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Terminal atrial standstill with ventricular escape rhythm in a neonatal calf with acute diarrhea

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Abstract: Here we present a case of terminal atrial standstill with ventricular escape rhythm associated with hyperkalemia and metabolic acidosis contributed to by diarrhea and lactate in a neonatal calf. This life-threatening arrhythmia disappeared and sinus rhythm was obtained by fluid therapy consisting of isotonic saline with $NaHCO_3$ and epinephrine.

Key words: Calf, terminal atrial standstill, hyperkalemia, metabolic acidosis

1. Introduction

Calfhood diseases have a major impact on the economic viability of cattle operations due to the direct costs of calf losses, treatment, and the long-term effects on performance (1,2). Diarrhea is generally the most common cause of morbidity and mortality in pre-weaned calves.

Metabolic acidosis is a frequent consequence of gastrointestinal diseases and is found in calves with dehydration. The so-called 'acidosis without dehydration syndrome' is found in clinically sick calves with minimal or no signs of dehydration (3).

Hyperkalemia is most commonly observed in neonatal calves that develop acute metabolic acidosis associated with secretory diarrhea caused by *E. coli* and may cause a variety of arrhythmias. Arrhythmias, although uncommon in calves, should signal the possibility of hyperkalemia when associated with signs of diarrhea, dehydration, and recumbency in neonatal calves (4).

Electrocardiographic (ECG) 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 (5). Atrial standstill and other arrhythmias have been documented in diarrheic calves having metabolic acidosis and hyperkalemia (4,6).

Because severe hyperkalemia may be associated with pathologic bradycardia, even without confirmatory blood work, the experienced clinician should be alert to the therapeutic need for fluids that will specifically address hyperkalemia in severely dehydrated, diarrheic calves with discordantly low heart rates for their systemic state. Hyperkalemia is corrected by the administration of glucose or sodium bicarbonate. Although the hypokalemic effect of glucose is well established in other species, controversial results are reported for sodium bicarbonate. Glucose lowers plasma K mainly through an insulin-dependent intracellular translocation of K, whereas $NaHCO_3$ (sodium bicarbonate) causes hypokalemia through hemodilution followed by intracellular translocation of K caused by a strong ion effect (7).

In this article, we describe a calf in which diarrhea and lactate were thought to have contributed to acidemia and hyperkalemia along with the associated electrocardiographic finding of terminal atrial standstill, which has disappeared following a fluid therapy consisting of isotonic saline with 150 mEq/L $NaHCO_3$ and epinephrine.

2. Case history

A 7-day-old Holstein dairy calf weighing 35 kg and suffering from diarrhea for 2 days was presented to the teaching hospital (Selçuk University, Faculty of Veterinary Medicine) and was the subject of this case report. Hematological analysis (Hemocell Counter MS4e, Melet Schloesing Laboratories, France) (complete blood count: cell counts, MCV, MCHC, PCV, Hb, and blood gas analysis (Gem Premier 3000, Instrumentation Laboratory, USA) (pH, PO_2, PCO_2, HCO_3, BE, lactate, Na, K) and electrocardiographic analysis (Petaş, Turkey) (lead V^{10}, 25 mm/s, 1 cm = 1 mV) were performed. Intravenous fluid therapy with isotonic saline (NaCl 0.9%, I.E., Ulubay®) with 150 mEq/L $NaHCO_3$ (Bikarvil, Vilsan®) and epinephrine

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(1 mg, Adrenalin, Biofarma®) was instituted. A total of 2 L of fluid was given over a 2 h period. Measurement of buffer need was based on a formula (bicarbonate requirement = body weight × base excess × 0.6).

3. Results and discussion

At the initial examination, the calf was recumbent, dehydrated (–8%), hypothermic (T = 36.8 °C), and had profuse watery diarrhea of 24-h duration. Poor pulse quality and occasional pulse deficits were revealed by palpation and auscultation, and there was no bradycardia (heart rate = 92 beats/min). Electrocardiographic analysis showed a regular rhythm, no P-waves, and consistent ventricular escape rhythm (Figure 1A).

