Clostridial disease in cattle: updates and developments

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Clostridial organisms are anaerobic, Gram-positive bacteria ubiquitous in the soil, where they can survive for many years. Most clostridial organisms can also be found in the gut of healthy cattle – they can live within the animal, causing no overt disease, and then be passed through the faeces to recontaminate the soil.

Table 1. List of *Clostridium* species, predisposing factors and clinical signs

	Clostridium species	Common name	Predisposing factors	Clinical signs	Comments
Histotoxic clostridia: produce exotoxins that cause a localised tissue necrosis and systemic toxaemia	*Clostridium chauvoei*	(True) blackleg	Damage to muscles – particularly of hindlimbs and neck.	Sudden death. Animals usually in good condition. May be swelling of affected muscle.	Rapid decomposition of carcase.
	(*Clostridium septicum*)	(False) blackleg			
	(*Clostridium novyi*)	(False) blackleg			
	C novyi type B	Black disease	Damage to liver by migrating fluke.	Sudden death.	Rapid decomposition of carcase with signs of previous fluke infestation.
	C septicum	Malignant oedema	Recent traumatic injury.	Marked pyrexia, dull and toxic.	Affected muscles appear dark red with some emphysema palpable. Surrounding area oedematous. Also identified in cases of abomasitis.
	Clostridium sordellii	Gas gangrene	Changes in diet or management.	Gas gangrene around wounds.	
	Clostridium haemolyticum	Bacillary haemoglobinuria	Higher risk in summer and autumn.	Dark red urine, fresh blood in faeces. Stand with arched back. Develops to recumbency with respiratory distress, then death.	On postmortem, jaundiced carcase with dark red/brown liver.
Neurotrophic clostridia: produce potent neurotoxins	*Clostridium tetani*	Lockjaw	Surgical wounds, navel, site of trauma.	Stiffness and reluctance to move, progresses to bloat. Inability to open jaw, raised tail head and prolapse of third eyelid.	Progresses to recumbency prior to death within three to four days. Often euthanised on welfare grounds.
	Clostridium botulinum	Botulism	Access to decaying matter – that is, contaminated poultry litter.	Often sudden death. Can present as hindlimb weakness, which progresses cranially. Results in recumbency with lack of muscle tone. Protrusion of tongue.	Paralysis of diaphragm leads to respiratory arrest. Often euthanised on welfare grounds.
Enterotoxic clostridia: bacteria commonly found in gut in low numbers	*Clostridium perfringens* type A	Haemorrhagic enteritis	Generally young animals. Sudden dietary change.	Sudden death. Severe diarrhoea.	Haemorrhagic enteritis, ulceration of intestinal mucosa.
	C perfringens type D	Pulpy kidney	Generally young animals. Overeating or dietary change.	Sudden death. May show neurological signs and depression.	Rapid kidney decomposition is not a definitive finding on postmortem.

Table 1. List of *Clostridium* species, predisposing factors and clinical signs.

The clinical syndromes associated with *Clostridium* species occur when uncontrolled growth is present due to favourable conditions, leading to excess production of toxins that, more often than not, are fatal. To multiply, most clostridia require a reduction in oxygen supply, which can often occur at sites of bruising or injury. The subsequent toxin production can lead to death within hours. When conditions are less favourable, the clostridial organisms form protective spores to allow survival in the soil or inside the body.

Diagnosis

Diagnosis of clostridial disease is important as the cases often present as sudden death. Accurate diagnosis is necessary to rule out other diseases, such as anthrax (which is notifiable), and to determine which preventive measures may be useful.

Samples submitted for postmortem examination should be from recently dead or dying animals. Clostridia have a tendency to multiply rapidly in carcases postmortem, which could potentially lead to a diagnosis of clostridia when this was not, in fact, the cause of death. Certain postmortem findings can be very indicative of specific clostridial disease syndromes. **Table 1** lists the range of clostridia causing disease in cattle and the main clinical signs.

