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AN OUTBREAK OF SARCOCYSTOSIS IN A CATTLE HERD

By

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LANDSVERK, THOR: *An outbreak of sarcocystosis in a cattle herd.* Acta vet. scand. 1979, *20*, 238—244. — In a cattle herd, 3 steers had anorexia and variable emaciation; 1 showed additional muscle stiffness, pyrexia, became recumbent and was killed in extremis. Gross changes in tissue specimens from this steer included grey-white foci in the skeletal muscle and a thickened, fibrous pleura. By histology interstitial pneumonia, myocarditis and necrotizing myositis were seen. There were wide-spread vascular lesions with endothelial damage, thrombosis and periarterial infiltrations. Sarcocystis-like schizonts were found in several organs, and different stages of thin-walled sarcocysts were seen in the myocardium. Examination of organs of 6 other steers in the same herd slaughtered 2 months later showed myocarditis, myositis, interstitial pneumonia and occasional vascular occlusions. There were a few schizonts in the spleen and lungs, and thin-walled sarcocysts were seen in the myocardium and skeletal muscle. It is concluded that development of vascular lesions probably represents an important step in the pathogenesis of sarcocystosis.

sarcocystis; cattle; myocarditis; myositis; vascular lesions.

Since the first demonstration of the life cycle and pathogenicity of Sarcocystis cruzi (syn. S. bovicanis) in cattle (*Fayer & Johnson* 1973) outbreaks of spontaneous disease associated with proliferative stages of sarcocystis have been reported (*Meads* 1976, *Frelier et al.* 1977, *Schmitz & Wolf* 1977, *Clegg et al.* 1978). However, Dalmeny disease described as early as in 1963 (*Corner et al.*) probably was sarcocystosis. In Norway spontaneous sarcocystosis has been reported in a sheep (*Landsverk et al.* 1978), but not in cattle. This paper describes a spontaneous disease in cattle apparently caused by a sarcocystis infection. Special attention is drawn to vascular lesions not previously reported. A preliminary account of parts of this report has been given elsewhere (*Landsverk & Anderssen* 1978).

CASE HISTORY

An outbreak of disease occurred in a cattle herd in Telemark in January 1978. The main stable in which the disease was seen, had 16 steers and 1 heifer tied up in stalls in 2 rows. A 1 year old steer got seriously ill with mild pyrexia, anorexia, emaciation, muscle stiffness and signs of abdominal pain. Treatment with penicillin-streptomycin and E vitamin had apparently no effect on the course of the disease, and the steer became recumbent and was killed in a moribund condition. Necropsy performed on the farm did not reveal any obvious cause of the disease, and tissue specimens were submitted to the National Veterinary Institute. Two other steers showed milder and transient symptoms. After the outbreak the remaining steers received an additional treatment with Tokosel®. A 1 year old German Sheep Dog and some cats had daily access to the stable.

MATERIALS AND METHODS

Tissue specimens from myocardium, skeletal muscle, liver and spleen from the steer killed in extremis (Steer 1) were received. Lungs, hearts, pieces of the muscular diaphragma and spleen of 6 steers (Nos. 2—7) from the same row of stalls as Steer 1 including 1 who had shown symptoms were collected at slaughter 2 months later. Sections from all these tissues were fixed in 25 % neutral, buffered formalin, processed routinely and stained with haematoxylin and eosin (HE) and elastin van Gieson.

Routine bacteriological examination was carried out on specimens from liver, lungs and spleen from Steer 1.

Analysis for selenium was done on liver tissue from Steer 1 by a fluorometric method (*Ihnat* 1974).

RESULTS

Steer 1

The liver selenium level was 0.05 μg/g liver wet weight. No pathogenic bacteria were isolated from the tissues.

By gross examination the skeletal muscle specimen had grey-white foci 3—4 mm in diameter. The lung specimen was somewhat firm and had a thickened fibrous pleura.

Histological examination of the lung showed thickened alveolar and intralobular septa with infiltration of mononuclear cells, lymphocytes and histocytes predominating. The lymphonodular

tissue was hyperplastic. The medium-sized and smaller arteries were frequently thrombosed, showed disruption of endothelium and internal elastic membrane, and there were accumulations of lymphocytes in the perivascular tissue (Fig. 1).

