Hypokalemia, Muscle Weakness, and Recumbency in Dairy Cattle*

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ABSTRACT
Seventeen cases of severe hypokalemia (serum or plasma potassium \( \leq 2.1 \) mEq/L) in association with profound muscle weakness and recumbency in lactating dairy cattle were included in a retrospective study. The cattle were from 15 different farms. Eleven of the 17 animals were recumbent at presentation while the remaining six became recumbent within 6 hours of admission. Both multiparous cows (n = 11) and first calf heifers (n = 6) were included. The median days in milk was 21 (range: 5 to 110), and chronic, recurrent ketosis (15 of the 17 cases) was the most common preexistent condition. Potential musculoskeletal and neurologic causes of recumbency were ruled out on the basis of physical examination and ancillary diagnostics. Ten of the 17 animals were euthanized and underwent full necropsy examination that demonstrated ischemic muscle damage and varying degrees of hepatic lipidosis. Aggressive potassium supplementation was instituted in all 17 cases either orally, intravenously, or by a combination of both routes. In the seven individuals that survived, potassium supplementation was administered orally and intravenously in five, orally only in one, and intravenously only in one.

INTRODUCTION
Hypokalemia in cattle is commonly encountered secondary to anorexia and many primary
conditions of the gastrointestinal and urinary systems. However, the types of clinical signs seen with these conditions are rarely attributed specifically to hypokalemia, which is an electrolyte abnormality not typically associated with muscle weakness, recumbency, and the downer cow syndrome. Recently, a syndrome of severe muscle weakness, recumbency, and hypokalemia was reported in association with ketosis and the intramuscular administration of isoﬂupredone acetate (Predef® 2X, Pharmacia and Upjohn, Kalamazoo, MI) to lactating dairy cattle.1 Hypokalemia also has been documented as a potential cause of muscle weakness in cattle of varying ages, independent of corticosteroid administration.2 The purpose of this retrospective study was to characterize the history, presentation, and treatment of severe hypokalemia in dairy cattle with muscle weakness and recumbency.

MATERIALS AND METHODS

Subjects
Six first-calf heifers and 11 multiparous cows were included in this study, which was conducted between 1991 and 1998. The cattle were from 15 different farms. Eleven of the 17 animals were recumbent at presentation while the remaining six became recumbent within 6 hours of admission.

Criteria for Selection of Cases
Medical records of cattle presented to the large animal hospital at the New York State College of Veterinary Medicine at Cornell University between 1991 and 1998 were reviewed retrospectively. Cattle that demonstrated a serum or plasma potassium of ≤2.1 mEq/L at admission and that were concurrently normocalcemic, normomagnesemic, and not hypoglycemic were eligible for the study. Information obtained from the medical record for each animal included age, breed, days in milk (DIM) at presentation, medical and treatment history from the current lactation, selected biochemical data at presentation, the route of potassium supplementation during hospitalization, case outcome, and postmortem findings where applicable.

Case Management
Treatment of the cattle in this report included aggressive potassium supplementation, intravenous fluid support, and nursing care. The Table details whether potassium supplementation was intravenous, oral, or both for each animal. The amount of potassium administered to each animal varied substantially between individuals. Intravenous potassium typically was administered in the form of supplemental potassium chloride added to polyionic fluids at rates that varied from 60 to 315 mEq/h. The dose and frequency of oral potassium supplementation also were highly variable, with individual animals receiving between 120 g (1600 mEq) three times daily and 500 g (6800 mEq) twice daily diluted in warm tap water by orogastric tube. Several of the animals that received the highest levels of oral potassium supplementation developed moderate to severe diarrhea. In animals that died and in those that survived, restoration of normokalemia was challenging. The median duration of potassium supplementation required to achieve normokalemia in those that recovered was 3 days, with a range of 1 to 7 days. A variety of devices and techniques including hip lifters, slings, and a flotation tank were used to assist downer cattle with standing. In some cases more than one device was used during management.

