AN OVERVIEW OF CLOSTRIDIUM SPECIES IN CATTLE AND SHEEP

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Categories: Vets

Date: April 15, 2013

SARA PEDERSEN looks at the diseases caused by pathogenic Clostridia that can be fatal and devastate a herd, and discusses management strategies.

CLOSTRIDIAL diseases are of great concern to the cattle and sheep industries due to their usually fatal outcome, which can result in catastrophic losses when outbreaks occur.

Due to their sudden onset and severe nature, they often cause death before any clinical signs are seen. Even if disease is diagnosed early in its course, treatment is very rarely successful.

The Clostridium species that cause disease are large, Gram-positive, rod-shaped, spore-bearing bacteria. The majority are saphrophytes and found growing in water, soil and decomposing animal and plant matter. Others are normal inhabitants of the intestinal tract, such as Clostridium perfringens, and play a large part in postmortem carcase decomposition, as they rapidly multiply and invade the blood and tissue. Therefore, it is important to be able to distinguish them from species causing primary clostridial disease.

The pathogenic clostridia can be divided into three categories: the enterotoxaemias, neurotrophic clostridia and histotoxic clostridia.

Enterotoxaemias

These are a group of diseases that are due to members of the C perfringens family. Although there
are five different types, only types B, C and D are of significance in sheep in the UK and types A and D in cattle. Spores are frequently found in the soil, faeces and intestines of normal animals and germinate, then rapidly multiply under certain conditions, such as abrupt diet changes.

*Clostridium perfringens* type A is part of the normal bacterial flora of the intestinal tract in many normal animals, but has been reported to cause fatal haemorrhagic enteritis in calves and adult cattle when conditions in the intestine become favourable for germination.

Diagnosis is made based on postmortem findings of extensive necrosis of the intestines and the presence of large quantities of alpha toxin in the intestinal contents.

Type B is the cause of lamb dysentery characterised by haemorrhagic enteritis in stronger animals less than two weeks of age, typically towards the end of the lambing period. Although in most cases lambs are found dead, if symptoms are seen they include signs of abdominal pain, blood-stained faeces and, in rare cases, central nervous system (CNS) signs due to a focal encephalomalacia.

Struck is the rarest of the enterotoxaemias and due to *Clostridium perfringens* type C. Associated with sporadic deaths, it causes sudden onset abdominal pain, which is rapidly followed by death. There are five subgroups, of which subgroup one causes disease in adult sheep and subgroup two affects calves and young lambs, but as yet has only been reported in the US.

*Clostridium perfringens* type D is the most common of the enterotoxaemias and causes pulpy kidney disease. Both sheep and cattle are affected, although it is more common in lambs between the ages of four to 10 weeks and growing lambs of six months of age and older. Adults can also be sporadically affected, particularly rams on a rising plane of nutrition prior to tupping. As with most clostridial diseases, the first sign is often sudden death, although diarrhoea and CNS signs are present in the very small proportion of animals that survive for a short period of time.

**Neurotrophic clostridia**

This group contains *C. tetani* and *C. botulinum*, both of which produce potent neurotoxins. *C. tetani* neurotoxins result in tetanus, which occurs sporadically in cattle and sheep of all ages. There are several potential routes of infection, including surgical wounds (for example, castration or tail docking), the navel or traumatised tissues, such as those of the genital tract following parturition. Post-infection, the spores germinate and produce a tetanospasmin toxin that is subsequently taken up by nerve endings. The incubation period post-infection is usually three to 10 days, but has been reported to be up to several weeks. Clinical signs include stiffness and reluctance to move, progressing to bloat (more obvious in cattle), trismus (“lockjaw”), a raised tailhead and prolapse of the third eyelid. In the final stages of disease, the animal becomes recumbent, before developing tetanic spasms. Death usually occurs within three to four days after the onset of signs, although the majority of animals will be euthanised prior to this on welfare grounds.
In contrast to *C. tetani*, the toxins released by *C. botulinum* spores cause spastic paralysis known as botulism. There are seven different types of botulinum toxin (A to G), but types C and D are responsible for the majority of cases, with as little as 10µg being sufficient to kill a cow. *C. botulinum* is found in decaying vegetable matter and carcases, with the majority of outbreaks being associated with poultry litter contaminated with carcases. Clinical signs occur within one to seven days of infection. In the early stages of disease (if death is not the first sign), animals develop hindlimb weakness, which progresses to the forelimbs, head and neck resulting in recumbency with a lack of muscle tone being evident, which may include the tongue muscles allowing it to protrude from the mouth. Paralysis of the diaphragm muscles results in respiratory arrest, but affected animals are often euthanised long before this stage.