Arterial and capillary endothelium was infected with schizonts which sometimes appeared as a basophilic finely granular cytoplasmic content (Fig. 2) or as cyst-like structures containing distinct bodies (Fig. 3). Occasionally, a rosette-like orientation of elongated bodies was seen.

The myocardium showed infiltration of mononuclear cells in the interstitial and perivascular tissue. The most heavy infiltrations were located beneath the epicardium. Some muscle fibers had young cysts containing only metrocytes (Fig. 4) or merozoites with some metrocytes in the periphery (Fig. 5). In other fibers there were mature sarcocysts containing only merozoites. The capsule of the sarcocysts measured about 1 μ in thickness (Fig. 6).

The liver showed infiltration of mononuclear cells in the portal areas. Scattered schizonts were seen in the lining of the sinusoids.

In the spleen there were large numbers of schizonts sometimes forming vascular occlusions. Arterial thrombosis with perivascular mononuclear infiltration could be seen.

Figure 1. Longitudinal section of a pulmonary artery with necrotic material in an area of the wall (arrows) and endothelial disruption. Infiltrating mononuclear cells in the perivascular tissue. Steer 1, HE, × 550.

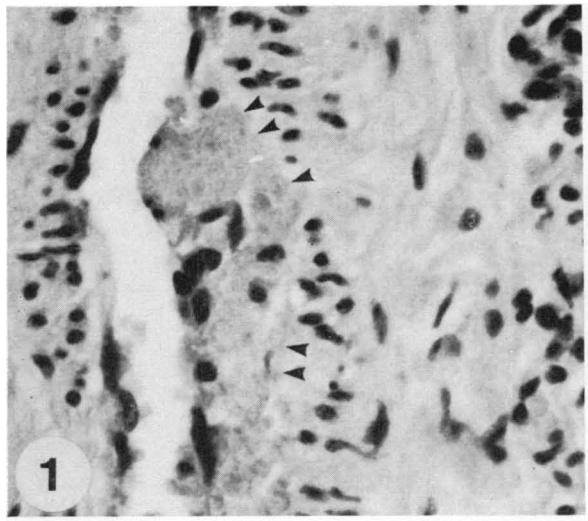
Figure 2. Early schizonts (ES) in a capillary of skeletal muscle. E = endothelial cells. Steer 1, HE, × 1700.

Figure 3. A mature schizont (MS) containing distinct bodies (merozoites). The structures marked with ES probably represent an early schizont. Spleen, Steer 1, HE, × 1445.

Figure 4. A young sarcocyst in a myocardial muscle fiber. The cyst contains metrocytes (MC) only. Steer 1, HE, × 1700.

Figure 5. A young sarcocyst in a myocardial muscle fiber. The cyst has thin capsule (black arrows) and contains merozoites (ME) and some metrocytes (MC) in the periphery. Steer 1, HE, × 1445.

