Eosinophilic myositis/lymphadenitis in slaughter cattle

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Sarcocystis species are obligatory, heteroxenous, coccidian parasites of muscles of animals. The sexual cycle develops in epithelial cells of the small intestine of definitive carnivorous hosts and humans. Asexual reproduction occurs in the endothelium and myocytes of the intermediate hosts, namely cattle, sheep, goats, swine, horses, and mice. Cattle become infected by ingesting herbage contaminated by sporocysts in the feces of carnivores or humans. Dogs, cats and humans are usually infected by consumption of raw or semicooked muscle, viscera, or commercial pet foods (1). Beaver *et al* (2) reviewed 40 cases of *Sarcocystis* infection in humans; three sarcocysts resembled those found in monkeys and one resembled sarcocysts in cattle.

Cattle can be infected with *Sarcocystis cruzi* (canine cycle), *S. hirsuta* (feline cycle), and/or *S. hominis* (primate cycle) (3). In cattle, *S. cruzi* infection can cause acute disease characterized by fever, anorexia, drooling, immunosuppression, lymphadenitis, vasculitis, microthrombosis, hemorrhage, anemia, icterus, serous atrophy of fat, pneumonia, encephalitis, endometritis, placentitis, abortion, and death (4–9). Survivors may develop chronic disease with unthriftiness and wasting. Eosinophilic myositis, which may be associated with sarcocystosis, can be detected during routine meat inspection (1,10) or when cutting the carcass into retail portions.

Sarcocystosis can be induced in calves by the feeding of sporocysts from the feces of dogs, coyotes, and cats (3,11,12). Diagnosis of sarcocystosis can be made by indirect hemagglutination, agar gel diffusion, indirect fluorescent antibody technique, ELISA, and peroxidase-antiperoxidase tests on formalin-fixed tissues (13–16). Prevalence of sarcocystosis in cattle at slaughter in New Zealand was found to be 64% (17); in Poland up to 97%, from which 36% were pathogenic for humans (18); and in Czechoslovakia, the average infection rate in cows was 91% (19).

Although many cases of eosinophilic myositis are thought to be due to sarcocystosis, there has been insufficient histological evidence to support this contention. Infiltration of eosinophils around mature sarcocysts, ulcerated sarcocystic capsules, and granulomatous eosinophilic lymphadenitis from natural cases have not been reported previously.

Samples of bovine hearts, skeletal muscles and lymph nodes were collected at abattoirs across Canada. Tissues were fixed in 10% neutral buffered formalin, shipped to this laboratory, processed routinely, and stained with hematoxylin and eosin (H & E). Selected samples were serially cut (up to 200 sections). Of 25 submissions (19 eosinophilic myositis and 6 eosinophilic lymphadenitis) reviewed in detail, 11 were from adult animals, 11 were from two-year-old cattle, and the age was not indicated in three cases.

There were 681 carcasses and 219 portions condemned because of eosinophilic myositis from 1984 to 1987 in registered establishments in Canada. The condemnations were based on finding green zones of various sizes in the skeletal muscles and heart (Figure 1a) at postmortem examination.

These findings strongly suggest that most cases of eosinophilic myositis are due to *Sarcocystis* infection

Microscopically, eosinophilic epicarditis, myocardial granulomas surrounded by eosinophils, and the presence of sarcocysts with and without inflammation were common (Figures 2 and 3). A few mature sarcocysts were surrounded by groups of eosinophils (Figure 4) or wide zones of eosinophils. Degenerated sarcocysts had capsules that were eroded and were surrounded by Splendore-Hoeppli reaction, multinucleated giant cells, and eosinophils (Figure 5). A few cases had mineralized muscle fibers surrounded by multinucleated giant cells and eosinophils. Also there were eosinophilic necrotic zones, each surrounded successively by a thin band of radiating fibroblasts and then by a wide zone of eosinophils (Figure 6). Macroscopically, a few lesions of eosinophilic myositis had 1–3 cm, focal or linear, green-grey zones. Microscopically, these zones were composed of eosinophils invading and replacing the muscle fibers (Figure 7). Thirteen of the 19 submissions of eosinophilic myositis were associated with sarcocysts. On routine examination, six cases had no sarcocysts, but three mature sarcocysts were found in two of 50 H & E sections in the sixth submission.

