AFRIVET BUSINESS MANAGEMENT

Veld talk

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CLOSTRIDIAL DISEASES OF CATTLE

FEATURES

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The importance of clostridial diseases

Diseases caused by *Clostridium* bacteria are responsible for large numbers of losses in cattle. The bacteria are widespread in nature and in the intestines of animals and people, but not all cause disease. Some are involved in the spoilage of food and the decomposition of carcasses. The clostridia that cause animal diseases occur in the environment and cannot be eradicated. Control must focus on prevention by vaccination as clostridial diseases have a rapid course and seldom allow time for diagnosis and treatment.

Gas gangrene (Quarter evil)

The syndrome known as blackquarter or quarter evil is a gangrenous infection of the muscles of the body. There are a number of different clostridia which can cause the syndrome but *C. chauvoei* is the most common cause. Other clostridia which cause quarter evil are *C. septicum, C. novyi* and *C. sordelli.*
The disease occurs mostly in young cattle of after weaning age until roughly 3 years of age but cases can be seen in older animals. The organisms penetrate the muscles of cattle in the form of spores and remain dormant until the conditions in the muscle become suitable for their germination (it is still unknown what the precipitating factor is). The bacteria germinate and produce toxins which kill off the muscle tissue, and spread throughout the body causing death of the animal.
The large muscles of the hindquarter are usually affected but occasionally the heart, diaphragm and tongue muscles are affected.
Cattle with quarter evil show stiffness and discomfort but the disease is seldom diagnosed at this stage and animals die acutely. Characteristically animals that have died of quarter evil have swollen carcasses due to gas formation and rapid decomposition. On post-mortem the affected muscles are dark red to black when cut, and have a rancid smell in cases of *C. chauvoei* infection.
The diagnosis of quarter evil is confirmed on post mortem by taking bacterial samples of muscle tissue. The most economic and effective control is done by vaccinating with multicomponent clostridial vaccines, such as Covexin 10 which contain all the causative strains.
Acute deaths caused by clostridial gas gangrene can be confused with anthrax cases. If anthrax is suspected, a blood smear must be examined by a vet be-

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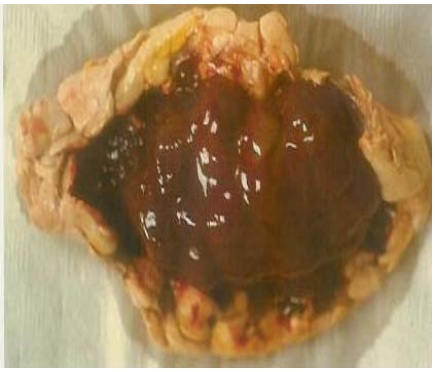
fore the carcass can be opened up. The muscle lesions caused by gas gangrene may also be confused with snakebite wounds.

***Clostridium perfringens* intestinal infections in calves**

Calves on a high level of nutrition can develop gastro-intestinal conditions caused by *C. perfringens* bacteria. A syndrome which is typified by sudden deaths and abomosal ulcers and termed "red gut" has been tentatively associated with *C. perfringens* type A. Although this organism has been included in multiclostridial vaccines, the manufacturers don't claim protection against this syndrome.
Newborn calves can develop necrotic enteritis as a result of *C. perfringens* type C. They show depression, weakness and may develop bloody diarrhoea. On post-mortem examination bleeding and necrosis of the gut may be seen.
***C. perfringens* type D, the cause of pulpy kidney in sheep, can cause an "overeating syndrome" in calves although it is fairly rare. On post mortem the calves show kidney and brain damage. The syndromes caused by types C and D, can be prevented by vaccination with multicomponent clostridial vaccines such as Covexin 10.**

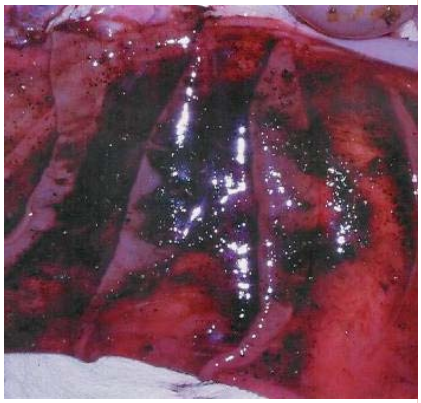
Photo 1: The kidney of a calf that died of "overeating syndrome" caused by C. perfringens D.



