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Porcine Enteritis Associated with *Eimeria spinosa* Henry, 1931 Infection

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ABSTRACT: Coccidia of the genus *Eimeria* are present in most pigs raised on dirt in the United States. They are generally considered non-pathogenic in weaned pigs. Oocysts of *Eimeria spinosa* Henry, 1931 were observed in tissue sections and intestinal contents of a weaned male pig that died suddenly on a farm in Iowa. Microscopically, necrotizing enteritis associated with many thick-walled coccidial oocysts was present in intestinal sections. Examination of intestinal contents demonstrated oocysts that were thick-walled and had small projections on the surface of the oocyst wall, characteristic of *E. spinosa* Henry, 1931 of swine. Twenty-five oocysts in intestinal contents measured 20.4 by 14.2 μm. No pathogenic bacteria were detected in the pig by culture methods, but lesions suggestive of salmonellosis were observed in some tissues. The specific cause of death was not determined; however, *E. spinosa* infection was considered to have contributed to the death of this pig. The results suggest that *E. spinosa* may be pathogenic for pigs.

Coccidia of the genus *Eimeria* are common in swine raised on dirt or pasture in the United States (Vetterling, 1966; Greiner et al., 1982; Lindsay et al., 1984); however, intestinal disease in swine due to *Eimeria* spp. infection is not common (Hill et al., 1985). *Eimeria spinosa* was described by Henry (1931) from swine and is a member of the rough-walled group of porcine *Eimeria* species as later defined by Vetterling (1965). Koudela and Vitovec (1992) described the complete life cycle of *E. spinosa* and supplemented the previous experimental studies on this coccidium by Wiesenmüller (1962) and Ernst (1987). Because clinical porcine coccidiosis caused by *Eimeria* species is rare (Hill et al., 1985), we report coccidiosis caused by *E. spinosa* in a weaned pig from Iowa.

Fresh and formalin-fixed small intestine and intestinal contents were collected from a weaned male pig that died on a farm in Iowa. Samples were sent to and examined by diagnosticians at the Veterinary Science Department, South Dakota State University, Brookings, South Dakota. The submitting veterinarian described hemorrhage in the lung and necrosis in the ileum and jejunum. Several attempts to obtain additional clinical history from the veterinarian were unsuccessful. Examination of hematoxylin and eosin–stained histological sections indicated severe intestinal coccidiosis. Examination of intestinal contents revealed numerous rough-walled coccidial oocysts.

Intestinal sections demonstrated severe diffuse necrotizing enteritis characterized by necrosis of the luminal one-half to two-thirds of the mucosa. In some areas, necrosis extended to the submucosa (Fig. 1). There was extensive mucus accumulation in crypts and lumen with mild to moderate amounts of inflammation. Inflammatory cells were mostly mononuclear, with some eosinophils and lesser numbers of neutrophils. In some areas with deeper mucosal necrosis, there was thrombosis of blood vessels in the lamina propria. Mixed with the necrotic debris, mucus, and inflammatory cells of the lumen were large numbers of coccidial oocysts (Fig. 2). A variety of bacteria were present throughout mucosal lesions, suggesting opportunistic or postmortem overgrowth. Severity of changes in the submucosa varied directly with the severity of the lesions of corresponding luminal mucosa. They were characterized by congestion, thrombosis, and inflammatory cell accumulation. Inflammatory cells were the same as those found in the mucosa. There was moderate lymphoid cell depletion in Peyer’s patches. The Peyer’s patch domes had severe depletion, with necrosis of covering epithelium and large numbers of coccidia present. Crypt epithelium appeared to be herniated deep into muscularis mucosa, forming cysts partially lined by crypt epithelium and filled with mucus, necrotic debris, and many coccidia. Microscopic changes in sections of colon examined were much milder than in intestine; the most prominent change was thrombosis of blood vessels in the lamina propria and to a lesser extent in submucosa.

The superficial mucosa was autolytic, and its epithelium was missing. No coccidia were seen in the colon. Attempts were made to isolate bacteria from the fresh intestinal and colonic material. Aerobic bacterial cultures did not produce bacterial pathogens. Dark-field examinations of preparations were negative for bacterial pathogens.

Lesions in other tissues consisted of splenic congestion, multifocal hepatitis, and pulmonary thrombosis, which are suggestive of bacter-
emia, specifically the presence of Salmonella; however, attempts to culture bacterial pathogens from lung, liver, and spleen were negative.

Intestinal contents were stored in 2.5% (w/v) potassium dichromate and sent to the Center for Molecular Medicine and Infectious Diseases, Department of Biomedical Sciences and Pathobiology, Virginia–Maryland Regional College of Veterinary Medicine, Virginia Tech, Blacksburg, Virginia, for morphological analysis. Oocysts (n = 25) were examined and photographed using an Olympus BX60 microscope equipped with differential contrast optics and a calibrated ocular micrometer.

No sporulated oocysts were observed. Oocysts had thick walls that contained spinelike projections (Fig. 3). The oocyst wall had 2 layers. Variable numbers of globules were present in the oocyst. The 25 oocysts measured 20.4 ± 1.96 by 14.2 ± 1.96 μm (range, 17–26 by 12–16 μm).

Vetterling (1965) described the coccidia of swine present in the United States and placed E. spinosa in the rough-walled group. This group contains Eimeria perminuta Henry, 1931, Eimeria polita Peltéryd, 1949 (syn. Eimeria cerdonis Vetterling, 1965), and Eimeria scabra Henry, 1931. The oocysts of E. perminuta (13.3 by 11.7 μm) are much smaller than those of E. spinosa, and the oocysts of E. polita (28.9 by 21.3 μm) and E. scabra (31.9 by 22.5 μm) are much larger (Vetterling, 1965).

Henry (1931) remarked that unsporulated E. spinosa oocysts contained large granules. In the present study, we described these as globules (Fig. 3).

*Eimeria spinosa* is generally considered nonpathogenic for pigs. Henry (1931) described *E. spinosa* from the cecal contents of a 5-mo-old pig that was clinically normal and from the cecum of a pig with enteritis. She did not describe microscopic lesions in these pigs. Wiesenäüter (1962) described clinical disease and death in a pig experimentally infected with 1.2 × 10^6 E. spinosa oocysts; however, this pig was weak and sickly prior to experimental infection. Ernst (1987) did not believe that *E. spinosa* was pathogenic for young pigs fed up to 1 × 10^6 oocysts. Koudela and Vitovec (1992) described microscopic changes but not clinical disease in pigs experimentally infected with 5 × 10^5 E. spinosa oocysts. The lesions consisted of inflammatory cell infiltration in the lamina propria of the posterior jejunum, Peyer’s patch activation, and sporadic erosions scattered at the villous tips. No villous atrophy was seen, but large numbers of endogenous stages were present (Koudela and Vitovec, 1992). The lesions described by these authors are much less severe than those we observed in the present study. We did not isolate *Salmonella* sp. from the pig tissues we examined, but there is a chance it was present and enhanced the *E. spinosa* infection we observed.

**LITERATURE CITED**


