Case Report

Listeria monocytogenes septicaemia in 2 neonatal foals

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Introduction

Listeriosis is caused by a Gram-positive bacterium, Listeria monocytogenes, that commonly affects ruminants, fowl and man, but it is rarely reported in horses (Wallace and Hathcock 1995; Wilkins et al. 2000; Jose-Cunilleras and Hinchcliff 2001; George 2002). Clinical forms of the disease include septicaemia of neonates, abortion and the neurological form (George 2002).

In horses, listeriosis has been associated with diarrhoea in a 21-day-old Appaloosa filly (Wallace and Hathcock 1995); ataxia in a 1-month-old Arabian foal (Clark et al. 1978); diarrhoea and bronchopneumonia in a 6-day-old Thoroughbred foal (Wilkins et al. 2000); anorexia, fever, depression and death in 2 horses (McCain and Robinson 1976); septicaemia and death in a 5-month-old colt (Roberts 1968); fever, depression and mild colic in 4 Welsh and 2 Shetland ponies (Emerson and Jarvis 1968); abortion in a mare (Welsh 1983); depression and diarrhoea in a 2-day-old Standardbred foal and a 3-week-old Paint filly; and seizures in a 5-day-old Quarter Horse filly (Jose-Cunilleras and Hinchcliff 2001).

This report describes 2 foals in which Listeria monocytogenes was associated with septicaemia and secondary multisystemic infections.

Case details

Case 1

History

A 7-day-old Appaloosa colt was presented to the Veterinary Teaching Hospital with a history of lameness and lethargy of 2 days’ duration. The mare had been purchased 2 months earlier and had an unknown vaccination and anthelmintic history. The birth was unattended and the colt was found under a fence and unable to rise. The colt was given colostrum and ceftiofur by the referring veterinarian and had improved considerably and was reported to be doing well until 2 days prior to presentation.

Clinical findings

At presentation the colt weighed 35 kg and was ambulatory but weak, depressed and in poor body condition. Heart rate was 100 beats/min, respiratory rate 24 breaths/min and rectal temperature 38.9°C (102°F). The foal was lame on the right hindlimb (grade 3/5 lameness) and effusion was noted in the right tarsocrural joint. Palpation of the umbilicus was normal. Mucous membranes were pink with a capillary refill time of less then 2 secs. Gastrointestinal borborygmi were present and lung auscultation was normal. The foal was estimated to be 5% dehydrated.

Haematology revealed leucocytosis (19.8 x 10⁹ cells/l; reference range [rr] 6.3–13.6 x 10⁹ cells/l) characterised by neutrophilia (17.8 x 10⁹ cells/l; rr 4.35–10.55 x 10⁹ cells/l), lymphopenia (0.79 x 10⁹ cells/l; rr 1.43–2.28 x 10⁹ cells/l), monocytosis (0.79 x 10⁹ cells/l; rr 0.03–0.54 x 10⁹ cells/l), left shift (0.396 x 10⁹ bands/l; rr 0.00–0.15 x 10⁹ bands/l) and hyperfibrinogenaemia (9.0 g/l; rr 1.5–4.5 g/l). Serum biochemistry showed mild hyperglycaemia (8.83 mmol/l; rr 6.38–8.66 mmol/l). Venous blood gas was unremarkable. A blood sample was obtained for culture. IgG concentration in the plasma was 4.0–8.0 g/l, showing failure of passive transfer.

Arthrocentesis of the right tarsocrural joint was performed and the synovial fluid submitted for cytology and aerobic and anaerobic culture. The fluid was cloudy and had a total nucleated cell count of 47.6 x 10⁹ cells/l (rr <5.0 x 10⁹ cells/l) and total protein 28 g/l (rr 16–20 g/l). Cytology showed a predominance of neutrophils. A tentative diagnosis of septic arthritis was made based on the increased number of white blood cells in the fluid, increased percentage of neutrophils and increased protein. The joint was flushed with 1 litre 0.9% NaCl and amikacin (65 mg) was administered intra-articularly.

