NEONATAL SEPTICAEMIA IN FOALS

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NIAMH COLLINS explains the importance of early recognition and management of this potentially fatal condition

NEONATAL septicaemia is a serious and often fatal disease affecting foals. It is initiated by pathogenic micro-organisms, or their associated toxins, and results in a systemic inflammatory response syndrome that can cause disorders in multiple organ systems.

Bacteria are the most common initiators of septicaemia in neonatal foals. As early recognition and appropriate therapeutic interventions are critical to the successful management of these cases, this article presents an overview of predisposing factors, clinical signs, diagnostic tests and appropriate treatment.

Predisposing factors

• Maternal factors: maternal illness (such as colic), placentitis, premature placental separation, running milk prior to parturition, poor lactation or dystocia.

• Abnormal gestation length: prematurity and dysmaturity.

• Other factors: failure of passive transfer, poor hygiene, overcrowding or poor ventilation.

Clinical signs

Clinical signs of postnatally acquired infections usually begin within 48 to 96 hours of age. Infections resulting from *Actinobacillus equuli* may become apparent as early as 18 to 24 hours of life. Signs associated with in-utero acquired infections usually exist at birth or appear within the first 24 hours of life.

The clinical signs of early sepsis in the neonatal foal are often subtle but rapidly progressive. Depression, weakness, poor feeding, lethargy and excessive periods in recumbency (Figure 1) may be reported.

The foal may have milk staining on its forehead, as depressed foals often stand under the udder but fail to suckle properly. As time progresses, the lack of intake will result in dehydration and hypoglycaemia. Mucous membranes are often bright or injected and capillary refill time may be rapid. Other common findings are tachycardia and tachypnoea. Body temperature abnormalities are variable in neonatal septicaemia; hyperthermia, hypothermia or normal rectal temperature may be present. Petechiation of the pinnae of the ears, sclera, vulva or buccal mucous membranes suggests septicaemia and/or disseminated intravascular coagulation.

If untreated, these signs may progress to septic shock, in which the deterioration in cardiovascular function becomes obvious and can manifest clinically by cyanosis (Figure 2), muddy mucous membranes, severe tachycardia, weak peripheral pulses, cold extremities and death.

Other localising signs may be apparent, such as palpable joint distensions or lameness in association with pain and/or oedema over a physis. The joint distensions may or may not be associated with lameness and, notably, the absence of lameness does not rule out septic arthritis.

Diarrhoea or enteritis is also a common early presenting sign. Other gastrointestinal signs associated with sepsis include ileus, abdominal distension (Figure 3) and colic. The external umbilicus can be normal on palpation, but ultrasonographic examination can demonstrate infection of the internal umbilical remnants. Uveitis, seizures and respiratory distress may also be seen.

Laboratory findings

Leukopaenia and neutropaenia are common in acute sepsis. Additionally, a degenerative left shift is frequently present and cytological examination of a blood smear may show evidence of toxicity in the neutrophils such as Döhle bodies, toxic granulation and vacuolisation. Hyperfibrinogenaemia, azotaemia, hyperlactaemia and hypoglycaemia are also common.

Clotting times may be prolonged. IgG concentrations are often low. Arterial blood gas analyses may show acidaemia and hypoxaemia. Positive blood cultures are definitive proof of bacteraemia, but at least 48 hours is usually required before results are available. Blood cultures are taken from a large vein (such as the jugular, cephalic or saphenous) following surgical clipping and aseptic preparation; 5-10ml of blood (without added anti-coagulant) should be placed in an appropriate

blood culture bottle and submitted to the laboratory for culture (aerobic and anaerobic) and sensitivity.

Not every foal with confirmed sepsis will have positive blood cultures and treatment should not be delayed pending culture results. The failure to obtain positive blood cultures in foals that are later confirmed to have neonatal septicaemia is thought to be due to a number of potential factors, including prior antibiotic therapy and low numbers of bacteria in the peripheral blood.

Use of the “sepsis score” had been recommended to predict the likelihood of sepsis in neonatal foals prior to obtaining blood culture results (Brewer and Koterba, 1988). This scoring system is based on historical, clinical and laboratory variables and uses a score greater than 11 as a predictor of sepsis in foals less than 13 days of age. Some authors have expressed concern about the sensitivity and specificity of this scoring system (Stewart et al, 2002; Corley and Furr, 2003).

Diagnostic investigation

Ultrasonography of the internal umbilical remnants may show infection of the umbilical vein (Figure 4) – either of the two umbilical arteries or an urachal infection. About 21 per cent of septicaemic foals have a patent urachus without involvement of other umbilical structures; however, this usually responds to medical management with or without topical treatment. Umbilical infections may resolve with long-term antibiotic therapy or may require surgical resection.