Biochemical abnormalities included metabolic acidosis (pH = 7.06, HCO_3 = 10.4 mEq/L, BE = –19 mEq/L, lactate = 7.9 mEq/L, pCO_2 = 40 mmHg), hyperkalemia (K = 9.5 mEq/L), and hyponatremia (Na = 117 mEq/L). Ionized Ca and glucose levels were 1.13 mEq/L and 113 mg/dL, respectively. There were abnormal complete blood count values (WBC = 20.24 × 10^3/µL, lymphocytes = 42.7%, granulocytes = 53.8%, monocytes = 3.5%, RBC = 4.6 × 10^6/µL, MCV = 31.5 fL, MCHC = 37.5 g/dL, Hb = 5.4 g/dL, PCV = 18%, RDW = 13.9%, platelets = 657 × 10^3/µL) (reference ranges: calf temperature = 38.5–39.5 °C, calf pulse rate = 80–120/min, WBC = 4–12 × 10^3/µL, lymphocytes = 45%–80%, granulocytes = 10%–30%, monocytes = 0%–0.6%, RBC = 6–11 × 10^6/µL, MCV = 40–60 fL, MCHC = 30–40 g/dL, Hb = 6–15 g/dL, PCV = 25–50%, RDW = 8%–12%, platelets = 100–800 × 10^3/µL, pH = 7.35–7.5, HCO_3 = 24–34 mEq/L, BE = ±2 mEq/L, lactate = 0.6–2.2 mEq/L, pCO_2 = 41–50 mmHg, Na = 134–145 mEq/L, K = 3.9–5.3 mEq/L, Ica = 1 mEq/L, and glucose = 31–77 mg/dL) (4,8).

After fluid therapy blood pH, hyperkalemia, hyponatremia, and lactate concentration normalized at 7.42 mEq/L, 4.3 mEq/L, 135 mEq/L, and 3.6 mEq/L, respectively. Electrocardiograms were continuously monitored. Narrowing of R and T waves was observed (Figure 1B)

