

Joint ill in young foals in focus

The term 'joint ill' refers to bacterial infection affecting one or more joints – sometimes involving the adjacent bone – and is a relatively common and potentially fatal condition in young foals. For the best outcome, prompt diagnosis and aggressive treatment are essential, and so this is one problem that all owners and breeders must be on the lookout for during the first weeks of a foal's life.

Bacteria can enter the body in a number of ways: via the umbilicus (navel) at or soon after birth; via the respiratory or gastro-intestinal tract from the environment, dam or other animal; or via a wound. For example, some young foals may develop an abscess in the remnants of the umbilical cord (urachal abscess) as a result of direct contamination, which may then act as a source of bacteria for the development of joint ill. Additionally, infection of a wound sustained in those early days of life could also potentially act as a source of infection for the development of joint ill, so it is important that neither should be ignored.

The bacteria involved vary from 'normal' skin or environmental contaminants to specific pathogens such as Salmonella spp.

No matter how they enter the body – and the route might not be obvious – the bacteria are taken up by the circulation and spread around the body in the blood (bacteraemia).

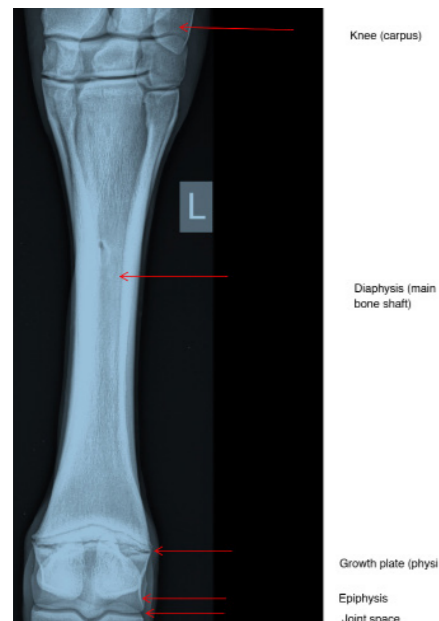


Figure 1 Normal cannon and fetlock of foal



Foals are most susceptible to joint ill during the first 21 days of life

This alone might make the foal very ill (septicaemia) and is, in itself, a life-threatening condition. However, in many cases, this bacteraemic phase does not cause illness and goes unnoticed, but the bacteria can localise in or adjacent to one or more of the foal's joint(s), to later manifest as joint ill. The 'seeding' of this infection usually occurs in the growth plate (physis), the adjacent bone (diaphysis or epiphysis, Fig 1), in the bone near the joint surface or in the membrane lining the joint (synovium). It is believed that these sites are predisposed to involvement because of their rich but complicated blood supply and the slowing of blood flow through these regions.

What are the signs to look out for?

Once they settle in the joint tissues, the bacteria multiply and cause cell death and inflammation, triggering the release of more inflammatory products such as prostaglandins and cytokines. While these inflammatory products are needed to help fight the infection, some of them, along with toxins released by the bacteria and chemicals released by killed cells, are also responsible for extending and worsening the damage

to the joint and adjacent structures. This damage and inflammation normally results in a degree of lameness, with swelling (Fig 2), heat and pain in the affected region(s). The lameness is usually sudden in onset, leading many to believe that the cause is traumatic rather than infectious.

Joint ill is usually seen within the first 21 days of life, although it can be seen in slightly older foals. If you have a foal which becomes lame, you should call your vet immediately to either rule out or confirm joint infection, as severe damage to the cartilage can occur in as little as 48 hours. Certain joints appear to be more commonly affected than others, with fetlocks, stifles and hocks being among the most frequently involved.

Predisposing factors

There are a few predisposing factors, but the most important one is partial or complete failure of passive transfer. All foals are born without an effective immune system or circulating antibodies to fight infection. As such, they rely on being able to ingest the first milk – colostrum – within a few hours of birth from their dam. Colostrum contains concentrated antibodies from the mare's circulation



Figure 2 Fetlock x-ray showing joint swelling (red arrow)

that are absorbed from the foal's intestine without being digested, as long as they are ingested within 12 to 18 hours of birth. These ingested antibodies provide the foal with some protection against infection until their own immune system matures, however some foals do not attain adequate circulating levels of antibody. This might be for a number of reasons, including:

- They could not/did not ingest enough colostrum early enough (the gut 'closes' to antibodies at around 12 hours of age and any colostrum ingested after this time will be digested like any other protein)
- The mare did not produce adequate colostrum, either in volume or quality
- The antibodies the foal did ingest were 'mopped up' by a pre-existing infection
- The foal could not absorb the antibodies

Other factors

Other predisposing factors include a dirty, contaminated or unhygienic environment; poor hygiene around the time of foaling such as using dirty hands, bandages or other equipment; illness, such as placentitis in the mare; physical weakness or disability in the foal; other illness such as infectious diarrhoea.

Diagnosis

Diagnosis is based on clinical signs, laboratory tests and imaging. Clinical signs include acute onset of lameness which might be mild or severe (or in between). There might be obvious heat and swelling of the affected area(s) and/or pain on manipulation and flexion of the affected joint. If the joint

Test Haem + (#3748) HBINFC	
Sample Serum, EDTA, Heparin, Glucose, Citrate	
Sample Quality	Suitable for testing
Urea	mmol/l
Ref Range Equine Thoroughbred Foal	Urea Nitrogen 110 µmol/l [97-138]
Total Protein	48 g/l [42-66]
SAA	1,669.7 mg/l [0-5.4]
Albumin	24 g/l [26-37]
Bile Acids	µmol/l
Globulin	24 g/l [15-33]
Plasma Fibrin	7.5 g/l [1.5-4.2]
Calcium	mmol/l
Phosphate	mmol/l

Figure 3 Inflammatory markers increase in joint ill

itself is infected, there will be effusion (increased fluid) in the joint evident as bulging of the joint capsule. There might be swelling of the adjacent tissues. The foal will usually have an elevated temperature and might also appear depressed and inappetent, although these symptoms are

inconsistent. These signs are not always obvious early in the condition and careful and/or repeated examination might be important.