Thoracic radiography and ultrasonography may confirm the presence of pneumonia (Figure 5). The pneumonia in septic foals may be due to haematogenous spread secondary to sepsis, or aspiration pneumonia secondary to generalised weakness and reduced sucking ability. Clinical examination may show increased respiratory rate and effort, abnormal lung sounds and increased rectal temperature. Arterial blood gas analysis is a useful clinical indicator of respiratory function. In cases with severe hypercapnia in addition to hypoxaemia, mechanical ventilation may be required.

About 26-33 per cent of neonatal septicaemia cases will develop joint infections and 11-12 per cent will develop bone infections – highlighting the importance of thorough orthopaedic examination in foals with suspected sepsis. Bone infections normally occur at the epiphysis of long bones, the metaphyseal side of growth plates, costochondral junctions and articular facets of vertebral bodies. In foals with palpable joint effusions, synoviocentesis should be performed. Radiographic examination may show lytic osseous lesions in cases where the infection involves the underlying subchondral bone (Figure 6). The prognosis for survival and athletic performance decreases considerably with multiple sites of infection.

Ophthalmic examination may show evidence of uveitis (manifested clinically as miosis, blepharoedema, corneal oedema, conjunctival hyperaemia and ciliary injection, and aqueous flare), which is often due to ocular extension of the systemic disease process. Corneal ulceration is also common in recumbent neonatal foals (Figure 7). This may be secondary to trauma or due to entropion

secondary to dehydration. Corneal ulceration is often not associated with signs of obvious ocular pain, such as blepharospasm and excessive lacrimation, normally seen in the adult horse. Accordingly, at least daily fluorescein staining of the eyes is recommended in the recumbent neonatal foal.

Meningitis is a rare complication in neonatal foals. This is usually manifested by a combination of severe depression (which can be hard to identify in collapsed, unresponsive, septicaemic animals), seizures, nystagmus, strabismus, head tilt and extensor rigidity, depending on the area of brain involvement. A major differential diagnosis is perinatal asphyxia syndrome (or hypoxic ischaemic encephalopathy) – a much more common disease in neonatal foals. Seizures may also occur secondary to electrolyte and acid-base imbalances, and pyrexia. A cerebrospinal fluid sample can be obtained from the atlanto-occipital space. In cases with meningitis, cytology will show an abnormally increased white blood cell count with a predominance of neutrophils (often degenerate). A Gram's stain may reveal bacteria.

Treatment

Broad-spectrum antibiotic therapy should be instituted pending blood culture results. Septicaemia is most commonly caused by gram-negative bacteria (with *Escherichia coli* the predominant organism). However, infection due to gram-positive bacteria and mixed bacterial infections also occur (Marsh and Palmer, 2001; Corley et al, 2007).

Cefquinome is a reasonable initial choice in neonatal septicaemia. Other possibilities include combinations of crystalline penicillin and amikacin or crystalline penicillin and gentamicin. Amikacin and gentamicin have potential for nephrotoxicity and should be avoided in foals with azotaemia. Higher levels of resistance of bacterial isolates to gentamicin than amikacin have been reported. Therapeutic drug monitoring is advisable for both gentamicin and amikacin to ensure adequate drug levels are achieved and also to confirm adequate drug clearance.

Widespread bacterial resistance to trimethoprim sulphonamides significantly reduces this drug's usefulness, and enrofloxacin should be avoided as it has been associated with arthropathy in foals. Antibiotics should be continued for at least two weeks in the septicaemic foal without focal sites of infection. Cases with localising signs, such as pneumonia or osteomyelitis, may require antimicrobial therapy for four to six weeks.

Supportive fluid therapy and/or inotropes and vasopressors are often required. Addition of dextrose to the intravenous fluids may be required if the foal is hypoglycaemic. Hyperimmune plasma is a very useful therapeutic agent in these foals; typically, two litres will be required for a 50kg foal with septicaemia.

Gastric ulceration, usually of the glandular part of the stomach, can occur in sick neonatal foals; omeprazole, ranitidine or sucralfate can be used. Anti-endotoxic treatments, such as flunixin and

polymixin B, are commonly used in adult horses, but these drugs have nephrotoxic potential in neonatal foals and are not used routinely. In cases with respiratory dysfunction, provision of humidified intranasal oxygen is advisable (Figure 8).

Enteral and/or total parenteral nutrition is often necessary. Septic arthritis has many treatment options including through-andthrough needle lavage, arthroscopic lavage and intra-articular medication with antibiotics such as amikacin or gentamicin.

Prognosis

The reported short-term survival for septicaemic neonates varies considerably between different studies, but is typically between 45 and 67 per cent.

Prevention

Key management steps in the prevention of neonatal septicaemia include the following:

• Maintaining a clean foaling environment; disinfection of the stable between mares; provision of plenty of fresh bedding.

• Washing down (and then drying) of the mare's perineum, udder and hindlegs after foaling, but before the foal begins its udder-seeking behaviour.

• Ensuring the foal receives adequate, good-quality colostrum. Foals with weak suck reflexes should receive colostrum via stomach tubing (bottle feeding colostrum in weak foals carries a significant risk of aspiration pneumonia). IgG concentrations should be measured when the foal is 18-36 hours old and hyperimmune plasma should be administered if necessary.

