Urinary Tract Disorders in Cattle

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Acute Renal Failure in Cattle

Acute Tubular Necrosis

Acute tubular necrosis is the most common pathologic cause of acute renal failure (ARF) in cattle. This results from renal ischemia or nephrotoxins. Renal ischemia occurs most frequently in cattle with acute severe mastitis, septic metritis, abomasal torsion, salmonella, or other severe septic processes. The most common toxins causing acute tubular necrosis in cattle are:

Quercus spp. (oak)
Halogeton glomerultos
Sarcobatus vermiculatus (greasewood)
Oxalis spp. (soursob)
Anaranthus spp. (pigwood)
Kochia spp.
Rumex (sorrel, dock)
Chenopodium album (lambs quarter)
Rheum Rhaporticum (rhubarb)
Salsola pestfiter (Russian thistle)
Lantana camara
Isotropis

The drugs most frequently resulting in acute tubular necrosis in cattle are aminoglycosides and oxytetracycline. There is a long list of chemicals which may produce acute tubular necrosis but these are rarely seen to cause renal failure in cattle. Hemoglobin or myoglobin may under certain circumstances cause renal failure in cattle as may certain mycotoxins, e.g. okra toxins, citrinin.

Clinical Signs: Clinical signs of ARF (acute tubular necrosis) in cattle are relatively nonspecific and in many cases, particularly those resulting in renal ischemia, a preexisting disorder may mask the renal problem. The most common clinical signs are marked depression, anorexia, and recumbency. The kidneys are often palpably enlarged in cases of acute nephrosis. In acute oak toxicity, which is probably the most common cause of ARF in cattle throughout the United States, cows are weak, markedly depressed, they may have hematochezia or very dark colored manure, and may bleed from the nostrils.

Clinical Pathology: Cattle with ARF caused by acute tubular necrosis are azotemic and are almost always hypochloremic, hypocalcemic, hyponatremic, hyperphosphatemic and hypermagnesemic. The serum potassium may vary but is usually normal or even hypokalemic. The urine usually has a lower than expected specific gravity for a dehydrated animal (less than 1.022), and microscopic hematuria is usually present.

Epidemiology: Acorn or oak bud poisoning is the most frequent in the autumn and spring. It often occurs in the autumn in association with sprouted acorns and the weaning of calves in a cow/calf operation onto a pasture of sprouting acorns. Oak bud poisoning occurs in the spring and may be associated with late winter snows covering other available grazing material.

Aminoglycoside nephrotoxicity is most frequent in dehydrated patients. Oxytetracycline nephrotoxicity is usually associated with overdosing of the drug, although it may occur at normal doses in septic cattle.

Treatment: Treatment is aimed at restoring intravascular fluid volume by the administration of intravenous fluids containing sodium chloride and calcium and potassium when needed. The animal should be removed from the source of the toxin. Most cattle with acute tubular nephrosis are polyuric, and if the disease process has not be present for several days, the prognosis is usually good if the predisposing cause can be corrected. The necessity to correct the predisposing cause is most important in those cases with tubular ischemia associated with sepsis. If the animal starts producing sufficient quantities of urine shortly after intravenous fluids are begun, then the possibility of oliguric or anuric renal failure should be considered and appropriate steps taken if the value of the animal warrants such therapy. In those cases, intravenous mannitol (0.25 g/kg) or dopamine (3–5.0 ug/kg/min) administered intravenously along with furosemide (1.0 mg/kg I.V.) every two hours should be given in an attempt to convert the oliguric state to a polyuric state.

Amyloidosis

Amyloidosis is a disease complex resulting in the deposition of twisted beta-pleated sheet fibrils formed from various proteins (amyloid) in the kidney, liver, adrenal gland and gastrointestinal tract. Cattle usually present with a nephrotic syndrome because of extensive renal/glomerular amyloid deposits.

