Dictyocaulus

This genus living in the bronchi of cattle, sheep, horses and donkeys is the major cause of parasitic bronchitis in these hosts.

Hosts:

Ruminants, horses and donkeys.

Site:

Trachea and bronchi, particularly of the diaphragmatic lobes.

Species:

*Dictyocaulus viviparus* cattle and deer

*D. filaria* sheep and goats

*D. arnfieldi* donkeys and horses.

Distribution:

Worldwide, but especially important in temperate climates.

IDENTIFICATION

The adults are slender thread-like worms up to 8.0cm in length. Their location in the trachea and bronchi and their size are diagnostic.

Since *D. viviparus* is the most pathogenic of the three species it is presented in detail. ***Dictyocaulus viviparus***

*Dictyocaulus viviparus* is the cause of parasitic bronchitis in cattle, also known as husk, hoose, verminous pneumonia or dictyocaulosis. The disease is characterized by bronchitis and pneumonia and typically affects young cattle during their first grazing season on permanent or semi-permanent pastures. The disease is prevalent in temperate areas with high rainfall.

LIFE CYCLE

The female worms are ovo-viviparous, producing eggs containing fully developed larvae, which hatch almost immediately. The L1 migrate up the trachea, are swallowed and pass out in the faeces. The larvae are unique in that they are present in fresh faeces, are characteristically sluggish, and their intestinal cells are filled with dark

brown food granules (Fig. 2.19). In consequence the preparasitic stages do not require to feed. Under optimal conditions the L3 stage is reached within 5 days, but usually takes longer in the field. The L3 leave the faecal pat to reach the herbage either by their own motility or through the agency of the ubiquitous fungus, Pilobolus. After ingestion, the L3 penetrate the intestinal mucosa and pass to the mesenteric lymph nodes where they moult. The L4 then travel via the lymph and blood to the lungs, and break out of the capillaries into the alveoli about 1 week after infection. The final moult occurs in the bronchioles a few days later and the young adults then move up the bronchi and mature. The prepatent period is around 3–4 weeks.

PATHOGENESIS

This may be divided into four phases:

(1) Penetration phase: days 1-7 During this period the larvae are making their way to the lungs and pulmonary lesions are not yet apparent.

(2) Prepatent phase: days 8-25

This phase starts with the appearance of larvae within the alveoli where they cause alveolitis. This is followed by bronchiolitis and finally bronchitis as the larvae become immature adults and move up the bronchi. Cellular infiltrates of neutrophils, eosinophils and macrophages temporarily plug the lumina of the bronchioles and cause collapse of other groups of alveoli. This lesion is largely responsible for the first clinical signs.

Towards the end of this phase bronchitis develops, characterized by mucus containing immature lungworms in the airways, which may only be seen with the aid of a low-power microscope, and cellular infiltration of the epithelium.

Heavily infected animals, whose lungs contain several thousand developing worms, may die from day 15 onwards due to respiratory failure following the development of severe interstitial emphysema and pulmonary oedema.

(3) Patent phase: days 26-60

This is associated with two main lesions.

First, a parasitic bronchitis characterized by the presence of hundreds or even thousands of adult worms in the frothy white mucus in the lumina of the bronchi. The bronchial epithelium is hyperplastic and heavily infiltrated by inflammatory cells, particularly eosinophils.

Secondly, the presence of dark red collapsed areas around infected bronchi. This is a parasitic pneumonia caused by the aspiration of eggs and L1, into the alveoli. These 'foreign bodies' quickly provoke dense infiltrates of polymorphs, macrophages and multinucleated giant cells around them .

Depending on the extent of the infection there may be varying degrees of interstitial emphysema and oedema.

(4) Postpatent phase: days 61-90

In untreated calves, this is normally the recovery phase after the adult lungworms have been expelled. Although the clinical signs are abating the bronchi are still inflamed and residual lesions such as bronchial and peribronchial fibrosis may persist for several weeks or months. Eventually the broncho-pulmonary system becomes completely normal and coughing ceases. However, in about 25% of animals which have been heavily infected, there is a flare-up of clinical signs during this phase which is frequently fatal.

CLINICAL SIGNS

Within any affected group, differing degrees of clinical severity are usually apparent; typically a few animals are mildly affected, most are moderately affected and a few are severely affected. Mildly affected animals cough intermittently, particularly when exercised.

Moderately affected animals have frequent bouts of coughing at rest, tachypnoea (>60 respirations per minute) and hyperpnoea. Frequently, squeaks and crackles over the posterior lung lobes are heard on auscultation.

Severely affected animals show severe tachypnoea (>80 respirations per minute) and dyspnoea and frequently adopt the classic 'air-hunger' position of mouth breathing with the head and neck outstretched. There is usually a deep harsh cough, squeaks and crackles over the posterior lung lobes, salivation, anorexia and sometimes mild pyrexia. Often the smallest calves are most severely affected.

