

## **EQUINE GRASS SICKNESS**

### ***What Causes Grass Sickness?***

Grass sickness has been causing high mortality of horses at grass since the early 1900s. Despite a great deal of work by veterinary surgeons over the years, the definite cause remains unknown but recent research indicates that the highly neurotoxic bacterium *Clostridium botulinum* type C is involved. It is hypothesised that this bacterium is one of many microorganisms that make up a normal component of the gastrointestinal tract of horses. In the healthy horse it is carried harmlessly, being controlled by the local gut immune system. The presence of antibodies both to the type C neurotoxin and the bacterium itself have been detected in the gut, in the systemic circulation of normal healthy animals, and in the colostrum and milk of lactating mares.

Disease is thought to be triggered by a change in nutrition, which is followed by either a vast increase in the number of *C. botulinum* type C organisms and/or there is a massive production of toxin within the gut. The end result of either of these processes is that the horse's immune system is unable to cope. A recent study of 30 horses with EGS and 77 healthy control horses showed that type C neurotoxin could be detected in 74% of horses with acute EGS disease, and 67% of sub acute and chronic cases, often in massive amounts, compared to 10% of controls, where the level was usually just above the limits of detection.

### ***Signs of Grass Sickness***

Grass sickness is a disease of horses, ponies and donkeys in which there is damage to parts of the nervous system which control involuntary functions, producing the main symptom of gut paralysis.

Grass sickness occurs in three main forms; acute, subacute and chronic, but there is considerable overlap in the symptoms seen in the three forms. The major symptoms relate to partial or complete paralysis of the digestive tract from the oesophagus downwards.

In ***acute grass sickness***, the symptoms are severe, appear suddenly and the horse will die or need to be put down for humane reasons within two days of the onset. Severe gut paralysis leads to signs of severe colic including rolling, pawing at the ground and looking at the flanks, difficulty in swallowing and drooling of saliva. The stomach may become grossly distended with foul-smelling fluid which may start to pour down the nose. Further down the gut, constipation occurs. If any dung is passed, the pellets are small, hard and may show a 'cheesy' coating of mucus. Fine muscle tremors and patchy sweating may occur. In this form, the disease is fatal and the horse should be put down once the diagnosis is made.

In **subacute grass sickness**, the symptoms are similar to those of the acute disease but are less severe. Accumulation of fluid in the stomach may not occur but the horse is likely to show difficulty swallowing, mild to moderate colic, sweating, muscle tremors and rapid weight loss. Small amounts of food may still be consumed. Such cases may die or require euthanasia within a week.

In **chronic grass sickness**, the symptoms develop more slowly and only some cases show mild, intermittent colic. The horse's appetite is likely to be reduced and there will be varying degrees of difficulty in swallowing but salivation, accumulation of fluid in the stomach and severe constipation are not a feature. One of the major symptoms is rapid and severe weight loss which may lead to emaciation. Previously, it was thought that nearly all such cases died and that the few which survived made only a partial recovery and were subsequently useless for work. This is now known to be incorrect.