Reported cases

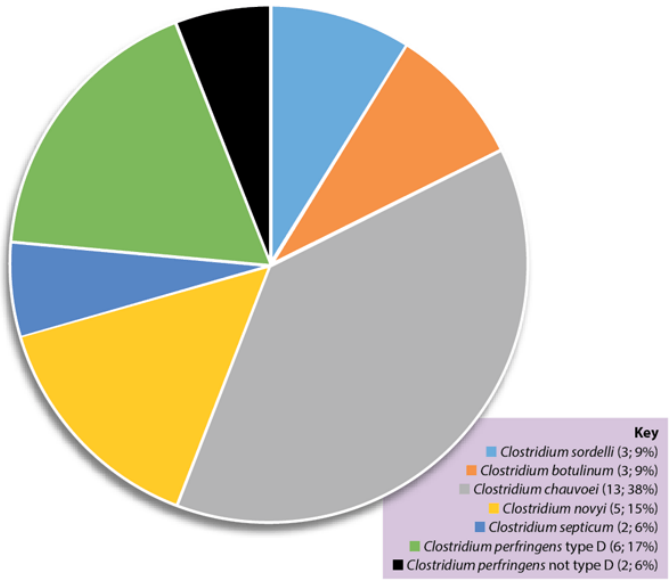

Figure 1. Cases of clostridial disease diagnosed by the APHA services in 2016 (does not include non-carcase submissions; 40 further cases of this type).

Both the APHA and Scotland's Rural College provide full postmortem services for livestock. Once anthrax has been ruled out from a case of sudden death, carcases can be submitted for diagnostics. It is imperative carcases are delivered quickly to prevent excessive decomposition and overgrowth of non-significant organisms. In 2016, both services received submissions where the cause of death was diagnosed as clostridial disease. **Figures 1 and 2** show charts demonstrating the number (and percentage) of clostridial cases seen in cattle in 2016.

Clinical syndromes

Blackleg

Blackleg (gangrenous myositis) is caused by *Clostridium chauvoei*. Clostridial spores will enter through skin wounds or contaminated equipment. These spores can then be activated when the host undergoes muscle trauma. Common causes of trauma are bulling injuries or injuries from feed barriers.

The site of infection may indicate the cause of the trauma and can help improve management practices. Outbreaks have also been reported after disturbance of the ground, causing cattle to be exposed to higher numbers of spores.

Affected cattle are often found dead, but can also demonstrate pyrexia, muscle stiffness and pain. Postmortem examination will reveal swollen or necrotic muscle tissue. Treatment with penicillin may be successful, but only if started in the very early stages of disease.

Black disease

Black disease (infectious necrotic hepatitis) is caused by *Clostridium novyi* type B. In the UK, disease is often secondary to the migration of immature fluke. It is, therefore, more likely to be seen during late summer/early autumn. Clinical signs are not often seen antemortem – more commonly, the cattle are found dead with no warning.

Postmortem examination may reveal necrosis of the liver with signs of fluke migration. Peritoneal and pleural fluids may be dark in colour. Treatment with penicillin is rarely successful, so prevention of disease, including a well-managed fluke control programme, is vital.

Malignant oedema and gas gangrene

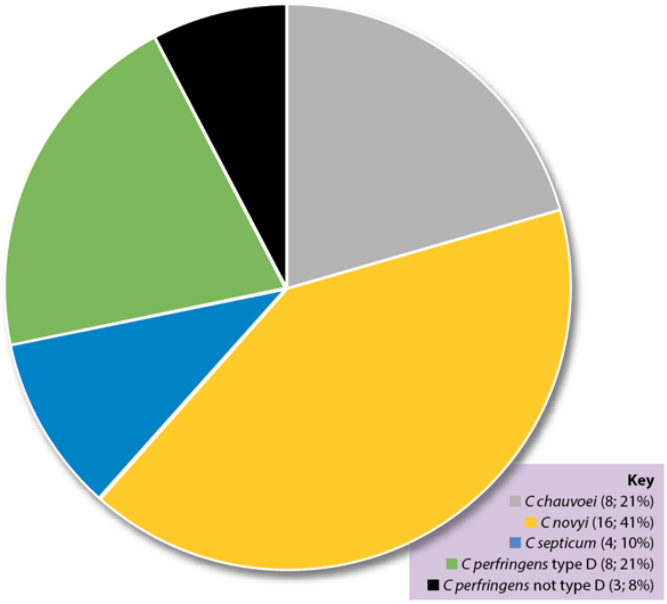

Figure 2. Cases of clostridial disease diagnosed by Scotland's Rural College services in 2016 (not complete).