Figure 6. A mature sarcocyst in a myocardial muscle fiber. The cyst has merozoites only, and the capsule is thin (arrows). Steer 1, HE, × 1445.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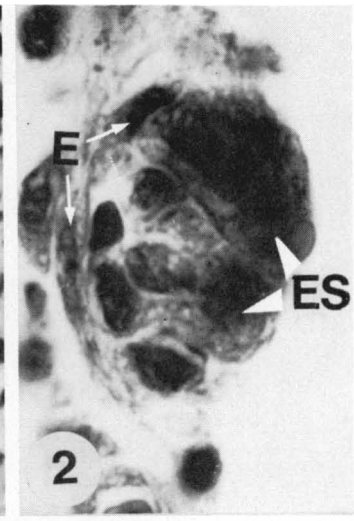
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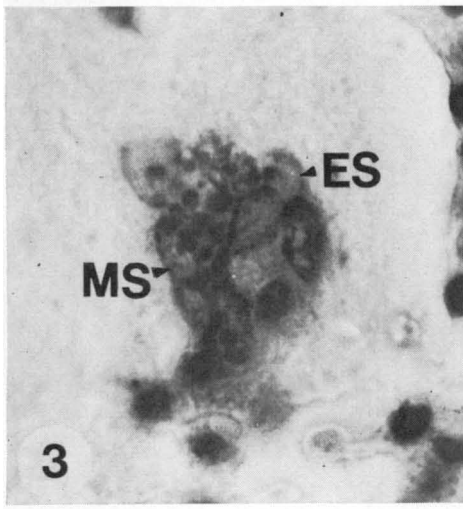
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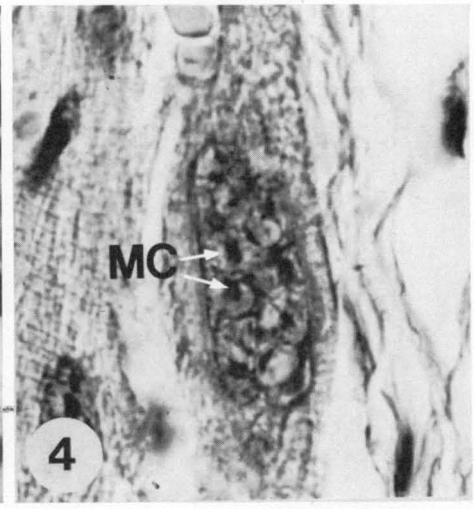
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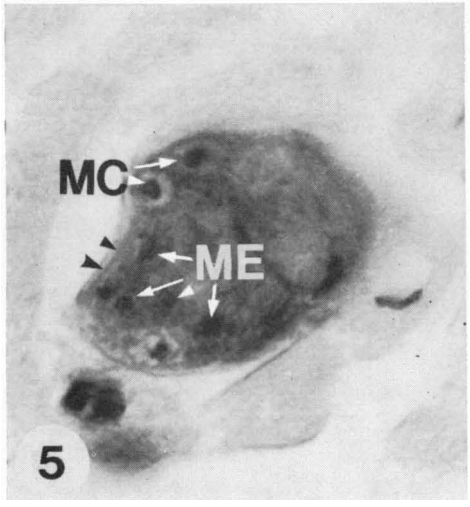
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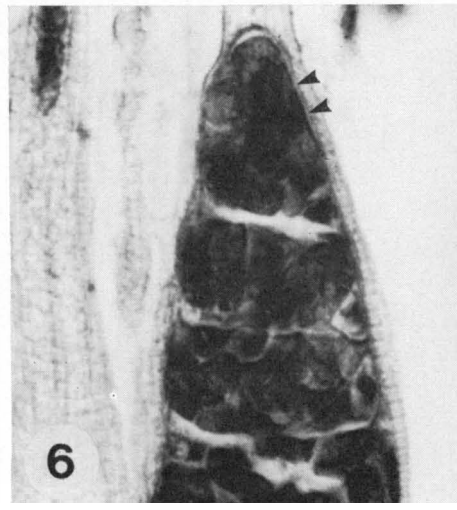
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In the skeletal muscle there were large necrotic foci with a brim of polymorphonuclear leukocytes in the periphery. The leukocytes had an eosinophilic cytoplasm; however, granules were difficult to discern, possibly due to tissue necrosis. Outside the totally necrotic areas muscle fibers showed varying degrees of degeneration and mononuclear infiltration. Thrombosed arteries were sometimes seen and some capillaries were occluded by schizonts (Fig. 2).

Steers 2—7

The lesions in the organs were similar for all these animals:

By gross examination the only changes were a slight fibrous thickening of the lung pleura and somewhat firm lungs.

By histology the lungs had thickened alveolar and interlobular septa infiltrated by mononuclear cells, in some areas with development of fibrosis. There was lymphonodular hyperplasia and some areas had a moderate infiltration of eosinophils. Arteries had proliferation of endothelium and occasional infiltrations of eosinophils in the wall. Minor vessels sometimes showed thrombosis. Schizonts were occasionally seen in the alveolar septa; lung worms were never found.

The myocardium had focal infiltrations of mononuclear cells and mostly mature and relatively large sarcocysts.

The muscular diaphragma showed degeneration of some fibers with sarcolemma proliferation and infiltration of mononuclear leukocytes and a few neutrophils. Occasional aggregations of eosinophils were seen. Sarcocysts appeared less frequently than in the myocardium and were mostly mature.

