**Ostertagiasis**

Cattle can be parasitised by over 18 species of gastrointestinal nematodes,infection causing parasitic gastroenteritis (PGE). The most economically important gastrointestinal nematode in cattle is *Ostertagia ostertagi* and whilst the diagnosis, epidemiology, treatment and control are described in detail for this parasite, details are similar for other gastrointestinal nematodes.

Species:

*Ostertcigia ostertagi* cattle

*O. circumcincta* sheep and goats

*O. trifurcata* sheep and goats

*Ostertagia ostertagi*

Synonym: Ostertagia lyrata, Skrjabinagia lyrata

Common name: Brown stomach worm

Predilection site: Abomasum

Parasite class: Nematoda

Superfamily: Trichostrongyloidea

Description, gross: Adults are slender, reddish brown worms with a short buccal cavity. Males measure 6–8 mm and females 8–9 mm in length.

**Description**, microscopic: The cuticle in the anterior region is striated

transversely whereas the rest of the body is unstriated and bears around 30 longitudinal ridges. The brown spicules are slightly curved and divided in the posterior region to terminate in three stubby hooked processes (Fig.2.3a). In the female, the vulva is sited about 1.5 mm from the posterior and is covered with a flap. The tail tapers gradually and ends in a slender, rounded tip.

Fig. 2.3 Spicules of Ostertagia species. (a) O. ostertagi. (b) O. leptospicularis.



Hosts: Cattle, deer and very occasionally goats

**Life cycle**: The life cycle is direct. Eggs are passed in the faeces, and under optimal conditions, develop within the faecal pat to the infective third stage within 2 weeks. When moist conditions prevail, the L3 migrate from the faeces on to the herbage. After ingestion, the L3 exsheaths in the rumen and further development takes place in the lumen of an abomasal gland. Two parasitic moults occur before the L5 emerges from the gland around 18 days after infection to become sexually mature on the mucosal surface. The entire parasitic life cycle usually takes 3 weeks, but under certain circumstances many of the ingested L3 become arrested in development at the early fourth larval stage (EL4) for periods of up to 6 months (also referred to as hypobiosis).

**Geographical distribution**: Worldwide. Ostertagia is especially important in temperate climates and in subtropical regions with winter rainfall.

**Pathogenesis**: Large populations of O. ostertagi can induce extensive pathological and biochemical changes and these are maximal when the parasites are emerging from the gastric glands (about 18 days after infection) but these may be delayed for several months when arrested larval development occurs.

In heavy infections of 40 000 or more adult worms the principal effects of these changes are first, a reduction in the acidity of the abomasal fluid, the pH increasing from 2.0 up to 7.0. This results in a failure to activate pepsinogen to pepsin. There is also a loss of bacteriostatic effect in the abomasum. Secondly, there is an enhanced permeability of the abomasal epithelium to macromolecules.

The results of these changes are a leakage of pepsinogen into the circulation, leading to elevated plasma concentrations, and the loss of plasma proteins into the gut lumen, eventually leading to hypoalbuminaemia. In addition, in response to the presence of the adult parasites, the zymogen cells secrete increased amounts of pepsin directly into the circulation.

Although reduced feed consumption and diarrhoea affect liveweight gain they do not wholly account for the loss in production. Current evidence suggests that this is primarily because of substantial leakage of endogenous protein into the gastrointestinal tract. Despite some reabsorption, this leads to a disturbance in post-absorptive nitrogen and energy metabolism due to the increased demands for the synthesis of vital proteins, such as albumin and the immunoglobulins, which occur at the expense of muscle protein and fat deposition.

**Clinical signs**: Bovine ostertagiosis occurs in two clinical forms. In temperate climates with cold winters the seasonal occurence of these is as follows:

Type I disease is usually seen in calves grazed intensively during their first grazing season, as the result of larvae ingested 3–4 weeks previously; in the northern hemisphere this normally occurs from mid-July onwards. In type I disease, the morbidity is usually high, often exceeding 75%, but mortality is rare provided treatment is instituted early.

Type II disease occurs in yearlings, usually in late winter or spring following their first grazing season and results from the maturation of larvae ingested during the previous autumn and subsequently become arrested in their development at the EL4 stage. Hypoalbuminaemia is more marked, often leading to submandibular oedema. In type II the prevalence of clinical disease is comparatively low and often only a proportion of animals in the group are affected; mortality in such animals can be high unless early treatment with an anthelmintic effective against both arrested and developing larval stages is instituted.

