PEER REVIEWED

Case Report–Scrotal Hydrocele Secondary to Ascites Caused by Liver Fluke Infection in an Adult Beef Bull

Kevin E. Washburn, *DVM, DABVP;* **Robert N. Streeter,** *DVM, MS, DACVIM*
Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74078

Abstract

A five-year-old, 2000 lb (909 kg) beef bull was presented to the Oklahoma State University Food Animal Clinic because of weight loss during the previous eight weeks, and lameness and increasing recumbency of two weeks' duration. Physical examination revealed a subsolar abscess, oral lesions, mild diarrhea, modest generalized lymphadenopathy and an enlarged left scrotum. Further diagnostic testing revealed liver fluke infestation, scrotal hydrocele, ascites and a positive gp-51 bovine leukemia virus serology. The ascites, weight loss, diarrhea, lymphadenopathy and hydrocele were attributed to migration of immature liver flukes. The lameness and recumbency were caused by a subsolar abscess and, therefore, unrelated to the primary problem. Bovine papular stomatitis was suspected to have caused the oral lesions, due to their physical appearance and rapid resolution. The bull was treated twice, at eight-week intervals, with ivermectin which contained clorsulon. Body condition markedly improved over the next 24 days, and the scrotal hydrocele resolved.

Résumé

Un taureau de boucherie de cinq ans pesant 2000 lbs (909 kg) a été admis à la clinique d'animaux de consommation de l'Oklahoma State University en raison d'une perte de poids durant les dernières huit semaines. De plus, l'animal avait des problèmes de boiterie et passait plus de temps couché depuis les deux dernières semaines. L'examen physique a révélé un abcès plantaire, des lésions buccales, une faible diarrhée, une lymphadénopathie généralisée modérée et une augmentation de la taille du scrotum à gauche. Des tests diagnostics plus poussés ont indiqué une infestation par des douves du foie, un hydrocèle scrotal, de l'ascite et une sérologie positive dévoilant la présence du virus de leucémie bovine gp-51. L'ascite, la perte de poids, la lymphadénopathie et l'hydrocèle ont été attribués à la migration des douves du foie immatures. La boiterie et le problème de décubitus étaient causés par l'abcès plantaire et n'étaient donc pas reliés au principal problème. La stomatite papuleuse bovine était probablement à l'origine des lésions buccales en raison de leur apparence et de leur rapide résorption. Le taureau a été traité à deux reprises à huit semaines d'intervalle avec de l'ivermectin contenant du clorsulon. L'état corporel s'est grandement amélioré lors des 24 jours suivants et l'hydrocèle scrotal a disparu.

Introduction

While liver fluke infestation in cattle is commonly subclinical, *Fasciola hepatica* infection can cause significant economic losses. Manifestations of fascioliasis range from mild to severe. Cattle that have been repeatedly exposed to the parasite develop a certain degree of resistance to infection, so naïve or newly introduced animals are more susceptible to clinical disease.[13] However, concurrent infection with *Clostridium hemolyticum* or *Clostridium novyi* Type B results in life-threatening disease, regardless of susceptibility to fluke infection.

This report describes weight loss, diarrhea, ascites and internal lymphadenopathy as clinical signs of liver fluke infestation in an adult bull. Clinical and laboratory findings warranted ruling out lymphosarcoma, Johne's disease, traumatic reticuloperitonitis and bovine viral diarrhea virus (BVDV) infection.

History

A five-year-old, 2000 lb (909 kg) registered Limousin bull was presented to the Oklahoma State University Food Animal Clinic in mid-summer with lameness of two weeks' duration. The bull was purchased 11 weeks prior to presentation, and kept in an 80-acre bermuda grass pasture with 25 cows. The pasture bordered a river and contained low-lying areas that are frequently flooded. Lameness in the right front

foot was noted and treated by the owner with 100 mL oxytetracycline (200 mg/mL) IM one week prior to presentation. The lameness continued, and the bull became increasingly recumbent as a result. The bull reportedly had a good appetite, but had lost weight during the previous eight weeks. Herd mates were unaffected. The bull had neither been vaccinated nor treated for parasites since purchase.