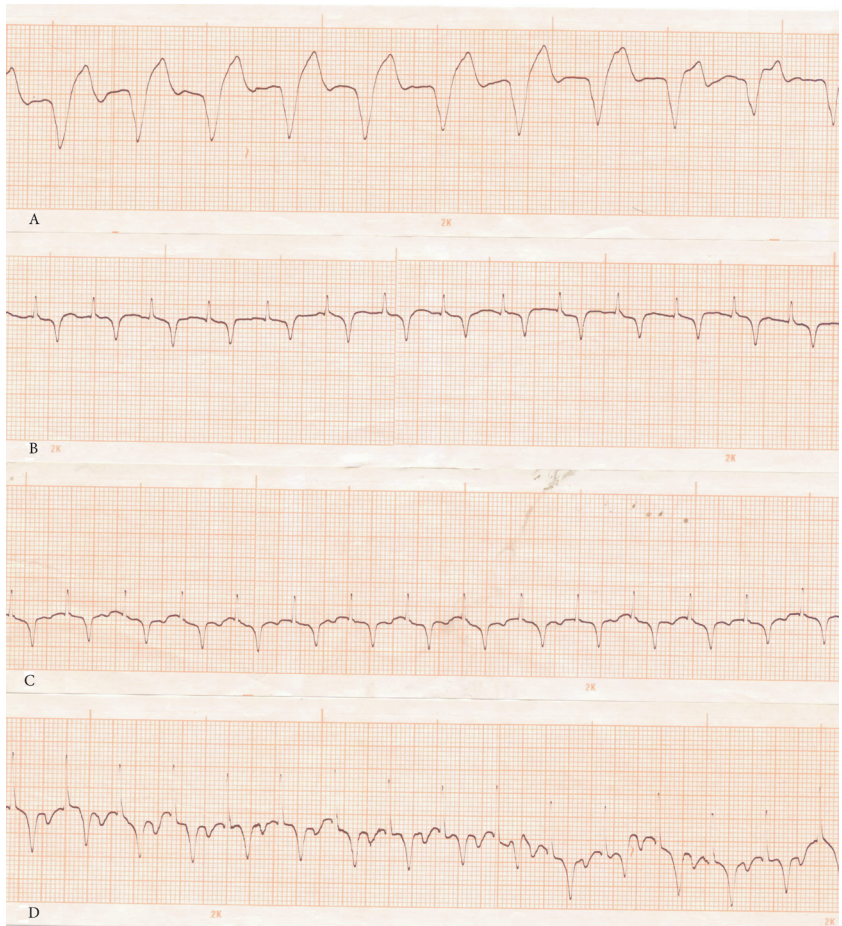

Figure 1. A: Terminal atrial standstill with ventricular escape rhythm (V10, 25 mm/s, 1 cm = 1 mV). B: Narrowing R and T waves approximately 1 h later. C: P waves began to appear until 2 h after the fluid therapy. D: A sinus rhythm was recorded over 2 h later.

approximately 1 h later. P waves began to appear (Figure 1C) and a sinus rhythm was recorded over 2 h later (Figure 1D), at which point the calf was able to stand.

Cardiac arrhythmias or bradycardia associated with hyperkalemia is primarily observed in neonates having severely acute diarrhea. Enterotoxigenic *E. coli* causing secretory diarrhea, metabolic acidosis, low plasma bicarbonate values, and hyperkalemia appears to be the most common causative organism. Rotavirus or coronavirus also may be involved in calf diarrhea, but they seldom produce as profound a metabolic acidosis as *E. coli* (4). Studies have demonstrated that D-lactate is a major component of the metabolic acidosis generally present in calves with diarrhea. The importance of bacterial overgrowth in the intestines of calves with diarrhea gained more attention when the role of D-lactate (the anion of D-lactic acid) in the development of metabolic acidosis was discovered (9–14). Trefz et al. (15) observed good correlations between base excess values and concentrations of D-lactate with the clinical parameters posture, behavior, and palpebral reflex. In the present diarrheic calf, lactate was an important component of the metabolic acidosis.

Hyperkalemia reduces the resting membrane potential, which initially makes cells more excitable, but gradually (with further elevation in potassium and further reduction in resting membrane potential) the cells become less excitable. Atrial myocytes seem more sensitive to these effects than those within the ventricles. Cardiac conduction is affected, and several characteristic ECG findings evolve in a typical sequence that correlates well with increasing K values. ECG changes include peaking of the T wave, shortening and widening of the P wave, prolongation of the PR interval, eventual disappearance of the P wave, widening of the QRS complex, and irregular R-R intervals (4). Further progression may lead to AV block, escape beats, ventricular fibrillation, asystole, and death. In atrial standstill there is an absence of any atrial activity, which can be confirmed by fluoroscopy or echocardiography (there is no A wave on an M-mode of the mitral valve or no atrial contraction inflow on Doppler studies). This occurs due to a failure of atrial muscle depolarization, such as when the SA node may produce an impulse, but the atria are not depolarized and remain inactive. If this occurs due to hyperkalemia, the impulses are conducted from the SA node by internodal pathways to the AV node; thus there is a sinoventricular rhythm. When the dominant pacemaker tissue, usually the SA node, fails to discharge for a long period the pacemaker tissue with a slower intrinsic rate (junctional or ventricular) may then discharge (i.e. they 'escape' the control of the SA node). This is commonly seen in association with bradyarrhythmias (i.e. sinus bradycardia, sinus arrest, AV block, and atrial standstill). Escape complexes are sometimes referred to as rescue beats, because if they did not occur death would be imminent. Atrial standstill due to hyperkalemia is usually life threatening and can occur in association with a 'dying' heart and is termed terminal atrial standstill (16). Idioventricular rhythm is the presence of only the ventricular escape complexes on an ECG and is also typically a terminal rhythm (17). An atrial standstill (absence of P-waves) and negative spiked T waves were recorded from a calf with plasma potassium of 9.6 mEq/L. In this record, narrow premature complexes were either supraventricular or high ventricular in origin. There was also a premature ventricular complex or a supraventricular impulse conducted with aberrancy (5). In the present calf, a terminal atrial standstill with ventricular escape rhythm consisting of highly wide R and T waves without bradycardia was recorded in the ECG (Figure 1A). This arrhythmia could be converted into ventricular fibrillation without intervention of the fluid therapy in a short time. We earlier observed the latter arrhythmia in another calf suffering from diarrhea and having severely hyperkalemia (12 mEq/L) (18).

