All tied up

We may be yet to pinpoint exactly why horses suffer from azoturia, but as vet Linda Belton MRCVS says, we can do much to prevent these painful attacks.

Azoturia is a condition that affects the muscles of the horse’s hindquarters and back. During an attack, these muscles become inflamed, resulting in pain and stiffness.

Over the years, azoturia has also been known by a variety of other names, such as ‘set fast’ or ‘Monday morning disease’ – a name that comes from the fact that many working horses that had had Sunday off were then affected by azoturia on returning to work on the Monday morning. We still see this condition occurring in horses that are fed a high-energy diet even when rested from their normal exercise regime.

More technically, azoturia is known as rhabdomyolysis. This is a term used to describe the damage occurring to the horse’s muscle fibres, which results in the release of a pigment, known as myoglobin, from the affected muscles. It is this pigment that produces urine discolouration in horses with azoturia. The urine becomes brown, or may even turn toward red depending on the severity of the case.

Most cases of rhabdomyolysis relate to exercise and thus are termed exertional rhabdomyolysis (ER). There is also the recognised condition of recurrent ER (RER), which we will discuss later.

What causes the problem?
Why horses get azoturia is not fully understood. The classically described situation, of a horse fed a high-cereal diet when not in work, is a common cause. Other cases are seen in horses in regular work and obviously, not every horse suffers from azoturia following rest.

Some individuals are highly susceptible; notably young fillies, where a reduction in exercise for even a day can lead to an attack. There is some evidence that their hormonal cycles may be a contributory factor.

Other causes include electrolyte imbalances in the body. The most common of these involve sodium chloride (salt) and calcium. Dietary supplementation in the form of salt and calcium carbonate may be helpful. Electrolyte levels are assessed by examination of paired blood and urine samples, but this cannot be carried out within two weeks of an episode of azoturia.

Showing the signs
The clinical signs of azoturia depend on the severity of the attack. Mild cases appear stiff behind after exercise. In more severe cases, the horse may be reluctant to move and may show signs of pain that can be confused with colic. The horse may sweat and paw the ground and, in severe cases, may be unable to move or even collapse.

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Episodes of azoturia may occur at exercise or post-exercise. If you suspect your horse is tying up, stop exercise at once. An affected horse will feel harder to keep moving. Very early signs may just involve a change in action and should not be ignored.

If the horse can walk, lead him back to his stable. Do not make him walk if he is reluctant to do so, as this may worsen the muscle damage. It may be necessary to get a horsebox to transport him home. Site the box carefully to reduce the incline of the ramp for both loading and unloading.

Exercise-related attacks
Recurrent exertional rhabdomyolysis (RER) can have a variety of underlying causes, some of which are, as yet, unknown. The investigation of RER involves muscle biopsies, as well as electrolyte testing, as examination of the sampled muscle tissue can help in determining an underlying cause of the problem.

Muscle biopsies can be taken in the standing, sedated horse, under local anaesthetic. The site of the biopsy will depend on which conditions the horse is being tested for. A common site is the semimembranosus muscle, the large muscle that runs from the top of the horse’s hindleg to the side of the tail. It may be necessary to take more than one biopsy.
The PSSM problem

One of the possible underlying conditions in horses that are suffering from RER is polysaccharide storage myopathy (PSSM). Affected horses have sporadic episodes of azoturia, and the condition is often triggered by rest for a few days prior to exercise.

Unlike other forms of azoturia, subclinical episodes – that is, those with no overt signs – occur and thus these horses may have persistently elevated CK enzyme levels. A definitive diagnosis of PSSM depends on particular laboratory findings of the biopsied muscle.

PSSM is a condition affecting the storage of glycogen, the chief storage substance for carbohydrates (sugars), within the body. Horses affected with PSSM tend to oversynthesise glycogen. When such horses are fed a high-starch meal, they store a higher proportion of the absorbed glucose in their muscles than normal horses. Why this causes cells to become damaged with exercise is unclear at present.

PSSM is most commonly found in horses related to the warmblood, Quarter Horse and draught horse breeds, although other breeds and crossbreeds can be affected. Caring for horses with PSSM depends on changes to their feeding and management.

Regular daily exercise and as much turnout as possible are important, while minimising stress to the horse and sticking to a routine are also beneficial.

Horses with PSSM require low-starch, high-fibre diets. Many are good doers, and good-quality hay with vitamin and mineral supplements will form the basis of their diets. If more energy is required, it is best provided in the form of fat, such as soya oil.

A high-fat diet decreases blood glucose and insulin concentrations, as well as insulin-sensitive glucose absorption. This may be the mechanism by which a high-fat diet reduces the muscle glycogen concentration, which then reduces muscle damage. Even a small amount of supplementary fat can have considerable benefit. There are some commercially available feeds that are suitable.

It is not always possible for horses with PSSM to perform well at high exercise levels, but many lead enjoyable working lives.

The treatment required depends on the severity of the azoturia attack. Moderate to severe cases require prompt veterinary attention. Initial treatment involves the use of anti-inflammatory agents to reduce pain and try to prevent further release of the damaging muscle pigments that cause myoglobinuria.

Myoglobin can cause damage to the kidneys and more severe cases require intravenous fluid therapy – especially if the horse is dehydrated due to exercise. Some severe cases may require more intensive supportive therapy if, for example, the horse has collapsed.

The vet will usually take blood samples from an affected horse to measure the level of muscle enzymes present in the blood. Damage to muscles results in an increase in the level of creatine kinase (CK) and aspartate aminotransferase (AST). Not only will an elevation in these enzymes confirm the diagnosis, but it can also give a guide to the severity of the muscle damage and thus the likely recovery time.

Further blood samples are used to monitor recovery and determine when the horse is ready to return to work. Many horses will look normal with a few days’ rest after an episode of azoturia. However, their muscle enzyme levels often remain elevated and, if put back into work at this time, will be more likely to suffer a recurrence with further muscle damage.

Returning to action

Once the muscle enzyme levels have returned to normal, a gradual return to work is recommended, on a low-energy diet. Initial blood samples, if taken quickly after the onset of an attack, may not give the maximum muscle enzyme level that the horse will reach, and this should be borne in mind when comparing subsequent results.

Exercise testing can be useful when a susceptible horse returns to work. Preventing muscle damage is the aim. Blood samples for muscle enzyme levels are taken before exercise and again, two to six hours after its completion. The initial muscle enzyme levels should be normal – if the levels more than double in response to the exercise, then this is an early warning sign of problems with exercise-related muscle damage.

Some horses, despite regular exercise, develop a tendency to recurrent episodes of azoturia, a condition known as RER (see ‘Exercise-related attacks’, page 81).

The calcium factor

Other horses with RER may have abnormalities in their calcium regulation. When a muscle contracts, calcium is released from muscle storage sites and then taken back up into these for relaxation. The threshold level of calcium release that leads to muscle contracture is lower in some horses with RER, suggesting that abnormal intracellular calcium regulation is the cause of this form of RER.

These concentrations of calcium are very small compared to the amount of calcium in the rest of the horse’s body and are independent of the dietary calcium level. This threshold is similar to a human muscle disease known as malignant hyperthermia.

Malignant hyperthermia can be prevented in people with a drug called dantrolene, which decreases the release of calcium and is useful in preventing muscle damage in horses that have this form of RER. However, it is unlicensed for use in horses and long-term treatment is expensive.

There is much debate about the genetic component of all these muscle diseases and some may be hereditary, although there is, as yet, limited proof. Think before breeding from a mare with any such disease. Just because she cannot perform does not make her an ideal broodmare candidate!