Calves may show clinical signs during the prepatent period and occasionally a massive infection can cause severe dyspnoea of sudden onset often followed by death in 24-48 hours.

Most animals gradually recover although complete return to normality may take weeks or months. However, a proportion of convalescing calves suddenly develop severe respiratory signs, unassociated with pyrexia, which usually terminates fatally 1-4 days later. This syndrome of postpatent parasitic bronchitis has been described above.

EPIDEMIOLOGY

Generally only calves in their first grazing season are clinically affected, since on farms where the disease is endemic older animals have a strong acquired immunity.

In endemic areas in the northern hemisphere infection may persist from year to year in two ways:

(1) Overwintered larvae: L3, may survive on pasture from autumn until late spring in sufficient numbers to initiate infection or occasionally to cause disease.

(2) Carrier animals: small numbers of adult worms can survive in the bronchi of infected animals, particularly yearlings, until the next grazing season. Until recently it was assumed that they all persisted as adults, but it has now been shown that the chilling of infective larvae before administration to calves will produce arrested L5; hypobiosis at this stage has also been observed in naturally infected calves in Switzer(and. Austria and Canada, although the extent to which this occurs naturally after ingestion of larvae in late autumn and its significance in the transmission of the infection has not yet been fully established.

The dispersal of larvae from the faecal pat during the grazing season appears to be effected by a fungus rather than by simple migration. This fungus, Pilobolus, is commonly found growing on the surface of bovine faecal pats about one week after these have been deposited. The larvae of *D. viviparus*, crawling on the surface of the pats, migrate in large numbers up the stalks of the fungi on to, and even inside, the sporangium or seed capsule. When the sporangium is discharged it is projected a distance of up to 3 m in still air to land on the surrounding herbage.

DIAGNOSIS

Usually the clinical signs, the time of the year and a history of grazing on permanent or semipermanent pastures are sufficient to enable a diagnosis to be made.

Larvae are found (50–1000/g) only in the faeces of patent cases so that faecal samples should be obtained from the rectum of a number of affected individuals. At necropsy, worms will often be apparent in the opened bronchi and their size is diagnostic. A lungworm ELISA can be used to detect antibodies to *D. viviparus*. Seroconversion takes 4–6 weeks and titres persist for 4–7 months. Serology can be helpful in cases of reinfection husk, as it will often detect larval stages. Cross-reactivity occurs with intestinal nematode species so test sensitivity and specificity requires validation and setting of appropriate optical density (OD) cut-off values when interpreting results.

TREATMENT

The anthelmintics available for the treatment of bovine parasitic bronchitis are the modern benzimidazoles, levamisole or the avermectin/ milbemycins. These drugs have been shown to be effective against all stages of lungworms with a consequent amelioration of clinical signs. In the past diethylcarbamazine was widely used, but it has been largely superseded by the drugs mentioned above.

For maximum efficiency all of these drugs should be used as early as possible in the treatment of the disease since clinical signs associated with pulmonary pathology are not rapidly resolved by mere removal of adult lungworms.

.CONTROL

The best method of preventing parasitic bronchitis is to immunize all young calves with lungworm vaccine. This live vaccine, consisting of larvae attenuated by irradiation, is currently only available in Europe and is given orally to calves aged eight weeks or more. Two doses of vaccine are given at an interval of four weeks and, in order to allow a high level of immunity to develop, vaccinated calves should be protected from challenge until two weeks after their second dose.

Although vaccination is effective in preventing clinical disease, it does not completely prevent the establishment of small numbers of lungworms. Consequently, pastures may remain contaminated, albeit at a very low level. For this reason it is important that all of the calves on any farm should be vaccinated whether they go to pasture in the spring or later in the year. Also once a vaccination programme has been undertaken it must be continued annually for each calf crop. Although the limited pasture larval contamination will serve to boost the immunity of vaccinated calves it can lead to clinical disease in susceptible animals.

The vaccination programme for dairy calves should, if possible, be completed before they go to grass in the spring or early summer. However, these or suckled calves can be vaccinated successfully at grass provided the vaccine is given in spring or early summer, that is, prior to encountering a significant larval challenge.

Control of parasitic bronchitis in first year grazing calves has been achieved by the use of prophylactic anthelmintic regimens either by strategic early season treatments or by the administration of rumen boluses, as recommended in the control of bovine ostertagiosis (p. 16). The danger of these measures however is that through rigorous control in the first grazing season, exposure to lungworm larvae is so curtailed that cattle remain susceptible to husk during their second season: in such situations it may be advisable to consider vaccination prior to their second year at grass.

It is also worth noting that, because of the unpre-dictable epidemiology, the technique commonly used in ostertagiosis of `dose and move' in midsummer does not prevent parasitic bronchitis.



Fig. 2.19 First stage larvae of *Dictyocaulus viviparus.*