Macroscopically, six submissions of mesenteric lymph nodes contained 1–3 mm diameter green or grey zones. Histologically, these lesions were similar to the granulomas observed in skeletal muscles. These necrotic areas were located in the germinal centers (Figure 8). Capsules of a few mesenteric lymph nodes were infiltrated by eosinophils. Eosinophilic vasculitis in lymph nodes was also present and rarely were there first generation meronts in the lumina of small blood vessels (Figure 9).

Of 16 submissions of muscle diagnosed as steatosis (Figure 1b) from 1985–1988, sarcocysts were found in four submissions. In addition, one submission of cardiac eosinophilic granuloma had sarcocysts in the myocardium; the diaphragm of the same case had linear fatty infiltration, with eosinophilic granulomas surrounded by many eosinophils (Figure 10).

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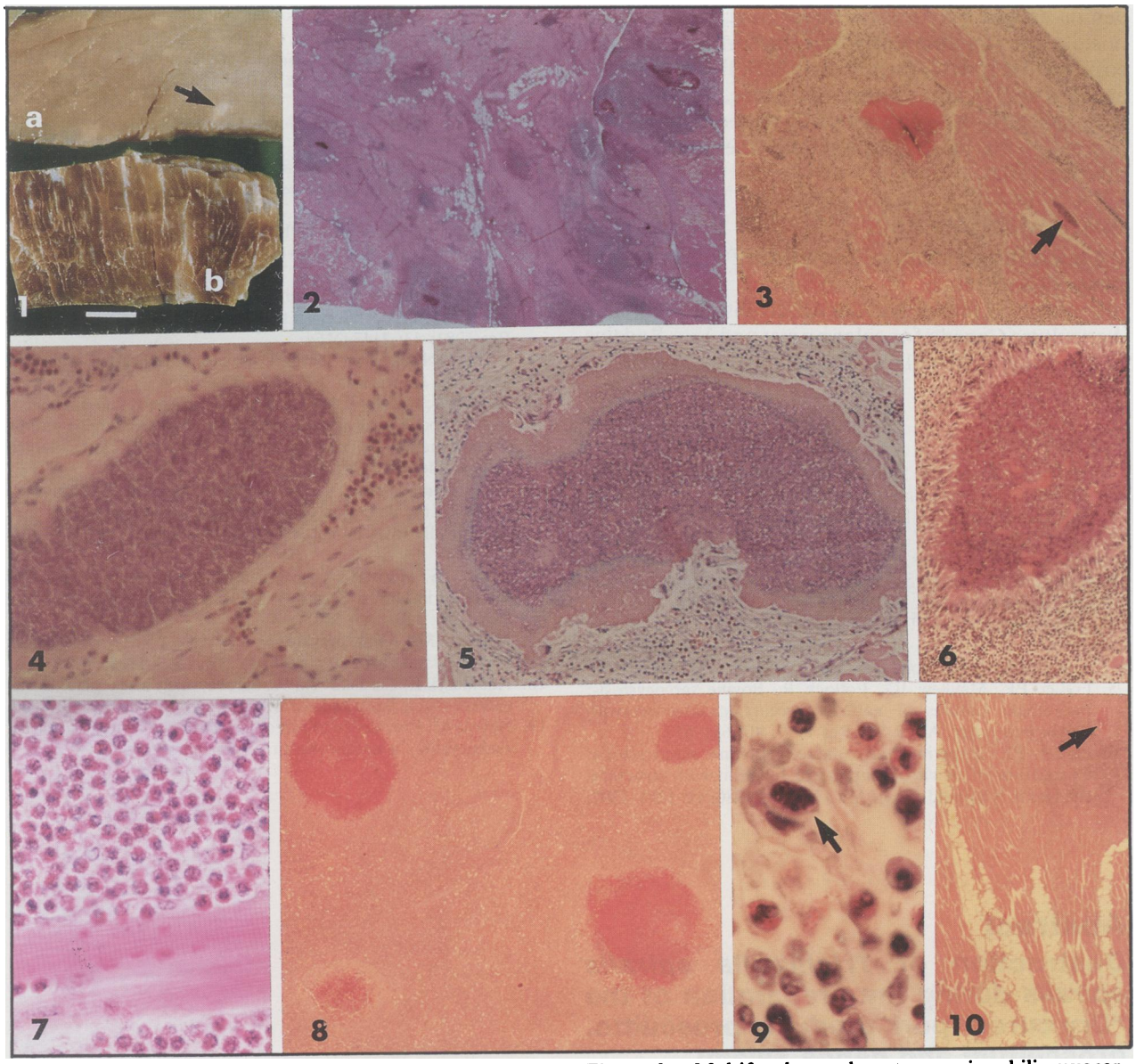

Figure 1. Multifocal green-grey granulomas (arrow) in the heart (a); and linear fatty infiltration of muscle (b). Formalin-fixed tissue. Bar = 1 cm.