• Appropriate treatment of the external umbilical remnants. Dipping of the umbilicus in a four per cent chlorhexidine solution is thought to be more effective than povidone iodine solutions.

References

- Brewer B D and Koterba A M (1988). Development of a scoring system for the early diagnosis of equine neonatal sepsis, *Equine Vet J* **20**: 18-22.
- Corley K T T et al (2007). Bacteraemia in neonatal foals: clinicopathological differences between Gram-positive and Gram-negative infections, and single organism and mixed infections, *Equine Vet J* 39: 84-89.
- Corley K T T and Furr M O (2003). Evaluations of a score designed to predict sepsis in foals, *J Vet Emerg Crit Care* **13**: 149-155.
- Marsh P S and Palmer J E (2001). Bacterial isolates from blood and their susceptibility

patterns in critically ill foals: 543 cases (1991-1998), *J Am Vet Med Assoc* **218**: 1,608-1,610.
- Stewart A J et al (2002). *Actinobacillus* sp bacteraemia in foals: clinical signs and prognosis, *J Vet Intern Med* **16**: 464-471.

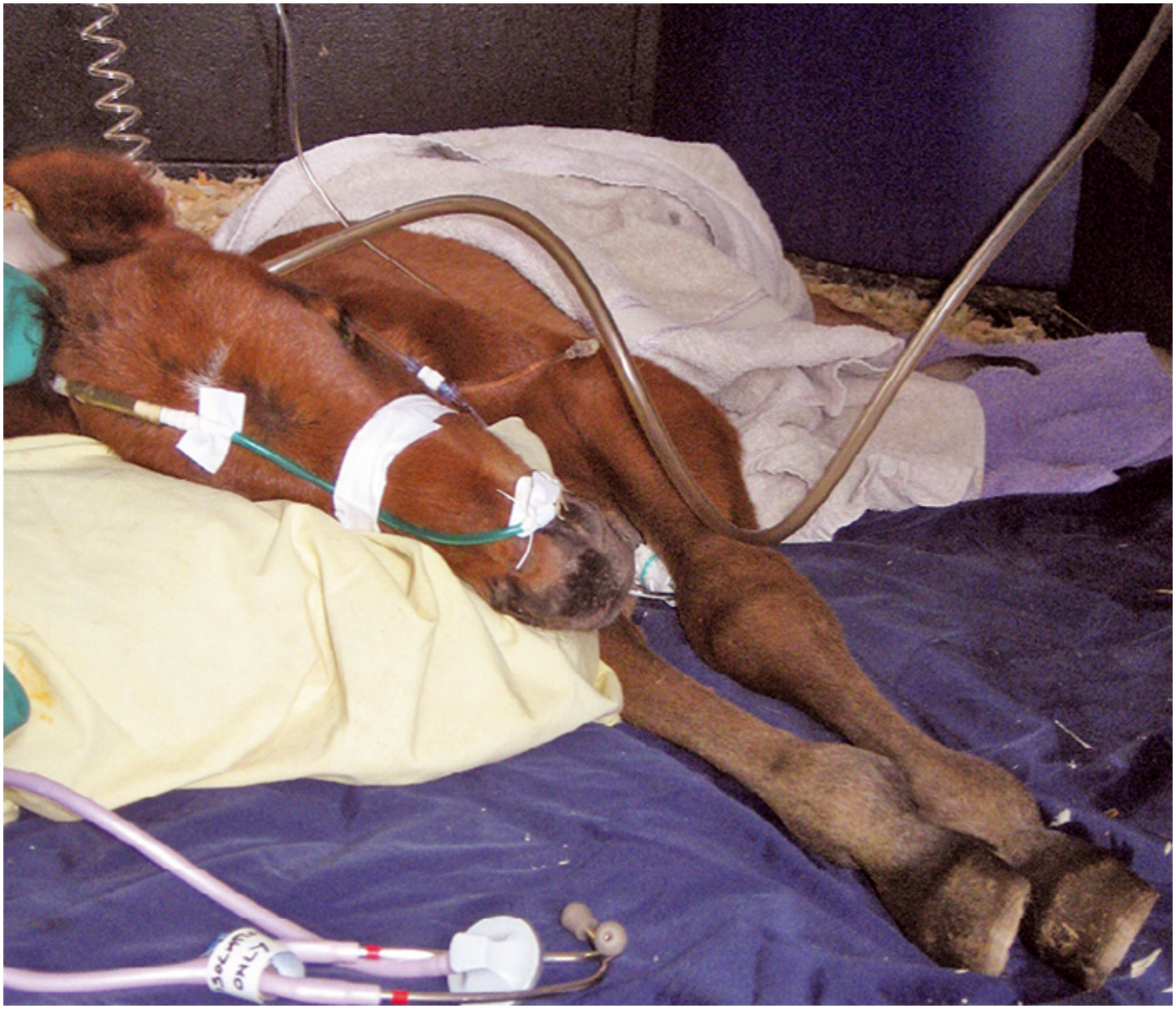

Figure 1. Initial clinical signs in sepsis may include excessive lethargy and sleeping, which can progress to recumbency and collapse if left untreated.