Diarrhea is generally the most common cause of morbidity and mortality in pre-weaned calves (19–21). Calves less than 2 weeks of age that have developed acute diarrhea, are recumbent and dehydrated, and have bradycardia or arrhythmia should be suspected of being hyperkalemic. Obviously only an acid-base and electrolyte analysis of the blood and an ECG can confirm this. However, these may not be available in the field. The consequences of underestimating the life-threatening importance of the heart and K relationship in these patients are severe. Calves suspected to be hyperkalemic based on history, physical signs, and arrhythmia or bradycardia should receive alkalinizing fluids and dextrose. As a rule, the diarrheic calf that can stand should receive oral fluids; otherwise, intravenous fluid therapy is desirable (22). Being neonates, hypoglycemia may contribute to bradycardia when this sign is present. Buffer solutions containing sodium bicarbonate have safely been used recently to improve the acid–base status in acidotic perinatal calves (23). One way to treat metabolic acidosis and hyperkalemia is by IV infusions of 5% dextrose solution containing 150 mEq $NaHCO_3$/L. Usually 1 to 3 L is necessary, depending on the magnitude of the metabolic acidosis and bicarbonate deficit. Glucose and bicarbonate help transport K back into cells, and the glucose also treats or prevents potential hypoglycemia. Once the acute crisis has been resolved, the calf may be safely treated with balanced electrolyte solutions containing potassium. Calves with diarrhea, despite having plasma hyperkalemia, have total body potassium deficits and require potassium supplementation. This may be true even in the acute phase of the disease, but when serum K is 5.0 to 8.0 mEq/L there is no time to worry about a 'total body potassium deficit'.

Hundreds of calves were treated as suggested above, and those with a venous blood pH of 7.0 or greater have a good to excellent prognosis unless they have had failure of passive transfer of immunoglobulins and subsequent septicemia. Specific insulin therapy as an adjunct to bicarbonate and glucose to correct hyperkalemia is not necessary in calves (24). IV administrations of isotonic (1.3%) and hypertonic (8.4%) sodium bicarbonate solutions are similarly effective in the correction of strong ion (metabolic) acidosis in severely dehydrated, diarrheic calves (25).

Because the present calf was hyponatremic and not hypoglycemic we used a fluid therapy consisting of isotonic saline with 150 mEq/L $NaHCO_3$. As a vasopressor, epinephrine (1 mg) was added to the fluid because terminal atrial standstill with ventricular escape rhythm could occur in association with a 'dying' heart. No studies exist comparing the efficacy of various pressors in critically ill animals, and the choice of drug is often based on individual preference and personal experience. A sinus rhythm and standing up were achieved after the fluid therapy (Figure 1D).

Electrocardiographic analysis can provide rapid insight into the cause of an arrhythmia and supports a diagnosis of acidotic and hyperkalemic terminal atrial standstill with ventricular escape rhythm contributed to by diarrhea and lactate. Treatment is directed at reducing serum K concentration, increasing blood pH and volume, and correcting concurrent metabolic derangement.