Case Outcome
Ten of the 17 animals in this study were euthanized at the owner’s request. The remaining seven animals recovered and were discharged from the hospital. The decision to euthanize cattle was based on continued recumbency for
more than 72 hours duration and/or financial considerations. All euthanized animals underwent a full necropsy examination to rule out other potential causes of weakness or recumbency such as traumatic musculoskeletal injuries, spinal neoplasia, or severe systemic disease. Gross and histologic evidence of myopathy was identified in weight-bearing muscles of all euthanized animals. In addition, in cases 6, 8, and 17, muscle tissue was obtained from non–weight-bearing muscles (diaphragm, cervical, and intercostal musculature) and demonstrated lesions of acute, multifocal, myofiber degeneration, and necrosis consistent with hypokalemic myopathy in humans and cats (Figure 1). Moderate (cases 5, 6, 7, 8, 11, 13, and 17) to severe (cases 2, 4, and 10) hepatic lipidosis also was noted at necropsy.

■ RESULTS

The Table gives case details including the age, breed, DIM, and selected biochemical data at presentation for each animal. The Table also summarizes medical history for the current lactation and whether the animal was recumbent at presentation (eleven cases)—and if so, for how long—or able to stand but profoundly weak (six cases). All cattle that were profoundly weak at admission became recumbent within 6 hours of admission to the hospital. The Table also summarizes the serum or plasma potassium at presentation, the mode of potassium supplementation during hospitalization (K suppl.), and the case outcomes.

Cases tended to be concentrated in the first 45 days of lactation with a median DIM of 21 and a range of 5 to 110. There were six first-calf heifers and 11 multiparous cows. Fifteen of the 17 cases had a history of chronic ketosis during the current lactation, where chronic ketosis is defined as moderate to severe ketonemia or ketonuria for 5 days or more that had been treated on the farm on at least three separate occasions. Preadmission therapy of the 15 animals with a history of chronic ketosis was quite variable but all of them had received both 500 mL of 50% dextrose intravenously and between 6 and 18 oz of propylene glycol on at least three separate occasions. Ten of these 15 animals had received isoflupredone acetate parenterally on multiple occasions; four others had received dexamethasone parenterally or orally on multiple occasions; and one animal had received both dexamethasone and isoflupredone acetate parenterally on multiple occasions. The two animals with no history of chronic ketosis (cases 12 and 13) had a history of clinical mastitis that had been treated with intramammary infusions of 20 to 40 mg isoflupredone acetate after each of 15 consecutive milkings. Cases 2, 3, 10, 11, and 15 had received 10 to 20 mg isoflupredone acetate parenterally on three separate occasions; case 4 had received 20 mg isoflupredone acetate parenterally on four occasions; cases 6, 8, and 14 had received 10 to 20 mg isoflupredone acetate parenterally on five occasions; and case 17 had received 10 mg isoflupredone acetate parenterally on six occasions. Cases 1 and 9 had re-
<table>
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<th>Case</th>
<th>Age (y)</th>
<th>Breed</th>
<th>DIM</th>
<th>Medical History</th>
<th>Status at Admission</th>
<th>Medical Suppl.</th>
<th>K (3.9–5.8 mEq/L)</th>
<th>AST (48–107 IU/L)</th>
<th>CK (83–1357 IU/L)</th>
<th>GGT (13–39 IU/L)</th>
<th>BUN (10–29 mg/dL)</th>
<th>P (3.9–9.2 mg/dL)</th>
<th>Cl (96–104 mEq/L)</th>
<th>Venous pH</th>
<th>Base Excess (0–6)</th>
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<td>15</td>
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<td>18,93</td>
<td>91</td>
<td>9</td>
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Cases 2, 3, and 8 had a history of insulin administration as part of the therapy for chronic ketosis. Adjunct therapy for ketosis included niacin boluses in five animals (cases 2, 3, 6, 10, and 17) and multivitamin preparations in nine cases (cases 2, 3, 5, 6, 8, 10, 14, 16, and 17). The clinical signs at presentation observed in the cattle of this report included muscle fasciculations, weakness, difficulty rising, and recumbency. Five of the animals (cases 2, 7, 15, 16, and 17) were documented to be in atrial fibrillation at initial presentation. Eleven of the 17 animals were recumbent and unable to stand at presentation, while the six remaining animals were profoundly weak and became recumbent within 6 hours of admission. In 13 of the 17 animals, the weakness was so severe at some point during hospitalization that they could not rise from lateral recumbency and required external support to maintain a sternal position (Figure 2). Of these 13 animals, eight were euthanized and five recovered. Of the four animals that could maintain sternal recumbency unassisted (cases 2, 11, 15, and 17), two were ultimately euthanized and two recovered, suggesting that the ability to remain in sternal recumbency was not related to recovery.