Figure 2. A foal presenting in terminal septicaemic shock with severe cardiovascular collapse and cyanosis. Cold extremities and muddy mucous membranes may also be seen.

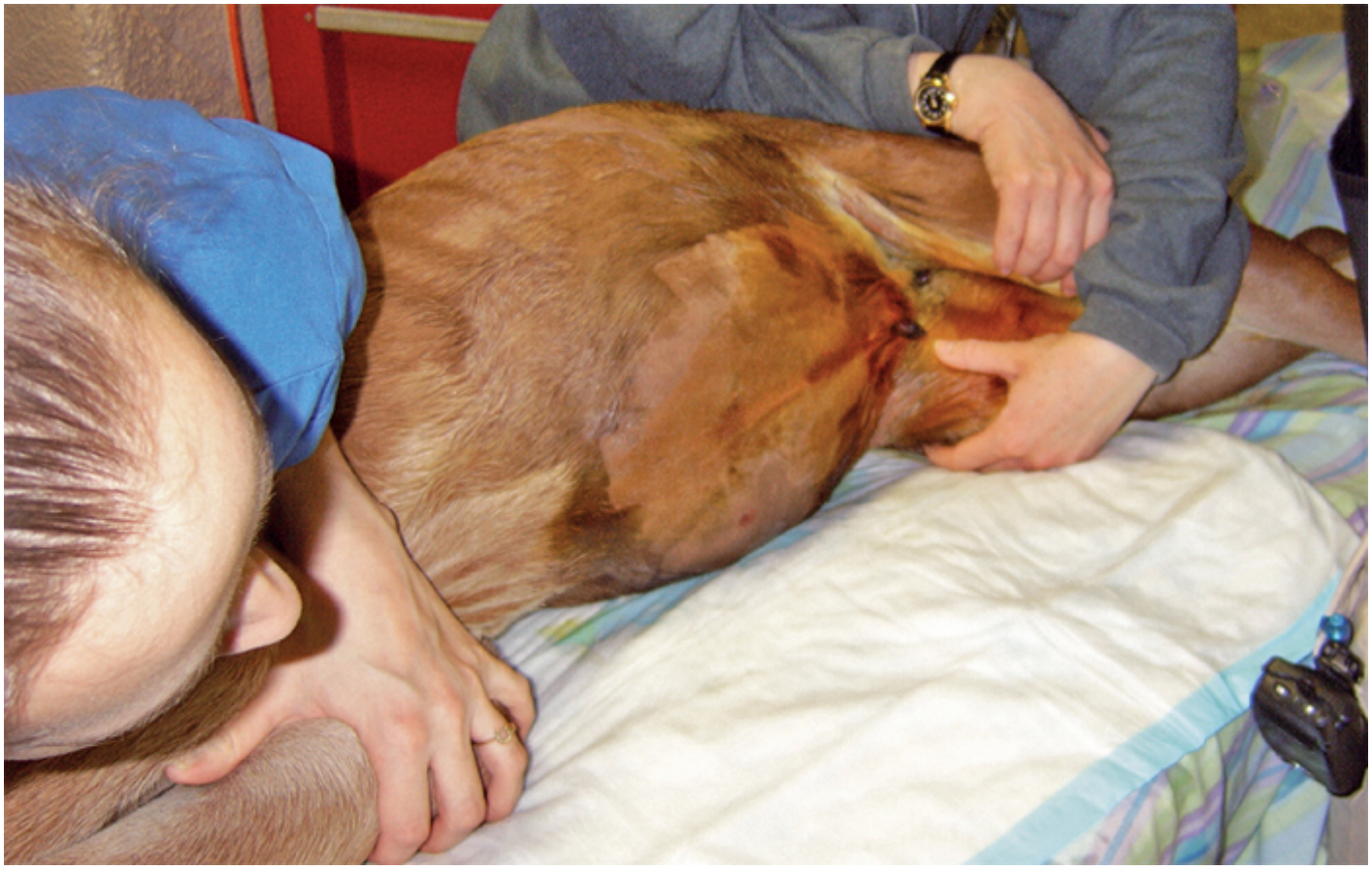

Figure 3. Severe abdominal distension in a foal with ileus and peritonitis secondary to septicaemia.

