Hyperkalemic Atrial Standstill in Neonatal Calf Diarrhea

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Hyperkalemia has been associated with cardiac abnormalities and muscular disorders. Hyperkalemia is a common problem associated with the acid-base and electrolyte disturbances that occur in neonatal calves having acute diarrhea. Occasional calves with acute neonatal diarrhea, metabolic acidosis, and hyperkalemia have cardiac rate or rhythm abnormalities. Bradycardia observed in three such calves was found to represent atrial standstill and was attributed to hyperkalemia. (Journal of Veterinary Internal Medicine 1992; 6:294–297)

INFECTIOUS DIARRHEA is a frequent and often fatal disease affecting calves in the first weeks of life.[1–3] Numerous causative organisms have been reported,[4] including enterotoxigenic *Escherichia coli,* rotavirus, coronavirus, *Salmonella* spp, *Clostridia* spp, and cryptosporidia.[5–9] The profuse diarrhea associated with infection by these organisms can lead to significant losses in bicarbonate, electrolytes, and water and severe life-threatening metabolic disturbances.[10]

Concomitant metabolic acidosis can cause significant transcellular electrolyte shifts whereby potassium ions leave the cell to enter the extracellular fluid in exchange for hydrogen ions.[11–14] Dehydration can further contribute to this hyperkalemic state by decreasing renal blood flow, thereby decreasing potassium excretion.[11,15] In response to hypovolemia, sodium reabsorption in the proximal tubule results in decreased delivery of sodium and water to the distal tubular sodium-dependent potassium secretory site, which results in potassium accumulation.[16] As the serum potassium increases (>5.5 mEq/L), aberrations in cardiac excitability occur and are manifested as progressive atrial standstill and, if left untreated, ultimately as ventricular fibrillation or asystole.[17–23] In this article, we describe three calves in which diarrhea was believed to contribute to hyperkalemia along with the associated electrocardiographic finding of atrial standstill.

Calf 1

A 3-day-old Jersey bull calf weighing 28.6 kg was examined at the New York State College of Veterinary Medicine as a representative of a recent farm outbreak of neonatal calf diarrhea during which seven calves had died. The calf had received colostrum, an *E. coli* bacterin, and a selenium injection at birth. Before arrival, this calf had been treated with scour boluses, oral electrolytes, and penicillin.

At initial examination, the calf was recumbent, dehydrated (~8%), hypothermic (T = 97.0°F/36.11°C), and had profuse watery diarrhea of 12 hours duration. Auscultation revealed bradycardia (heart rate = 88 beats/min), poor pulse quality, and occasional pulse deficits. Electrocardiographic analysis showed an irregular rhythm, no P-waves, and inconsistent supraventricular premature complexes with aberrant ventricular conduction (Figure 1A). A serum biochemical profile indicated marked hyperkalemia (K = 9.6 mEq/L), azotemia (BUN = 105.0 mg/dL), increased creatinine concentration (6.1 mg/dL), and an increased anion gap (34.0 mEq/L).

An intravenous catheter was placed in the jugular vein and therapy with 5% dextrose and 150 mEq $NaHCO_3$/L was instituted. Electrocardiographic tracings were done every 15 minutes until a normal sinus rhythm was recorded 45 minutes later, at which point the calf was able to stand. Two heavily encapsulated species of *E. coli* and *Clostridium perfringens* were isolated from the feces. Fecal electron microscopy for viruses and blood cultures were negative. Fluid therapy was maintained until the serum potassium and creatinine were normal.

Calf 2

A 2-week-old Holstein heifer weighing 84 kg was presented because of diarrhea of 48 hours duration. Six other neonatal calves on the farm also were affected. Before admission, the calf had been treated with gentomi-

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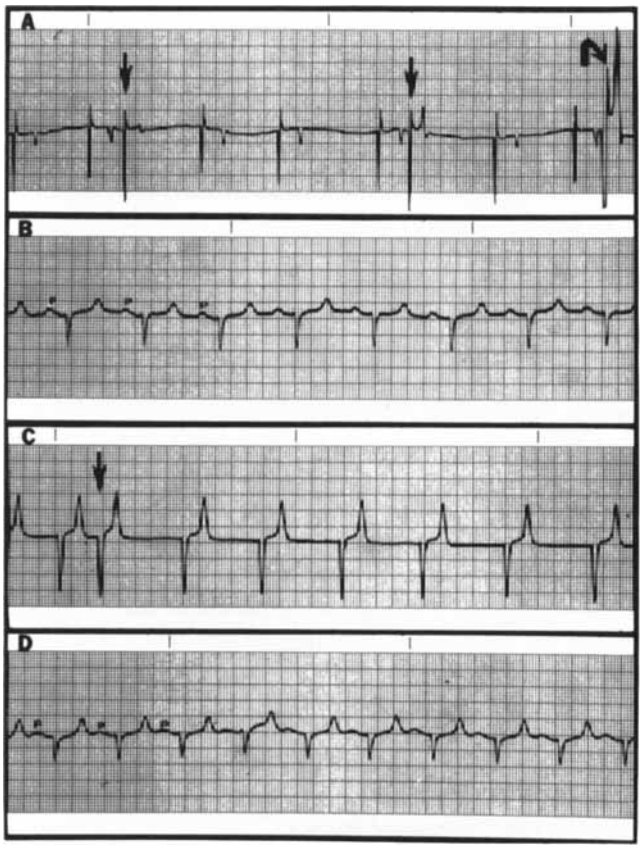

