Ruptured urinary bladder after dystocia in a cow

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A 415-kg Hereford cow was admitted to the veterinary teaching hospital for evaluation of dystocia of 12 hours duration. Uterine contractions were absent, and a cold swollen limb of a calf protruded from the vulva.

Physical examination revealed a weak, lethargic cow with a rectal temperature of 40.8 C and tachycardia (80 beats/min). Vaginal examination revealed the calf to be in a cranial dorsal sacral position with 1 forelimb flexed at the carpus. The flexion was corrected, and an attempt was made to extract the calf, but it was unsuccessful because of the size of the fetus.

Cesarean section was performed via left paralumbar celiotomy to remove a 49-kg dead calf from the right uterine horn. After surgery, the cow was treated with penicillin G (22,000 IU/kg of body weight, IM, q 12 h), flunixin meglumine (1 mg/kg, IM, q 12 h), and oxytocin (40 IU, IM).

On the day after surgery, the cow remained lethargic and anorectic. Few rumen contractions were detected, and urination was not observed. Differential diagnoses included peritonitis, ruminal or intestinal atony, metritis, and metabolic derangement.

Treatment included 8 L of a balanced electrolyte solution administered via orogastric tube. In addition, a 26% calcium borogluconate solution[a] (500 ml, SC) was administered to alleviate presumed hypocalcemia.

On day 2, rumen motility had decreased and a dull ping was ausculted over a distended left paralumbar fossa. An ammonia odor was detected in the oral cavity, suggestive of azotemia. Analysis of ruminal fluid revealed a pH of 7.0 and low protozoal activity. A CBC and plasma fibrinogen concentration (800 mg/dl) were suggestive of an inflammatory process. Paracentesis yielded a small volume of clear fluid that contained 1,000 leukocytes/μl (80% segmented neutrophils and 20% macrophages) and a protein concentration of 1.4 g/dl. Attempts to collect a voided urine sample were unsuccessful because the cow had not been observed to urinate, although there was evidence of urine in the stall.

Bilateral ventral abdominal distention was apparent by day 3. A CBC supported a continuing focus of inflammation, with a neutrophilia and a left shift and a further increase in serum fibrinogen; (1,300 mg/dl). Serum abnormalities included hypochloremia, hypocalcemia, hyperphosphatemia, and increased creatinine. An exploratory laparotomy through a left paralumbar incision was performed. The abdmoninal cavity contained 10 L of clear yellow fluid, and several fibrinous adhesions were found between the urinary bladder and the body wall. A 12-F Foley catheter was passed into the bladder via the urethra. Injection of cold sterile 0.9% NaCl (saline) solution into the bladder confirmed a 4-cm, full-thickness tear in the ventral wall of the bladder, immediately cranial to the urethral orifice.

Intravenous administration of physiologic saline solution was initiated, and approximately 10 L of abdominal fluid was drained. The bladder tear was repaired with 4 vertical mattress sutures. The Foley catheter was left in place to prevent bladder distention and minimize leakage through the tear. Treatment after surgery included continuation of the flunixin meglumine, procaine penicillin, and calcium borogluconate solution as well as administration of 8 L of a balanced electrolyte solution given via orogastric tube. The next day the cow's attitude, appetite, and rumen motility improved. Urine was observed dripping from the end of the Foley catheter.

Peritoneal fluid (26.4 mg/dl) and serum (14 mg/dl) creatinine concentrations at surgery were consistent with uroperitoneum. Hyperphosphatemia, hypochloremia, and hypocalcemia also were indicative of uroperitoneum. Three days after the bladder repair, 50 ml of new methylene-blue dye was infused into the bladder through the urinary catheter. Paracentesis that was performed 1 hour later recovered no dye; thus, the Foley catheter was removed. Complete resolution of the leu-

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[a]Norcalciphos, Norden Laboratories, Lincoln, Neb.

kocytosis, azotemia, and serum electrolyte abnormalities was noticed 5 days after surgery. The owner was instructed to allow a 60-day drug withdrawal time for slaughter. The cow was discharged 6 days after the bladder repair and lost to further follow-up examination.

