Acute fasciolosis in cattle in southern Brazil

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This study describes the epidemiological and pathological aspects of an outbreak of acute fasciolosis in cattle in southern Brazil. Fifteen out of 70 three-year-old pregnant cows lost weight in the 30-40 days prior to calving. Clinical signs included diarrhea, weakness, mild anemia and jaundice. Dark yellow fluid in the abdominal cavity was observed at necropsy. Fibrin and clotted blood were adhered to the pericardium and lung, primarily in the diaphragmatic lobes. The liver was enlarged, and the capsular surface was irregular with clear areas and petechiae. At the cut surface, the liver was irregular, firm and edematous, and several hemorrhagic channels could be observed. Areas of fibrosis through the parenchyma and whitish thrombi occluding the great vessels were also observed. The livers of 10 cows that did not die were condemned at slaughter for lesions of fasciolosis similar to those observed at necropsy. Microscopically, the liver showed areas of coagulation necrosis, extensive hemorrhages in the streaks or foci and disruption of the parenchyma with neutrophil and eosinophil infiltration. Fibrosis and bile duct proliferation were also observed. Immature Fasciola hepatica flukes were observed in the parenchyma surrounded by degenerated hepatocytes, neutrophils, eosinophils, and hemorrhages. The outbreak occurred on a farm located in an area endemic for fasciolosis, although the acute form of the disease is not common in cattle in this region. It is likely that the cows were infected by F. hepatica metacercariae released in the late fall or early spring in the rice stubble where the herd was grazing prior to calving. Although mortality due to fasciolosis in cattle is infrequent, outbreaks can occur and treatments that are effective in both the immature and adult forms of the parasite should be administered to prevent economic losses.

INDEX TERMS: Fasciolosis, Fasciola hepatica, acute fasciolosis, fluke, cattle.

RESUMO.- [Fasciolose aguda em bovinos no Sul do Brasil.] Descrevem-se os aspectos epidemiológicos e patológicos de um surto de fasciolose aguda diagnosticado em bovinos na região sul do Rio Grande do Sul. De um lote de 70 vacas de três anos de idade 15 apresentaram perda de peso 30-40 dias antes da parição. Dessas, 10 vacas abortaram e 5 morreram. Os sinais clínicos caracterizaram-se por diarreia, fraqueza, anemia discreta e icterícia. Na necropsia havia líquido escuro na cavidade abdominal. Na cavidade torácica havia fibrina e coágulos de sangue aderidos ao pericárdio e pulmões, principalmente nos lobos diafragmáticos. O fígado estava aumentado de tamanho a superfi cie capsular estava irregular com áreas claras e petequias. A superfície de corte estava irregular, firme e edematosa e podiam ser observadas estrías hemorrágicas através do parênquima. Áreas de fibrose e trombos esbranquiçados ocluindo vasos sanguíneos foram, também, observados. Os fígados das 10 vacas que não morreram foram condenados no abate por lesões de fasciolose similares às observadas na necropsia. Microscopicamente, o fígado apresentava áreas de necrose de coagulação, foci de hemorragia acen-
INTRODUÇÃO

Fasciolose é um dos mais importantes parasitas em rebanhos bovinos e ovineos em todo o mundo. O parasita é transmitido por metacercárias liberadas de seus hospedeiros intermediários no final do outono ou início da primavera (Müller et al. 2012). Em Santa Vitória do Palmar, a única espécie que tem sido reportada é L. viatrix (Müller 2007). Fasciolose com alta mortalidade em bovinos ocorreu em regiões de baixa altitude. A fasciolose não é letal em bovinos, porém, pode resultar em perdas econômicas. Embora a forma aguda da doença não seja muito comum em bovinos nesta região, é provável que as vacas tenham se infectado pelas metacercárias liberadas do hospedeiro intermediário no final do outono ou início da primavera. A mortalidade em bovinos devido à fasciolose é infequente, entretanto, surtos podem ocorrer e a utilização de fármacos eficazes para controlar as formas imaturas ou adultas deste parasita deve ser administrada aos bovinos para evitar eventual perdas econômicas.

TERMOS DE INDEXAÇÃO: Fasciolose, Fasciola hepatica, fasciolose aguda, bovinos.