In the spleen there was some hyperplasia of lymphoid tissue and endothelial proliferation and thrombosis of arteries. In proximity to vessels trabecular mononuclear infiltration occurred. A few schizonts were seen.

DISCUSSION

The described outbreak of disease in cattle with myocarditis, myositis and interstitial pneumonia is consistent with earlier descriptions of sarcocystis infection in cattle (*Johnson et al.* 1975, *Frelier et al.* 1977). However, especially the interstitial pneumonia might be caused by a number of other agents. Damage by sarcocystis has been attributed to the schizont stages (*Johnson et al.*). The schizonts reported here had an appearance simi-

lar to that of sarcocystis (*Fayer & Johnson* 1973, *Markus et al.* 1974). It must be admitted that schizonts were few in some of the steers and might have been misinterpreted. However, it is questionable whether the presence of schizonts should be required for the diagnosis of sarcocystosis. Schizonts were found only 26—33 days post inoculation in experimental infection (*Johnson et al.*) and, therefore, assessment of cyst age would be of considerable supportive value for the diagnosis in less acute cases. In this report the presence of young cysts, i.e. cysts containing metrocytes only or metrocytes beneath the capsule (*Mehlhorn et al.* 1975), is considered as a support for the identity of the schizonts. Under natural conditions the infection occurs probably more than once in the same animal. This may explain the presence of both mature sarcocysts and schizonts at the same time. The thin capsule of the mature cysts reported here is according to *Mehlhorn et al.* characteristic for S. cruzi (syn. S. bovicanis) in cattle contrasting the thick capsules of S. hirsuta (syn. S. bovifelis) and S. hominis (syn. S. bovihominis). S. cruzi is recognized as the pathogenic species for cattle (*Fayer & Johnson*). The capsule type demonstrated here represents a further support of the presumption of sarcocystis as the causative agent for the reported disease.

The liver selenium level in the one steer examined indicates a probable deficiency (*Frøslie*, personal communication 1978). Selenium deficiency may cause myopathy. However, the possible role of selenium deficiency in this case cannot be decided.

In recent papers the vascular lesions in sarcocystosis have received relatively little attention. However, as early as 1957 *Hansen & Mostafa* suggested a relationship between periarteritis nodosa and sarcocystis in a calf. *Landsverk et al.* (1978) reported periarterial infiltrations in a sheep with sarcocystosis. In the present study the intimal reaction of vessels was more marked than the perivascular, possibly because of the affinity of sarcocystis for endothelial cells (*Fayer & Johnson*). The endothelial lesions have probably triggered the thrombosis seen here, as in nosematosis, which is another protozoan disease causing vascular damage (*Nordstoga & Westbye* 1976). Thrombosis and vascular occlusions caused by the protozoon itself when obstructing capillaries, are probably important for the development of lesions in sarcocystosis.

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SAMMENDRAG

Et utbrudd av sarkocystose i en storfébesetning.

I en besetning av 16 fóringsokser og en kvige ble 3 syke med anoreksi og moderat feber. Én av oksene magret av, ble stiv i muskulaturen og ble til slutt liggende. Den ble avlivet i moribund tilstand. Forandringer i innsendte organprøver omfattet grå-hvite foci i skjelettmuskulaturen og fortykket fibrøs pleura. Histologisk såes interstitiell pneumoni, myokarditt og nekrotiserende myositt. Det var utbredt endotelcelleskade i karene med trombosering og perivaskulære celleinfiltrater. Sarkocystis-lignende schizonter ble funnet i flere organer, og forskjellige stadier av tynnveggede sarkocyster såes i myokardiet. Ved undersøkelse av organer fra 6 av oksene i samme båsrekke, slaktet 2 måneder senere, fantes myokarditt, myositt og interstitiell pneumoni, samt karforandringer som ovenfor. Tynnveggede sarkocyster ble funnet i myokard og skjelettmuskulatur, og noen få schizonter såes også i lunge og milt. Det er konkludert med at utviklingen av karskader trolig utgjør et viktig steg i patogenesen ved sarkocystose.

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