Abdominal ultrasonography was performed to rule out umbilical infection as a source of septicaemia. It revealed normal umbilical structures and an increased volume of hyperechoic peritoneal fluid. A sample of peritoneal fluid was submitted for cytology and aerobic and anaerobic culture. Cytology revealed an elevated total nucleated cell count of 52.2 x 10⁹ cells/l (rr <5.0 x 10⁹ cells/l), increased protein of

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The colt was treated with potassium penicillin (22,000 iu/kg bwt i.v. q. 6 h), amikacin (15 mg/kg bwt i.v. q. 12 h), flunixin meglumine (0.5 mg/kg bwt i.v. q. 12 h), omeprazole (4.4 mg/kg bwt per os s.i.d.), equine plasma (1 litre i.v.) and a single dose of vitamin E and selenium (1 ml i.m.: 68iu vitamin E and 2.5 mg selenium). Flunixin meglumine was administered to provide analgesia. Omeprazole was administered in an attempt to prevent gastric ulcers, even though the increase in gastric pH does not occur until approximately 3–4 days after initiation of treatment. The drug is also not approved for use in foals at this age. Fluid therapy consisted of 450 ml of a sterile, isotonic balanced salt solution (Normosol) administered i.v. q. 3 h to provide maintenance requirements. The colt was assisted to stand and nurse every hour.

The colt improved in the initial 24 h of hospitalisation and was able to stand and nurse on his own. Rectal temperature decreased to 38.3°C (101°F) and remained at this level throughout hospitalisation, indicating improvement of clinical signs or response to treatment with anti-inflammatory drugs. Radiographs of the carpal and tarsal bones showed adequate calcification. No signs of osteomyelitis or septic physis were identified involving the right tarsocrural joint. Thoracic radiographs and urine analysis were within normal limits.

The foal developed a patent urachus on Day 3, suspected on observation of urine dripping from the umbilicus and confirmed by ultrasound examination. Ultrasonography also revealed absence of the hyperechoic peritoneal fluid identified previously on presentation. The colt underwent general anaesthesia on Day 3 for surgical resection of the urachus, abdominal lavage, arthrocentesis and lavage of the right tarsocrural joint. The owner opted for surgical resection of the patent urachus over medical treatment. Cytological examination of the synovial fluid revealed a decreased total nucleated cell count of 16.1 x 10^9 cells/l, consisting primarily of nondegenerate neutrophils and a decreased concentration of protein of 27 g/l. Regional perfusion with potassium penicillin (22,000 iu/kg bwt i.v.) was performed on the right hindlimb daily for 3 days. The umbilical structures removed surgically were submitted for culture and sensitivity. The umbilical vein and umbilical arteries appeared grossly normal and the urachus was patent.

Culture results from the synovial fluid, peritoneal fluid and blood yielded *Listeria monocytogenes*. The organism was sensitive to amikacin, cephalothin, chloramphenicol, enrofloxacin, erythromycin, gentamicin, penicillin, tetracycline, ticarcillin and sulpha (20 mg/kg bwt per os q. 12 h for 10 days), flunixin meglumine (0.5 mg/kg bwt per os q. 24 h for 4 days) and omeprazole (4.4 mg/kg bwt per os q. 24 h for 10 days). The colt was reported to be clinically healthy 6 months later.

In an attempt to investigate the source of the *Listeria monocytogenes* infection, the mare's milk and faeces were submitted for culture on Day 5. No *Listeria* bacteria were isolated from these samples.

### Outcome

The colt continued to thrive and was discharged after 10 days of hospitalisation with instructions to administer trimethoprim-sulpha (20 mg/kg bwt per os q. 12 h for 10 days), flunixin meglumine (0.5 mg/kg bwt per os q. 24 h for 4 days) and omeprazole (4.4 mg/kg bwt per os q. 24 h for 10 days). The colt was reported to be clinically healthy 6 months later.