Clinical Findings: Renal amyloidosis occurs sporadically in cattle greater than four years of age and affected cattle almost always have chronic projectile diarrhea, weight loss, and ventral edema. Gross enlargement of the kidneys can usually be appreciated on rectal examination although the kidneys retain their normal shape and lobation. This

is important in differentiating renal amyloidosis from chronic pyelonenephritis or hydronephrosis, or lymphosarcoma infiltration of the kidney. Clinical signs of amyloidosis may appear very similar to those seen in cattle with Johne's disease.

Clinical Pathology: The most marked laboratory finding is heavy proteinuria and systemic hypoproteinemia. Serum creatinine values are usually elevated in cattle with clinical signs of renal amyloidosis but may be in only high normal range if cases are detected early.

Treatment: The prognosis in infected cattle is poor, and salvage is usually recommended. I have tried treating two cows with intravenous DMSO, but neither attempts were successful.

Urinary Tract Infections

Infection of the urinary tract occurs sporadically in cattle and may result in lower urinary tract infection, e.g. cystitis, or upper urinary tract infection, e.g. pyelonephritis. *Corynebacterium renale* and *Escherichia coli* are the organisms which are most frequently responsible for either upper or lower urinary tract infections.

Clinical Signs: Cattle with subacute pyelonephritis have fever, dysuria, gross hematuria, and pyuria, and may show signs of abdominal pain. When the condition becomes chronic, severe weight loss and chronically depressed milk production occur, and the gross urine changes may become less remarkable. In pyelonephritis, symmetrical involvement of both kidneys is unusual, but if the left kidney is affected it may appear enlarged and painful upon rectal palpation. The ureter may also be palpably enlarged on the affected side. Cattle with cystitis may have minimal clinical signs other than crystalluria, and occasionally hematuria and dysuria.

Clinical Pathology: Urinalysis in either acute or chronic cases of urinary tract infection reveals pyuria and bacteriuria ($>10^4$ organisms/ml) and urine culture results in growth of the responsible organisms. *C. renale* is the most common organism to be cultured in subacute pyelonephritis while *E. coli* is the most common one to be cultured in chronic pyelonephritis. Azotemia is usually evident in subacute cases of subacute pyelonephritis, but much of this may be prerenal azotemia. With pyelonephritis, either acute or chronic, an inflammatory leukogram and elevated fibrinogen would be expected.

Epidemiology: Cystitis and pyelonephritis usually occur in adult cows, and this is considered to be associated with the short, wide urethra of the female. *C. renale* may be spread from one cow to another cow within the herd. Cystitis may occur in younger calves but is much less common than in adult cows. Clinical signs of dysuria, pollikiuria in young calves may also be associated with umbilical abscesses and a pelvic bladder.

Treatment: The prognosis for life and return to production for most cattle with pyelonephritis is good if they are treated adequately and early. The prognosis for cows with cystitis is good if bladder atony is not present. The antibiotic of choice for *C. renale* is penicillin. In cases of subacute pyelonephritis, which is almost always caused by *C. renale*, intravenous penicillin over the first couple of days would be recommended in order to allow more rapid penetration of the kidney with the penicillin. After the cow has shown signs of improvement, therapy may be switched to intramuscular procaine penicillin and should be continued for 2–4 weeks. Intravenous fluid therapy is also necessary in some cases of subacute pyelonephritis in order to combat dehydration and provide diuresis, which is important in flushing obstructed tubules. In chronic pyelonephritis, the selection of antimicrobial agents is based on culture and sensitivity of the causative organism, but the limitations of antibiotics effective *in vitro* must be taken into account. In cases of chronic pyelonephritis that do not respond to the appropriate antibiotic, an ultrasound examination of the kidneys would be recommended in order to rule out nephrolithiasis. Valuable cattle with unilateral pyelonephritis which has resulted in gross abscessation of the kidney are best treated by a nephrectomy. But an accurate assessment of the functioning ability of the remaining kidney is essential before surgery. This can usually be done simply by looking at the serum creatinine. If one kidney is believed to be totally nonfunctional in association with the abscessation and the serum creatinine is less than 2.0 mg/dl, then one can be relatively sure that the opposite kidney has maintained most if its functioning nephrons. Calves with dysuria associated with urachal abscesses are best treated by surgical removal of the infected urachus and the diseased bladder.