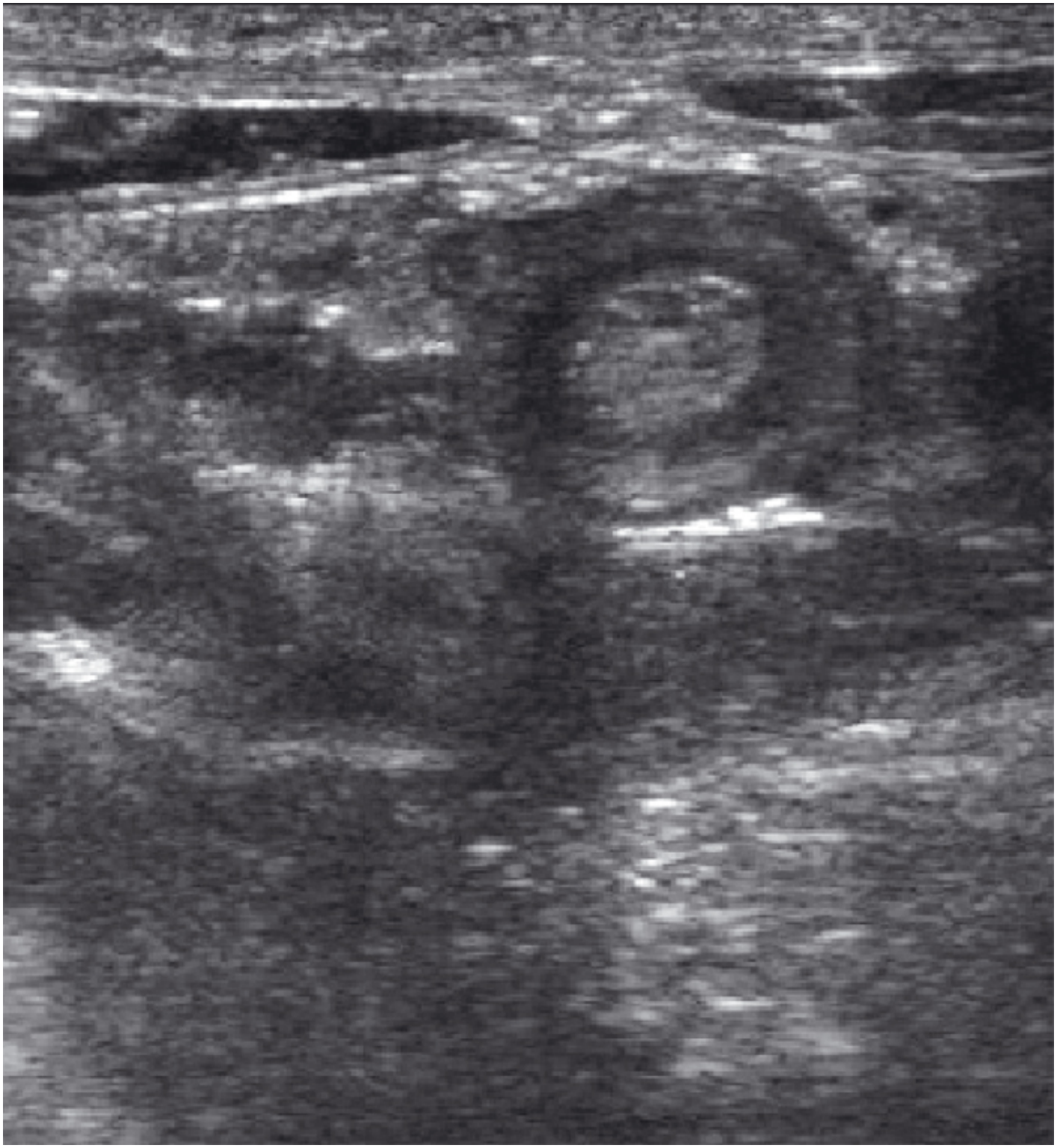

Figure 4. Ultrasonogram of a foal's infected umbilical vein (omphalophlebitis). This image was taken just cranial to the umbilical stump and demonstrates dilation of the umbilical vein, which contains echogenic material. Thickening of the vein wall is also apparent.

