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Clinical findings in 10 foals with bacterial meningoencephalitis

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Summary

Reasons for performing the study: Bacterial meningoencephalitis is a severe complication in septic foals and there is scarce and often unclear information in the equine literature.
Objectives: To report the most frequent clinical signs, clinicopathological findings, causative agents, treatments given and outcome of a group of foals with confirmed bacterial meningoencephalitis.
Methods: Foals aged <6 months of age admitted to the Universitat Autonoma de Barcelona (2004–2009) with confirmed bacterial meningoencephalitis were retrospectively included in the study. Diagnosis of bacterial meningoencephalitis was made by cerebrospinal fluid (CSF) culture, CSF analysis consistent with bacterial infection, observation of bacteria in CSF cytology or *post mortem* confirmation.
Results: Nine neonates and one 5-month-old foal were included. The most frequently observed clinical signs were alterations in mental status (10/10), recumbency (8/10), weakness (8/10), abnormal pupillary light reflexes (6/10), decreased suckling-reflex (6/9), seizures and/or nystagmus (4/10). Common clinicopathological alterations included hyperfibrinogenaemia (8/9), hyperlactataemia (7/7), and neutropenia (5/10) or neutrophilia (5/10). Most neonates (8/9) developed bacterial meningoencephalitis despite having a sepsis score near the cut-off value (median = 12). On CSF analysis, pleocytosis (9/9), increased total protein concentration (5/6) and intracellular bacteria (6/9) were detected. The most frequently isolated bacterium was *Escherichia coli*. Once bacterial meningoencephalitis was diagnosed, antimicrobial therapy was switched to third and fourth generation cephalosporins.
Conclusions: The diagnosis of bacterial meningoencephalitis is established based on CSF analysis and culture. Clinical recognition of bacterial meningoencephalitis is difficult and can be easily overlooked. Moreover, severe sepsis is not necessary to develop bacterial meningoencephalitis.
Potential relevance: CSF analysis should be considered more often in sick newborn foals with signs indicative of central nervous system (CNS) involvement. Cerebrospinal fluid (CSF) cytology and culture would help to confirm or rule out unnoticed bacterial meningoencephalitis, and to choose appropriate antimicrobial therapy.

Keywords: horse; neonate; septic meningitis; sepsis; neurological disease

Introduction

Bacterial infection of the equine central nervous system (CNS) is an uncommon condition and has been reported to affect 2.5% of horses with neurological disease (Furr 2008). Bacterial meningoencephalitis appears to be more frequent in neonatal foals. It is reported as a complication of sepsis in as many as 8–10% of septic foals (Koterba *et al*. 1984; Brewer and Koterba 1990) and is associated with a high mortality rates (Koterba *et al*. 1984; Brewer and Koterba 1990; Sanchez *et al*. 2008). However, information about bacterial meningoencephalitis in equine patients is scarce, and some authors consider that there is a lack of comprehensive case series of bacterial meningitis in these animals (Mitchell *et al*. 2007). Reported clinical signs of bacterial meningoencephalitis, such as lethargy, weakness, fever and seizures (Pellegrini-Masini and Livesey 2006; Furr 2008) are nonspecific and frequently present in other common neonatal diseases.

Early diagnosis of bacterial meningoencephalitis is essential for selection of appropriate therapy, and for increasing the likelihood of survival. However, clinical signs consistent with bacterial meningoencephalitis are often observed in many critically ill foals with other diseases, such as hypoxic-ischaemic encephalopathy (HIE). Thus, recognition of bacterial meningoencephalitis based on clinical signs can be difficult. In human medicine, it is well known that meningitis can easily go unrecognised in septic neonates, so cerebrospinal fluid (CSF) is routinely collected in septic infants (Heath *et al*. 2003).

The objective of this retrospective case series was to review the clinical signs, causative agents, laboratory findings and outcome in foals with confirmed bacterial meningoencephalitis admitted to a referral equine hospital.