Urolithiasis

Urinary calculi occur sporadically in cattle in most parts of the world, but are of particular importance in feedlots and in grazing cattle in western North America. Calculi become clinically important when obstruction of the urinary tract occurs. Obstructive urolithiasis is almost exclusively a disease in males and primarily affects steers.

Clinical Signs: There are three common clinical entities which occur in urolithiasis syndrome. These are urethral rupture, urethral obstruction and rupture of the bladder. In a rare case, rupture of the kidney may also occur. The clinical signs of urethral obstruction are associated with the urethral pain. The animal is usually restless, may often switch its tail, and exhibit stranguria. The prepuce often hangs down an inordinate amount of time. Dribbling of blood-stained urine often occurs and a pulsation of the urethra at the ischial arch can be felt. The preputial hairs should be palpated because sand-like calculi may be

found on them. The most common site for calculi to lodge in the urethra is at the proximal sigmoid flexure. Rectal examination will reveal a distended bladder, if the bladder has not ruptured. The clinical signs of urethral rupture are similar to those of urethral obstruction, except there is an often marked subcutaneous swelling along the sheath and ventral abdominal wall. The clinical signs of bladder rupture may include relief of those signs associated with urethral obstruction. The animal becomes anorexic, depressed and uremic-smelling within 2–3 days after the bladder has ruptured. Bilaterally symmetrical ventral abdominal distension may be noticed and a fluid wave can be detected by abdominal ballotment. The bladder is not palpable per rectum and the abdomen may have an empty feel to it. A ruptured bladder is not always found in association with urethral obstruction; it may also be seen infrequently in cows post partum.

Diagnosis: The diagnosis of urethral obstruction is based on history and signalment along with clinical examination. The finding of stranguria and/or tenesmus along with the passage of no urine or small amounts of blood-tinged urine is highly suggestive of urethral calculi in male cattle. Palpation of the urethra and bladder will usually provide definitive information. The diagnosis of ruptured urethra is also based upon historical information and signalment, along with the characteristic "water belly" swelling associated with the ruptured urethra. Aspiration from the swelling usually reveals a clear fluid that may or may not smell of urine. A comparison of the creatinine in the aspirated fluid with the serum creatinine will usually reveal some elevation in the creatinine from the aspirated fluid over in comparison with the serum creatinine. Diagnosis of a ruptured bladder is based upon clinical signs, rectal examination findings and comparison of peritoneal fluid creatinine to serum creatinine. The ratio of the two should be greater than 2:1 with uroperitoneum.

Clinical Pathology: Serum creatinine and BUN are elevated in cattle with ruptured bladder or a ruptured urethra. Serum sodium and chloride values are decreased with a ruptured bladder and may be decreased with a ruptured urethra. Serum phosphorus is usually elevated with either ruptured bladder, ruptured urethra, or a urethral obstruction.

Epidemiology: In cattle the predominant type of calculi is struvite calculi, composed of magnesium ammonium phosphate. High concentrate feeds high in phosphorus predispose to the formation of struvite calculi. Silica calculi are found in pastured cattle grazing in fields with high levels of silica. Both forms of calculi, struvite or silica, are predisposed to by decreased water intake and a decreased urine volume production. This allows continued supersaturation of the urine with crystalloids and the development of the calculi. The pH of the urine, vitamin A levels in the diet, and the amount of sodium chloride or magnesium in the diet have also been incriminated in calculi formation. It would appear that calculi form to an equal extent in all cattle, but castrated males are more susceptible to obstructive urolithiasis because of the decreased urethral diameter in young castrated bulls.

Treatment: A favorable response to treatment of urethral obstruction with tranquilization has been reported. This may allow sufficient relaxation of the urethra such that passage of the calculi may occur. Urethrostomy is performed in cases where the urethra has ruptured as a salvage procedure. In those cases an incision should also be made over the area of the central cellulitis to allow drainage. The prognosis for surgical repair of a ruptured bladder in steers associated with ureteral calculi is poor, but in those rare cases that occur in post partum cattle it is fair to good.

