LECTURE 05

(Superfamily) :- TRICHOSTRONGYLOIDEA

Genus: TRICHOSTRONGYLUS

Trichostrongylus also causes the gastroenteritis in ruminants.

One species, T. axei is also responsible for gastritis in horses.

T. tenuis is responsible for severe enteritis in game birds.

Hosts

Ruminants, horses, pigs, rabbits and fowl

Site

Small intestine, except T. axei and T. tenuis.

Distribution

Worldwide

SPECIES

► T. axei abomasum of ruminants and stomach of horses and pigs

► T. colubrimormis ruminants

► T. vitrinus sheep and goats

► T.capricola sheep and goats

► T. retortaeformis rabbits

► T. tenuis small intestine and caeca of game birds

IDENTIFICATION

Gross

The adults are small and hair like

Less than 7.0 mm long

Difficult to see with the naked eye

Microscopic

□ The worms have no obious buccal capsule.

□ A most useful character is the distinct excretory notch in the oesophageal region.

□ The spicules are thick and unbranched and in case of T. axei are also unequal in length.

□ In female the tail is bluntly tapered and there is no vulval flap.

LIFE CYCLE

□ The life cycle is direct and typical to other strongyloides except the exsheathment of L3 of

intestinal species occurs in the abomasum.

□ The development from egg to infective stage takes 1-2 weeks.

□ The parasitic phase is non migratory.

□ The prepatent periods in ruminants is 2-3 weeks.

□ In hoses the prepatent period is of 25 days and in game birds of 10 days only.

PATHOGENESIS

□ After ingestion the L3 penetrate between the epithelial glands of the mucosa with

formation of tunnels beneath the epithelium.

□ After 10-12 days of infection, the tunnels ruptures to liberate the young worms.

□ Due to this rupture, there is considerable hemorrhage and edema and plasma proteins

are lost into the lumen of the gut.

□ Grossly there is enteritis particularly in the duodenum.

• The villi become distorted and flattened, reducing the area available for absorption of

nutrients and fluids.

• In heavy infection diarrhea occurs.

• Diarrhea and plasma protein loss results in weight loss.

• In case of T. axei the changes induced in the gastric mucosa are similar to those of

Ostertagia with an alteration in pH and increased permeability of the mucosa.

• One difference is that the worms penetrate between the glands rather than into the

glands as in Ostertagia.

• Coalescence of the nodular lesions often results in plaques or ring like lesions.

CLINICAL SIGNS

Heavy infection

Rapid weight loss and diarrhea

Low infection

Inappetance and poor growth rates

Soft faeces

It is often difficult to distinguish the effects of low infection from malnutrition.

DIAGNOSIS

• Clinical signs

• Seasonal occurrence of disease

• Lesions at post mortem examination

• Faecal egg counts

□ Faecal cultures are necessary for generic identification of the larvae.

TREATMENT AND CONTROL

Like that of ostertagiosis

T. tenuis infection

In game birds, the heavy infections produce an acute and fatal haemmorhagic typhilitis.

Lighter infections results in a chronic syndrome characterized by anemia and emaciation.

Treatment

Levamisole in drinking water

Pens should be moved regularly to prevent the accumulation of the larvae.

Thanx for Patience

FORMATED BY

M. SAJJAD HUSSAIN

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PRESENTED BY

M. SAJJAD HUSSAIN

dvmdoctors@gmail.comesulting in the

average egg count reaching 2000 eggs/g faeces. This was followed by a sharp

fall in spite of a rapid rise in pasture larval count during early July to a peak of

1300 larvae on 30 July.

At the beginning of the experiment, paddock C had a simitar level of residual

infeetion to paddock A, and as for paddock B, the spring rise contamination did

not result in a significant rise in pasture larval count in early June. A sharp

rise took place in early July reaching a peak of 2500 larvae/kg on 30 July. In

spite of low levels of pasture larval contamination during their early grazing

period, the average faecal egg count of the lambs reached 2600 on 25 June after

which a steady decline occurred.

Paddock D remained almost free from larvae of 0. circumdncta throughout the

observation and the faecal egg counts of the lambs reached a maximum of only

24 eggs/g faeces.

DISCUSSION

The state of affairs depicted in Fig. 1 is not dissimilar to that shown in Fig. 3

of Gibson and Everett (1973) which also i l l u s t r a t e d the effects of residual contamination.

The picture for spring rise contamination was, however, quite

different. Gibson and Everett (1973) describe a diphasic pasture larval count

with peaks in mid-June and mid-August. The line is reproduced in Fig. 2 for

comparison with the results of the current experiment, in which the f i r s t peak

was absent. Silverman and Campbell (1959) have shown that eggs of Ostertagia spp

from sheep take a minimum of 30 days to reach the infective stage when kept at

a constant temperature of ll°C. At 7.2°C some development occurred but the

infective stage was not reached. Pandey (1972) showed that eggs of the related

species 0. osterta~a developed between 10 and 35°C, the optimum temperature

being 25oc. It is generally conceded that the eggs and larvae of parasitic

nematodes require a minimum temperature of 10°C for development to proceed.

From Fig. 4 it will be observed that in 1973 the daily average soil temperature

did not reach and remain above 10oc until 10 May whereas in 1971 this occurred

on 2 May, there having been a period of 4 days in April when the soil temperature

was over 10°C. From Fig. 4 it will also be observed that temperatures were

generally higher in 1971 than in 1973. These lower temperatures in 1973 were

almost certainly the factor responsible for slowing down the development of the

eggs spread on the pasture in April and eliminating the f i r s t peak of larva[

infection due to the spring rise normally seen in June. The rainfall over the

relevant period in the two years was similar and was not likely to have been a

factor. The second peak resulting from infection generated by the iambs occurred

as normal in early August and some of the slowly developing eggs of the spring

rise contamination may have contributed larvae to this peak. On paddocks where

both residual and contamination derived from the spring rise occur, a diphasie

peak is observed - see for example Fig. 4 of Gibson and Everett (1973). Figure

3 shows that in the current experiment only a single peak was observed on

paddock C. This is the result of the delayed development of the eggs producing

the residual contamination the reason for which is discussed above.

From Table 1 it is seen that the control group (paddock D) gained 97 kg during

the experiment. The lambs on paddock A exposed to residual infection gained

approximately the same amount, namely 102.75 kg. This is perhaps surprising

since the lambs on this paddock carried a total of 302,080 worms at autopsy

whereas those on paddock D harbotr ed only 3,050 worms, On paddocks B and C

weight gains 77.5 and 79.75 kg were recorded with worm burdens of 482,890 and54

Paddock B was free from trichostrongylid larvae when the Iambs were turned

out and very few larvae (a peak of 124/kg) resulted from the spring rise contamination.

In spite of this the Iambs acquired Ostertagia infections resulting in the

average egg count reaching 2000 eggs/g faeces. This was followed by a sharp

fall in spite of a rapid rise in pasture larval count during early July to a peak of

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