Materials and methods

Case selection

Data from medical records of all newborn foals (<21 days old) and foals aged 21 days to 6 months, admitted to the Universitat Autonoma de Barcelona from January 2004 to December 2009, with a confirmed diagnosis of bacterial meningoencephalitis were reviewed.

Diagnosis of bacterial meningoencephalitis was confirmed by a board-certified neurologist and was based on either bacterial growth on CSF culture, consistent CSF analysis, or *post mortem* confirmation. Cerebrospinal fluid (CSF) analysis was considered consistent with bacterial meningoencephalitis when neutrophilic pleocytosis or intracellular bacteria were detected. *Post mortem* diagnosis was reached by the presence of either purulent CSF or histopathological evidence of bacterial infection in the CNS. Moreover, diagnosis of sepsis in newborn foals was reached when at least one of the following conditions occurred: positive blood culture, increased sepsis score (>11) or *post mortem* findings consistent with sepsis.

Medical record review

Data obtained from medical records included case details, clinical signs, neurological and ophthalmological examination findings (confirmed by a boarded neurologist and ophthalmologist, respectively), and clinicopathological results obtained when clinical signs consistent with bacterial meningoencephalitis were detected (total white blood cell count, neutrophil count, packed cell volume; total plasma protein [TP], fibrinogen, lactate, sodium, potassium and chloride concentrations; serum IgG concentration; CSF total protein concentration, cell count and cytology; and blood and CSF cultures). Other information included was: sepsis score (neonatal foals) (Brewer and Koterba 1988), concurrent diseases during hospitalisation, treatments given, outcome (survival to discharge from the hospital, euthanasia or death due to complications from sepsis or bacterial meningoencephalitis), and *post mortem* findings. Information regarding the organisms isolated from blood and CSF samples, including bacterial identification and antimicrobial sensitivity, was also obtained.

Results

Nine of 173 septic newborn foals (from a total of 309 sick neonates admitted during the study period) fulfilled the inclusion criteria (*Foals 1–9*). In addition, a 5-month-old foal (*Foal 10*) of 174 admitted foals (>21 days and

TABLE 1: Main results of cerebral spinal fluid (CSF) analysis of the 10 foals with bacterial meningoencephalitis

	CSF analysis*				
Foal	TNCC (×10^6 cells/l)	Type of pleocytosis	TP (g/l)	Other observations	CSF culture
Ref. value	<7		1.20	None	Negative
1	136	Mixed (27% neutrophils, 46% macrophages, 27% lymphocytes)	NP	Bacteria	NP
2	5810	Neutrophilic	4.73	Xanthochromic	Negative
3	940	Neutrophilic	NP	Marked degeneration	NP
4	NP	NP	NP	NP	NP
5	380	Neutrophilic	1.85	Xanthochromic, rods	*Escherichia coli*
6	Increased	-	7.69	Cocci	*Escherichia coli*
7	Increased	-	4.73	Rods	Negative
8	13	Neutrophilic	NP	Rods	NP
9	13	Neutrophilic	1.49	Xanthochromic	NP
10	15	Neutrophilic	0.77	One rod	NP
Mean	1216	-	3.95	-	-
(range)	(13–5810)	-	(0.76–7.69)	-	-

TNCC = total nucleated cell count, TP = total protein concentration; NP = not performed; WBC= white blood cell count.

<6 months) also met the inclusion criteria. Six foals were Andalusians, 2 were Arabians and 2 were crossbreds. There were 4 females and 6 males. Median age at the time of diagnosis of bacterial meningoencephalitis was 2 days for septic neonates.

Diagnosis of bacterial meningoencephalitis was reached by bacterial growth on CSF culture, consistent CSF analysis and *post mortem* confirmation in 2 foals; consistent CSF analysis and *post mortem* findings in 3 foals; consistent CSF analysis only in 4 foals, and *post mortem* confirmation only in one foal (Table 1). Diagnosis was made based on CSF analysis collected immediately after death in 3 foals.