The Table gives selected biochemical data obtained at admission from all 17 animals. In addition to severe hypokalemia, other pertinent findings on serum biochemistry included elevations in the muscle-specific enzyme creatine kinase in all animals (17 cases). In addi-
tion, all 17 animals had elevations in the levels of aspartate aminotransferase (AST), which is released into serum subsequent to hepatocellular injury. Eight animals also had elevations in serum activity of the enzyme γ-glutamyl transpeptidase (GGT), which is specific to biliary epithelium. It is not possible to say what proportion of the elevation in the serum activity of AST was the result of myopathy or of hepatocellular origin. Only one cow (case 17) had normal acid–base status and serum chloride levels on admission. The other animals demonstrated hypochloremic metabolic alkalosis (nine cases), hypochloremic metabolic acidosis (five cases), or normochloremic metabolic acidosis (two cases). All seven of the animals demonstrating metabolic acidosis at presentation were ultimately euthanized compared with only two of the nine animals with metabolic alkalosis. Hypophosphatemia was documented in four animals at presentation. On presentation all cattle demonstrated ketonuria that was at least moderate (40 mg/dL) on urine dipstick examination.

**DISCUSSION**

There are a number of important factors that could have contributed to the development of hypokalemia in the cattle in this study. The typical forage-based diets offered to dairy cattle are relatively high in potassium, and consequently urinary potassium excretion is high. Adaptation to a high-potassium diet significantly contributes to the development of hypokalemia with any condition that causes a reduction in voluntary feed intake because urinary potassium excretion cannot be reduced sufficiently or rapidly enough to maintain external potassium balance. However, hypokalemia from anorexia alone tends to be mild and asymptomatic. Physiologically, hypokalemia also can be the consequence of the intracellular movement of potassium resulting from metabolic alkalosis or insulin release secondary to hyperglycemia or as a pathologic result of increased potassium loss through the alimentary or urinary systems in cases of intestinal or renal disease. However, it is worth remembering that only about 10% of the body’s total potassium is found extracellularly, with the plasma level representing only 0.4% of the total. For these reasons plasma or serum potassium measurement can be an imprecise means of establishing whole body potassium status. Case 17 provides an example of an individual with one of the higher serum potassium measurements at admission but clinical evidence of severe hypokalemia, resulting in a complete inability to rise from lateral recumbency, and histologic features in non–weight-bearing muscles consistent with hypokalemic myopathy in other species. The severity of the hypokalemia at presentation, the chronicity of illness, and the histologic lesions consistent with hypokalemic myopathy in non–weight-bearing muscles such as those of the diaphragm, neck, and intercostal area are suggestive of severe whole body potassium depletion in several animals in this report.

Hypochloremia and metabolic alkalosis were the most common serum electrolyte and acid–
base abnormalities accompanying hypokalemia at presentation in the animals in this report, though metabolic acidosis was identified in seven animals. All seven animals that demonstrated metabolic acidosis at presentation were ultimately euthanized. Five of the seven animals exhibiting metabolic acidosis had been recumbent for several hours to days before admission. Three of these plus the two with metabolic acidosis that were still able to stand on admission also had a documented increase in the anion gap consistent with an increase in unmeasured anions. Possible sources of these unmeasured anions would include both lactate, potentially from muscle damage from intermittent or complete recumbency; poor tissue perfusion; or a combination of both; and ketoacids (all individuals demonstrated urine ketone measurements of at least 40 mg/dL on admission). Paradoxically, one of the two individuals with metabolic acidosis but a normal anion gap also had the highest creatine kinase at admission (case 10). However, all seven animals with metabolic acidosis at presentation were ultimately euthanized, suggesting that high creatine kinase is a negative prognostic sign.