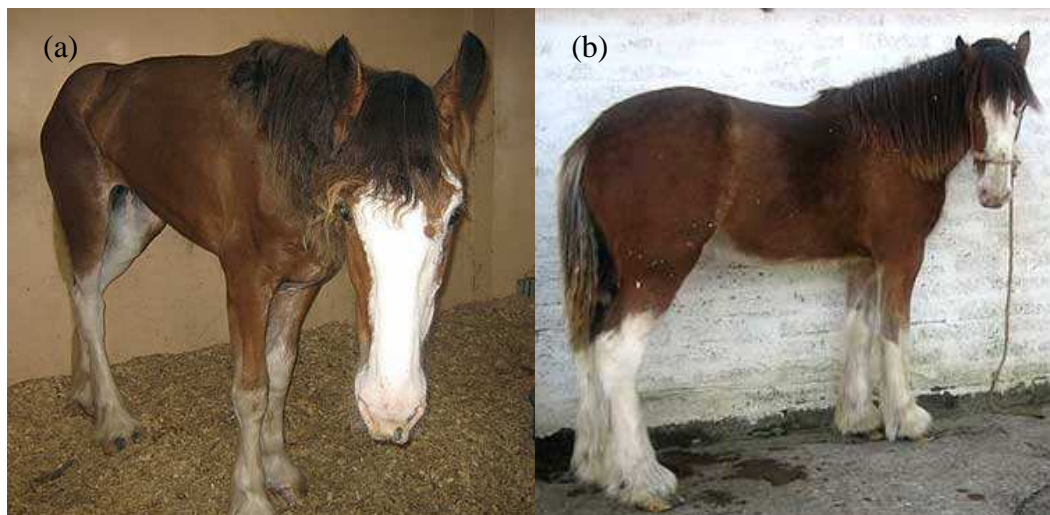


Fig. 1: Clydesdale horse suffering from chronic grass sickness (a) and post recovery (b).

### ***Diagnosis of Grass Sickness***

The symptoms described above may seem quite clear-cut but unfortunately not all affected animals show all these signs and it can sometimes be very difficult for the veterinary surgeon to distinguish grass sickness from other causes of colic, difficulty in swallowing and weight loss. There is no non-invasive test for diagnosing the disease in the live animal although certain diagnostic tests can be helpful, when considered together with the symptoms.

A definite diagnosis can be made only by microscopic examination of nerve tissue after death or by surgical removal of a piece of small intestine by opening the abdomen. Characteristic degenerative changes in the nerve cells can then be demonstrated in the tissues.

### ***Treatment of Grass Sickness Cases***

As previously stated, treatment should not be considered in acute and subacute cases. However, in chronic cases, if the animals are not in much pain, can still eat at least a small amount and are still interested in life, treatment can be attempted. The correct selection of potentially treatable cases using these criteria is essential and requires experience as not all chronic cases are treatable.

Treatment of chronic cases involves dedicated nursing and the provision of palatable, easily swallowed food e.g. chopped vegetables, grass and high energy concentrates soaked in molasses. It is essential that high energy foods are consumed as chronic cases fed roughages and succulents alone will invariably die. Nursing is vital and provides the mainstay of management. The patients require constant stimulation by human contact, frequent grooming to prevent them becoming scurfy and sticky with sweat and, in some cases, rugging which has been found to reduce sweating and prevent hypothermia.

The recent recovery rate for carefully selected cases at the Dick Vet Equine Hospital, Easter Bush, is now approximately 70%. Contrary to commonly held views, a follow-up study has shown that 41% of these recovered cases were back to work including hunting, racing, eventing, 33% were being hacked, preparing for competitive work or being used for breeding and the other 26% (the more recent cases) were still gaining weight and recovering at the time of the survey. None of the survivors were described as being of no use. This represents a major improvement in the prognosis for such cases compared with the situation before the late 1980s.

### ***Potential management practices which may reduce the risk of grass sickness***

Several studies have identified certain “risk factors” associated with an increased incidence of grass sickness and certain “protective factors” associated with a decreased incidence of grass sickness. These can be sub-divided into:

1. Horse-related factors
2. Premises-related factors
3. Management-related factors

Therefore following the occurrence of a case (or several cases), it may be worth considering which of these factors can be manipulated with a view to reducing the risk of the disease occurring in other horses. This may involve the implementation of “protective” factors and/or the avoidance of “risk” factors. Unfortunately, out of all the recognised “protective” and “risk” factors, there are only a few which can be manipulated by altering the day-to-day management of the “at-risk” horses.

### **Risk avoidance**

- Minimise exposure to pastures where previous cases have occurred
- Minimise any pasture/soil disturbance (e.g. harrowing/mechanical faeces removal/pipe-laying/construction work etc.)
- Minimise soil exposure (e.g. close grazing/poaching of fields)
- Avoid any sudden changes in diet (quantity and/or feed type)

- Avoid the “over-use” of ivermectin-based wormers (e.g. rotate with other classes of wormers)

If circumstances dictate that the above changes have to be prioritised, then they should be prioritised towards stock which possess the greatest “horse-related risk factors” (young adults, new arrivals, horses in “show condition”) and at “peak” seasonal (spring and early summer) and climatic (cool/dry weather) periods.

#### **Implementation of potential protective factors**

- Co-graze with ruminants
- Regular grass cutting on pastures
- Hand removal of droppings
- Supplementary forage feeding (hay/haylage)

More information can be found at <http://www.grassickness.org.uk>