### Case 2

#### History

A 7-day-old American Quarter Horse colt presented to the Veterinary Teaching Hospital with a history of depression and anorexia of 8 h duration. The colt became progressively weak and was unable to stand. The referring veterinarian had noticed a swollen umbilicus.

#### Clinical findings

On presentation, the foal weighed 47 kg and was depressed, weak and unable to stand. Rectal temperature was 38.0°C (100.4°F), respiratory rate 44 breaths/min and heart rate 92 beats/min. Mucous membranes were pink with a capillary refill time >3 secs. Dehydration was estimated to be approximately 8%. The colt appeared painful with a tense abdomen. Lung auscultation was within normal limits. Gastrointestinal borborygmi were decreased in all 4 quadrants. The umbilicus was swollen and constantly dribbling urine. Abdominal ultrasound revealed many thickened loops of small intestine. Small intestinal intussusception was suspected based on ultrasonography. The liver appeared to have several hyperechoic areas (measuring approximately 1–2 mm) throughout the parenchyma. An umbilical abscess was identified within the umbilical vein. The urachus was patent.

Haematology revealed a left shift (1.1 x 10^9 bands/l; rr 0.00–0.15 x 10^9 bands/l). A chemistry profile showed hypoglycaemia (2.05 mmol/l; rr 6.38–8.66 mmol/l), increased AST (1120 u/l; rr 200–450 u/l), increased GGT (45 u/l; rr 10–38 u/l) and hyperbilirubinaemia (56.43 µmol/l; rr 11.97–42.75 µmol/l) characterised by increased indirect bilirubin (49.59 µmol/l; rr 8.55–34.2 µmol/l). Venous blood gas revealed metabolic and respiratory acidosis (pH 7.06; base excess -12.3 mmol/l; HCO3 17.7 mmol/l; total CO2 19.6 mmol/l; pCO2 65.2 mmHg). IgG concentration was less than 2.0 g/l, indicating failure of passive transfer.

#### Treatment

Initial stabilisation was attempted with fluid therapy consisting of a sterile, isotonic balanced salt solution (Normosol) at a rate of 150 ml/kg bwt/day with 50 mmol/l bicarbonate added.
to 1 litre, dextrose 5% at a rate of 75 ml/kg bwt/day, potassium penicillin (22,000 iu/kg bwt i.v. q. 6 h), amikacin (15 mg/kg bwt i.v. q. 12 h), flunixin meglumine (1.1 mg/kg bwt i.v. q. 12 h) and oxygen therapy administered via indwelling nasopharyngeal tube at a rate of 5 Vmin. The owner declined surgery due to financial restrictions.

**Outcome**

The colt progressively deteriorated over the next 2 h and developed a fever of 40.9°C (105.7°F). Respiratory rate increased to 116 breaths/min and heart rate to 204 beats/min. The colt was subjected to euthanasia 3 h after initial presentation.

**Necropsy findings**

Necropsy revealed a patent urachus and a 1 cm diameter abscess in the umbilical vein located 5 cm distal to the umbilicus. The liver contained numerous small abscesses (1–2 mm) within the parenchyma (Fig 1). Two intussusceptions (25 and 30 cm long) were identified in the small intestine. The intussusceptum and intussucipiens were necrotic and haemorrhagic in both lesions. Listeria monocytogenes was isolated from the umbilical vein abscess and from the hepatic abscesses.

**Discussion**

Septicaemia constitutes a major cause of mortality in the neonatal foal (Koterba et al. 1984). Foals are particularly susceptible to septicaemia because of failure of passive transfer, prematurity, perinatal stress and contamination of the environment with pathogenic bacteria (Marsh and Palmer 2001; Vaala and House 2002). The bacteria responsible for neonatal equine septicaemia were found to consist predominantly of Gram-negative organisms, *Escherichia coli* being the most common bacteria isolated (Koterba et al. 1984; Raisis et al. 1995; Vaala and House 2002). However, a recent study evaluating blood cultures from 543 critically ill foals concluded that Gram-positive organisms were isolated in a greater prevalence than previously reported and *Listeria monocytogenes* accounted for 2% of the isolates (Marsh and Palmer 2001).

