Mastery of Potassium Status and Their Consequences of Hypokalemia in Dairy Cattle

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Abstract

Low levels of total body potassium leads to muscle weakness and this condition may or may not be associated with low plasma potassium concentration. In most of the clinical cases, hypokalemia is observed on a serum biochemistry profile in animals without potassium depletion. So the clinical significance of hypokalemia cannot be ascertained without considering the other electrolytes as well as the acid-base status. Moreover, the physical examination and complete history dictate whether intervention is necessary in cattle with hypokalemia. The purpose of this review is to identify potential problems related to hypokalemia and their treatment.

Key words: *Bovine, Recumbency, Anorexia, Electrolytes*

Depending on soil and fertilisation, diet of dairy cows is generally rich in potassium. Thus for a long time hypokalemia was not in the focus of research. Furthermore hypokalemia is not frequently diagnosed because the measurement of serum potassium content needs special attention. Sattler et al., (1998) observed that the serum potassium concentration is maintained in the narrow range between 3.5 and 5.8 mmol/L in cattle and other mammals. He also stated that the majority of potassium is located intracellular, little cell damages may lead to large overestimation of serum potassium content. In recent years, measurement methods have been improved, and hypokalemia has increasingly been diagnosed in cows in conjunction with metabolic and mineralic disbalances, abomasal displacement, ketosis and recumbency.

Potassium Homeostasis

Potassium has multifactorial physiologic roles in normal metabolism and homeostasis. It functions at the cellular level as the principle intracellular cation and plays an important role in osmotic pressure regulation and water balance. There is no specific endocrine control of potassium homeostasis and as a result cattle are very heavily dependent upon intake to maintain adequate potassium levels. Due to the high potassium content of the forage based ruminant diet potassium is normally present in relatively high concentrations in the intestinal lumen so that absorption occurs principally by passive diffusion through the lateral intercellular spaces (Cunningham, 1997).

Although potassium absorption in ruminants can occur throughout the intestinal tract the principle sites of absorption are the small intestine and colon. Potassium excretion is

predominantly through the kidneys and to a lesser degree via obligate losses through the gastrointestinal tract. In the lactating dairy cow approximately 13% of the absorbed dietary potassium is secreted in milk, smaller amounts are lost in sweat and saliva (Ward, 1966). Increased secretion of aldosterone is a normal endocrine response to increased plasma potassium or decreased plasma sodium and will also result in increased kaluresis. Hyperaldosteronism has not been documented in cattle but exogenous, iatrogenic mineralocorticoid excess may be a significant risk factor for the development of hypokalemia Sielman et al., (1997). Use of non-potassium-sparing diuretics such as furosemide would also act to increase urinary potassium losses.

Internal potassium balance refers to the distribution of potassium between the intracellular fluid (ICF) compartment and extracellular fluid (ECF) compartment. Acid-base balance has a significant effect on the distribution of potassium between these compartments, with acidosis causing the movement of potassium from the ICF to the ECF and resulting in hyperkalemia, and alkalosis causing potassium movement in the other direction resulting in hypokalemia. Sattler and Fecteau suggested that the insulin also facilitates the movement of potassium from the ECF to the ICF. Therefore, the administration of dextrose or insulin may result in hypokalemia or an amelioration of hyperkalemia if it exists. The electrophysiologic role of potassium in the generation of action potentials in cardiac pace maker tissue, the conduction of impulses throughout the heart and contractile events in the myocardium are of particular clinical relevance because both hyperkalemia and hypokalemia can have effects on cardiac rhythm and contractility (Ganong, 1993; Mandal, 1997)

Clinical Signs

The identification of the concomitant disease is essential and should not be overlooked because it influences the recovery and prognosis.

Some animals are presented with an obscure GI problem: anorexia, little to no feces, reluctance to move, and rapid return to recumbency after stimulation to get up, mimicking colic.

In most cases, the animal rapidly becomes incapable of getting up and severe paresis develops.

This constellation of signs creates some confusion in the management of the case; specifically, whether surgery is indicated.

