INTRAVENOUS DEXTROSE ADMINISTRATION REDUCES PLASMA PHOSPHORUS CONCENTRATION IN DAIRY COWS

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TAKE HOME MESSAGES

- Rapid intravenous administration of 500 mL of 50% dextrose solution to lactating dairy cows causes a marked (35%) drop in plasma phosphorus concentration that lasts approximately 90 minutes.

- Continuous infusion of 50% dextrose solution by slow intravenous drip for several days causes a similar but more prolonged (36 hour) reduction in plasma phosphorus concentration.

- Intravenous dextrose should be used cautiously in post-calving or off-feed cows, as these animals are already at risk for hypophosphatemia. Treated cows should be monitored for adverse effects associated with hypophosphatemia (eg, weakness or recumbency).

INTRODUCTION

Phosphorus is a critical nutrient for many cellular functions and an important structural component of bones and teeth. Normal plasma phosphorus concentration is maintained when phosphorus loss and utilization are balanced by phosphorus intake from feed and mobilization from bone. At calving time, lactating dairy cows experience a sudden loss of phosphorus into milk and are inefficient at mobilizing phosphorus from bone. Feed intake is often reduced before calving, which, in addition to reducing phosphorus intake, causes negative energy balance and predisposes the cow to ketosis and fatty liver syndrome. At the same time, phosphorus is being shunted from the cow to the fetus to support fetal growth. Reduced feed intake often continues after calving, particularly if the cow develops a disorder(s) such as LDA, metritis, or mastitis, or experiences ketosis or fatty liver syndrome. All of these factors combine to put periparturient cows at risk for low plasma phosphorus concentration (hypophosphatemia).

The clinical effects of hypophosphatemia in cattle are uncertain. However, hypophosphatemia frequently accompanies hypocalcemia in cows with milk fever. Hypophosphatemia has been implicated in non-responsive milk fever cases and is thought to contribute to “downer cow syndrome”.

A mainstay of treatment for ketosis and fatty liver syndrome is intravenous administration of 50% dextrose solution. Intravenous dextrose is also frequently given to off-feed cows or as a supportive treatment for post-calving disorders. Some producers routinely administer dextrose to cows after calving to “boost energy”. Five hundred milliliters of 50% dextrose is the standard dose (repeated as judged necessary) and is administered as a bolus by rapid infusion into the
jugular vein. However, in cows with fatty liver syndrome or persistent ketosis, dextrose may be administered as a slow intravenous drip for hours to days.

Intravenous dextrose administration is known to cause hypophosphatemia in simple-stomached species such as rodents, dogs, and people. Fatal hypophosphatemia has been reported in starved people who were administered dextrose without supplementing phosphorus. The effect of intravenous dextrose administration on plasma phosphorus concentration in dairy cattle and other ruminants has not been reported. We hypothesized that lactating cows would experience a similar drop in plasma phosphorus concentration when administered dextrose. However, lactating cows have a large phosphorus demand for milk and excrete phosphorus mainly in saliva, rather than in urine as is the case in simple-stomached species. These factors could impact the magnitude and duration of hypophosphatemia.

**MATERIALS AND METHODS**

Two studies were conducted. Study 1 involved rapid intravenous administration of 500 mL of 50% dextrose solution to 6 healthy cows in early lactation. Plasma concentrations of glucose, insulin, and phosphorus were monitored for 12 hours after treatment. Urinary and salivary phosphorus concentrations and hourly urine volume were also determined. Study 2 involved slow intravenous administration of 50% dextrose solution (0.3 g/kg/hour) to 4 healthy lactating cows for 5 days. In addition to monitoring plasma, urinary, and salivary parameters, phosphorus intake was measured and phosphorus losses in milk and feces quantified. In both studies, each cow also received a sham treatment in which the cow was instrumented and sampled but not given dextrose; this allowed cows to serve as their own controls.

**RESULTS AND DISCUSSION**

Plasma glucose, insulin, and phosphorus concentrations were stable during sham treatment in both studies. Plasma phosphorus concentration declined rapidly after administering a 500-mL bolus of 50% dextrose solution, dropping by 35% in 1 hour and remaining low for 90 minutes. Salivary and urinary phosphorus concentrations and hourly urine volume remained stable, but glucose was detected in the urine for up to 6 hours. The amount of glucose excreted in urine in the 12 hours following dextrose administration was less than 5% of the administered dose, indicating that the dextrose was utilized by the cow.

When 50% dextrose was infused continuously for 5 days, plasma phosphorus concentration again dropped rapidly, reaching a nadir 24 hours after starting the infusion and remaining low for 36 hours. Urinary phosphorus excretion did not change during dextrose infusion but feed intake dropped, resulting in a 25 to 50% decrease in phosphorus intake. This was followed by a reduction in fecal output and fecal phosphorus loss. Milk yield increased at 12 hours, followed by a progressive decline, but milk phosphorus loss was stable. Plasma phosphorus concentration began rebounding within minutes after discontinuing the dextrose infusion, peaking hours later. Feed intake and milk yield subsequently returned to normal.

In both studies, the immediate drop in plasma phosphorus concentration accompanying intravenous dextrose administration appeared to be driven by an increase in plasma insulin concentration in response to hyperglycemia. Hyperinsulinemia probably caused a rapid shift of phosphorus from plasma into cells, as has been demonstrated in people. With long-term dextrose
administration, the reduction in feed intake probably contributed to hypophosphatemia but did not occur quickly enough to account for the immediate drop.

CONCLUSIONS AND RECOMMENDATIONS

Our studies confirm that dairy cows experience a marked decline in plasma phosphorus concentration when administered intravenous dextrose solution. The response occurs regardless of whether administration is by bolus or continuous infusion. Although clinical abnormalities did not accompany hypophosphatemia in our studies, we used healthy cows that had normal plasma phosphorus concentrations before treatment. On farms, dextrose is usually given to post-calving cows that are off-feed and may already have low plasma phosphorus concentrations. Dextrose should be administered cautiously in these cows and avoided when not indicated. Cows that are stressed at calving or have conditions such as mastitis are often hyperglycemic rather than hypoglycemic and are unlikely to benefit from additional glucose administration. A cow-side glucometer is a practical tool for measuring blood glucose concentration and, along with a urine ketone test, can identify cows that need dextrose. When dextrose is given by continuous intravenous infusion over several days, veterinarians should monitor plasma phosphorus concentration and supplement phosphate if signs of hypophosphatemia develop.