Figure 2. Multifocal granulomatous eosinophilic myocarditis with zonal fatty infiltration. H & E.

Figure 3. Myocardial eosinophilic granuloma with epicarditis, and sarcocysts (arrow). H & E.

Figure 4. Mature sarcocyst with a thick capsule surrounded by groups of eosinophils. H & E.

Figure 5. Degenerated sarcocyst with ulcerated capsule, surrounded by Splendore-Hoeppli reaction, giant cells and eosinophils. H & E.

Figure 6. Degenerated sarcocyst surrounded by fibroblasts at right angles and eosinophils. H & E.

Figure 7. Diffuse eosinophilic myositis with the invasion of muscle fibers by eosinophils. H & E.

Figure 8. Eosinophilic granulomas of various sizes in the mesenteric lymph node. H & E.

Figure 9. First generation meront in a small blood vessel of the cortex of a mesenteric lymph node (arrow). H & E.

Figure 10. Fatty infiltration and sarcocystic eosinophilic granuloma (arrow) surrounded by eosinophils in the diaphragm. H & E.

Our macroscopic and microscopic findings of eosinophilic myositis and lymphadenitis in slaughter cattle support and extend the causal role of *Sarcocystis* infection reported previously (1,5,10). Histologically, sarcocysts were associated with lesions in a high proportion of our cases. Some sarcocysts were mature, ruptured or degenerated, and surrounded by eosinophils. These findings strongly suggest that most cases of eosinophilic myositis are due to *Sarcocystis* infection. However, in some cases it may not be possible to find sarcocysts because they are no longer present in the end stage of sarcocystosis or samples submitted are insufficient. It has been hypothesized that eosinophilic myositis is caused by toxins released from sarcocysts (1).

Although *Trichinella spiralis* may produce eosinophilic myositis, it is not generally considered important as a differential diagnosis because herbivores usually do not consume infected dead rodents. Nutritional myopathy is characterized microscopically by myofibrillar degeneration, lysis, mineralization, and infiltration by macrophages. Eosinophilic lympha-

denitis caused by parasitic larval migration may be mistaken for sarcocystosis, but the former has large eosinophilic zones which usually lack the fibroblasts at right angles around necrotic zones. Tuberculous lymphadenitis has characteristic caseous zones of necrosis with mineralization and the presence of mycobacteria.

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Abstract

Destruction of *Trichinella spiralis spiralis* during the preparation of the "Dry cured" pork products prosciutto, prosciuttini and genoa salami

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Genoa salami, proscuittini (pork butt) and proscuitto (Italian ham) were prepared from pork carcasses that were heavily infected experimentally with *Trichinella spiralis spiralis*. Genoa salami was prepared with salt concentrations of 2.0%, 2.75% and 3.3%. Proscuitto was prepared by two procedures approved by Agriculture Canada. At various times post-preparation, samples of the various cured products were taken and examined by pepsin digestion and rat bioassay for the presence of viable trichinae. Water activity and pH of th cured meat were also determined.

Curing of the various products was shown to destroy the *Trichinella* larvae. Pepsin digestion revealed that larvae progressively became loosely coiled, uncoiled and more subject to digestion (ghost larvae) during the curing process. Rat bioassay revealed the presence of viable trichinae in the proscuitto prepared using a sodium chloride salt mixture at day 34 but not at day 48 postpreparation. All other bioassays carried out on Genoa salami between 13 and 42 days postpreparation, on proscuittini between days 27 and 69 and on proscuitto between days 34 and 69 were negative for viable trichinae.

Under the conditions of this study, preparing Genoa salami with salt concentrations as low as 2% did not appear to affect the destruction of *Trichinella* larvae.

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