

Neonatal Isoerythrolysis

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 includes preventing foal from sucking and blood transfusions when needed
- Preventable by blood-typing mares before breeding

THE DISEASE

Neonatal isoerythrolysis (NI) is caused by an incompatibility of blood types between 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 beneficial) 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 pyrexemic. Peracute cases progress rapidly – cardiovascular collapse, neurological disorders and multi-organ failure can cause death within hours.



Fig 1. Jaundice of the oral mucous membranes in a NI foal.



Fig 2. Jaundiced sclera ('white' of the eye).

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 red blood cells from the dam and the foal – a positive result would demonstrate maternal antibodies binding to the foal's red blood cells.



Fig 3 As the blood sample begins to settle, you can see that the plasma is a bright red colour due to the presence of RBC break-down products.

Fig 4 The urine is dark brown because the kidney has filtered out the RBC break-down products.

TREATMENT AND MANAGEMENT

Foals suffering from NI must be rapidly identified and prevented from sucking for 48-72 hours. After this time, the mare will stop producing colostrum and the foal's gastrointestinal tract will no longer be able to absorb antibodies. The foal should be given an alternative source of nutrition (milk replacer or milk from another mare).

A blood sample is taken to assess the foal's packed cell volume (PCV, an indicator of red blood cell count). Foals with low PCV (less than 12%) are given a blood transfusion – this may be washed red blood cells from the mare, or blood from a donor horse that has been matched to the foal. Washed mare cells are preferable because the red blood cells from the transfusion should not be affected by the antibodies ingested from the mare's colostrum. "Washing" removes any of the harmful antibodies the mare may have in her plasma. Ideally, if other horses are to be used, cross matching is used to check the foal will not react to the transfusion. If facilities for cross matching are not available, the donor horse is generally a gelding as one can rarely be sure a donor mare is not NI-prone.

Foals with a mild anaemia (> 15%) and relatively limited clinical signs may be managed simply by preventing further suckling and limiting stress and exertion. Because the foal is comprised, broad-spectrum antibiotics should be given. Additional supportive care may be needed, such as intravenous fluids, oxygen therapy and nursing care. Iron-binding drugs are also sometimes used to combat the effects of the RBC destruction that releases lots of iron.



Fig 5 Blood transfusion may be necessary in severe cases.

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. There is a screening test that gives a "rough-guide" whereby the pregnant dam's blood is tested against a panel of 10 other horses, but this test should not be relied on totally. A test can be performed immediately post-foaling (before the foal sucks). If positive, the foal should not be allowed to suck for 48-72 hours. It should be given colostrum or plasma from another source to ensure adequate transfer of immunity. Careful record keeping about past problems in young foals and risk factors such as blood transfusion is most reliable. Once a mare has had one NI foal, she is likely to have more, and typically each year, the foal is more severely affected.