## KEY POINTS

- This disease of young foals is characterised by a foal that is normal at birth rapidly becoming depressed, collapsed and jaundiced
- Caused by antibodies passed from a mare to a foal in colostrum that attack and destroy the foal's red blood cells
- Treatment of clinical disease includes close monitoring and blood transfusion
- Prevention relies on preventing ingestion of mare's colostrum in at risk foals

## THE DISEASE

Neonatal isoerythrolysis (NI) is caused by an incompatibility of blood types between and a mare and her foal, in which the foal's red blood cells (RBC) are destroyed by antibodies ingested from the mare's colostrum. In order for NI to occur, certain events must have taken place previously:

- The foal must have inherited a red blood cell antigen from its sire that is not possessed by the mare (i.e. it is 'foreign' to the mare).
- The mare must have been previously exposed to blood containing the foreign antigen - this usually occurs at a previous foaling, but may occur due to a blood transfusion or due to placental leakage (antigen will not cross a normal placenta).
- The mare must then have developed antibodies against the foreign RBC antigen.

After the foal is born, the mare naturally passes these harmful antibodies (along with many others that are benefcial) to the foal in the colostrum – when the foal ingests and absorbs the antibodies they bind to the foal's RBC and destroy them. The foal's gastrointestinal tract will only absorb antibodies for 24 hours – after this, the gut "closes" and no further antibodies are absorbed.

## EQUINE BLOOD GROUPS

There are 8 major blood groups in horses (A, C, D, K, P, Q, T and U) with 32 distinct red blood cell antigens. The majority of NI cases involve the antigens Aa and Qa - mares that are Aa and Qa negative are at a higher risk of producing a foal with NI. The prevalence of NI in Thoroughbreds is 1-2%.

## CLINICAL SIGNS

Antibodies cannot cross the placenta, meaning foals will be unaffected during pregnancy and will appear normal at birth. Signs can develop from 5 hours to 5 days post-foaling. The severity of the clinical signs depends on the severity of the anaemia, which in turn depends on the amount and quality of colostrum ingested.

Cases with a mild anaemia may show signs of lethargy, jaundice, an increased respiratory rate and an increased heart rate. Acute cases with a more severe anaemia may progress to become depressed, anorexic, dehydrated and pyrexic. Peracute cases progress rapidly – cardiovascular collapse, neurological disorders and multi-organ failure can cause death within hours.

## DIAGNOSIS

A tentative diagnosis of NI can be made for any young foal with lethargy, jaundice and anaemia (PCV <15%). The breakdown of RBC also discolours the plasma and sometimes the urine. A definitive diagnosis is made by cross-matching the mare's serum with red blood cells from the foal- agglutination indicates the presence of antibodies against the foal's red blood cells in the mare's serum or colostrum. A Coombs test can also be used to look for the presence of antibodies bound to the foal's red blood cells.

## TREATMENT AND MANAGEMENT

In mare's which have no history of producing a foal with NI the disease is usually recognised too late to prevent any further absorption of colostral antibodies. The peak of colostral antibody absorption occurs within the first 6 hours after foaling and the gut is effectively 'closed' by 24 hours.

Foals which are recognised to have clinical disease should be monitored closely. Their packed cell volume and clinical status should be monitored closely. There are no absolute guidelines to predict the need for a blood transfusion but the severity of anaemia and the speed in decline of PCV can be useful. In general the younger the foal at the time of detection the more concerning the disease is as it usually indicates greater absorption of antibodies. In foals which are showing signs of clinical deterioration and have a low PCV (15% often used as a guideline) blood transfusion is indicated. Measuring blood lactate can also be useful to assess when the degree of anaemia is affecting tissue perfusion. Blood transfusion is lifesaving in clinically affected foals but is not without risks (transfusion reaction, liver injury from iron overload etc). Foals with a mild anaemia (> 15%) and relatively limited clinical signs may be managed simply by preventing further suckling and limiting stress and exertion.

To provide a blood transfusion washed red blood cells can be given from the mare. "Washing" removes antibodies and the mare's red blood cells will be compatible with the foals. If this is not possible then a cross matched donor should be used. If cross matching is not possible a healthy young gelding would usually be considered the best choice.

Foals with NI may also need additional supportive care. Iron-binding drugs are also sometimes used to combat the effects of the RBC destruction that releases lots of iron.

## PREVENTION AND CONTROL

In the past, NI could be prevented by blood-typing mares prior to breeding, or shortly before foaling. Only mares that lack Aa and Qa antigen can form antibodies against it. These mares should ideally be matched to stallions that are also negative. Some breeds have a high prevalence of Aa and Qa antigens (e.g. Thoroughbreds) meaning a suitable stallion may be difficult to find. However, with the advent of DNA- based parentage testing, there are no longer any labs in UK which offer blood typing.

The haemolytic antibody test can be used as a screening test.  This tests the pregnant dam's blood against a panel of 10 other horses. If this is positive it suggests there is as increased likelihood of the mare producing a foal with an incompatible blood group.

After foaling the jaundiced foal agglutination test can be performed. This must be performed before the foal is allowed to nurse. This combines the mare's colostrum and foal's red blood cells and looks for agglutination. A dilution of 1:16 or greater suggests that there is a significant risk of the foal developing NI if allowed to consume the mare's colostrum. A cross match can also be performed between the mare's serum and foal's red blood cells to look for agglutination. If this is positive it again suggests an increased risk of NI.

Foals which have to be prevented from nursing the mare should be provided with an alternative source of colostrum and then milk. It is usually safe to allow these foals to nurse from the mare by 24 hours of age. These foals should be monitored closely and be screened to ensure adequate passive transfer of immunity. Careful records should be kept because mare's which have produced a foal with NI are likely to produce future foals with the condition.