Clinical signs

On physical examination, 2 neonates had fever (39.4°C and 39.1°C), 4 were tachypnoeic (42–46 breaths/min) and 2 were tachycardic (150–155 beats/min).

The only neurological sign present in all foals was altered mental status. Mental status was described as mildly obtunded (2/10), markedly obtunded (3/10), stuporous (3/10) or comatose (2/10). Seven of the 9 neonates and the 5-month-old foal were recumbent and showed generalised weakness on admission. Two foals showed gait and posture alterations (ataxia and head tilt), and one of them was circling. Other neurological deficits detected were: spontaneous nystagmus (4/10), seizures (3/10), *status epilepticus* (1/10), abnormal respiratory pattern (4/10) with respiratory arrest (2/10), tremors (1/10) and ventrolateral positional strabismus (1/10). Some animals also had decreased (4/10) or absent (2/10) pupillary light reflexes. Of these, one showed anisocoria, and another was mydriatic with lack of pupillary light reflexes.

Clinicopathological findings

Complete blood cell count (CBC) displayed leucocytosis (>12×10^9 cells/l) in 5 foals. Of these, 3 had leucocyte counts above 20×10^9 cells/l, with marked neutrophilia (>10×10^9 cells/l). The remaining foals had severe leucopenia (<2.1×10^9 cells/l) with neutropenia (<1.8×10^9 cells/l). Plasma fibrinogen concentration was increased in 7/8 foals. Blood lactate concentration was measured in 7 foals, being increased in all of them (Table 2).

When clinical signs of bacterial meningoencephalitis were first detected, median values of PCV and total plasma protein concentration were 35% (range 24–54%) and 59 g/l (range 53–71 g/l), respectively. Plasma creatinine was measured in 8 foals and it was normal. Plasma glucose concentration was measured in 8 cases, being low in 4 neonates (values lower than 1.4 mmol/l in 3 animals), and normal in the remaining 4. Blood electrolyte concentrations (Na^+, K^+, Cl^-) were determined in 8/9 neonates and hyponatraemia (<130 mmol/l) was detected in 2 foals (124 and 127 mmol/l, respectively). The remaining animals (including the 5-month-old filly) did not show alterations in electrolyte concentrations.

Serum immunoglobulin G concentration was determined in 8/9 neonates. Failure of transfer of passive immunity (FTPI) (<0.4 g/l) was detected in 3 of these foals and partial failure (0.4–0.8 g/l) in one.

The sepsis score (Brewer and Koterba 1988) was calculated in all neonates upon admission, and in one case when neurological signs were detected (median value = 12, range 9–20). Two neonates had values below 11, 6 neonates had values between 11 and 13, and only one had a higher sepsis score (20).

Cerebrospinal fluid was obtained under general anaesthesia through atlanto-occipital puncture (6/9) or immediately after death/euthanasia (3/9). The CSF analysis showed pleocytosis (total nucleated cell count, TNCC> 7×10^6 cells/l) (Corley and Stephen 2008) in all neonatal foals, and increased total protein concentration (TP>1.2 g/l) (Corley and Stephen 2008) in the 5 cases that had it determined (Table 1). The 5-month-old filly had an increased total nucleated cell count (TNCC) (15×10^6 cells/l), but normal TP (0.765 g/l). Intracellular bacteria were observed in the CSF of 6 horses (5 neonates and the 5-month-old filly).

Concurrent diseases

All newborn foals had a diagnosis of sepsis and one or more of the following problems: polyarthritis (4/9), pneumonia (4/9), omphalitis (4/9) and enteritis (1/9). Other diagnoses reached in the neonates were FTPI (4/9), perinatal asphyxia syndrome (4/9), uroperitoneum (3/9), immaturity (3/10) and oral candidiasis (1/9). Pneumonia, myositis, colitis, unilateral mild uveitis and bilateral optic neuritis were diagnosed in the 5-month-old filly.