Malignant oedema and gas gangrene is caused by *Clostridium septicum* (also *C novyi* and *Clostridium sordellii*). It often occurs following IM injections of products that may damage the local

tissues, causing activation of latent spores. But it can also be the result of trauma. Extensive oedema of the affected muscle is observed alongside pain around the site of infection. This will often lead to lameness. Clinical signs rapidly progress to death in 48 hours.

Postmortem examination typically reveals enlarged local lymph nodes, subserosal haemorrhage in the abdominal organs and an excess of serosanguineous fluid in the peritoneal and pleural cavities. Treatment with penicillin is rarely successful.

Bacillary haemoglobinuria

Bacillary haemoglobinuria (redwater disease) is caused by *Clostridium haemolyticum*. Spores are activated by liver damage, leading to toxin release. Toxins then attack red blood cells, leading to signs of anaemia. Changes in the urine to a dark red colour will occur and fresh blood may be visible in faeces. Cattle show a reluctance to move, which may progress to recumbency and death within 24 hours.

Postmortem examination may reveal a jaundiced carcase. Signs of anaemia and petechiae may be present on multiple surfaces. The liver may show signs of fluke migration or areas of necrosis.

Treatment with penicillin is rarely successful, unless treatment begins prior to excessive liver damage.

Tetanus

Tetanus is caused by *Clostridium tetani*. Activation of spores leads to release of potent neurotoxin (tetanospasm toxin). Infection most commonly occurs through wounds associated with rubber ring castration, but can occur following surgical castration or other surgical sites – particularly where conditions are far from optimal.

Following infection, it may take anywhere from 3 to 10 days for clinical signs to occur. These signs include hindlimb stiffness with difficulty walking, standing with limbs abducted, having a startled expression with protrusion of eyes and ears pointed caudally. The animal will be unable to open its jaw and a moderate degree of bloat will be present. Commonly, the infected animal will also demonstrate a raised tail head.

The condition generally worsens over two to five days, progressing to recumbency and seizures (tetanic spasms), and, finally, death, related to respiratory failure.

Postmortem findings are often unremarkable, so diagnosis is based on clinical signs and a history of recent wounds/trauma.

Treatment is rarely successful, but may require placement of a rumen trocar to relieve bloat. A

rumenotomy may be necessary to enable supply of food and water. Tetanus antitoxin may be used, the recommended dose being 50 units/kg IV followed by IM doses twice a day. If available, IV penicillin can be given followed by IM doses twice a day. NSAIDs must be used to provide analgesia. Sedatives such as acepromazine are useful as muscle relaxants and animals should be kept on their own in darkened, bedded sheds to reduce overstimulation and potential further trauma.

Often, tetanus cases do not recover sufficiently and will be euthanised on welfare grounds. The underlying cause of infection should be investigated and management practices improved accordingly.

Idiopathic tetanus

Idiopathic tetanus is due to overgrowth of C tetani normally present in the gastrointestinal tract, leading to the release of sufficient toxin to cause disease. Generally, the main sign is bloat, with other clinical signs less obvious. It may respond better than classical tetanus to supportive treatment.

Botulism

Botulism is caused by *Clostridium botulinum*. The disease is caused by ingestion of pre-formed toxins. Access to decaying vegetable matter and animal carcases is the main source for ingestion. Poultry litter is often regarded as a common source of contamination. Clinical signs can occur from 1 to 14 days following ingestion of toxins. Sudden death is often the first sign, but farmers may report cattle showing hindlimb weakness. Some cases will remain bright and signs will progress no further, with the animals recovering in two to three weeks.

In more severe cases, hindlimb weakness progresses to full body weakness and recumbency. These animals will have difficulty swallowing, with paralysis of the tongue leading to permanent protrusion from the mouth. Due to complete loss of tone in the neck musculature, the head will be pressed against the chest. Paralysis of the diaphragm will result in respiratory arrest.

Postmortem findings are non-specific, although suspect contaminated material may be found in the stomachs. Diagnosis is based on clinical signs, progression of disease and access to contaminated material. No effective treatment exists and animals should be monitored closely for signs of suffering. Unaffected cattle should be removed from those areas where the likely contamination occurred. In most cases, the animal will be euthanised on welfare grounds once generalised paralysis has occurred.

