Hypokalemia Syndrome in Cattle

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KEYWORDS

• Bovine • Hypokalemia • Recumbency • Electrolytes • Anorexia • Potassium

KEY POINTS

- Risk factors for hypokalemia syndrome include the early lactation period, anorexia, and repeated administration of isoflupredone.
- Treatment includes basic supportive care for recumbent animals and aggressive potassium replacement therapy.
- Prognosis is guarded for recumbent cattle and worsens if hypokalemic myopathy or complications of recumbency occur.

INTRODUCTION

Total body potassium depletion leads to muscle weakness and may or may not be associated with low plasma potassium concentration (hypokalemia). More often, hypokalemia is observed on a serum biochemistry profile in animals without potassium depletion (potassium redistribution). 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.

NORMAL POTASSIUM BALANCE

Potassium is mostly intracellular. Serum potassium concentration is a poor indicator of the potassium status of the animal. Determination of intracellular potassium concentration in erythrocytes or muscle cells is a more accurate way to assess potassium depletion, but with current technology it is not clinically feasible in most cases.[1,2]

The concentration of potassium in plasma depends on external potassium balance and internal potassium balance. External potassium balance refers to potassium

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intake and absorption from the gastrointestinal (GI) tract, and potassium excretion by the kidneys. The primary source of potassium is the forage portion of the normal ruminant diet. An animal with a normal appetite usually has a normal serum potassium concentration. Almost all ingested potassium (more than 300 g per day for a 600-kg cow) is absorbed and reaches the intracellular fluid compartment. Because they have a forage-based diet, lactating dairy cows may eat more than 10 times their daily potassium requirement. For this reason, cattle have renal excretory mechanisms that are well developed to eliminate the excess potassium load from the body. When intake is interrupted, the excretory mechanisms may not respond rapidly enough to avoid potassium depletion. Cows with partial and/or total anorexia frequently have moderate hypokalemia. This hypokalemia is not coupled with clinical signs of weakness. Other abnormalities such as diarrhea, third space loss, and alkalosis can exacerbate potassium loss from the intracellular and extracellular fluid compartments.

Some corticosteroids with mineralocorticoid effects are known to lead to hypokalemia (eg, isoflupredone acetate) by increasing potassium excretion in the kidneys. Diuretic drugs (eg, furosemide) may also contribute to renal potassium loss. Following relief of a urinary obstruction, a diuretic phase occurs and may lead to significant potassium loss.

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. 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 CLINICAL SYNDROME

Introduction

Hypokalemia syndrome has been reported.[3–6] Lactating dairy cows as well as younger animals may develop the disease. At present, except for animals treated with repeated isoflupredone acetate administrations, the exact determinants causing hypokalemia syndrome remain uncertain.

Risk Factors

Lactating dairy cows less than 60 days in milk seem to be at greatest risk. Systemic illness causing anorexia of several days' duration is a risk factor.[4] Repeated systemic or intramammary administration of isoflupredone acetate can cause the syndrome. The mineralocorticoid activity of isoflupredone acetate disturbs both the internal and external potassium balance.[7] Repeated doses can reduce serum potassium concentration by 70%.[8] Food-restricted dairy cattle receiving repeated doses of isoflupredone acetate developed the syndrome in an experimental model.[9] Following the label directions concerning dose and duration seems to be important. Although it is a less potent mineralocorticoid, use of dexamethasone was also reported as a potential cause in some cases.[4,6]

Multiple treatments of dextrose and insulin are also reported to be associated with the syndrome.[3–6] In young animals, the repeated administration of isoflupredone acetate to treat pneumonia and intravenous (IV) fluid administration have been reported to initiate the syndrome.[4]