Proceedings of the 47th British Equine Veterinary Association Congress
BEVA

Sep. 10 – 13, 2008
Liverpool, United Kingdom

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BEVA CONGRESS
British Equine Veterinary Association
9-12th September - Birmingham, UK

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Proliferative enteropathy (PE) is now a commonly diagnosed condition in older nursing foals and weanlings in several areas of North America. The first publication confirming the association between *Lawsonia intracellularis* and PE in a North American foal was in 1996. There are now numerous cases reported in the North American literature, a small number in Europe, and one in Australia. In a recent abstract in the AAEP proceedings, there were 57 cases reported in a single large Kentucky practice over a 2 year period. Although the geographic distribution of the disease has not been established, a review of published cases would indicate that there is likely some regional distribution to the disease. *Lawsonia intracellularis* has been best characterised in pigs as producing a protein-losing enteropathy similar to what occurs in foals, but there is virtually no epidemiological evidence to suggest that pigs are the source of the infection for foals. The organism is probably carried in the intestinal tract of several wild and/or domestic animal species, permitting contamination of horse farms and interspecies faecal-oral transmission. Rabbits, deer, ostrich, dogs, foxes, guinea pigs and hamsters are some of the other species known to have clinical disease caused by *Lawsonia intracellularis*. One report indicates that the organism only survives for 2 weeks in the environment and is readily killed by quaternary ammonium disinfectants.

When a clinical case is diagnosed on a farm, it is likely that other weanling foals and some adults on the farm will be seropositive; we have found this on a miniature horse farm. Small outbreaks have been reported on 2 Canadian farms. Genetic predisposition to the disease, infective dose, age at infection, immune response and stress factors may all be involved in determining which infected foals develop clinical signs. Clinical disease appears to be restricted to 3–11-month-old foals with peak incidence of clinical disease occurring near or right after weaning. The most common clinical findings include weight loss, diarrhoea, and ventral oedema. Ventral oedema is the most common clinical sign. The ventral oedema, which most often affects the ventral abdomen and sometimes the legs and/or head, is a physiological oedema caused by hypoproteinaemia and decreased plasma oncotic pressure. The enteric protein loss (protein-losing enteropathy) is caused by proliferation of the crypt cells and inflammation of the *lamina propria* in the distal small intestine. Two confirmed cases in our hospital have also had relatively nonpainful effusion of several joints (no *Rhodococcus* was found). One other case had severe cellulitis of the oedematous ventral abdomen caused by a *Staphylococcus aureus*. At our hospital, diarrhoea has been present in over half the PE cases, but in a large Kentucky study, <30% of the cases had diarrhoea. Loss of bodyweight occurs in most affected foals, a result of intestinal malabsorption. Some foals have become so weak they could not rise. Fever and colic occur in a few PE foals in association with the intestinal pathology.

Clinical pathology findings are characteristic. Hypoalbuminaemia and hypoproteinaemia would be expected in all cases. As a result of the oedema and in some cases diarrhoea, hyponatraemia and hypochloraemia are other common biochemistry findings. Serum total calcium is low because of the hypoalbuminaemia. A neutrophilic leucocytosis and increased plasma fibrinogen are present in most foals, but in general are not as high as in foals with *Rhodococcus equi*-related disease. Increased muscle enzymes (CK and AST) are present in the serum/plasma of many affected foals and I do not have a good explanation for this finding. A tentative diagnosis is made by consideration of age, clinical findings, typical laboratory findings, and ruling out other diseases (parasitism, other bacterial causes of enteritis, and abdominal abscessation) that may cause similar clinical signs. Both PCR testing of the faeces and serology should be performed as neither single test is highly sensitive. Faecal inhibitors may cause false negative results with PCR faecal testing in some
foals and the humoral immune response against the organism appears to be weak decreasing the sensitivity of the serological assay.

Proven antimicrobial treatments have not been uniformly established, but intravenously administered tetracycline for 5–7 days followed by oral doxycycline has been associated with a favourable outcome in many cases. Oral chloramphenicol or macrolides (azithromycin or clarithromycin combined with metronidazole and/or rifampicin) seems to have been effective in several cases. Supportive care including plasma, hetastarch, crystalloids, nutritional support, and nursing care may be as important as the antibiotic treatments.

Preventive measures will be difficult to recommend until reservoir hosts are better identified. An oral live attenuated vaccine is available for swine and could be used on horse farms with a high incidence of PE in foals.

FURTHER READING


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