Septic arthritis is a frequent sequela of neonatal septicaemia (Fenger 1998; Madigan and House 2002). In foals, bacteria that produce septic arthritis include *E. coli*, *Klebsiella* spp., *Actinobacillus equuli*, *Salmonella* spp., *Rhodococcus equi* and *Streptococcus* spp. (Madigan and House 2002). With treatment, the prognosis for survival of foals with septic arthritis was favourable as reported in one study where 73/93 foals survived after hospitalisation (Steel et al. 1999). Septic arthritis was diagnosed in Case 1 in our report and was treated successfully with joint lavage, regional limb perfusion with antibiotics and systemic broad-spectrum antimicrobial drugs. Studies have shown that the development of septic arthritis in foals significantly reduces the likelihood of starting on a racecourse when compared with controls (Steel et al. 1999; Meijer et al. 2000; Smith et al. 2004), and carries a poor prognosis for athletic performance. However, the foal in Case 1 was not intended to be used as an athletic animal.

None of the previous reports in the literature had described listeriosis in equine patients causing septic arthritis and peritonitis as described in Case 1. Clinical presentations of listeriosis in equine neonates included 4 cases of diarrhoea (Wallace and Hathcock 1995; Wilkins et al. 2000; Jose-Cunilleras and Hinchcliff 2001), 2 with neurological signs (Clark et al. 1978; Jose-Cunilleras and Hinchcliff 2001) and one in respiratory distress (Wilkins et al. 2000).

Reports of post mortem examination of horses and foals with listeriosis described lesions affecting the liver (Emerson et al. 1968; Roberts 1968; McCain and Robinson 1976; Wilkins et al. 2000), lung (Emerson and Jarvis 1968; Wilkins et al. 2000), meninges (Clark et al. 1978; Wilkins et al. 2000), brain (Clark et al. 1978), heart (McCain and Robinson 1976; Clark et al. 1978), adrenal glands (Clark et al. 1978), skeletal muscle (McCain and Robinson 1976), spleen and kidney (Emerson and Jarvis 1968). There was no description of *Listeria* sp. affecting the joints or peritoneal cavity.

Listeriosis is caused by ingestion of the organism *Listeria monocytogenes* from a contaminated environment, in faeces and poorly prepared silage (Radostits et al. 2000; Jose-Cunilleras and Hinchcliff 2001; Pugh 2002). Outbreaks in man have been associated with ingestion of contaminated milk (Wallace and Hathcock 1995; Jose-Cunilleras and Hinchcliff 2001; George 2002). It was speculated in one report that the source of infection may have been from the mare’s milk (Wallace and Hathcock 1995). However, in our Case 1 culture of the mare’s milk yielded no growth. This finding suggests that the milk was an unlikely source of infection, unless the mare was shedding the organism intermittently or the sample was not representative of the mammary secretion.

The authors speculate that another possible source of infection in Case 1 was through the umbilicus. However, culture of the umbilical structures was negative. The isolation of *Listeria monocytogenes* from the umbilicus in Case 2 demonstrated a possible site of entry of the bacteria in neonates. The
environment would be the most likely source of the organism. No previous reports of listeriosis in equine neonates have isolated *Listeria monocytogenes* from the umbilical structures (Roberts 1968; Clark et al. 1978; Wallace and Hathcock 1995; Wilkins et al. 2000; Jose-Cunilleras and Hinchcliff 2001). The sensitivity results of *Listeria monocytogenes* isolated from Cases 1 and 2 were similar to those in another report (Jose-Cunilleras and Hinchcliff 2001). Isolates were sensitive to amikacin, cephalothin, chloramphenicol, gentamicin, penicillin, sulphamethoxazole with trimethoprim, tetracycline and ticarcillin, and were resistant to cefotiofur.