Hepatic lipidosis was present at postmortem in all ten of the euthanized animals, and seven of these animals also had elevations in the hepatobiliary enzyme GGT at admission. A mild degree of hepatic lipidosis is expected and considered normal during the period of negative energy balance in early lactation. However, chronic ketosis would have predisposed the cattle in this study to more significant hepatic lipid infiltration and could have contributed to some of the clinical signs and biochemical abnormalities detected because severe hepatic lipidosis can lead to severe anorexia, weakness, recumbency, and death. Metabolic acidosis also would be an expected acid–base abnormality with advanced hepatic lipidosis and fulminating hepatic failure. Metabolic acidosis and biochemical evidence of advanced liver disease (elevations in GGT, low BUN) alongside hepatic lipidosis that was histologically classified as severe were documented in cases 4 and 10.

Hypophosphatemia was documented in four of the animals in this study and was a feature of 40% of the cows with hypokalemic myopathy and weakness in the study by Sielman et al.1 Hypophosphatemia also was documented in 35% of the cattle demonstrating hypokalemia in the study by Sattler et al.2 The potential contributory role that hypophosphatemia may have played in the development of weakness and recumbency in the cattle of this study is uncertain. The clinical signs of severe weakness were seen with both hypophosphatemic (four animals) and normophosphatemic (13 animals) cattle. Because no specific phosphorus supplementation was given to animals that recovered and their clinical signs improved with specific restoration of normokalemia only, it is unlikely that there was a significant contribution from low serum phosphorus.

Recurrent ketosis was an antecedent condition in 15 of the 17 animals in this study, a finding consistent with the report by Sielman et al.1 The two animals (cases 12 and 13) that did not have associated ketosis were representative of a herd problem of weakness and recumbency in lactating cows and heifers that was associated with clinical mastitis. During a 7-week period, 10 lactating animals on this farm exhibited signs of progressive weakness and recumbency. All animals had received intramammary infusions of 20 to 40 mg isoflupredone acetate three times a day as therapy for clinical mastitis for 5 to 7 days immediately before the development of clinical signs. Although detailed biochemical information is only available for cases 12 and 13, all 10 animals showed clinical signs consistent with those in other animals in this study, including

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profound weakness with an inability to support themselves in sternal recumbency.

Although all of the cows in the report by Sielman et al\(^1\) had received the corticosteroid isoflupredone acetate as part of the treatment for the ketosis, this was not a feature of all the cattle in this study. Twelve of the 17 animals documented here had received isoflupredone only, while four had received dexamethasone only and one had received both isoflupredone and dexamethasone. It should be noted that in all cases where isoflupredone acetate was used, the amount used, dose frequency, or route of administration were contrary to the manufacturer’s current recommendations. The package insert stipulates that 10 to 20 mg of isoflupredone acetate should be administered by intramuscular injection, and that treatment can be repeated in 12 to 24 hours if necessary. The potential for enhanced renal and gastrointestinal potassium losses resulting from some mineralocorticoid activity can exist after isoflupredone acetate administration, particularly when the product is used repeatedly. Although the specific mineralocorticoid activity of isoflupredone acetate is unknown, it has been shown to possess as potent a mineralocorticoid effect as aldosterone both in vivo and in vitro in an adrenalectomized rat model.\(^13\) By comparison, dexamethasone is considered to have minimal mineralocorticoid effect. Values of 55%, 126%, 81%, 128%, and 122% for the urinary fractional excretion of potassium were obtained from cases 7, 8, 10, 14, and 17, respectively, before intravenous or oral potassium supplementation. Each of the five animals had received multiple doses of isoflupredone acetate in the days before presentation. However, in calculating these fractional excretion values, urine potassium measurement was performed using an ion-selective electrode technique, which has been demonstrated consistently to underestimate urine potassium concentrations in cattle, putatively from chelation by a low–molecular-weight compound.\(^14,15\) Therefore the values calculated in these five animals may actually underestimate the true fractional potassium excretion values. Of the several studies reporting values for urinary fractional excretion of potassium in cattle,\(^16–18\) only one gives data for lactating dairy cattle in early lactation, citing a reference range of 26.9% to 120%.\(^18\) Based on the data from cases 7, 8, 10, 14, and 17, it would appear that these animals had values for urinary fractional excretion of potassium that were either within or above the normal reference range for healthy cattle in early lactation. Four of the animals in this report had received dexamethasone only—a steroid with minimal to no mineralocorticoid activity—which suggests that severe hypokalemia with muscle weakness certainly can occur in cattle independent of isoflupredone administration.