A diagnosis of sepsis was established based on positive blood cultures and sepsis scores above 11 in 4/9 foals, while it was based only on positive blood cultures in 2/9 foals, and only on sepsis scores above 11 in 3/9 animals. Ophthalmological examinations were performed in 4 neonates and the 5-month-old filly. Three of the neonates had ocular signs consistent with bilateral anterior uveitis. The other neonate and the older foal had ocular signs consistent with bilateral optic neuritis (papillary oedema, peripapillary exudate and haemorrhage or peripapillary retinal elevation).

Bacteriology

Six of the 9 newborn foals had positive blood cultures. Two neonates with negative blood cultures had positive CSF cultures. Blood samples for culture were collected upon admission, but CSF cultures were performed later, once bacterial meningoencephalitis was clinically suspected. The most commonly isolated organism was *Escherichia coli* (4/9 blood cultures, and 2/4 CSF cultures) (Table 1). Other isolated bacteria were *Salmonella* (2 cases: one blood and one synovial fluid culture) and *Klebsiella oxytoca* (one case; blood culture). All *E. coli* isolates were sensitive to amikacin and ceftiofur, but some of them were also sensitive to ceftriaxone (4/5), cephalexin (4/6), cefotaxime (4/6) and amoxicillin-clavulanic acid (3/5). Only 50% of these isolates were sensitive to tetracycline, enrofloxacin, cefoperazone and trimethoprim-sulphamethoxazole.

TABLE 2: Blood clinicopathological data, diagnosis and CNS *post mortem* findings of the 10 foals with bacterial meningoencephalitis

	Blood and plasma biochemistry							
Foal	Blood culture	WBC ($\times10^9$ cells/l)	Neutrophil count ($\times10^9$ cells/l)	Fibrinogen (g/l)	Lactate (mmol/l)	Sepsis score	Concurrent diseases	*Post mortem* findings
Ref. value	Negative	5.4–14.3	2.30–8.60	<0.4	<2	<11		
1	*Klebsiella oxytoca*	27.18	19.57	0.6	2.4	12	Sepsis (polyarthritis, pneumonia)	Meningeal inflammatory infiltrate
2	Negative	17.95	13.82	0.8	NP	13	Sepsis (polyarthritis, omphalitis)	Discharged
3	*Escherichia coli*	1.69	0.47	0.6	4.8	12	Sepsis (pneumonia), PAS, FTPI, immaturity, uveitis	Mononuclear and polymorphonuclear meningeal infiltrates
4	*Escherichia coli*	1.14	0.78	NP	4.8	10	Sepsis (pneumonia), PAS, immaturity, UP, uveitis	Purulent meningitis
5	Negative	22.66	17.90	0.5	2.1	13	Sepsis (omphalitis), oral candidiasis	Purulent meningoencephalitis
6	Negative	0.68	0.16	0.5	NP	20	Sepsis (polyarthritis), PAS, FTPI, UP, prematurity	Purulent meningoencephalitis
7	*Salmonella* spp.	2.10	1.80	1.0	NP	11	Sepsis (enteritis)	NP
8	*Escherichia coli*	1.08	0.40	NP	9.3	13	Sepsis (polyarthritis, enteritis, omphalitis), FTPI	Encephalic perivascular rods
9	*Escherichia coli*	12.09	10.40	0.4	8.8	9	Sepsis (pneumonia, omphalitis), PAS, FTPI, UP, optic neuritis, uveitis	Meningeal congestion
10	NP	20.11	16.21	0.3	3.2	NP	Pneumonia, myositis, optic neuritis, colitis	Discharged
Mean	-	10.67	8.11	0.59	5	12.3		
(range)	-	(0.68–27.18)	(0.40–19.570)	(0.3–1.0)	(2.1–9.3)	(9–20)		

NP = not performed; WBC = white blood cell count; UP = uroperitoneum; PAS = perinatal asphyxia syndrome; FTPI = failure of transfer of passive immunity.