Urinary bladder rupture is a rare complication of bovine dystocia.[1,2] It is more commonly a consequence of obstructive urolithiasis in feedlot steers.[3-5] Fetal position during prolonged dystocia could obstruct the urethra and cause bladder distention. Subsequent surgical manipulation of the fetus against a compromised bladder may have resulted in rupture.

Metabolic abnormalities associated with uroperitoneum in cattle include azotemia, variable alterations in sodium and potassium concentrations, hypochloremia, hypocalcemia, and hyperphosphatemia.[2-8] The most consistent electrolyte abnormality detected in cattle with postrenal obstruction is hypochloremia.[2-8]

Urine normally has low sodium and chloride concentrations, but as it accumulates in the peritoneal cavity, there is a net diffusion of these ions from the serum into the peritoneal fluid. In addition, sequestration of chloride in the abomasum because of gastrointestinal stasis may have contributed to the hypochloremia seen in the cow of this report. An increased serum total CO_2 or bicarbonate concentration would have supported this as a possible mechanism. Hyponatremia and normonatremia have been reported in association with bladder rupture.[2,4-8] In 1 study involving experimentally induced ruptures of the bladder in 5 cattle, all developed hyponatremia.[6] Serum sodium concentrations remained within the normal range in the cow of this report. Sodium/potassium exchange by the salivary gland and the intestinal mucosa are 2 mechanisms that conserve sodium.[7] Administration of sodium in fluids given orally may have limited the decrease in plasma sodium concentration.

Potassium is primarily excreted by the kidney in most species, and hyperkalemia is commonly associated with uroperitoneum in dogs, cats, and foals.[3,5] Variable serum potassium concentrations have been detected in cattle with postrenal obstruction with or without cystorrhexis.[2-6,9] The intracellular movement of potassium in response to a hypochloremic metabolic alkalosis may be an important factor, as well as a decreased potassium intake with anorexia. Cattle have additional excretory routes through the salivary glands and intestine.[2,7] An aldosterone-mediated increase in salivary potassium and decrease in salivary sodium excretion has been detected in nephrectomized cattle and in 1 other reported case of urinary bladder rupture in a cow.[2,6]

Hypocalcemia and hyperphosphatemia have been reported with cystorrhexis.[2-7] Salivary excretion of phosphorus is believed to be the primary excretory route, with renal excretion of phosphorus of minor importance; however, hyperphosphatemia frequently is detected with bladder rupture.[4-6] Hyperphosphatemia was found to be a prognostic indicator of survival rate with postrenal disorders in a study by Donecker and Bellamy.[4] Decreased salivary excretion attributable to decreased salivary production may be a mechanism for hyperphosphatemia with uroperitoneum. Additionally, hypocalcemia resulting from anorexia is associated with an increase in serum phosphorus concentration in noncattle. It is difficult to assess which factors are most important in determining serum phosphorus concentration.

The ratio of concentrations of peritoneal fluid creatinine to serum creatinine was the most useful diagnostic indicator for uroperitoneum in 1 study.[6] Creatinine equilibrates more slowly than urea across the peritoneal membrane and is therefore likely to be in higher concentration in the peritoneal cavity.

The ventral location of the bladder tear in the cow of this report precluded healing by second intention. A complete seal was not achieved with the surgical repair, and the Foley catheter was left in place to minimize bladder distention and urine leakage. Drainage of accumulated urine from the peritoneal cavity facilitated abdominal exploration and localization of the bladder tear. Fluids were initiated IV, and drainage was performed over a 15-minute period. Pulse and attitude were observed closely to monitor for possible development of hypovolemia during drainage. Complications of the bladder tear included adhesions of the bladder to the uterus and body wall. Adhesions could result in problems in future pregnancies and parturitions; thus, prognosis for return to reproductive soundness was guarded.

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