**Histotoxic clostridia**

The *Clostridium* species in this group all produce exotoxins that cause a localised tissue necrosis and systemic toxaemia.

- **Blackleg (gangrenous myositis)**

Blackleg is the most common clostridial disease seen in cattle, but is less common in sheep. It mostly affects young cattle at grass during the summer months, but it can affect animals of all ages throughout the year and has also been reported in housed cattle.

Disease is due to ingestion and activation of *C. chauvoei* spores. Most cases are found dead, but if signs are observed they include depression and lameness. Large doses of penicillin can be effective, but only if given in the very early stages. Following death, the carcase is bloated and affected muscle areas are crepitant, palpably spongy and when incised they appear blackened, dry and have a rancid odour. Fluorescent antibody testing (FAT) can be used to confirm diagnosis.

- **Malignant oedema**

Sometimes referred to as “false blackleg”, malignant oedema is caused by various combinations of clostridial organisms. *C. septicum* is most frequently involved in cattle and sheep, although *C. novyi* type A is most commonly the cause of “big head” in rams where lesions are restricted to the head only and usually a result of fighting. Clinical signs appear soon after infection of a wound with clostridia from soil contamination. The affected muscles may appear dark red and there will be extensive oedema of the surrounding tissues, with crepitus due to emphysema. Affected animals have a marked pyrexia and will be dull and toxic, with death following within one to two days. Unlike other clostridial diseases, early aggressive antibiotic treatment has been reported to be successful in sheep.

- **Braxy**
Also due to *C septicum*, braxy occurs in autumn and winter with ingestion of frost-affected feed being the suspected cause. It usually affects older lambs born the previous spring and causes sudden onset pyrexia with severe abdominal pain due to abomasitis, leading to coma and death. Treatment is unsuccessful.

- **Black disease**

Also known as infectious hepatitis, this is caused by *C novyi* type B and is associated with immature liver fluke larvae migrating through the liver and creating the ideal environment for spores to germinate and multiply. Late summer/early autumn are the typical risk periods. Clinical signs are rarely observed and animals are commonly found dead.

- **Bacillary haemoglobinuria**

Caused by *C haemolyticum*, this is predominantly a disease of cattle, but is occasionally seen in sheep with summer and autumn being the greatest risk periods. Affected animals have dark red urine and fresh blood in their faeces, an arched back and are reluctant to move. Recumbency follows with respiratory distress and death within 24 hours. Postmortem signs include jaundice and a mahogany-coloured liver, which will have at least one area of focal necrosis. It can be differentiated from black disease via toxin-antitoxin tests.

- **Clostridium sordellii**

While it has traditionally been associated with wound infections leading to gas gangrene, *C sordellii* has more recently been linked to acute abomasitis in young lambs of three to 10 weeks of age, sudden death of finishing lambs and fatal metritis in ewes. It has also been identified in cases of bovine abomasitis. Disease appears to be triggered by changes in diet or management.

**Control strategies**

Although some measures can be taken to reduce the risk of different clostridial diseases, such as fluke control to prevent black disease, due to the fact clostridia are almost ubiquitous in the environment, it is virtually impossible to eliminate risk. No control measures are as effective as vaccination; however, vaccine selection is important.

Usually the first sign of clostridial disease is the discovery of one or more dead animals. At this stage, it is important to try to determine a definitive diagnosis to allow future control and prevention of further cases. For example, suspected cases of blackleg could be malignant oedema and vaccination against *C chauvoei* only would be ineffective. Where enterotoxaemias are suspected, vaccination in the face of an outbreak has proved effective. Another strategy is to prophylactically treat all at-risk animals with long acting penicillin to prevent further losses. However, this is not a guarantee and not without risk due to the stress of handling during treatment, which could
accelerate disease in already infected animals.

There are several clostridial vaccines licensed for use in cattle and sheep and all are extremely cost-effective when balancing their cost against potential losses. Not all vaccines protect against the same clostridia species or their toxins and this is an important consideration when deciding on a vaccination policy.

The only species for which there is no general licence is *C botulinum*. However, this can be obtained under special treatment certificate (STC) from the VMD.

All of the clostridial diseases discussed can cause catastrophic losses. Due to the rapid demise of affected animals and the fact treatment is so unrewarding, outbreaks are a source of great distress to the farmer. Although certain measures can be taken to reduce the risk of disease, none are as effective as vaccination.

References/further reading

- NADIS bulletin: Clostridial disease in cattle ([www.nadis.org.uk](http://www.nadis.org.uk))