Clinical and Laboratory Findings

The bull was lame on the right front limb (lameness score of 3 on a 5-point scale). Body condition score was 4 (9-point scale). Mentation was normal. Mucous membranes were moist and pink, and capillary refill time was less than 2 seconds. The sclera were normal. The hard palate contained three raised, circular lesions, approximately 1 cm in diameter, with umbilicated centers. No lesions were noted on the tongue or lower gingiva.

Pre-scapular and pre-femoral lymph nodes were slightly enlarged. Vital signs, as well as rumen contractions, succussion and ballottement, were within normal reference ranges. The left scrotal sac was enlarged and fluctuant, but not painful or palpably warm. Palpable abnormalities were not detected in either epididymis. Scrotal circumference was 44 cm.

The bull had dark-green diarrhea with no obvious blood or fibrin. Rectal examination revealed modestly enlarged internal iliac lymph nodes, but no abnormalities in the other palpable structures. The bull urinated normally during the examination. All other aspects of the physical examination were normal.

On day 1, the right front foot was examined to determine the cause of lameness. A small subsolar abscess was discovered in the toe of the medial claw. The lesion was opened, drained and cleaned with 2% chlorhexidine scrub. A block was placed on the lateral claw, and the foot was bandaged. The lameness subsequently resolved during the hospitalization period.

To diagnose the cause of other abnormalities found during physical examination, a complete blood count (CBC) with fibrinogen, serum chemistry, acid-fast stained rectal mucosa scraping, BVDV isolation, bovine leukemia virus (BLV) gp-51 AGID and Johne's ELISA were performed. In light of the history and environment, fecal examination for liver fluke eggs with a commercial test kit[a] was performed. Ultrasonography of the abdomen and left scrotal sac was also performed.

Abnormalities of the CBC included mild neutrophilia (4,092/ul; normal 1,000-4,000/ ul) and eosinophilia (2,600/ul; normal 100-2,500 ul). Pertinent serum chemistry findings were elevations in creatinine (2.0 mg/dl; normal 0.5-1.1 mg/dl); total protein (8.3 g/dl; normal 5.8-7.5 g/dl); globulin (5.5 g/dl; normal 3.0-3.48 g/dl); LDH (4,744 IU/l; normal 2,853-4,319 IU/l); GGT (70 IU/l; normal 22-64 IU/l); and AST (213 IU/l; normal 58-100 IU/l). Albumin was low (2.2 g/dl; normal 2.4-3.5 g/dl), as was the albumin/globulin ratio (0.41; normal 0.84-0.94).

Because the bull urinated normally during physical examination and hydration appeared normal, a urinalysis was performed to determine if the elevated creatinine was of renal origin. Dilute urine (specific gravity 1.008; normal 1.020-1.050) in the face of elevated creatinine prompted a fractional excretion of sodium test to further define renal function. Fractional excretion of sodium was 0.6% (normal < 1%), indicating the kidneys were reabsorbing sodium normally.

Ultrasonography of the left scrotal sac revealed a hydrocele, and subsequent ultrasonography of the cranial abdomen displayed a large amount of anechoic fluid. Abdominocentesis revealed a modified transudate consisting of 62% neutrophils, 18% eosinophils, 20% mononuclear cells, specific gravity of 1.014 and total protein of <2.5 g/dl. Liver fluke eggs were found on fecal sedimentation, which prompted ultrasonography of the liver. No sonographic abnormalities were detected.

The bull was treated on day 1 with ivermectin (0.09 mg/lb; 0.2 mg/kg SQ) containing clorsulon[b] (0.91 mg/lb; 2.0 mg/kg SQ) to kill the mature liver flukes. Ceftiofur hydrochloride[c] was initiated (0.5 mg/lb; 1.1 mg/kg IM, q24 hrs for 7 days) due to the foot lesions and evidence of inflammation on the laboratory results. A fertility examination revealed the bull's semen failed to meet Society of Theriogenology criteria for a satisfactory breeder. The bull ate and drank normally while hospitalized.