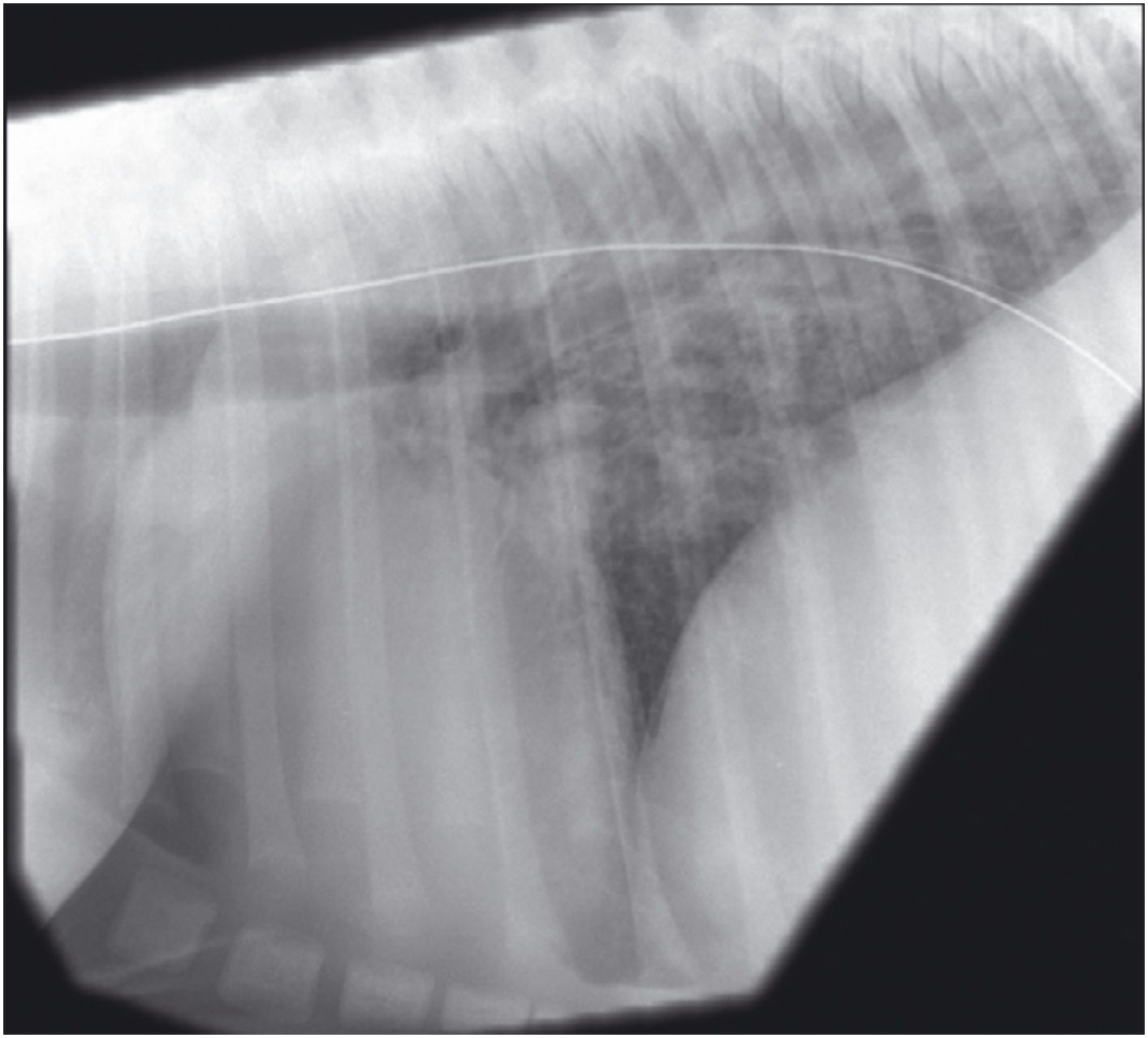

Figure 5. Lateral radiographic view of a one-day-old foal with interstitial pneumonia, consistent with haematogenous infiltration secondary to sepsis. An indwelling nasogastric tube has been placed to allow enteral feeding to take place.