The main clinical sign in both type I and type II disease is a profuse watery diarrhoea and in type I, where calves are at grass, this is usually persistent and has a characteristic bright green colour. In contrast, in the majority of animals with type II, the diarrhoea is often intermittent and anorexia and thirst are usually present. In both forms of the disease, the loss of body weight is considerable during the clinical phase and may reach 20% in 7–10 days.

**Diagnosis**: In young animals this is based on:

1. The clinical signs of inappetence, weight loss and diarrhoea.

2. The season. For example, in Europe type I occurs from July until September and type II from March to May.

3 The grazing history. In type I disease, the calves have usually been set-stocked in one area for several months; in contrast, type II disease often has a typical history of calves being grazed on a field from spring to mid-summer, then moved and brought back to the original field in the autumn. Affected farms usually also have a history of ostertagiosis in previous years.

4. Faecal egg counts. In type I disease these are usually more than

1000 eggs per gram (epg) and are a useful aid to diagnosis; in type II the count is highly variable, may even be negative and is of limited value.

5. Plasma pepsinogen levels. In clinically affected animals up to 2 years old these are usually in excess of 3.0 IU tyrosine (normal levels are 1.0 IU in non-parasitised calves). The test is less reliable in older cattle where high values are not necessarily correlated with large adult worm burdens but, instead, may reflect plasma leakage from a hypersensitive mucosa under heavy larval challenge.

6. Postmortem examination. Adult worms can be seen on close inspection of the abomasal surface. Adult worm burdens are typically in excess of 40 000, although lower numbers are often found in animals which have been diarrhoeic for several days prior to necropsy. Species differentiation is based on the structure of the male spicules (Fig. 2.3).

In older animals, laboratory diagnosis is more difficult since faecal egg

counts and plasma pepsinogen levels are less reliable.

**Pathology**: The developing parasites cause a reduction in the functional gastric gland mass; in particular the parietal cells, which produce hydrochloric acid, are replaced by rapidly dividing, undifferentiated, non-acid-secreting cells. Initially, these cellular changes occur in the parasitised gland (Fig. 2.4), but as it becomes distended by the growing worm these changes spread to the surrounding non-parasitised glands, the end result being a thickened hyperplastic gastric mucosa. Macroscopically, the lesion is a raised nodule with a visible central orifice; in heavy infections these nodules coalesce to produce an effect reminiscent of morocco leather (Fig. 2.5). The abomasal folds are often very oedematous and hyperaemic and sometimes necrosis and sloughing of the mucosal surface occurs .

Fig. 2.4 Ostertagia ostertagi emerging from a gastric gland.



Fig. 2.5 Abomasum showing the characteristic nodules produced by the

development of O. ostertagi larvae in the gastric glands.



**Treatment**: Type I disease responds well to treatment at the standard dosage rates with any of the modern benzimidazoles, the pro benzimidazoles (febantel, netobimin and thiophanate), levamisole, or the avermectins/milbemycins. All of these drugs are effective against developing larvae and adult stages. Following treatment, calves should be moved to pasture which has not been grazed by cattle in the same year.

For the successful treatment of type II disease it is necessary to use drugs which are effective against arrested larvae as well as developing larvae and adult stages. Only the modern benzimidazoles (such as albendazole, fenbendazole or oxfendazole) or theavermectins/ milbemycins are effective in the treatment of type II disease when used at standard dosage levels, although the pro-benzimidazoles are also effective at higher dose rates.

The field where the outbreak has originated may be grazed by sheep or rested until the following June.

In lactating dairy cattle, topical eprinomectin has the advantage that there is no milk withholding period.

**Control:**

Traditionally, ostertagiosis has been prevented by routinely treating young cattle with anthelmintics over the period when pasture larval levels are increasing. However, it has the disadvantage that since the calves are under continuous larval challenge their performance may be impaired. With this system, effective anthelmintic treatment at housing is also necessary using a drug effective against hypobiotic larvae in order to prevent type II disease.

The prevention of ostertagiosis by limiting exposure to infection is a more efficient method of control. This may be acheived by allowing young cattle sufficient exposure to larval infection to stimulate immunity but not sufficient to cause a loss in production. The provision of this ‘safe pasture’ may be achieved in two ways:

1. Using anthelmintics to limit pasture contamination with eggs during periods when the climate is optimal for development of the free-living larval stages, i.e. spring and summer in temperate climates, or autumn and winter in the sub-tropics.

2. Alternatively, by resting pasture or grazing it with another host, such as sheep, which are not susceptible to O. ostertagi, until most of the existing L3 on the pasture have died out.