***Clostridium perfringens* intestinal infections in adult cattle**

It has been suggested that the "red gut" syndrome seen in feedlot cattle in South Africa is due to a proliferation of *C. perfringens* type A in the intestine. Similar conditions have been described in the USA as "jejunal haemorrhagic syndrome". The disease is acute, characterised by loss of appetite, bloat, haemorrhage in the gut and rapid deaths. Predisposing factors are possibly the high intake of concentrates and low roughage intake. There is no successful treatment for the treatment for the condition. The use of vaccines for this condition is controversial although the organisms are included in certain vaccines. It is suggested that feed management can control the syndrome, namely consistent feeding especially with regard to the quantities, and restricting water intake after feeding.
***C. perfringens* type A is included in some multicomponent vaccines because the organism causes wound infections, the so-called gas gangrene which also occurs in humans.**

Photo 2: "Red gut" may be precipitated by certain feeding practices which cause an overgrowth of C. perfringens A.



Botulism

***C. botulinum* bacteria occur widely in the environment and grow in carcases. The bacteria produce a powerful toxin which is responsible for the clinical symptoms of botulism. Botulism in cattle occurs either in cattle on winter veld (*C.botulinum* type D) or when the feed of animals becomes contaminated by animal carcasses (*C. botulinum* type C).**
During late summer and

winter the phosphate levels of veld grass begin to drop. Cattle develop a phosphate hunger or "pica" which causes them to eat old bones or tortoise shells which can contain botulism toxin. In kraal conditions feed can be contaminated when small animals like rats, birds and cats die and contaminate the feed with botulism toxin. Broiler manure is very commonly contaminated because it almost always contains chicken carcasses.

Once cattle have ingested botulism toxin the symptoms will be seen in 2-6 days. The first signs that are noticed are weakness in the hind limbs, and then paralysis of the throat and jaw. Animals are unable to swallow and saliva streams from the mouth. Because they cannot swallow, attempts to dose animals will cause gangrenous pneumonia as the remedies will end up in the lungs. As the condition progresses, animals are eventually unable to stand and lie down with their heads held against their bodies. Eventually the breathing muscles are affected and the animal dies. A post-mortem will reveal nothing specific and confirmation can only be done by isolating the toxin from the gut.

Treatment with botulism antiserum can be attempted but it is seldom successful except in very early cases. The most economical control measure is vaccination. Supplementation with phosphate licks in winter will reduce eating of bones and will improve the condition of animals.

The symptoms of botulism can be confused with *Diplodea* poisoning (a fungus which grows on mealies), "krimpsiekte" plant poisoning, rabies, and three-day stiff-sickness.

Photo 3: A case of botulism in a bovine

Tetanus

Cattle are not very susceptible to *Clostridium tetani* bacteria, but cases do occur after surgical procedures such as dehorning. *C. tetani* bacteria occur in the faeces of animals and enter wounds very easily under unhygienic conditions. The bacteria grow well in deep wounds, where they produce a powerful toxin which affects the nervous system of the animal. Initially, affected animals show stiffness of the legs, which they hold far from their bodies as if trying to balance. The third eyelid may collapse across the eye, and the animals are constipated and depressed. In the terminal stages they develop spastic (jerky) paralysis. Eventually they die when the respiratory muscles become paralyzed.

Treatment of affected animals with antibiotics is usually too late as the toxin has already been produced. The use of tetanus antiserum is also usually not very effective unless given early in the course of the disease. Since cattle are less susceptible than sheep or horses, they are not usually vaccinated against tetanus but control measures such as stringent wound hygiene must be practiced when doing dehorning, hoof paring and any other surgical interventions. Sterile instruments and wound disinfection must be used. Tetanus in cattle can be confused with laminitis, and certain plant poisonings, and in the late stages with heartwater.

Photo 6: Tetanus in a calf

THE USE OF INACTIVATED VACCINES

Clostridial vaccines are all inactivated vaccines and when used there are certain guidelines that must be followed.

Handling: inactivated vaccines are more stable than live vaccines but nevertheless must be stored in a refrigerator at a temperature between 2 and 8°C, but never frozen. If this happens by accident the vaccine must be discarded. Do not accept vaccines that have been stored on co-op counters or transported without cool packs over long distances. Inactivated vaccines usually contain an adjuvant which settles whenever the bottles have been standing for some time. Before injecting the vaccine ensure the bottle is shaken thoroughly to suspend the adjuvant evenly. Once opened and used, vaccine bottles should be discarded because vaccine can become contaminated during use. Contaminated vaccine is a potential cause of infections and may even become ineffective.

Administration: inactivated vaccines seldom give effective protection after a single inoculation. They must be boosted a month after the initial injection and thereafter require an annual revaccination. Vaccination must be done timeously before the exposure of the animals to disease. Most clostridial vaccines are therefore given at weaning age. Cattle must only be fed chicken manure two weeks after the two initial vaccinations against botulism. Unlike many live vaccines inactivated vaccines will not be affected by the admistration of antibiotics.

Hygiene: as with all vaccines good needle and syringe hygiene are absolutely essential: needles and syringes must be sterilised in boiling water for a minimum of 15 minutes before use. Disinfection with alcohol or other disinfectants is not suitable. Infected needles and syringes can causes abcessation at injection sites and even fatal septicaemias.

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