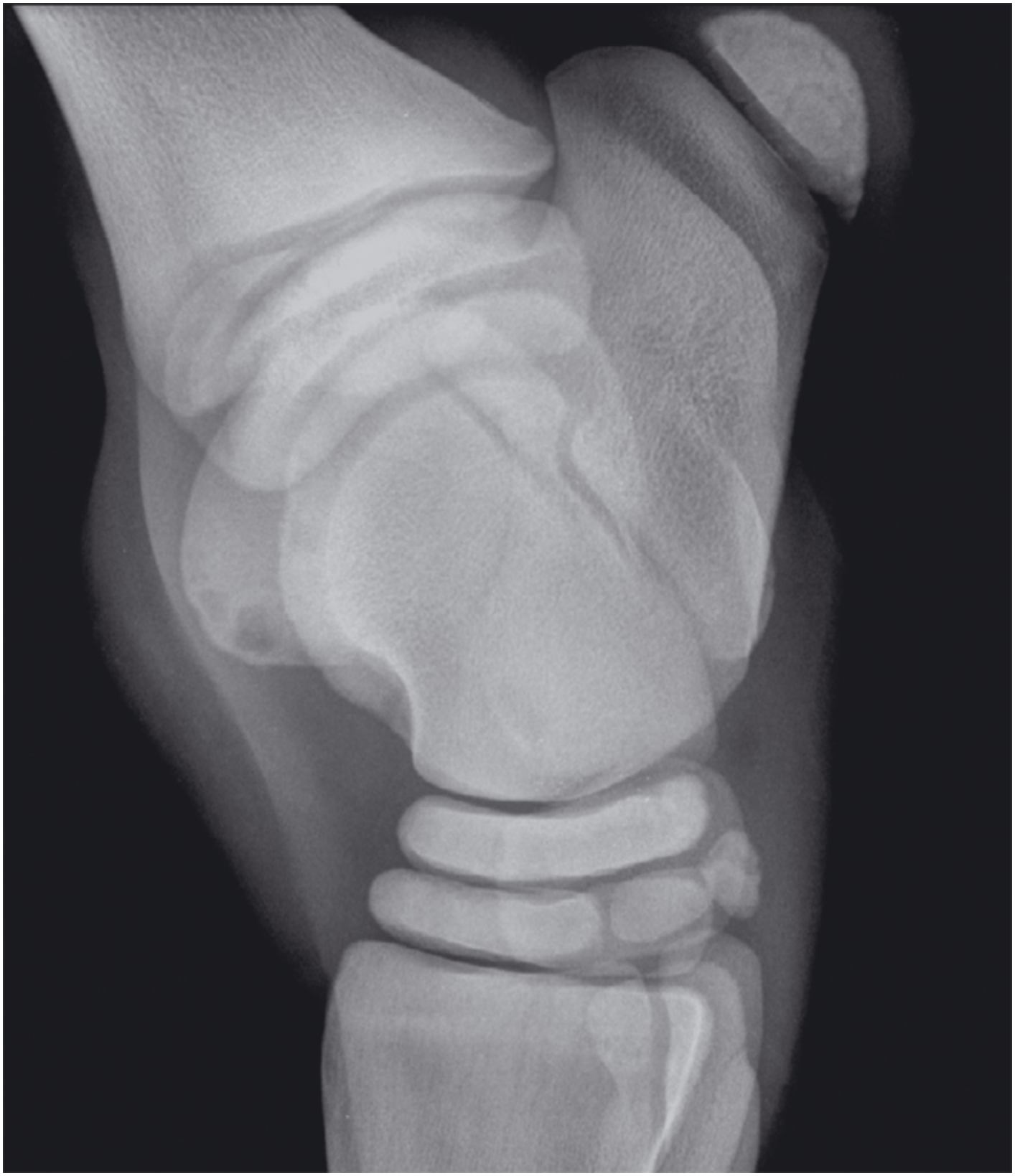

Figure 6. Two abnormal lytic lesions are observed on the lateral trochlear ridge of the talus of this neonatal foal with a tarsocrural joint infection. Soft tissue swelling is apparent due to the severe joint effusion.

