Ventricular tachycardia associated with exhaustive exercise in a horse

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- Exhaustive exercise in horses can lead to signs of colic as a result of fluid and electrolyte depletion.
- Cardiac arrhythmias may develop following disturbances in electrolyte (particularly potassium) status and tissue perfusion.

A 12-year-old Appaloosa gelding was referred to the veterinary teaching hospital because of colic. The horse, which had not been in a regular conditioning program, had been ridden approximately 40 miles through difficult terrain over 3 days, and signs of abdominal discomfort had been observed intermittently since the first day of the ride. On the evening of the third day, the signs of colic (lying down and muscle tremors) worsened and the horse was admitted to the referring veterinarian's hospital. Initial examination by the referring veterinarian revealed lethargy, tachycardia (96 beats/min), dark mucous membranes, prolonged capillary refill time, and lack of gastrointestinal tract sounds. Attempts to obtain fluid or gas via nasogastric intubation were unproductive. Examination per rectum revealed no abnormalities with the exception of displacement of the spleen away from the body wall. Initial laboratory data included a high PCV (55%) and high total plasma protein concentration (9.0 g/dl). Blood urea nitrogen (BUN) concentration was moderately high (77 mg/dl; reference range, 10 to 24 mg/dl) as was serum creatine kinase (CK) activity (2,700 U/L; reference range, 92 to 307 U/L). Results of abdominal fluid analysis were within reference limits. Because of the severity of dehydration (estimated at 10% by the referring veterinarian), bilateral jugular catheters were placed for administration of fluids (30 L of lactated Ringer's solution, IV, over the initial 6 hours of hospitalization) and 12 L of an isotonic electrolyte solution (equal mixture of sodium chloride and potassium chloride) were administered orally via a nasogastric tube. In addition, antibiotic treatment (procaine penicillin G, 22,000 U/kg, IM, q 12 h) was initiated and the horse received IV doses of analgesics (900 mg of ketoprofen, 5 mg of detomidine, 50 mg of xylazine, and 5 mg of butorphanol) at various times during the initial 6 hours of admission to control signs of abdominal pain. The gelding was observed to urinate twice during the course of fluid treatment. The first urine voided was not discolored, but a complete urinalysis was not performed by the referring veterinarian. Because of the normal appearance of the urine and only a mild increase in CK activity, a primary diagnosis of exertional rhabdomyolysis was not made at that time.

Reexamination of the horse the next morning (4 days after onset of clinical signs) revealed mild improvement. Capillary refill time and mucous membrane color had returned to normal, although tachycardia (72 beats/min) and azotemia (BUN, 72 mg/dl; creatinine concentration, 5.4 mg/dl; reference range for creatinine concentration, 0.7 to 1.8 mg/dl) persisted. Although signs of abdominal pain were less frequent, sounds associated with gastrointestinal tract motility remained infrequent and findings on palpation per rectum were unchanged. Fecal production was low but consistency of the feces was normal. Although the horse had drank approximately 40 L of water through the night, its appetite was poor. Treatment consisted of further fluid therapy (30 L lactated Ringer's solution, IV, and 8 L of a similar isotonic oral electrolyte solution administered via a nasogastric tube) over the subsequent 6 hours. Lack of further improvement and persistence of tachycardia prompted referral.

Physical examination on admission to the large animal teaching hospital revealed lethargy, a normal rectal temperature, tachycardia (63 beats/min), and tachypnea (50 breaths/min). Gastrointestinal tract sounds were present and transrectal palpation revealed displacement of the spleen away from the body wall. The PCV and total protein concentration were 48% and 6.6 g/dl, respectively. Additional abnormal laboratory findings included neutrophilia (8,240 segmented neutrophils/μl; reference range, 2,100 to 7,000 cells/μl) with a left shift (927 bands/μl; reference range, 0 to 200 cells/μl), lymphopenia (618 cells/μl; reference range, 1,300 to 5,700 cells/μl), and hyperglycemia (147 mg/dl; reference range, 53 to 83 mg/dl); findings were consistent with stress. Azotemia (BUN, 54 mg/dl; creatinine concentration, 3.8 mg/dl) was apparent and its persistence in the face of fluid treatment indicated a renal in addition to a prerenal component. Hyperalbuminemia (3.9 g/dl; reference range, 2.8 to 3.2 g/dl) was suggestive of a persisting plasma volume deficit, and hypochloremia (80 mEq/L; reference range, 99 to 109 mEq/L) was consistent with persisting deficits or renal compromise. Serum potassium concentration at admission was 2.7 mEq/L (reference range, 2.4 to 4.7 mEq/L) and serum sodium concentration was within reference limits. Increased activities of CK (2,294 U/L) and aspartate aminotransferase (1,040 U/L; reference range, 153 to 411 U/L) reflected mild myonecrosis consequent to the prolonged exercise. Results of abdominal fluid analysis were within reference limits.