MATERIALS AND METHODS

An outbreak of disease in cattle occurred in a farm located at Latitude 32°58’50” S and Longitude 52°55’42” W in the municipality of Santa Vitória do Palmar, Southern Brazil, was studied. A three years old herd of heifers calved between October and November 2011 began to lose weight 30-40 days before calving and died. The epidemiological and clinical data were gathered from the owner or from the practicing veterinarian by visits to the farm where the outbreak occurred. One heifer that was recumbent for 12 hour was euthanized and necropsied to macroscopic and histological evaluation. The organs were fixed in 10% buffered formalin, embedded in paraffin, cut into 4-6μm sections and stained with hematoxylin and eosin.
RESULTS
The cows were in a wetland during summer (in February 2011) and returned for calving near the facility in August to a paddock with rice stubble. The cows were treated with Nitroxynil in May and September prior to calving and with Ivermectin after calving. Between October and November 2011, 15 of the 70 three-year-old pregnant cows lost weight in the 30-40 days prior to calving. Ten cows aborted and 5 died. Clinical signs included diarrhea, weakness, mild anemia and jaundice.

The necropsied cow showed mild anemia and jaundice. Dark yellow fluid was present in the abdominal cavity, and a thick layer of fibrin caused adhesion among the peritoneum, intestines, diaphragm and liver. The liver was enlarged with rounded edges, and the capsular surface was irregular with adhered fibrin and red strips interspersed with clear areas and petechiae (Fig.1A). At the cut surface, the liver was irregular, firm and edematous with hemorrhagic channels and fibrosis in the hepatic parenchyma (Fig.1B). Ten affected cows that did not die, were sent to slaughter and their livers were condemned by similar fasciolosis lesion. There were several dark foci filled with debris and different stages of *F. hepatica*. There were several whitish thrombi occluding the great vessels (Fig.1B). The gallbladder was enlarged, and its wall was thick and edematous and contained several trematodes. The hepatic lymph nodes were enlarged, and the cut surface was hemorrhagic and edematous. Renal infarction was also observed. Fibrin and clotted blood were adhered to the pericardium and lung, primarily in the diaphragmatic lobes.

Microscopically, the liver showed areas of coagulation necrosis, extensive hemorrhages in streaks or foci (Fig.2A), and disruption of the parenchyma with neutrophil and eosinophil infiltration (Fig.2B). There was also fibrosis and bile duct proliferation in some areas. Several vessels were occluded by thrombi (Fig.3A). Immature *F. hepatica* flukes were observed in the parenchyma surrounded by fibrous tissue (Fig.3B) and degenerated hepatocytes, neutrophils, eosinophils and hemorrhages in several areas. Peritonitis with fibrino-hemorrhagic deposits was observed on the serous surfaces. Multifocal fibrosis, hemorrhages, and neutrophil infiltration were observed in the renal cortex, and hyaline casts were present in the renal tubules.

DISCUSSION
The diagnosis of acute fasciolosis was made based on macroscopic and histological lesions. Hepatitis with extensive hemorrhages associated with infiltration of neutrophils and eosino-
phils and the presence of immature *Fasciola hepatica* flukes in the liver parenchyma are characteristic of the disease.

The outbreak occurred on a farm located in an area endemic for fasciolosis in the southern Rio Grande do Sul state (Marques & Scrofeneker 2003, Müller 2007). However, the acute form of the disease has not been previously reported. It presents a flat topography with wetlands used mainly to cultivate irrigated rice and for sheep and cattle production. The water is pumped from Mirim lake to rice fields through irrigation channels, which are appropriate for the development of the intermediate host. Also the paddocks are frequently flooded favoring the spread and maintenance of the snails throughout the year (Müller et al. 1999). In Brazil this region has the highest percentage of liver condemnation by fasciolosis at slaughterhouses, varying from 18.6% (Dutra et al. 2010) to 19.6% (Cunha et al. 2007) in cattle, and from 2.27% to 14.57% in sheep (Ueno et al. 1982, Cunha et al. 2007).