References

1. Lorenz, I., Mee, J.F., Earley, B., More, S.J.: Calf health from birth to weaning. I. General aspects of disease prevention. Ir. Vet. J., 2011; 64: 10.
2. Lorenz, I., Fagan, J., More, S.J.: Calf health from birth to weaning. II. Management of diarrhoea in pre-weaned calves. Ir. Vet. J., 2011; 64: 9.
3. Naylor, J.M., Zello, G.A., Abeysekara, S.: Advances in oral and intravenous fluid therapy of calves with gastrointestinal disease. Proceedings of the 24th World Buiatrics Congress, Nice, 2006; 139–150.
4. Divers, T.J., Peek, S.F.: Rebhun's Diseases of Dairy Cattle. Elsevier Inc., St. Louis, MO, USA. 2008.
5. Weldon, A.D., Moise, N.S., Rebhun, W.C.: Hyperkalemic atrial standstill in neonatal calf diarrhea. J. Vet. Intern. Med., 1992; 6: 294–297. Erratum in: J Vet Intern Med 1992; 6: 324.
6. Smith, B.P.: Large Animal Internal Medicine. 4th Ed. Mosby Elsevier, New York. 2009.
7. Grünberg, W., Hartmann, H., Burfeind, O, Heuwieser, W., Staufenbiel, R.: Plasma potassium-lowering effect of oral glucose, sodium bicarbonate, and the combination thereof in healthy neonatal dairy calves. J. Dairy Sci., 2011; 94: 5646–5655.
8. Radostits, O.M., Gay, C.C., Hinchcliff, K.W., Constable, P.D.: Veterinary Medicine. 10th Ed. Saunders Elsevier, New York. 2006.
9. Ewaschuk, J.B., Naylor, J.M., Zello, G.A.: Anion gap correlates with serum D- and DL-lactate concentration in diarrheic neonatal calves. J. Vet. Intern. Med., 2003; 17: 940–942.
10. Ewaschuk, J.B., Naylor, J.M., Zello, G.A.: D-Lactate in human and ruminant metabolism. J. Nutr., 2005; 135: 1619–1625.
11. Lorenz, I:. Investigations on the influence of serum D-lactate levels on clinical signs in calves with metabolic acidosis. Vet. J., 2004; 168: 323–327.
12. Lorenz, I.: Influence of D-lactate on metabolic acidosis and on prognosis in neonatal calves with diarrhoea. J. Vet. Med. Physiol. Pathol. Clin. Med., 2004; 51: 425–428.
13. Lorenz, I.: D-Lactic acidosis in calves. Vet. J., 2009; 179: 197–203.
14. Constable, P.D.: Treatment of calf diarrhea: antimicrobial and ancillary treatments. Vet. Clin. North Am. Food Anim., 2009; 25: 101–120.
15. Trefz, F.M., Lorch, A., Feist, M., Sauter-Louis, C., Lorenz, I.: Metabolic acidosis in neonatal calf diarrhea: clinical findings and theoretical assessment of a simple treatment protocol. J. Vet. Intern. Med., 2012; 26: 162–170.
16. Martin, M.: Small Animal ECGs: An Introductory Guide. 2nd Ed. Blackwell Publishing Ltd, Oxford. 2007.
17. Aiello, S.E.: The Merck Veterinary Manual. 9th Ed. Merck & Co, Inc. Rahway, NJ, USA. 2005.
18. Başoğlu, A., Turgut, K.: A case of ventricular flutter and fibrillation in a calf suffering from diarrhea. Eurasian J. Vet. Sci., 1989; 5: 259–263.
19. Lorenz, I., Fagan, J., More, S.J.: Calf health from birth to weaning. II. Management of diarrhoea in pre-weaned calves. Ir. Vet. J., 2011; 14: 9.
20. Azizzadeh, M., Shooroki, H.F., Kamalabadi, A.S., Stevenson, M.A.: Factors affecting calf mortality in Iranian Holstein dairy herds. Prev. Vet. Med., 2012; 104: 335–340.
21. Torsein, M., Lindberg, A., Sandgren, C.H., Waller, K.P., Törnquist, M., Svensson, C.: Risk factors for calf mortality in large Swedish dairy herds. Prev. Vet. Med., 2011; 99: 136–147.
22. Grove-White, D.H.: Monitoring and management of acidosis in calf diarrhoea. J. R. Soc. Med., 1998; 91: 195–198.
23. Bleul, U.T., Schwantag, S.C., Kähn, W.K.: Effects of hypertonic sodium bicarbonate solution on electrolyte concentrations and enzyme activities in newborn calves with respiratory and metabolic acidosis. Am. J. Vet. Res., 2007; 68: 850–857.
24. Berchtold, J.: Treatment of calf diarrhea: Intravenous fluid therapy. Vet. Clin. Noth Am. Food Anim. Pract., 2009; 25: 73–99.
25. Coskun, A., Sen, I., Guzelbektes, H., Ok, M., Turgut, K., Canikli, S.: Comparison of the effects of intravenous administration of isotonic and hypertonic sodium bicarbonate solutions on venous acid-base status in dehydrated calves with strong ion acidosis. J. Am. Vet. Med. Assoc., 2009; 236: 1098–1103.