FIG. 1. Base apex electrocardiograms recorded from two calves (strips A and C: 1 cm = 1 mv; strips B and D ½ cm = 1 mv 25 mm/second). A: Electrocardiogram recorded from calf with plasma potassium of 9.6 mEq/L. Atrial standstill (absence of P-waves) and negative spiked T-waves are seen. Arrows point to narrow premature complexes that are either superventricular or high ventricular in origin. The curved arrow points to either a premature ventricular complex or a supraventricular impulse conducted with aberrancy. B: Electrocardiogram recorded from same calf as in A after therapy. Plasma potassium was 5.1 mEq/L. Sinus rhythm is evident, P-waves (p) are visible, and the T-wave has changed polarity and is no longer spiked. A 60-cycle artifact is also recorded. C: Electrocardiogram recorded from a calf with plasma potassium of 7.6 mEq/L. Atrial standstill with a supraventricular premature complex (arrow) and tall spiked T-waves are seen as well as variable R-R' interval and S-T segment elevation. D: Electrocardiogram recorded from the same calf as C after therapy. Plasma potassium was 4.1 mEq/L. Sinus rhythm with normal T-waves can be seen.

cin,* trimethoprim† and sulfadiozine and a commercial scour preparation. All calves had a K-99 *E. coli* bacterin at 2 days of age. Upon admission to the clinic, the calf was febrile (T = 103.2°F/39.56°C), depressed, recumbent, and approximately 8% dehydrated. On physical examination, an arrhythmia, weak peripheral pulses, and a slightly thickened umbilicus were found. Electrocardiography revealed bradycardia (heart rate = 89 beats/min) with no P waves present. Biochemical abnormalities included hyponatremia (Na^+ = 122 mEq/L), hyperkalemia (K^+ = 8.7 mEq/L), metabolic acidosis (pH = 7.27; HCO_3 = 10 mm/L; pCO_2 = 23 mmHg), an increased anion gap (2.5 mEq/L), and azotemia (BUN = 83 mg/dL; creatinine = 8.1 mg/dL).

Intravenous fluid therapy with 2 L of 5% dextrose with 150 mEq/L $NaHCO_3$ was given over a 1-hour period and ceftiofur‡ therapy (1 mg/lb twice daily) was instituted.

Electrocardiograms (not shown) were obtained at 15-minute intervals until a normal sinus rhythm was obtained approximately 1 hour later. Serum potassium was 5.4 mEq/L at this time. Fluid therapy was changed to lactated Ringer's solution to maintain a normal serum potassium concentration because of the possibility of total body potassium depletion associated with diarrhea.

Fecal culture yielded *Clostridium perfringens* and fecal flotation revealed *Cryptosporidium spp.* Electron microscopy was negative for viral particles.

Calf 3

A 12-day-old Holstein heifer weighing 30 kg was presented with diarrhea of 48 hours duration and a patent urachus. All calves on the premises had received an *E. coli* bacterin at birth, and this calf was the only one affected. The calf was recumbent, approximately 10% dehydrated, hypothermic (T = 98.6°F/37.0°C), and had an irregular heart rate (HR = 98 beats/min). A serum biochemical profile revealed hyponatremia (Na^+ = 124.0 mEq/L), hyperkalemia (K^+ = 7.5 mEq/L), hypochloremia (Cl = 93.0 mEq/L), an increased anion gap (23 mEq/L), and azotemia (BUN = 80.0 mg/dL) with increased creatinine concentration (2.6 mg/dL). The electrocardiogram showed an irregular rhythm and the absence of P-waves (Figure 1C).

Intravenous fluid therapy consisting of 2 L of 5% dextrose with 150 mEq/L $NaHCO_3$ was given over a 45-minute period. Electrocardiograms were performed at 15-minute intervals. A normal sinus rhythm was recorded after 30 minutes of therapy. Serum potassium normalized at 4.1 mEq/uL, at which time a balanced electrolyte solution was given.