In this outbreak, it is likely that the cows were infected by metacercariae released in the late fall or early spring in the rice stubble where the herd was grazing prior to calving. The number of cercariae liberated from *Lymnaea* spp. has been reported to increase considerably in the early spring due to reactivation of the cycle that stopped or was delayed during the winter (Acosta 1994). Ingestion of large numbers of metacercariae in a short period of time may induce acute illness (Radostits et al. 2007). The herd had been treated in May and September with Nitroxynil, but this drug does not act on the immature flukes (Radostits et al. 2007). The treatment of acute fasciolosis must be performed using flukicides that are effective at killing immature *F. hepatica* (Radostits et al. 2007). Because the cattle had remained in the infested area for a long period of time, many infections may have occurred, and the treatment performed in September only eliminated the adult parasites.

Young cattle acquire resistance to fasciolosis when exposed to repeated infestations by the parasite (Acosta 1994). The capsular response appears to stimulate a surface reaction with a consequent fibrous reaction at the capsule itself (Anderson et al. 1978). Clinical fasciolosis can occur in 3 categories of cattle: lactating cows with poor feeding; adult cattle with no previous experience with the parasite; and cattle that have been parasitized infrequently at low levels and are exposed to considerable challenges (Acosta 1994). The latter condition may have occurred in this outbreak. Acute fasciolosis and associated mortalities have been observed in sheep in the same region (Fiss et al. 2012). Acute fasciolosis has been rarely reported in cattle in other countries (Anonymous 2007).

Gross and histologic lesions were typical of acute fluke infection. Peritonitis is due to the migration of immature forms of the parasite in the abdominal cavity. Inflammation involves the parietal peritoneum and the visceral peritoneum, particularly in the liver, spleen, and omentum (Brown et al. 2007). Microscopically, extensive liver damage with hemorrhagic dark red tracts of necrotic liver parenchyma, the presence of immature flukes, and thrombosis of the hepatic vessels during the migratory phase are consistent features of the disease in all species (Dow et al. 1967). Thrombosis occurs over areas of localized phlebitis produced by fluke migration tracks, but in some instances, there is no evidence of damage to the vessel wall in calves (Dow et al. 1967).

Proliferation of fibrous tissue and bile ducts were observed. These chronic lesions were likely induced by previous infections because the farm is located in an area endemic for fasciolosis. In calves, the proliferation of fibroblasts and bile ducts has been observed between seven and eight weeks after a single experimental infection with 200-300 metacercariae (Ross et al. 1966, Dow et al. 1967).

Bovine fasciolosis is generally considered a subclinical disease with economic losses primarily resulting from decreased animal productivity. However, control of *F. hepatica* is complicated by the lack of available drugs with efficacy against parasites younger than 8 weeks and the complexity of the parasite's life cycle and epidemiology. Removal of flukes from infected animals provides an added health benefit, but preventing pasture contamination of infective stages and the subsequent transmission to cattle is the basis of most treatment recommendations (Kaplan 2001).

In conclusion, although mortality due to fasciolosis in cattle is infrequent, outbreaks can occur, and treatments that are efficient in both immature and adult forms of the parasite should be administered to prevent economic losses that go beyond condemnation of the liver at the slaughterhouse.

**REFERENCES**


Ueno S., Shiomi M. & Matsumoto M. 1993. Incidence of liver condemnation by fasciolosis at slaughterhouses, varying from 18.6% (Dutra et al. 2010) to 19.6% (Cunha et al. 2007) in cattle, and from 2.27% to 14.57% in sheep (Ueno et al. 1982, Cunha et al. 2007).

Fasciola hepatica is a species of fluke belonging to the order *Trematoda* and family *Fasciolidae*. It is commonly known as the liver fluke and is a major cause of economic loss to livestock industries worldwide. The life cycle of *F. hepatica* involves several stages: cercariae, metacercariae, and adult flukes. The adult flukes attach to the liver and release eggs that hatch in the small intestine, infecting new hosts. The cercariae are released by intermediate host snails into the water, where they encyst in a variety of aquatic and semi-aquatic insects, including beetles and flies. When these insects are ingested by definitive hosts, such as cattle or sheep, the parasites mature and cause disease. The disease is characterized by extensive liver damage, fibrosis, and inflammation, leading to reduced animal productivity and increased mortality. The disease can be controlled through the use of flukicides, which are effective at killing immature and adult forms of the parasite. However, the development of resistance to these drugs highlights the need for alternative control strategies. The control of *F. hepatica* is complicated by the lack of available drugs with efficacy against parasites younger than 8 weeks and the complexity of the parasite's life cycle and epidemiology.
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