On day 2, fluid was collected from the hydrocele for cytological examination. The fluid was classified as a modified transudate consisting of 40% neutrophils, 39% eosinophils and 21% mononuclear cells. Specific gravity was 1.014 and total protein was <2.5 g/dl. The Johne's ELISA and the acid-fast stained fecal smear results were reported negative on day 3.

On day 4, a right paralumbar celiotomy was performed. The visceral surface of the liver near the hilus contained two to three palpably sunken, but firm, linear tracts approximately 0.39 inch (1.0 cm) in width and 1.18 inches (3.0 cm) in length. A small nodule approximately 0.39 inch (1.0 cm) in diameter was also palpated on the most ventral aspect of the liver's visceral surface. Mesenteric lymph nodes were modestly enlarged, but not hemorrhagic. The ileum and reticular area were normal.

On day 5, negative BVDV isolation results from blood collected on day 1 were reported. At the same time, the bull had a BVDV titer of 1:48. A convalescent titer check for BVD was scheduled in 21 days to determine if an acute BVD infection could have caused the diarrhea and oral lesions. The gp-51 BLV ELISA test was reported positive on day 5, so blood was submitted for a p-24 AGID to check the possibility of lymphoma.

On day 6, the hydrocele was smaller in diameter. Liver fluke infestation was diagnosed as the cause of clinical findings other than the oral lesions and lame-

ness. The bull was discharged with instructions to return in one week for a foot bandage change and to remeasure scrotal hydrocele parameters. Long-term instructions were to deworm the bull again in two months with combination ivermectin and clorsulon at label dose to kill any matured flukes unaffected by the first treatment. It was recommended that the entire herd, especially newly introduced members, be treated for internal parasites at least annually with a product effective against liver flukes. A breeding soundness examination was recommended before the next breeding season to evaluate semen quality. Based on previous reports of bulls that recovered from hydrocele, the bull was given a fair prognosis for return to fertility.[8] The owner was advised that the bull was positive for BLV and would remain so for life. The owner was cautioned about procedures that would risk transferring infected lymphocytes from the bull to other herd members.

On day 13 the oral lesions, diarrhea and hydrocele were completely resolved. Scrotal circumference was 38 cm. The p-24 BLV antigen test reported negative. Blood taken 21 days after the first sample for convalescent BVDV serology revealed a titer of 1:96.

Discussion

Problems identified on history and physical examination were lameness, increased recumbency, weight loss, mild diarrhea, modest generalized lymphadenopathy, oral papular lesions and an enlarged left scrotal sac. The lameness and recumbency were determined on day 1 to be caused by a subsolar abscess, and these resolved with therapy.

Differentials for weight loss, diarrhea and generalized lymphadenopathy, in light of the history and physical examination, were parasitism, BVDV infection, Johne's disease, lymphosarcoma, septicemia and malignant catarrhal fever. Parasitism was concluded the most likely etiology for several reasons: fluke eggs were observed on fecal sedimentation; BVD virus isolation, Johne's ELISA test, and BLV p-24 serology were negative; and rectal temperature was normal. Negative p-24 serology has been used as an exclusion test to rule out malignant lymphoma in cattle with antibodies to the gp-51 antigen of BLV.[10]

Possible rule-outs for the oral lesions were BVDV, bovine papular stomatitis, vesicular stomatitis and malignant catarrhal fever. Negative BVDV isolation results, lack of seroconversion to BVDV, absence of fever, rapid resolution and clinical appearance (perfectly round, papular lesions with umbilicated centers) suggested that bovine papular stomatitis was the most likely cause of the oral lesions.

Scrotal hydrocele has been associated with thermal injury, neoplasia and trauma in horses,[7] and ascites in bulls.[1,5] While excess fluid in the abdominal cavity suggested that ascites may have led to the hydrocele, the abdominocentesis results (>60% neutrophils) pointed to the possibility of suppurative peritonitis. Scrotal hydrocele has also been associated with the migration of parasites through the vaginal tunic cavity and associated structures,[9] lending compelling evidence that migrating immature liver flukes caused the ascites and subsequent scrotal hydrocele. Cytology results of the hydrocele indicated that this fluid most likely came from the abdomen because of similar cell populations, specific gravity and protein levels.