Treatments

Bacterial meningoencephalitis was diagnosed during hospitalisation (n = 2) or on *post mortem* examination (n = 4) in 6 foals. These were initially treated with common broad spectrum antibiotics: penicillin plus amikacin (n = 3), ceftiofur (n = 1), ceftiofur plus amikacin (n = 1) and imipenem (n = 1). Once a diagnosis of bacterial meningoencephalitis was reached, one foal was subjected to euthanasia and the other was treated with ceftiofur (10 mg/kg bwt q. 8 h i.v.).

On the other hand, bacterial meningoencephalitis was diagnosed upon admission in 3 neonates, one of these was treated with ceftriaxone (25 mg/kg bwt q. 12 h i.v.), and 2 were treated with cefepime (11 mg/kg bwt q. 8 h i.v.). Bacterial meningoencephalitis was diagnosed upon admission and treated with trimethoprim-sulphadimidine (15 mg/kg bwt q. 12 h i.v.) and oral rifampicin (10 mg/kg bwt q. 12 h) in the 5-month-old filly.

Additionally, 8/9 neonates received fluid therapy: 6 of these received an isotonic (lactated Ringer's solution plus 10 ml/l of 7.5% NaCl solution i.v.) or slightly hypertonic solution (lactated Ringer's solution plus 20 ml/l of 7.5% NaCl solution i.v.). The 5-month-old filly received the same sodium-rich fluid therapy and a colloidal solution (6% hydroxyethyl starch, 10 ml/kg bwt i.v.). Six of the 9 neonates and the older filly received intranasal oxygen supplementation. Anticonvulsant therapy (diazepam and phenobarbital) was given to 4 newborn foals when needed to control seizure activity, and the 5-month-old filly was treated with phenobarbital (4 mg/kg bwt q. 12 h i.v.) to reduce intermittent hyperexcitability. Mannitol (1000 mg/kg i.v.) was administered to 4 neonates to control clinical signs consistent with increased intracranial pressure. Three neonates also received a single dose of corticosteroids (methylprednisolone sodium succinate 0.25–1 mg/kg bwt i.v.) to decrease meningeal inflammation and to ameliorate neurological signs. In addition, 7 foals received dimethyl sulphoxide (1000 mg/kg bwt q. 12 h i.v.). Other treatments administered included: plasma transfusion in neonatal foals with FTPI (4/9), nonsteroidal anti-inflammatory drugs (6/10, flunixin meglumine 0.25 mg/kg bwt q. 8 h or 0.5 mg/kg bwt q. 12 h i.v.), and omeprazole (10/10, 2 mg/kg bwt q. 24 h *per os*).

Outcome

One neonate and the 5-month-old filly survived. Two foals died due to respiratory arrest and the other 6 were subjected to euthanasia: 2 due to the owners' economical limitations; one because of unresponsive *status epilepticus*; one foal had frequent apnoeic periods; and 2 because of progressive neurological signs consistent with increased intracranial pressure (mydriasis with absent pupillary light reflexes, mental status deterioration and development of horizontal or vertical nystagmus) and seizures.

Discussion

The most commonly described findings in foals with bacterial meningoencephalitis are lethargy, weakness, fever, cervical pain, blindness and, in advanced cases, seizures and coma (Pellegrini-Masini and Livesey 2006; Furr 2008). Regarding laboratory tests, reported findings include FTPI, neutrophilia and hyperfibrinogenaemia, as well as pleocytosis and increased TP in the CSF (Moore 1995; Pellegrini-Masini and Livesey 2006). In this retrospective study, the main clinical findings in foals with bacterial meningoencephalitis were altered mental status, recumbency, altered pupillary light reflexes and decreased suckling reflex. Relevant observations acquired from this study include: 1) apart from altered mental status and seizures, other previously reported clinical signs observed frequently in foals with bacterial meningoencephalitis were uncommon in the animals in this study; 2) severe sepsis was not essential to develop bacterial meningoencephalitis; 3) marked CSF pleocytosis was detected in most, but not all cases; and 4) all CSF and blood cultures (8/9) rendered Gram-negative enterobacteria