Laboratory tests can be very useful when trying to differentiate infectious causes of lameness from others. Infection will usually result in elevated white cell numbers and inflammatory markers such as SAA and fibrinogen (Fig 3). Again, serial tests might be necessary. Once a joint is suspected of involvement, taking a sample of joint fluid using aseptic (sterile) technique will give specific information about any inflammation or infection in that joint. Infection causes elevation in white cell count and protein levels in the joint fluid. This might not be of great use if the infection is adjacent to but not within the joint.



Figure 4 Infection in bone of hock. The red arrow shows the area of bone damage

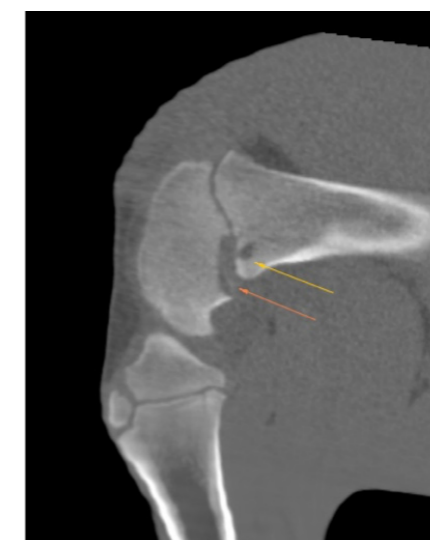


Figure 5 CT image of growth plate abscess (arrows) distal femur (stifle joint)

Imaging techniques which are of benefit include radiography (X-ray, Fig 4), magnetic resonance imaging (MRI), computed tomography (CT, Fig 5) and ultrasound scanning. In most cases, radiography will show lesions in the affected bone, but in early stages of infection these lesions might be very subtle and difficult to interpret, particularly if the growth plate (physis) is involved.

CT is particularly useful in foals, although a general anaesthetic is usually required. Unfortunately, as ultrasound cannot penetrate bone, it can be used only to visualise the outer layers of those structures which can be accessed via the skin. However, it is particularly useful to assess joint effusion and bone and cartilage surfaces (where accessible) and provide guidance for joint fluid sampling.

In virtually all cases, a combination of at least two of these is required for diagnosis and monitoring progress of the condition and for providing evidence for prognosis.

» Classification of joint infections

It is quite usual to attempt to classify the 'type' of joint infection based on the tissues/structures affected.

P-type: affects the growth plate (physis) or adjacent bone

I-Type: affects the physis or the joint, having spread from a peri-articular or subcutaneous abscess. This type is usually seen in the hip or stifle.

S-type: This is infection of the membrane lining the joint

E-type: Epiphyseal (bone ends) infection – often in slightly older foals and might be associated with systemic illness

T-type: This is when the cuboidal bones (the bones of the knee or hock) are involved. These might collapse if weakened by infection

Treatment

Treatment must be aggressive and must directly target those tissues involved. Systemic antibiotics will be administered and must be able to penetrate bone and/or joint fluid as well as being effective against the bacteria causing the infection. The 'gold standard' is to attempt to culture bacteria from the joint.

In practice, we would take a joint fluid sample as part of the diagnostic process and send that to the laboratory for evaluation (i.e. white cell count and protein levels), as well as for bacterial culture and testing for susceptibility to specific antibiotics. Unfortunately, culture results can take several days and treatment usually must be started

immediately after diagnosis has been made. For this reason specific broad spectrum or a combination of antibiotics are usually given along with anti-inflammatory analgesics and anti-ulcer medication.

If a joint is involved it must be flushed through with a large volume of sterile fluid (joint lavage) after careful aseptic skin preparation. This might need to be repeated on several occasions. In more complicated cases or joints or such as the hock and stifle, joint lavage is often performed using arthroscopic surgery so that damaged cartilage and bone can be removed before the joint is flushed through with a large volume of fluid. Antibiotics are instilled into the joint after flushing.

Intravenous regional perfusion with antibiotics (Fig 6) is also used to treat infections of the physis or epiphysis, particularly where there is no communication with the joint. This involves temporarily applying a tourniquet above the region to be treated and inserting a catheter into a vein. Antibiotics are injected via the catheter and remain in the targeted tissues because the tourniquet stops them from entering the systemic circulation.

Surgical debridement (removal of damaged tissue), wherever it might be, might be undertaken as this damaged tissue might act as a site for further multiplication of bacteria.

Box rest is essential to help prevent further damage caused by load bearing on damaged bone. Additional supportive treatment might include intravenous fluids or plasma and anti-oxidants.

Success of treatment is usually monitored using serial joint fluid samples, assessment of lameness and radiographic reassessment. Treatment can be prolonged and become very expensive



Figure 6 Intravenous regional perfusion

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and decisions may need to be made on a number of occasions as to whether to continue treatment and which treatment option to use. Repeated surgery and/or joint flushing might be required and unfortunately some cases cannot be 'cured', either because the damage is too severe or the infection cannot be controlled. In these cases euthanasia is the only option.

If left untreated joint infection results in untreatable and irreversible damage to the affected joint(s) and permanent, incurable lameness. The infection might also spread to adjacent tissues or burst through the skin. If treatment is not possible for financial or other reasons, the foal should be euthanised on humane grounds.

Foals that are successfully treated usually manage to regain full soundness and are able to be treated normally after a variable period of recuperation, during which they will have to have restricted exercise.