While thermal injury from increased recumbency while lame in mid-summer could also have contributed to the hydrocele, the large proportion of eosinophils in the hydrocele fluid strongly suggested parasite migration. In one report of scrotal hydrocele secondary to ascites in 28 bulls, mean percentages of eosinophils found in scrotal and abdominal fluid were 23.1% and 38%, respectively.[1] In this case, the scrotal fluid contained 39% eosinophils as opposed to 18% in the abdominal fluid. This discrepancy suggests that immature liver flukes might migrate aberrantly into the scrotal vaginal tunic. We conclude the scrotal hydrocele was most likely caused by ascites due to migration of immature liver flukes through the abdomen, migration of immature flukes into the scrotal vaginal tunic, thermal injury secondary to increased recumbency, or combinations of any of the above.

Exploratory laparotomy was performed because of the bull's value and the fact that abdominocentesis (>60% neutrophils), CBC (neutrophilia) and serum chemistry results (hyperglobulinemia, low albumin/globulin ratio) suggested chronic inflammation. Surgery also allowed examination of portions of the liver not visible by ultrasonography, as well as the ileum for evidence of Johne's disease. While a liver biopsy may have been indicated, exploratory laparotomy was a safe alternative that provided more information to narrow the differential list. Liver lesions found near the hilus on exploratory celiotomy were thought to be firm bile ducts, and were consistent with described gross lesions of fluke migration.[4] These lesions were undetectable by ultrasound, possibly due to their depth from the skin surface. Exploratory laparotomy findings made traumatic reticuloperitonitis, Johne's disease and lymphosarcoma unlikely, but confirmed significant liver fluke disease.

The elevated creatinine was attributed to the large muscle mass of the bull and was consistent with a previous report of elevation in serum creatinine in normal Holstein bulls greater than four years of age.[6]

Signs of fascioliasis in cattle are typical of liver dysfunction and include emaciation, weight loss, icterus, edema and/or ascites, constipation or diarrhea, anemia and rough hair coat. Common laboratory findings include eosinophilia, hypoalbuminemia, hyperglobulinemia, decreased albumin/globulin ratio, decreased packed

cell volume and elevated serum liver enzymes (GGT, LDH, AST).[13] The bull in this case demonstrated many of these clinical and laboratory abnormalities. To survive immunocompetent hosts, liver flukes can alter and suppress humoral and cell-mediated immune responses, thereby increasing susceptibility of the host to other diseases.[13] This may explain the concurrent presence of bovine papular stomatitis and external lymphadenopathy in a five-year-old bull. Internal lymphadenopathy was attributed to migration of immature flukes through the peritoneal cavity and liver parenchyma. A positive BLV gp-51 test, however, warranted additional testing (p-24) to rule out lymphoma.

The common means of diagnosing fascioliasis in cattle is fecal examination through sieving, centrifugal or gravity recovery methods.[3,11] Attempts to recover fluke eggs using fecal flotation solutions are unsuccessful, since fluke eggs fail to float in these solutions due to rupture of the operculum. Eggs were recovered in this case by using a fecal sieving and sedimentation technique. The Flukefinder® utilizes two progressively smaller sets of screens that remove fecal debris. Fecal material remaining in the smallest screen then undergoes sedimentation. After the supernatant is poured off, the fecal pellet is re-suspended and placed on a slide for examination. Serodiagnostic tests available for liver fluke infection include the ELISA and DOT-ELISA. The disadvantage to these tests is that antibodies to *Fasciola hepatica* remain after active infection.[12,14,15] Ultrasound-guided cholecystocentesis is another diagnostic tool that may be useful in detecting liver fluke infestation. The eggs of the common liver fluke pass through the bile ducts to the gall bladder, where they are stored until release into the small intestine.[4] Passing an ultrasound-guided needle into the gall bladder allows collection of eggs contained within the bile, a method which in one report proved superior to fecal examination for diagnosis of liver fluke infestation.[2]

Treatment and control of fascioliasis is based on using effective anthelmintic agents at strategic times of the year to minimize loss. In this case the bull was the only herd animal affected, which could be explained by the native herd's development of resistance to flukes so that clinical disease would be seen primarily in newly introduced, naïve animals.