Initial treatment after referral consisted of administration of fluids (lactated Ringer's solution, IV, at a rate of 5 L/h for the first 2 hours, followed by 2 L/h throughout the night) and frequent monitoring of vital parameters. During the first 12 hours after admission, muscle fasciculations were intermittently observed, and the heart rate varied between 54 and 104 beats/min despite rather mild signs of abdominal discomfort. Cardiac auscultation revealed no audible murmur but it was not possible to auscult the fourth heart sound during the periods

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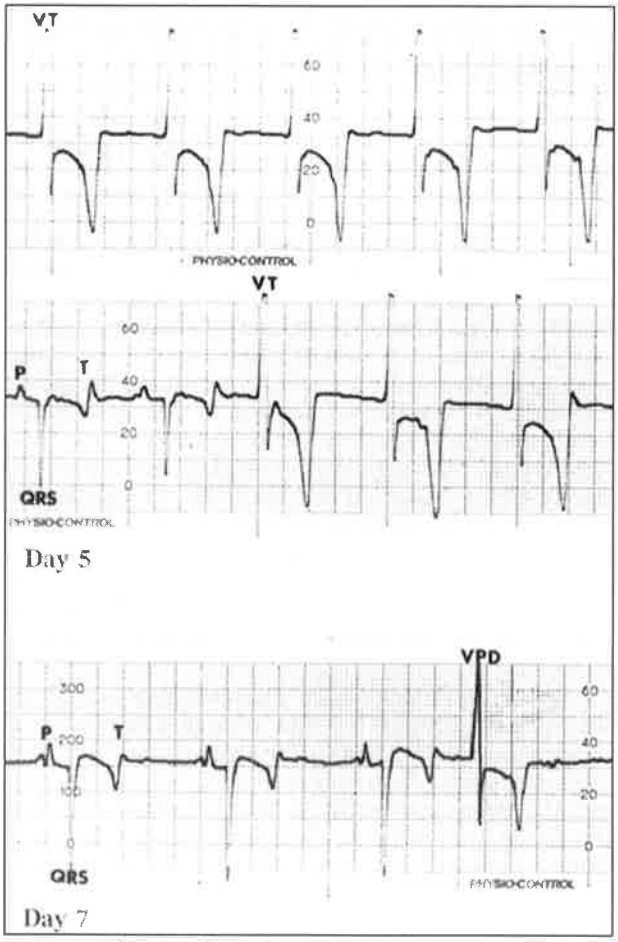

Figure 1—Electrocardiographic recordings from a horse with ventricular tachycardia after exhaustive exercise. Ventricular tachycardia (VT) and ventricular premature depolarization (VPD) were evident on ECG obtained 5 and 7 days, respectively, after onset of clinical signs. 10 mm = 1 mV; 25 mm/sec; P = P wave; QRS = QRS complex; T = T wave; top and middle tracings = day 5; bottom tracing = day 7.

of greatest tachycardia. Peripheral pulse pressures were within normal limits, and the apex beat was palpably normal. A cardiac arrhythmia was suspected, and electrocardiography revealed episodic unifocal ventricular tachycardia (Fig 1). Echocardiography was not performed because of owner financial constraints and the low possibility of detecting evidence of cardiac pathology.[1]

By the next morning (day 5 from onset of clinical signs), the horse's attitude had improved and it began to show interest in eating. Intravenous fluid administration was discontinued, but an isotonic oral electrolyte solution was made available at all times. Laboratory assessment revealed a PCV of 30% and a total protein concentration of 5.5 g/dl. A free-catch urine sample had a specific gravity of 1.016 and contained blood/urinary pigments. Although the findings could have been consistent with mild acute renal failure, the dilute urine and pigmenturia also could be explained by fluid treatment and myonecrosis. In either case, the BUN and creatinine concentrations had returned to within reference limits on day 5 and remained within normal limits throughout the remainder of hospitalization. Abnormal laboratory values detected on day 5 included mild leukopenia (total WBC count, 4,000 cells/μl, with 2,640 segmented neutrophils and 360 bands/μl), hypochloremia (89 mEq/L), hypokalemia (2.3 mEq/L), and slightly high activities of CK and aspartute aminotransferase. Because of the probability of persisting total body deficits of potassium and chloride, oral potassium chloride supplementation was initiated (25 g of KCl oral drench, q 12 h, on days 5 to 8 for a total dose of approximately 3,000 mEq or 10% of body stores).[2] Serum potassium concentrations steadily increased over the subsequent 3 days.

Mean heart rate decreased by the afternoon of day 6 (48 to 52 beats/min). Electrocardiography revealed less frequent paroxysms of ventricular tachycardia and a few isolated premature ventricular beats that were similar in waveform to those observed during the episodes of ventricular tachycardia (Fig 1). By day 8, cardiac rhythm had returned to normal and the horse was discharged on day 10. Based on the history and clinical presentation, signs of gastrointestinal and cardiac disease were attributed to dehydration and electrolyte depletion as a consequence of exhaustive exercise. In addition, decreased perfusion or mild endotoxemia were additional factors that may have contributed to the development of the arrhythmia. Follow-up communication 15 months later found no recurrence of the disorders.

