EXPERIMENTAL TRANSMISSION OF COCCIDIOSIS TO NEONATAL PIGLETS

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The coccidium Isospora suis has been incriminated as a cause of diarrhea in the newborn pig in recent years (Bergeland; Sangster; Stuart 1979; Morin; Sanford; Roberts). The disease was characterized clinically by a diarrhea variable in severity, dehydration and retarded growth in piglets from five to 15 days of age. Morbidity rates were variable with mortality rates usually under 20%. The most common lesion was a multifocal atrophy and blunting of the villi in the jejunum and ileum with coccidial organisms present in variable numbers of epithelial cells covering affected villi. Experimental reproduction of the disease confirming Isospora suis as a determinant cause of diarrhea in piglets has been reported recently (Stuart 1980). This paper reports the experimental reproduction of porcine neonatal coccidiosis with sporulated oocysts of Isospora suis.

Thirty-eight piglets were obtained from a specific pathogens free (SPF) herd with no history of neonatal diarrhea since the beginning of the exploitation; feces from several sows in the herd examined for the presence of coccidia were all negative. The piglets born naturally were allowed to suck their dam until two days of age and were then housed in individual isolation units. They were fed a commercial sow's milk replacer at a rate of 150 cc every eight hours. Twenty-eight piglets were inoculated at three days of age with 100,000 sporulated oocysts of Isospora suis; the inoculums were obtained from natural cases of porcine neonatal coccidiosis where no other enteropathogenic agents had been demonstrated. Ten piglets were used as normal controls. Fecal samples were examined for coccidia daily; the appetite, stool consistency and general state of health were also recorded. Groups of piglets were killed from three days to 12 days postinoculation and routine necropsies were performed. Histopathological examinations were performed on all levels of the digestive tract; bacteriological examinations were also conducted and the E. coli isolates obtained were verified for their enteropathogenicity. The direct fluorescent antibody technique was used on frozen sections for the detection of TGE virus and rotavirus and direct electron microscopic examinations of their intestinal contents by negative staining were also performed.

Most of the inoculated pigs were anorectic and depressed four days postinoculation (p.i.). Diarrhea was noted for the first time at four days (8 piglets), five days (13 piglets) and six days (2 piglets) p.i. The diarrhea was initially yellow colored, soft and milky and was becoming watery and profuse within 24 hours. Anorexia, depression, diarrhea or soft stools, dehydration and retarded growth were still present in pigs euthanised at 11 and 12 days p.i. Unsporulated oocysts were first observed in the feces at four days (3 piglets), five days (11 piglets), six days (2 piglets) and seven days (1 piglet) p.i. Ninety percent of the pigs kept alive more than four days p.i. have shed oocysts; the patent period could not be determined precisely with the design of our experiment but some of our pigs have shed oocysts for at least five to six days and counts ranged from 300 to 558,000 per gram of feces.

Gross lesions were characterized mainly by liquid and yellow colored intestinal contents with no remarkable changes in the intestinal mucosa. Microscopic lesions were restricted to the small intestine and more pronounced in the middle and lower jejunum and ileum. In piglets killed three days p.i., few foci of mild villous atrophy with erosions of the tip of few villi were observed; asexual stages of coccidia were rarely seen. Two of the three piglets killed four days p.i. had well developped lesions characterized by a multifocal villous atrophy and focal erosions of villi with adhered fibrinous exudate, necrotic debris and inflammatory cells. Many asexual stages of coccidia (meronts and merozoites) were present in epithelial cells lining more or less affected villi. Similar lesions were observed in piglets killed between five and eight days p.i.; the number of asexual forms present in immature epithelial cells covering atrophic villi had decreased significantly in pigs killed after five days postinoculation. Sexual forms (macrogametocytes and microgametocytes) were seen in significant numbers for the first time in pigs killed at five days p.i. Regeneration of affected villi seemed well underway in piglets necropsied from nine to 12 days p.i. and coccidial forms were difficult to find at that time. Bacteriological and virological examinations performed on the infected and control pigs gave negative results.

Clinical signs and microscopic lesions observed in our experimentally infected animals were similar to those reported in spontaneous cases of porcine neonatal coccidiosis. The incubation period was four to five days and the main clinical signs were depression, anorexia and a soft or watery, fetid, yellowish diarrhea which lasted for several days. The prepatent period varied from four to seven days and the most common was five days. The patent period could not be determined precisely with the type of experiments we have done but some of our pigs have shed oocysts for at least five to six days. These observations are similar to those reported recently by Lindsay. Microscopic lesions were characterized by a multifocal villous atrophy in the small intestine with the presence of coccidial organisms within the epithelium covering affected villi. These lesions seemed to result from the destruction of mature absorptive cells during the peak of asexual reproduction which occured around four to five days p.i. Regeneration of the villi seemed well underway in pigs killed nine to 12 days postinoculation (five to seven days after the beginning of diarrhea) and very few organisms were still present. Results of this work confirm Isospora suis as a determinant cause of neonatal diarrhea in piglets.

The authors acknowledge the support of doctor Serge Larivière.

Selected references: Bergeland, M.E.: Proc. Am. Ass. Vet. Lab. Diag. 1977, 151; Sangster, L.T., B.P. Stuart, D.J. Williams and A.M. Bedell: Vet. Med. small Anim. Clin. 1978, 73, 1319; Stuart, B.P. and D.S. Lindsay: J. Am. vet. med. Ass. 1979, 175, 328; Morin, M., Y. Robinson and D. Turgeon: Can. vet. J. 1980, 21, 65; Sanford, S.E. and G.K.A. Josephson: Can vet. J. 1981, 22, 282; Roberts, L., E. J. Walke, D. R. Snodgrass and K.W. Angus: Vet. Rec. 1980, 107, 156; Stuart, B.P., D.S. Lindsay, J.V. Ernst and H.S. Gosser: Vet. Pathol. 1980, 17, 84; Lindsay, D.S., B.P. Stuart, B.E. Wheat and J.V. Ernst: J. Parasitol. 1980, 66, 771.