A heavily encapsulated *E. coli* was isolated from the feces. Culture of the umbilicus, resected on the second day of hospitalization, yielded a different *E. coli,* as well as *Actinomyces pyogenes,* and *Clostridium* spp. Results of blood cultures, fecal flotation, and electron microscopy were negative.

Discussion

Cardiac arrhythmias in calves are not common; when they are detected, septicemia, endotoxemia, white muscle disease, congenital anomalies, ingestion of toxins, hypoglycemia, and electrolyte aberrations must be considered.[13,24] Changes in serum potassium concentrations,

* Gentocin, Schering Corp., Kenilworth, NJ.
† Tribrissen, Burroughs Wellcome, Co., Kansas City, MO.
‡ Naxcel (ceftiofur sodium), Upjohn Co., Kalamazoo, MI.

particularly hyperkalemia, can profoundly affect myocardial activity.[20–22] Typically, this is viewed electrocardiographically as a combination of broadening, flattening, or disappearance of the P-wave. A tall spiked T-wave usually is observed.[21–23,25] It must be emphasized, however, that the T-wave is a highly variable component of the electrocardiogram and that T-wave changes alone cannot accurately predict hyperkalemia.[25,26] T-wave changes can occur with normal serum K^+ concentrations.[6,12]

As serum K^+ concentration increases (>7.0 mEq/L), the atrial myocardium becomes refractory to activation. However, conduction of impulses from the S-A and A-V nodes continues as these specialized fibers are relatively resistant to hypercalcemia.[20,23,27,28] This differential effect on cardiac tissues is due to an increased sensitivity of atrial myocardium when compared with the ventricular myocytes and specialized tissues to depolarization by excess K^+. The increase in extracellular K^+ and subsequent drop in intracellular K^+ reduces the resting membrane potential, shortens the duration of the action potential,[20] and eventually decreases the excitability of the atrial myocyte. Continued increase in serum K^+ leads to ventricular conduction disturbances, as evidenced by widening or axis deviation of the QRS. Other ECG alterations, such as ST segment elevation, irregular R-R intervals, and premature complexes also have been described.[19,20] These changes also were evident in Figure 1C. The St-T change could be indicative of myocardial ischemia or could develop secondary to QRS alterations. Bundle of His studies in the dog have shown that A-V conduction failure does occur at higher K^+ concentrations,[19,20,29] indicating progressive A-V nodal block.[20] It has been shown that the cardiotoxic effects of hyperkalemia can be exacerbated by concurrent hyponatremia and/or hypocalcemia.[12,20,30] This, in conjunction with decreased intracellular K^+ concentrations could possibly explain the clinical finding of hyperkalemic atrial standstill in diarrhetic calves at lesser K^+ concentration than in experimentally induced hyperkalemic atrial standstill.[21–23,31]

Arrhythmia and/or bradycardia in a diarrhetic calf should alert the examiner to the possible presence of hyperkalemia. Assessment of acid-base status, serum electrolyte concentrations, and renal function can assist the diagnosis and management of the hyperkalemic patient. Hydration status, however, is best assessed clinically in calves[2] because of the wide variations in PCV and TP during the neonatal period.[2,32] Furthermore, electrocardiographic analysis can provide rapid insight into the cause of an arrhythmia and supports a diagnosis of hyperkalemic atrial standstill. Treatment is directed at reducing serum K^+ concentration and increasing volume, and correcting concurrent metabolic derangement.

Intravenous fluid therapy is paramount. The use of 5% dextrose and sodium bicarbonate is an effective initial treatment for hyperkalemia. Both dextrose and bicarbonate promote transcellular potassium transport from extracellular fluid.[11,16,33] Insulin also can be used to expedite potassium transfer into cells because it increases sodium potassium ATPase activity at the cell membrane level, a process independent of glucose uptake into the cell. However, due to the frequent presence of concurrent hypoglycemia, caution must be exercised when using insulin.

Calcium can also be used in severe life-threatening hyperkalemia because it antagonizes the cardiotoxic effects of potassium.[33,34] However, this is a transient effect, and further therapies must be employed to decrease serum potassium. Recent reports have suggested the efficacy of β_2 adrenergic agents such as salbutamol in treating hyperkalemic patients.[35–37] These reports suggest results similar to treatment with dextrose, alkalinizing agents, and insulin. Consideration should be given to these agents in animals with hyperkalemia.

Arrhythmias, although uncommon in calves, should signal the possibility of hyperkalemia when associated with signs of diarrhea, dehydration, and recumbency in neonatal calves. Electrocardiographic analysis can provide a rapid indication of hyperkalemia in situations in which serum electrolyte determination is unavailable or delayed. Electrocardiography can be further used to assess the effectiveness of therapy.

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