There is an uncertain association among chronic ketosis, recurrent corticosteroid administration, and clinically significant hypokalemia. Chronic ketosis or clinical mastitis could have resulted in a significant reduction in dietary potassium intake from prolonged anorexia in each of the animals in this report. Furthermore, hyperglycemia subsequent to exogenous glucose administration and enhanced gluconeogenesis after steroid administration would further reduce plasma potassium from intracellular potassium shifting subsequent to insulin release.\(^19,20\) However, it is also worth remembering that cases 12 and 13 had no history of chronic ketosis and therefore no history of exogenous dextrose, propylene glycol, or insulin administration and had experienced a relatively short period of mild anorexia (5 to 7 days). These two animals, along with several others from the same farm, had received intramammary isoflupredone acetate on multiple occasions and developed severe hypokalemia and weakness that was indistinguishable clinically from the signs in other cattle in this study. This situation suggests that severe hypokalemia
leading to muscle weakness can occur independent of chronic ketosis but that it can be predisposed to by overuse of isoflupredone acetate. The possibility that recurrent corticosteroid administration could contribute to severe potassium depletion, independent of mineralocorticoid activity, is based on case results of cattle that received repeated doses of dexamethasone alone. However, a mechanism for enhanced intracellular potassium loss due to either a direct or indirect effect of repeated corticosteroid administration is not apparent from a review of the literature.

Potassium supplementation for cattle with severe hypokalemia ideally should include both intravenous and oral administration, though from a practical standpoint oral supplementation is frequently the chosen route. It should be remembered that kaluresis will be a consequence of diuresis with all intravenous fluids; thus proprietary or homemade preparations always should contain supplemental potassium. However, in all cases of supplemental intravenous potassium administration, practitioners are cautioned not to exceed a maximum infusion rate of 0.5 mEq/kg/h, so as to avoid potential pathologic cardiac arrhythmias. It is the opinion of the authors, based on experience with the cattle of this study and others with less severe hypokalemia, that oral supplementation either alone or in combination with intravenous potassium supplementation more rapidly restores normokalemia than exclusively intravenous supplementation. Similar observations have been made by Sielman et al. and Sattler et al. Recommendations are not to exceed 0.5 lb potassium chloride orally twice daily because of the risks of inducing severe osmotic diarrhea at higher doses.

**CONCLUSION**

Hypokalemia should be considered in the differential diagnosis of weakness and recumbency in dairy cattle, particularly in animals with a history of prolonged anorexia associated with chronic refractory ketosis and corticosteroid administration during the first month of lactation. Although the cattle in this report tended to be concentrated in the first month of lactation, one animal was 110 days in milk. Based on this report, recurrent ketosis appears to be a particular risk factor for the development of severe hypokalemia, but the potential for other conditions to at least contribute to the development of this problem should not be overlooked. Although repeated use of the corticosteroid isoflupredone acetate previously has been associated with severe hypokalemia, this was a common, but not invariant feature, of the cattle in this study. Based on the observations in this retrospective study it is not possible to establish a cause and effect between any or all of the various therapeutic modalities that cattle had received before admission and the development of severe hypokalemia. Potential contributory factors to the development of clinically significant hypokalemia in the chronically ketotic cow include reduced potassium intake, intracellular shifting of potassium subsequent to metabolic alkalosis and hyperglycemia, kaluresis resulting from hyperglycemic osmotic diuresis, and increased potassium loss from the mineralocorticoid effects of exogenously administered corticosteroids. Excessive use of corticosteroids with mineralocorticoid activity for conditions other than ketosis also can predispose cattle to the development of severe hypokalemia. Therefore, oral potassium supplementation to at-risk animals, particularly those showing signs of early hypokalemia including muscle fasciculations and weakness, is recommended.

**REFERENCES**