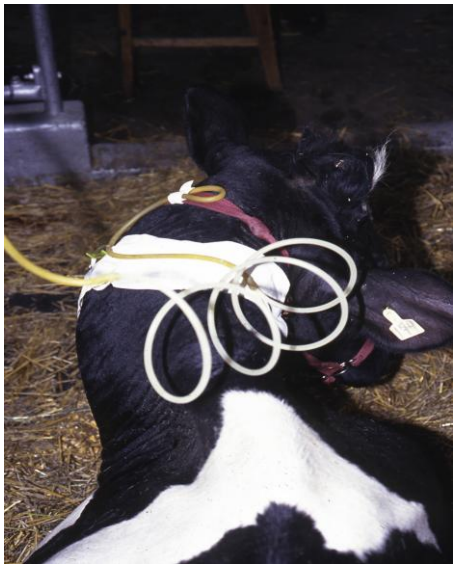

Figure Typical (or classic) S-shape position of the neck of a cow with hypokalemia. The neck muscle tone is so weak that the head cannot be held straight *(Image Courtesy: Sattler, N and Fecteau, G. (2014)*

After the initial phase of stiffness and tendency to lie down, most animals develop the following clinical signs:

- Severe apparent depression (generalized weakness with little resistance to any
- manipulations)
- Lack of tone of most muscle groups (tail tone to tongue tone are reduced)
- Tachycardia
- Abnormal neck posture (S-shaped neck)
- Recumbency
- GI stasis (forestomach and intestine) so no ruminal motility and little if any feces

Differential Diagnosis

In the early stages of the condition, hypokalemia syndrome could be confused with GI problems such as acute abdomen: intestinal ileus and intussusception or musculoskeletal/ neurologic problems such as cervical trauma, luxations, osteomyelitis or neoplasia, vertebral malformations, fractures, and torticollis. A complete history and thorough physical examination help rule out the other possibilities mentioned earlier. Other conditions to rule out when the animal is presented recumbent are hypocalcemia, botulism, and tick paralysis. It is important to consider hypokalemia syndrome as a differential diagnosis in recumbent postpartum cows because administration of calcium solution intravenously to normocalcemic cows carries a substantial risk.

Treatment of Hypokalemia

Restoration of normokalemia will follow prompt correction of the inciting primary disease. However, in the authors' experience traditional formulae for calculating the amount of supplemental potassium required to restore normokalemia in a severely hypokalemic cow tend to significantly underestimate the amount of potassium required for clinical improvement and resolution of the hypokalemia. These formulae are based upon calculating the difference between an individual's measured serum or plasma potassium and the normal reference range and multiplying this deficit first by the bodyweight (in kgs) and then by a factor that corrects for extracellular fluid space volume (0.3 in an adult). The refractoriness to conventional intravenous supplementation in severe cases of hypokalemia associated with profound muscle weakness or recumbency may be related to whole body potassium depletion but a categorical reason for this is uncertain at this time.

In cases of severe hypokalemia (< 2.3 mEq/L) potassium supplementation should ideally include both intravenous and oral administration, although for practitioners oral supplementation is frequently the chosen route. It should be remembered that kaluresis will be a consequence of diuresis with all intravenous fluids and so proprietary or homemade preparations should always contain supplemental potassium. Intravenous potassium can be administered in the form of supplemental potassium chloride added to polyionic fluids. The final concentration of potassium to be administered intravenously should vary with the severity of the hypokalemia, but is typically in the range of 30 to 100

mEq/L, at flow rates of between 2 and 4 L/hr.

However, in all cases of supplemental intravenous potassium administration practitioners are cautioned not to exceed a maximum infusion rate of 0.5 mEq/kg/hr, in order to avoid potential pathologic cardiac arrhythmias. It is the opinion of the authors 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.,(1997) and Sattler et al.,(1998). Due to the poor palatability of potassium salts and the low likelihood of adequate voluntary intake, oral supplementation is best done by orogastric tube. We suggest between 125 and 500 grams of potassium chloride in 15-20 litres of water twice daily. Recommendations would be not to exceed 500 grams (0.5 lb) of potassium chloride orally twice daily, due to the risks of inducing severe osmotic diarrhoea at higher doses.

The restoration of normokalemia in cows with severe (< 2.3 mEq/L) hypokalemia can be challenging and may require several days of aggressive potassium supplementation15. Case management may be complicated by recumbency and the subsequent potential for ischemic myopathy and peripheral nerve injury. The implications of recumbency of even a relatively short period of time accentuate the need for aggressive recognition and treatment of correctable electrolyte and mineral abnormalities and attention to management factors that avoid further injury and maximize the chances of recovery. Subsequently, oral potassium supplementation to at risk animals, particularly those showing signs of early hypokalemia including muscle fasciculations and weakness is recommended. Treatment is usually necessary for 3 to 5 days. It is probably safe to continue some supplementation until the appetite has returned to 100% (Peek et.al., 2000).

Prevention

Prevention is oriented toward supplementation of animals considered to be at risk. Dairy cattle that are chronically anorectic and treated with isoflupredone acetate and/or IV dextrose and insulin should received oral potassium supplementation. The optimal dosage regimen to administer to a normal patient considered at risk is empiric, but 100 g twice a day seems safe.

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.

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