Medical problems resulting from exhaustive exercise have collectively been referred to as exhausted horse syndrome.[3-6] Common signs include lethargy, dehydration (with lack of thirst), decreased appetite, high rectal temperature, tachycardia, and tachypnea. Factors incriminated in development of the exhausted horse syndrome include the horse's level of training, duration of the ride, and difficulty of the terrain. Other important factors that are often overlooked by the rider are environmental temperature and humidity.[3,6]

During demanding physical activity such as an endurance ride, a horse can generate 3,000 to 6,000 kcal/h through aerobic metabolism of fat and glycogen. Such energy expenditure produces a substantial heat load, and dissipation of this heat is essential for continued performance.[6-8] Heat loss during exercise occurs by 4 mechanisms: conduction, convection, radiation, and evaporation.[3,6] In horses, evaporative heat loss plays the greatest role, from the skin as well as across the respiratory tract.[6,8] Evaporative heat loss may be hampered by the progressive fluid and electrolyte depletion during prolonged exertion, which leads to an eventual decrease in sweating.[2,3,6,8] Dehydration has been reported to be the most consistent finding of exhausted horse syndrome, and affected horses may lose up to 7 to 10% of their body weight.[2,5,9,10] In addition to a loss of water, sweating results in total body depletion of sodium, chloride, potassium, calcium, and magnesium.[9-12] In concert with these fluid and electrolyte losses (the most substantial being chloride), progressive hyperventilation to increase evaporative heat loss across the respiratory tract leads to development of hypochloremic metabolic and respiratory alkalosis.[4,12]

Disturbances in electrolyte and acid-base status and tissue perfusion result in alteration of resting and threshold membrane potentials. Clinical signs may include neuromuscular irritability (synchronous diaphragmatic

flutter, muscle fasciculations, or cramping), gastrointestinal tract disturbances, or cardiac arrhythmias.[3-6]

Gastrointestinal tract abnormalities may be manifested as colic or diarrhea. Gastrointestinal tract sounds are decreased or absent, and it has been suggested that potassium depletion may contribute to ileus by hyperpolarizing the resting membrane potential.[4,5] In addition, decreased gastrointestinal tract perfusion may lead to absorption of endotoxin, which also decreases motility.[13] Findings from studies of racehorses have indicated a significant increase in lipopolysaccharide concentrations after races between 1,000 and 2,800 m. It is theorized that hypoxia, elevated core temperature, or alteration in the splanchnic blood flow allow for release of endotoxins into the portal circulation.[14] Further, King and Gerring[15] have demonstrated significant inhibition of gastric, left dorsal, and small colon motility following administration of low doses (0.1 μg/kg) of endotoxin.

Although less commonly recognized, cardiovascular abnormalities have been associated with exhausted horse syndrome, and have included arrhythmias and murmurs.[16] Other causes of arrhythmias in horses include myocardial disease/irritation, metabolic disturbances associated with gastrointestinal tract disorders, bacterial infection and immune-mediated sequelae, autonomic imbalance, and feed or drug toxicoses.[1,17,18] Rhythm disruptions attributable to electrolyte disturbances are attributed primarily to alterations in potassium balance.[19] Either a deficit or excess of potassium may lead to the development of ventricular arrhythmias. Hypokalemia can induce arrhythmias via hyperpolarization of the resting membrane potential, leading to an increase in action potential duration, prolongation of the refractory period, and conduction block.[19] Alternately, hyperkalemia may decrease the resting membrane potential, reduce the duration of the action potential, induce more rapid depolarization, and suppress automaticity.[19] In the horse described in this report, total body depletion of potassium consequent to sweat losses and reduced dietary intake likely contributed to the development of the arrhythmia. Unfortunately, specific assessment of potassium balance, via determination of muscle or red cell potassium contents or determination of urinary clearance ratios, was not pursued.

Hypoxic damage and reperfusion injury have been described as mechanisms of myocardial injury, leading to an increase in the arrhythmogenic potential of the myocardium.[20-21] Myocardial ischemia and subsequent reperfusion have been linked to the production of oxygen radicals and subsequent lipid peroxidation and disruption of myocardial cell membranes, resulting in electrical instability.[20-22] Because of financial constraints, cardiac muscle isoenzyme values were not obtained in this horse.

Although the most important aspect of treatment of exhausted horses is aggressive fluid treatment, the initial intravenous administration of fluids that are not supplemented with potassium may potentiate extracellular hypokalemia and contribute to neuromuscular irritability and the development of arrhythmias. In the horse of this report, correction of the dehydration and electrolyte losses led to resolution of the signs of abdominal pain and, over a period of several days, the cardiac arrhythmia.

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