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Book Review

CYATHOSTOME INFECTIONS IN THE HORSE

Pathogenesis of Cyathostome (Trichonema) Infections in the Horse. A Review. Colin P. Ogbourne. Commonwealth Inst. of Helminthology, CIH Miscellaneous publication No. 5, 1978, 25 pp., £4.00, ISBN 0-85198-434-7.

After the excellent review entitled "*Strongylus vulgaris* in the horse: its biology and veterinary importance" by C.P. Ogbourne and J.L. Duncan (1977), the Commonwealth Agricultural Bureaux published a study of literature about the pathogenesis of cyathostome infections in the horse by the former author. From recent reports, it may be concluded that the interest in cyathostome infections is on the increase and, therefore, this review can be a useful aid. The scientific value has been increased because of the proper use of Russian literature as well.

In the introduction the author states his preference for the taxonomic classification introduced by Lichtenfels in 1975. He replaced the genus *Trichonema* by four separate genera: *Cyathostomum*, *Cylicodontophorus*, *Cylicocyclus* and *Cylicostephanus*.

In the host, the development of Cyathostominae takes place in the large intestine. In every part of the large intestine the cyathostome population consists of a rather small number of different species. *Cyathostomum catinatum*, *Cylicocyclus nassatus*, *Cylicostephanus calicatus* and *Cylicostephanus minutus* are mainly found in the ventral colon. *Cylicocyclus insigne*, *Cylicostephanus longibursatus* and *Cylicostephanus goldi* are primarily found in the dorsal colon. The caecum, however, does not seem to be a predilection site for any prevalent cyathostome species.

Although little information is available about the life-cycle of the different cyathostome species in horses, it is clear that the complete development of the parasites takes place in caecum and colon, without migrating into other organs and tissues. The shortest time that cyathostome larvae stay in the gut wall appears to range from one to two months, depending on the species. Under certain circumstances, the larval phase of development can be greatly prolonged. The number of larvae in the gut wall can rise up to 60 per cm^2. Depending on the stage of development of the enclosed larvae and the species to which they belong, the nodules differ considerably in size, shape and colour. Cyathostominae have their characteristic location for larval development in the gut wall. In the mucosa, as well as in the submucosa, the larvae become surrounded by fibroblasts and encapsulated. Infiltrations of lymphocytes, eosinophils and neutrophils occur in the surrounding tissues. A great deal of the knowledge of the histopathology of larval cyathostome infections originates from the work of Mathieson (1964) who also provided several splendid photomicrographs to illustrate this review.

After emerging into the lumen of the large intestine, larvae of species which live as adults further down the gut must travel with the flow of food to their predilection site. They feed on the gut wall. Species with a small buccal capsule take in only the glandular epithelium, whereas larger species damage the deeper layers of the gut wall.

The presence of cyathostome larvae in the gut wall may lead to a catarrhal, haemorrhagic or sometimes fibrinous enteritis. The clinical symptoms caused by cyathostome infections are not very specific: loss of weight, delayed shedding of winter coat, unthriftiness, increased peristalsis, diarrhoea, poor appetite, emaciation, weakness, exhaustion and pyrexia. Affected horses are mostly less than five years old.

Strongylid infections can result in a normocytic, normochromic anaemia. Leucocytosis may occur, mostly as a result of neutrophilia, which is usually higher in more severely affected animals. Eosinophilia is directly related to the exposure to infection. Changes in the serum-protein spectrum are characteristic: especially a raised β-globulin (IgG(T)) level and a reduction in the amount of albumin.

Special attention is payed to the seasonal incidence of acute cyathostomiasis (Ref: cyathostominosis might be a better name for the disease). From winter to early summer, mostly young horses get sick and show general debility, progressive wasting, chronic diarrhoea, periodically elevated temperature, unsteadiness of gait, weakness and oedema. The mortality rate is high. Feacal egg counts are low or negative but numerous cyathostome larvae (4th and early 5th stages) can be found in the faeces. Unlike the 5th stage larvae, the 4th stage larvae are difficult or impossible to identify.

The seasonal incidence is probably the result of simultaneous resumption of development by a large number of inhibited fourth stage larvae. The disease shows, aetiologically and epidemiologically, great similarity with type II ostertagiasis in cattle and sheep.

The anthelmintics routinely used in horses are ineffective against the larvae in the gut wall. An exception seems to be fenbendazole in a single dose of 30 mg per kg bodyweight. Therefore, it might be possible to prevent outbreaks of acute cyathostomiasis by treating young horses with high doses of fenbendazole (and possibly other benzimidazole-derivates) at the end of the grazing season.

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