Prevention and Control: Prevention of urolithiasis is dependant upon dietary regulation of the important elements (magnesium, phosphorus, calcium) and the addition of salt. The addition of sodium chloride will not only increase water consumption but may also lead to the formation of magnesium chloride in the urine, which is more soluble than magnesium phosphate. Chloride ions may also bind to mucoproteins thereby decreasing silicate and phosphate binding. Free choice, fresh water at all times is important in the control and prevention of urethral calculi in feedlot animals. Ammonium chloride can be added to the diet in hopes of acidifying the normal alkaline urine, but this is highly variable from animal to animal and is met with only variable success.

Chronic Renal Failure

Chronic renal failure (CRF) in cattle most often occurs from nonreversible acute tubular nephrosis which progresses to interstitial nephritis and fibrosis or from chronic pyelonephritis or obstructive disease. Chronic glomerulonephritis has been important in cattle as a cause of CRF but is certainly not as common as is amyloidosis.

Clinical Signs: Cattle with CRF are usually cachectic and may have abnormally long hoof wall growth. If glomerulonephritis is present they may also have ventral body all edema. The diagnosis of CRF in cattle is best confirmed by history, complete physical examination, and an examination of clinical chemistries, and urinalysis. A renal biopsy may be needed to confirm the chronic disease process and the primary site of involvement (tubulointerstitial versus glomerular). There is very little treatment that can be offered for such cases.

Discolored Urine

Veterinarians are often called to examine cattle with discolored urine. The basic causes of discolored urine may either be humaturia, hemoglobinuria, myoglobinuria,

bilirubinuria, or porphyrinuria. Porphyrinuria is seen predominantly in Holstein calves and a few other breeds with congenital porphyrin defects. In those cases the urine is visibly discolored only after being exposed to sun light, so this does not create a real problem for differentiating discolored urine in cattle. Bilirubinuria is also quite rare in cattle and occurs only with obstructive biliary disease. Myoglobulinuria is not as common in cattle as in the horse, and is rarely seen as a cause of discolored urine except in the case of plant poisoning, e.g. coffee weed, or in a rare case of septic myositis. Therefore, hemoglobinuria and hematuria are the prime considerations for discolored urine in cattle.

Hematuria

Hematuria is defined as blood in the urine. Hematuria may appear as gross blood clots being passed at the beginning of urination, only at the end of urination, or may occur throughout urination. If the blood clots are more severe at the beginning of urination then urethral disease, e.g. calculus, must be considered highest on the differential list. If the hematuria is most noted at the end of urination then bladder hemorrhage, such as ulcerative cystitis due to *C. renale*, would be considered most likely. With *C. renale* pyelonephritis, blood may be seen intermittently throughout urination. Gross blood clots in the urine are not always noted with hematuria, and often the urine may have a uniform red discoloration very similar to hemoglobinuria. A determination of hematuria can usually be made by spinning down such urine samples and looking for microscopic evidence of red blood cells or sediment in the spun sample indicative of red cell sedimentation. The most common cause of the hematuria in cattle in the United States include *C. renale*, pyelonephritis or cystitis, urethral calculi, and renal infarcts. Acute tubular necrosis often leads to microscopic hematuria but no discoloration of the urine is seen.

Hemoglobinuria

There are many causes of hemoglobinuria in cattle. Hemoglobinuria almost always produces a uniform red discoloration of urine throughout the process of urination. There should be a minimal number of red blood cells within the urine and the plasma of affected animals should be pink, unlike those animals affected only with hematuria. The most common causes of hemoglobinuria in cattle include leptospirosis, water intoxication in calves, Heinz body hemolytic anemias (such as onion, rye grass, and kale), hypophosphatemic or post partum hemoglobinuria, isoimmune hemolytic anemia in calves in association with previous anaplasmosis vaccination, bacilliary hemoglobinuria, Black's disease, or drug-induced hemolytic anemia. It should be mentioned that anaplasmosis rarely, if ever, causes hemoglobinuria in cattle.

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