hyperkalemic patients. However, it has more recently been considered that administration of this solution in large volumes may contribute

to the extracellular movement of potassium because of the acidifying nature of saline solution.³² Although both approaches were used successfully in the dogs of the present report, data increasingly support administration of a balanced alkalinizing electrolyte solution instead of physiologic saline solution.^{5,32} Reduction of the risk for cardiac arrhythmias by increasing the cardiac threshold potential with calcium gluconate administration has also been described.³⁻⁵ Wide-complex or ventricular tachycardia can develop from hyperkalemia, but treatment with lidocaine is contraindicated.⁵

Considering that development of clinically relevant hyperkalemia appears to progress with time, duration of general anesthesia may be a risk factor. Proactively minimizing the duration of general anesthesia or staging anticipated procedures of long durations into multiple, shorter anesthetic events whenever possible may be beneficial. Additional considerations that may have merit in preventing or minimizing the development of perianesthetic hyperkalemia in Greyhounds include use of analgesia for potentially painful conditions and taking steps to alleviate anxiety (which in turn may minimize contributing factors such as hyperthermia and muscle fasciculation); appropriately positioning and padding these often athletic, well-muscled patients; avoiding or reversing α_2 -adrenergic receptor agonists; and providing mechanical ventilation if indicated to control respiratory acidosis.

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Footnotes

- a. Heartgard Plus, Merial Ltd, Duluth, Ga.
- b. Antech Diagnostics, Englewood, Colo.
- c. West-Ward Pharmaceuticals Corp, Eatontown, NJ.
- d. Akorn Inc, Lake Forest, Ill.
- e. Plasma-Lyte A, Hospira Inc, Lake Forest, Ill.
- f. Zoetis Inc, Kalamazoo, Mich.
- g. Bair Hugger, Arizant Healthcare Inc, Eden Prairie, Minn.
- h. Bionet Multi-parameter Patient Monitor, Bionet, Seoul, South Korea.
- i. Henry Schein Animal Health, Dublin, Ohio.
- j. I-Stat, Flextronics Manufacturing (Singapore) Pte Ltd, Abbott Point of Care Inc for Abaxis Inc, Union City, Calif.
- k. Hospira Inc, Lake Forest, Ill.
- l. Unasyn, Pfizer Inc, New York, NY.
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From this month's AJVR

Effects of storage over a 36-month period on coagulation factors in a canine plasma product obtained by use of plasmapheresis

Margret E. Donahue and Alberto L. Fernandez

OBJECTIVE

To evaluate stability of coagulation factors in canine plasma obtained by use of plasmapheresis and stored over a 36-month period.

SAMPLE

Canine plasma obtained by use of plasmapheresis acquired from a commercial blood bank.

PROCEDURES

Coagulation testing for fibrinogen concentration and activity of factors II, V, VII, VIII, and IX and von Willebrand factor was performed on canine plasma obtained by use of plasmapheresis. Samples were obtained for testing at 6-month intervals from plasma stored for up to 36 months.

RESULTS

A simple mixed linear regression model was created for each analysis. Median fibrinogen concentration was > 150 mg/dL for all time points, except at 467, 650, and 1,015 days of storage. Median value for factor VIII was > 70% only at 650 days. Median value for factor V was > 50% through 650 days. Median value for factors VII and X was > 50% through 833 days, and median value for factors II and VII was > 50% through 1,015 days. Median value for von Willebrand factor was > 50% for the entire study (1,198 days). Median value for factor X was always < 50%.

CONCLUSIONS AND CLINICAL RELEVANCE

Coagulation factors degraded over time at variable rates, and all labile factors remained at > 50% activity for longer than 1 year. Plasma collected by plasmapheresis potentially offers prolonged life span of some clotting factors. Plasmapheresis is an acceptable form of canine plasma collection for transfusion purposes, and further studies should be performed to determine all of its benefits. (*Am J Vet Res* 2019;80:578-585)



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