Bacterial meningitis is an uncommon condition in mature horses. It has been associated with infectious diseases involving the head (Smith *et al*. 2004; Mitchell *et al*. 2006), trauma (Cornelisse *et al*. 2001) or common variable immunodeficiency (Pellegrini-Masini *et al*. 2005). *Streptococcus equi equi* meningoencephalitis (Finno *et al*. 2006) or abscessation (Smith *et al*. 2004; Pellegrini-Masini and Livesey 2006), and *Rhodococcus equi*

abscessation (Janicek *et al*. 2006) have also been reported in young horses. On the other hand, bacterial meningoencephalitis is considered a complication that can appear in 8–10% of newborn foals with confirmed sepsis. This incidence rate was reported in the 1980s (Koterba *et al*. 1984; Brewer and Koterba 1990), but no further studies about bacterial meningoencephalitis in foals have been reported since. In more recent clinical studies on sepsis, meningitis was detected in only 2.6% of cases (Sanchez *et al*. 2008). In the present study the incidence of bacterial meningoencephalitis in septic neonates is 5.2%.

The increased permeability of the neonatal blood–brain barrier (BBB) together with FTPI are risk factors for bacterial meningoencephalitis in neonatal foals (Pellegrini-Masini and Livesey 2006; Mayhew 2009). However, in the present study only 4/9 newborn foals had FTPI. Another important point to take into account is the severity of sepsis. The sepsis score is based on predisposing factors and clinical parameters, and it has traditionally been considered a useful tool for the diagnosis of sepsis in critically ill foals (Mayhew 2009). This score was further re-evaluated in order to predict sepsis in foals (Corley and Furr 2003) and it was demonstrated to have lower sensitivity (67%) and specificity (79%) than originally described (93% and 86%, respectively). More recently, the sepsis score was used to assess severity of sepsis in neonatal foals and it was shown to be negatively correlated with survival (Peek *et al*. 2006). In the present study 8/9 neonates developed bacterial meningoencephalitis, even though their sepsis scores were either just below or above the cut-off value (values between 9 and 13).

Alternatively, severe systemic derangements (severe sepsis or HIE) can mask or mimic neurological signs of bacterial meningoencephalitis (BME) and falsely diminish the true incidence of the condition. This is a well-recognised problem in human neonates. In infants, lumbar CSF collection is recommended in cases with suspected or proven late onset sepsis (i.e. infection coming from extrauterine environment), and highly advisable in all neonates with possible sepsis (Heath *et al*. 2003). In the present study, 4 neonates with bacterial meningoencephalitis were not diagnosed *ante mortem*. One of them was diagnosed at necropsy and the other 3 when CSF was analysed immediately after death, and a large number of intracellular and extracellular bacteria were observed on cytological examination. These findings may suggest that the true incidence of bacterial meningoencephalitis is higher than clinically recognised.

The most frequently reported primary signs in foals with bacterial meningoencephalitis are hyperaesthesia, neck stiffness, muscle tremors, somnolence, seizures and blindness. Bacterial meningoencephalitis is also associated with fever and sluggish suckling reflex in newborn foals (Pellegrini-Masini and Livesey 2006; Mayhew 2009). Some of these signs were not observed or were anecdotal in the foals reported herein (i.e. tremors, blindness, cervical stiffness). Most of the typical bacterial meningoencephalitis signs are common to other neonatal diseases, especially HIE in foals. In fact, some of the foals of this study also had HIE, and bacterial meningoencephalitis was diagnosed *post mortem* in one of them because there was no clinical suspicion of the condition.

Neutrophilia (with or without a left shift) and hyperfibrinogenaemia are the most common clinicopathological findings in mature horses with infectious meningitis (Pellegrini-Masini and Livesey 2006). In newborn foals, low serum IgG concentration, hyperfibrinogenaemia, neutropenia or neutrophilia and electrolyte imbalances have been reported (Moore 1995). The clinicopathological alterations observed in the present study were similar to those previously reported.