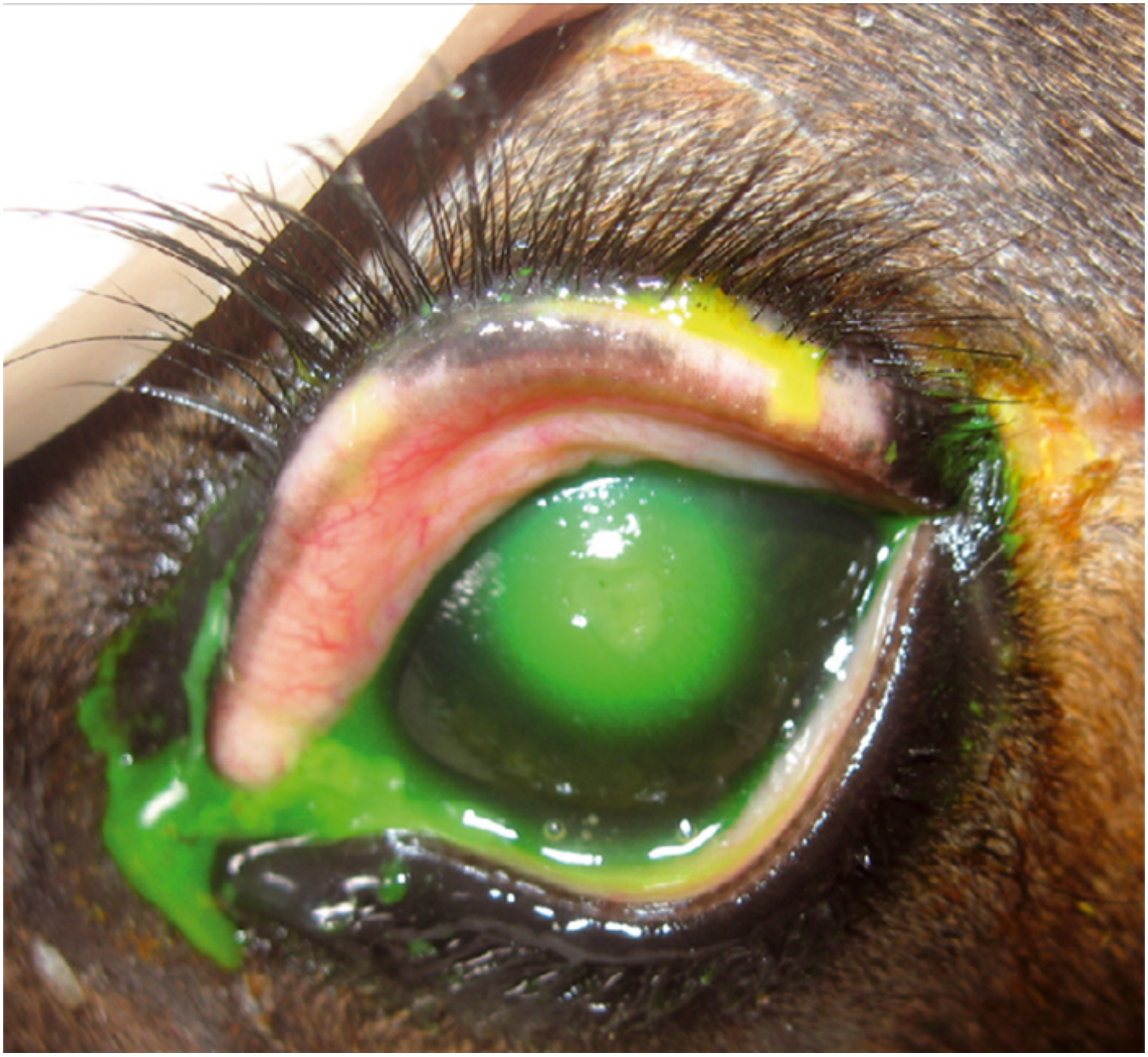

Figure 7. The corneal surface of this foal is stained with fluorescein dye, demonstrating the presence of a large corneal ulcer.

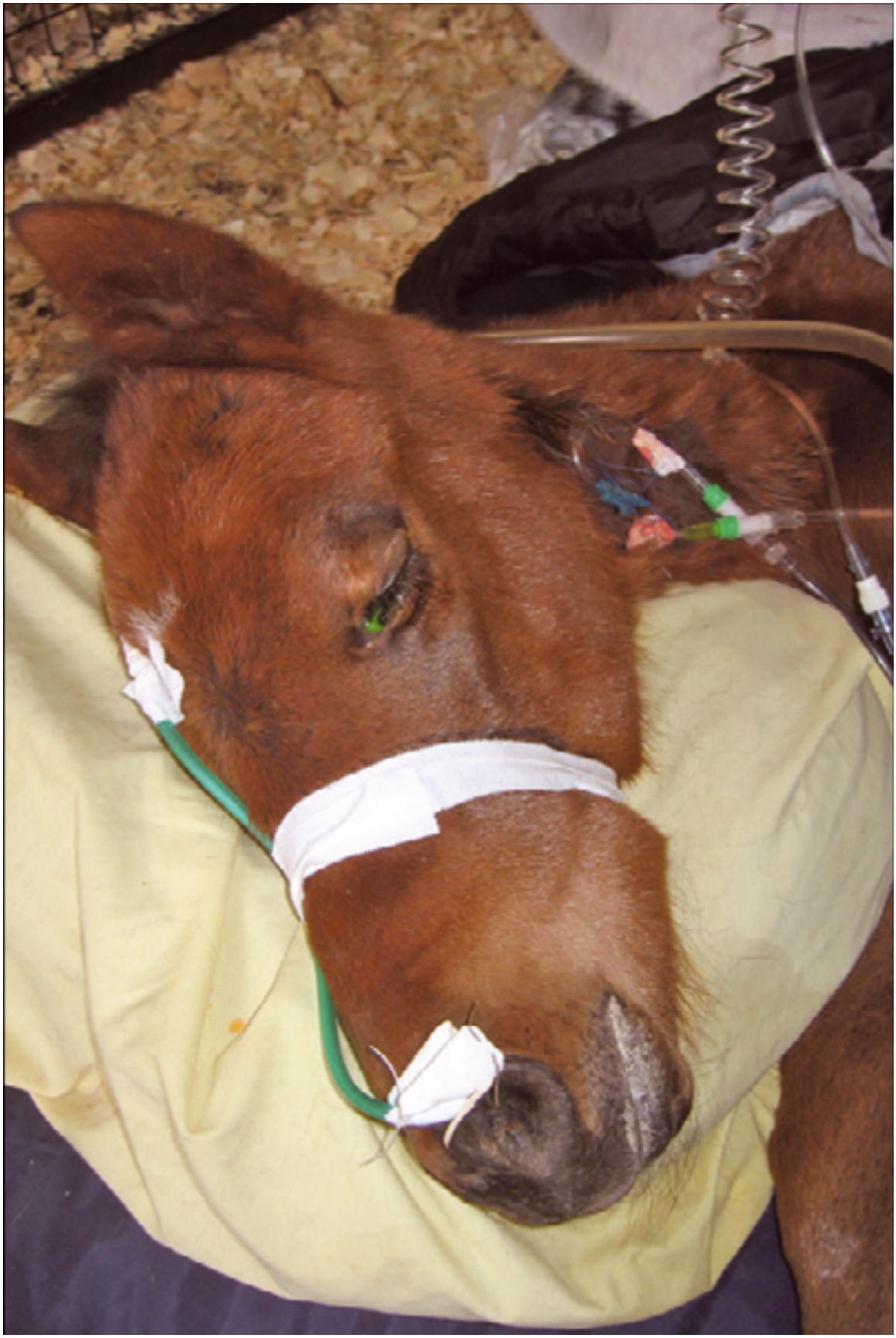

Figure 8. This neonatal foal with septicaemia is receiving humidified intranasal oxygen, intravenous fluids and antimicrobial therapy.