Haemorrhagic enteritis

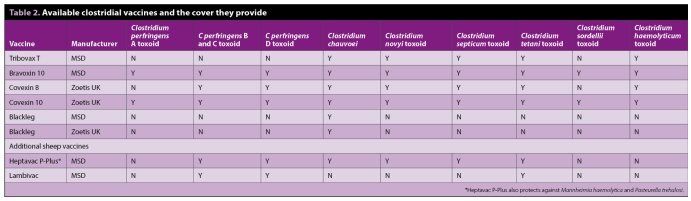

Table 2. **Available clostridial vaccines and the cover they provide**

Vaccine	Manufacturer	*Clostridium perfringens* A toxoid	*C perfringens* B and C toxoid	*C perfringens* D toxoid	*Clostridium chauvoei*	*Clostridium novyi* toxoid	*Clostridium septicum* toxoid	*Clostridium tetani* toxoid	*Clostridium sordellii* toxoid	*Clostridium haemolyticum* toxoid
Tribovax T	MSD	N	N	N	Y	Y	Y	Y	N	Y
Bravoxin 10	MSD	Y	Y	Y	Y	Y	Y	Y	Y	Y
Covexin 8	Zoetis UK	N	Y	Y	Y	Y	Y	Y	N	Y
Covexin 10	Zoetis UK	Y	Y	Y	Y	Y	Y	Y	Y	Y
Blackleg	MSD	N	N	N	Y	N	N	N	N	N
Blackleg	Zoetis UK	N	N	N	Y	N	N	N	N	N
Additional sheep vaccines										
Heptavac P-Plus*	MSD	N	Y	Y	Y	Y	Y	Y	N	N
Lambivac	MSD	N	Y	Y	N	N	N	Y	N	N

*Heptavac P-Plus also protects against *Mannheimia haemolytica* and *Pasteurella trehalosi*.

Table 2. Available clostridial vaccines and the cover they provide.

Haemorrhagic enteritis is caused by *Clostridium perfringens* type A, a commensal of the intestinal tract. Favourable conditions within the gut may allow germination and lead to the release of alpha toxins, causing disease. Infection can cause fatal haemorrhagic enteritis in both calves and adult cattle. Clinical signs may include abdominal distension, colic, depression, anorexia or sudden death.

At postmortem, examination signs of enteritis, abomasitis, haemorrhage and mucosal ulceration may be observed. If diagnosed early, treatment with penicillin and supportive care may improve outcomes in affected calves.

Overeating disease/pulpy kidney

Overeating disease/pulpy kidney is caused by *C perfringens* type D. Bacteria are normal commensals of the intestinal tract. Enterotoxaemia occurs as a result of beta toxin release. It is typically seen as sudden death in rapidly growing, well-fed calves and has been described in cattle of all ages on high grain rations. If found alive, animals may show CNS signs.

Postmortem examination reveals excessive pericardial fluid and glucosuria. It is important to note identifying friable “pulpy” kidneys is an inconsistent finding. Treatment is nearly always impossible due to rapid death. Dietary management may aid prevention.

Control of clostridial disease

Clostridial infection and disease is a risk in all livestock enterprises due to its ubiquitous nature in the environment. It is, therefore, an important consideration for all farmers.

The cost of clostridial disease – particularly due to sudden deaths – can be significant. The key to disease control is to manage the aforementioned risk factors and inciting causes. This may include altering feeding practices, diets or surgical techniques. It is also important farmers are aware of these diseases as, if treatment is to have most chance of success, it must be implemented quickly.

Farms at high risk of clostridial disease may benefit from the implementation of a vaccination strategy. Only five clostridial vaccines are licensed for use in cattle. These, alongside two others licensed in sheep, are shown in **Table 2**. These vaccines protect against a variety of varying

clostridial diseases and toxins, so it is important to select the best one for the problems identified on each farm.

Vaccine developments have led to the introduction of vaccines that provide protection against 10 clostridial strains. These may be particularly useful for producers in high-risk situations and can provide a cost-effective method of reducing losses.