Neutrophilic pleocytosis and increased TP are the most commonly reported findings in the CSF of horses with infectious meningitis, although cases with normal CSF cell counts have been described (Pellegrini-Masini and Livesey 2006). In the present study, 3 foals had slight increases in CSF TNCC. The TP concentration was determined in 2 of these foals and was normal or close to normal (1.49 and 0.77 g/l). However, intracellular bacteria were observed in 2 of the 3 foals with mild pleocytosis. As previously reported, severe neutrophilic pleocytosis and increased TP concentration were found in all the other foals that had a CSF analysis performed (Furr 2008).

Central nervous system infections in newborn foals are most commonly associated with bacteria causing generalised sepsis (Santschi and Foreman 1989; Moore 1995; Furr 2008). Hence, Gram-negative bacteria predominate in both neonatal bacterial meningoencephalitis and sepsis (Sanchez *et al*. 2008; Stewart *et al*. 2008). On the other hand, the most frequently isolated agents in CNS infections of older animals are Gram-positive bacteria, such as streptococcal species. *E. coli* is the most commonly reported bacterium in septic newborn foals with meningitis (Seino 2007; Furr 2008) and it is also a frequent isolate in human neonatal meningitides (Heath *et al*. 2003). Isolated agents from blood and CSF cultures from neonatal foals with bacterial meningoencephalitis include *Actinobacillus* spp., *Klebsiella* spp., *Streptococcus* spp. (Seino 2007) and less frequently *Salmonella* spp (Furr 2008). The previously reported incidence of these bacteria is similar to the one observed in the neonatal group of our study.

Cerebrospinal fluid culture was performed in only 4 cases of the present study. Although chances of bacterial growth on CSF samples are low after antibiotic therapy has been initiated, CSF culture should have been performed more often given the useful information obtained in the few positive cases. Furthermore, literature reports indicate no limitations in the ability to isolate bacteria from CSF within 1–2 h after initiation of antimicrobial treatment (Pellegrini-Masini and Livesey 2006).

The prognosis of bacterial meningoencephalitis has been reported to be fair to poor (Seino 2007). Reported survival rates in mature horses ranged from 0–60% in 2 small case series (Smith *et al*. 2004; Mitchell *et al*. 2006). In newborn foals, the reported survival rate is 0% (Brewer and Koterba 1990; Sanchez *et al*. 2008). In the present study, one neonatal foal was discharged with no neurological deficits and the 5-month-old filly was also discharged with mild neurological deficits (left thoracic limb paresis) that resolved one month after discharge. Most of the nonsurviving foals were subjected to euthanasia, 2 of them due to economical restraints, despite the neurological improvement observed in one of them. Others were subjected to euthanasia due to poor prognosis, but it is difficult to know whether any of them would have survived with intensive care and longer treatment. In addition, the study includes a small number of foals, so it is very likely that estimation of survival rates would be inaccurate.

In conclusion, clinical signs in newborn foals with bacterial meningoencephalitis are nonspecific and frequently similar to those of other common diseases affecting foals. Clinical signs and suspicion of bacterial meningoencephalitis may be overlooked in septic and HIE foals due to the vague and nonspecific clinical signs present in these animals. Appropriate antibiotic selection in these cases can also be difficult, because not all antibiotics cross the BBB. Antimicrobials commonly used in neonatal foals with sepsis (ceftiofur or penicillin combined with aminoglycosides) are not adequate to treat bacterial meningoencephalitis, given their lack of penetration into the CNS. Therefore early and accurate diagnosis of bacterial meningoencephalitis should be based on results of complementary diagnostic techniques, such as CSF analysis and culture, and it is essential to initiate proper antimicrobial treatment.

Authors' declaration of interests

No conflicts of interest have been declared.

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