Conclusions

Although liver fluke infestation in cattle is most commonly subclinical, this case demonstrates that conditions may occasionally become conducive to overt disease. The bull in this case was placed in a new environment where flukes were common, while the native herd had developed resistance to repeated fluke infection. Scrotal hydrocele and generalized lymphadenopathy are two clinical signs not generally associated with liver fluke infestation. However, laboratory findings suggested that ascites associated with scrotal hydrocele might occasionally be caused by migration of immature flukes through the abdomen and possibly even the vaginal tunic cavity. The diagnostic workup in this case was extensive due to many confounding clinical and laboratory findings that warranted ruling out lymphosarcoma, Johne's disease, traumatic reticuloperitonitis and BVDV.

Footnotes

[a]Flukefinder®, RC Dixon and RB Wescott, Washington State University, Pullman, WA 99164
[b]Ivomec Plus, Merial Limited, Iselin, NJ 08830
[c]Excenel, Pharmacia Animal Health, Kalamazoo, MI 49001

References

1. Abbitt B, Fiske RA, Craig TM, Bitter JW: Scrotal hydrocele secondary to ascites in 28 bulls. *J Am Vet Med Assoc* 207 (6): 753-756, 1995.
2. Braun U, Gerber D: Percutaneous ultrasound-guided cholecystocentesis in cows. *Am J Vet Res* 53(7):1079-1084, 1992.
3. Dixon RC, Westcott RB: A fast and accurate fecal examination technique for diagnosis of *Fasciola hepatica. Proc Am Assoc Vet Parasitol* 32:34, 1987.
4. Krull WH: Trematoda-trematodes or flukes, in Krull WH (ed): *Notes in Veterinary Parasitology.* Manhattan, University Press of Kansas, 1969, pp 243-257.
5. Ladds PW: The male genital system, in Jubb KVF, Kennedy PC, Palmer N (eds): *Pathology of Domestic Animals.* ed 2, Vol. 14. Orlando, Academic Press, 1985, p 409.
6. Monke DR, Kociba GJ, DeJarnette M, Anderson DE, Ayars WH: Reference values for selected hematologic and biochemical variables in Holstein bulls of various ages. *Am J Vet Res* 59(11):1386-1391, 1998.
7. Schumacher J, Varner DD: Surgical correction of abnormalities affecting the reproductive organs of stallions, in Younquist RS (ed): *Current Therapy in Large Animal Theriogenology.* Philadelphia, WB Saunders, 1997, p 27.
8. Shore MD, Bretzlaff KN, Thompson JA, Magee DD: Outcome of scrotal hydrocele in 26 bulls. *J Am Vet Med Assoc* 207(6):757-760, 1995.
9. Teuscher H: Diseases of the male genital organs and hermaphroditism, in Dietz O, Wiesner E (eds): *Diseases of the Horse.* New York, Karger, 1982, p 310.
10. Thurmond MC, Holmberg CA, Picanso JP: Antibodies to bovine leukemia virus and presence of malignant lymphoma in slaughtered California dairy cattle. *J Natl Cancer Inst* 74:711-714, 1985.
11. Theinpoint D, Rochette F, Vanparijs OFJ: Diagnosing helminthiasis through coprological examination. Beerse, Belgium, Janssen Research Foundation, 1979.
12. Westcott RB, Farrell CJ, Shen DT: Diagnosis of naturally occurring *Fasciola hepatica* infections in cattle with an enzyme-linked immunosorbent assay. *Am J Vet Res* 45(1):179, 1984.
13. Zimmerman GL: Liver flukes in ruminants, in Reinhardt RW (ed): *Large Animal Internal Medicine.* St. Louis, CV Mosby Company, 1990, pp 856-858.
14. Zimmerman GL, Jen LW, Cero JE, *et al*: Diagnosis of *Fasciola hepatica* infections in sheep by an immunosorbent assay. *Am J Vet Res* 43(12):2097, 1982.
15. Zimmerman GL, Nelson MJ, Clark CRB: Diagnosis of ovine fascioliasis by a dot enzyme-linked immunosorbent assay: a rapid microdiagnostic technique. *Am J Vet Res* 46(7):1513, 1985.