The prognosis and response to treatment in Case 1 were favourable, as described in a few recent reports (Wallace and Hathcock 1995; Jose-Cunilleras and Hinchcliff 2001). This provides evidence that the outcome can be favourable with an early diagnosis, prompt treatment with appropriate antibiotics and supportive care. The poor outcome in Case 2 was probably caused by the 2 sites of intussusception in the small intestine and the extensive hepatic injury.

**Manufacturer's address**

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**References**


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**Clinical Commentary**

**Listeriosis in neonatal foals: just like any other bacteraemia?**

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Within the last 6 years there have been several reports describing the clinical manifestation of *Listeria monocytogenes* sepsis/bacteraemia in neonatal foals, including this most recent report (above) describing 2 cases in foals 7 days of age at presentation (Wilkins et al. 2000; Cunilleras and Hinchcliff 2001; Monteiro et al. 2006). In addition, in at least one report of bacteria isolated from blood cultures obtained from neonatal foals *Listeria monocytogenes* was present in
In man, clinical presentation of sepsis without definitive localising signs is most common in hosts with immune compromise; immune compromise is generally decreased or impaired cell-mediated immunity and is observed within neonates, pregnant women and the elderly. In these populations, infection with *Listeria monocytogenes* is an important cause of life-threatening infections including sepsis and meningoencephalitis. The patient often appears severely ill with fever, nausea, vomiting and malaise. Sepsis may progress to disseminated intravascular coagulation, acute respiratory distress syndrome and multi-organ system failure (Doganay 2003). The clinical features of listerial sepsis are similar to those of other types of bacterial sepsis and its diagnosis is based on a positive blood culture.

It appears as if the clinical presentation of listerial infection in foals follows a similar pattern, in that there is no predicted presentation form and infection appears to result in multiple organ dysfunction, ranging from pneumonia and diarrhoea to joint and peritoneal infections. In man, listerial infections in infants are separated into early- and late-onset infection, with early-onset infection most commonly associated with maternal infection and chorioamnionitis (Posfay-Barbe and Wald 2004). The seriousness of maternal and perinatal infection of pregnant women and their fetuses with *Listeria monocytogenes* has led to food restriction for mothers during pregnancy. Prohibited foods include luncheon meats such as bologna, soft cheeses such as Camembert (Normandy) and Brie (Meaux) and other meat and fish products (Jemmi et al. 2002; Sanaa et al. 2004). In this case, the gastrointestinal tract is the route of entry to the mother, immunocompromised by pregnancy, who then experiences bacteraemia, sepsis and subsequent infection of the placenta and/or fetus. Within all the currently available reports of listerial infection in foals, none appear to be associated with maternal, and subsequent *in utero*, infection and the cases are most likely to be due to infection by an alternate route. The slightly older age of presentation for these cases, ranging from 2 to 21 days for equine neonate, with most presenting between 5 and 10 days of age, suggests an environmental rather than maternal source and is more akin to late-onset sepsis described in human infants. The route by which *Listeria monocytogenes* gains access to the foal can be debated. In general, there are 3 primary routes of infection considered important in the neonatal foal: the umbilicus, gastrointestinal tract and respiratory tract. Certainly, infection of skin wounds can provide another means of entry, but as a general observation the skin is probably less important. In one of the 2 cases presented by Monteiro et al. (2006) it appears as if the umbilical route may have played a role, while the route of infection is unclear for the other, the general rule for the other cases reported to date. However, because *Listeria monocytogenes* has the ability to attach to and enter mammalian cells, the gastrointestinal route seems most likely (Boyle and Findlay 2003).

The clinical course for *Listeria monocytogenes* infection in the equine neonate appears to be related to the extent of organ dysfunction at the time of presentation and the early use of appropriate antimicrobial treatment. The cases presented by Monteiro et al. (2006) were treated with penicillin and amikacin, reportedly effective against their isolate, but only the foal with the less severe, more localised, clinical signs survived. Similar to some other equine pathogens, *Rhodococcus equi* among them, *Listeria monocytogenes* is able to live and replicate intracellularly, which may affect the *in vitro* efficacy of some antimicrobial agents (Boyle and Findlay 2003).

**References**


