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Brain abscesses as a metastatic manifestation of strangles: symptomatology and the use of magnetic resonance imaging as a diagnostic aid

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Summary

Reasons for performing study: **The occurrence of unexpectedly high numbers of horses with neurological signs during two outbreaks of strangles required prompt in-depth researching of these cases, including the exploration of magnetic resonance imaging (MRI) as a possible diagnostic technique.**

Objectives: **To describe the case series and assess the usefulness of MRI as an imaging modality for cases suspected of space-occupying lesions in the cerebral cavity.**

Methods: **Four cases suspected of suffering from cerebral damage due to *Streptococcus equi* subsp. *equi* infection were examined clinically, pathologically, bacteriologically, by clinical chemistry (3 cases) and MRI (2 cases). In one case, MRI findings were compared to images acquired using computer tomography (CT).**

Results: **In all cases, cerebral abscesses positive for *Streptococcus equi* subsp. *equi* were found, which explained the clinical signs. Although the lesions could be visualised with CT, MRI images were superior in representing the exact anatomic reality of the soft tissue lesions.**

Conclusions: **The diagnosis of bastard strangles characterised by metastatic brain abscesses was confirmed. MRI appeared to be an excellent tool for the imaging of cerebral lesions in the horse.**

Potential clinical relevance: **The high incidence of neurological complications could not be explained but possibly indicated a change in virulence of certain strains of *Streptococcus equi* subsp. *equi*. MRI images were very detailed, permitting visualisation of much smaller lesions than demonstrated in this study and this could allow prompt clinical intervention in less advanced cases with a better prognosis. Further, MRI could assist in the surgical treatment of brain abscesses, as has been described earlier for CT.**

Introduction

Strangles, a purulent pharyngitis and lymphadenitis of the upper respiratory tract, is highly contagious for naive horses; unique to equids, ubiquitous and caused exclusively by *Streptococcus equi* subspecies *equi*. (Bazeley and Battle 1940). Formerly, the causative agent was supposed to show no serological or genetic variation (Timoney 1993), but recently repetitive sequence-base polymerase chain reaction (rep-PCR) was used to delineate *Streptococcus equi* subsp. *equi* into rep-PCR subtypes, evidencing true genetic variation (Al-Ghandi *et al.* 2000). Takai *et al.* (2000) demonstrated differences in genomic sequence with pulsed-field gel electrophoresis. Further, Chanter *et al.* (2000) discovered greater occurrence of variants of *Streptococcus equi* subsp. *equi* among carriers within cases of clinical strangles and suggested that truncation of the M-protein could be linked to a reduction in virulence.

In most uncomplicated cases, *Streptococcus equi* causes a focal transient lymphadenopathy and abscessation of the mandibular and retropharygeal lymph nodes (Sweeney *et al.* 1987a; Timoney 1993; Foreman 1999). In some cases, metastatic abscesses develop and other organs may become infected. This condition is called 'bastard' strangles. Metastatic abscessation has been described in the lungs, mesentery, liver (Ford and Lokai 1980; Kaplan and Moore 1996), spleen, kidney (Jubb and Kennedy 1970) and brain (Ford and Lokai 1980; DeLahunta 1977; Nation 1978; Raphel 1982; Kaplan and Moore 1996). These metastatic abscesses may result in upper respiratory obstruction (Ford and Lokai 1980; Rooney 1979), guttural pouch empyema (Nation 1978, Rooney 1979) laryngeal hemiplegia (Pillers 1954), panophthalmitis (Kaplan and Moore 1996), periorbital (Rossdale and Ricketts 1974) and paravertebral abscesses (Rooney 1979); arthritis (Dalgleish *et al.* 1993; Meijer *et al.* 2001), tenosynovitis (Rossdale and Ricketts 1974), purpura haemorrhagica (Biggers and Ingram 1948; Jubb and Kennedy 1970; Rooney 1979; Sweeney *et al.* 1987b,c), myocarditis (Ford and Lokai 1980), endocarditis (Kaplan and Moore 1996), mastitis and changes in electrocardiographic findings (Bergsten and Persson 1966).

Metastatic abscesses in the central nervous system are rare. CT was used successfully to localise and treat intracerebral abscesses caused by *Streptococcus equi* subsp. *equi* in 2 horses (Allen *et al.* 1987; Cornelisse *et al.* 2001).

Here we report 4 cases of cranial abscesses caused by *Streptococcus equi* subsp. *equi*. In 2 cases, magnetic resonance imaging (MR) was used to confirm the presence and define the extent of the brain abscesses. In one case, the use of MR imaging was compared with CT.

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TABLE 1: Biochemical findings in *Cases 2*, *3* and *4*

Parameter	*Case 2*	*Case 3*	*Case 4*	Normal range
PCV (%)	0.27	0.28	0.36	0.36–0.42
WBC (x 10^9/l)	22.1	20.0	24.3	7.0–10.0
Neutrophils (%)	92	67	92	35–60
Lymphocytes (%)	8	27	8	30–35
Total protein (g/l)	89	64	67	60–90
Albumin (%)	32.2	46.2	45.4	35–55
α (%)	18.3	23.0	20.5	15–20
β (%)	29.9	13.4	19.8	0–22
γ (%)	28.6	17.4	14.3	0–25
Bilirubin (µmol/l)	10.7	15.0	38.8	0–40
BUN (mmol/l)	3.3	3.0	5.6	2–8
Creatinine ((µmol/l)	69	98	112	106–168

Case details

History

Cases 1 and *2:* A group of Shetland ponies purchased for research purposes and consisting of 9 intact males, age 2–7 years (mean 3.7). After regrouping, clinical signs of strangles developed in all 9 animals. Six out of 9 ponies recovered without treatment. One pony stayed febrile, anorectic, continued to lose weight and was subjected to euthanasia. Pathology revealed abscessation of the abdominal lymph nodes. Two ponies (age 3 and 4 years; *Cases 1* and *2)* developed neurological signs, approximately 1 month after onset of the first clinical signs in each case.

Cases 3 and *4:* A group of 16 Arabian foals from a studfarm where an outbreak of strangles was ongoing. Four foals from this group showed clinical signs of metastatic (bastard) strangles. Two animals recovered after treatment. The other 2, *Cases 3* and *4*, developed neurological signs and were referred to the Equine Referral Centre in Emmeloord and Department of Equine Sciences, Faculty of Veterinary Medicine, University of Utrecht, respectively.

Clinical findings and treatment

Case 1: The 3-year-old Shetland pony showed abscessation of the right retropharyngeal lymph node. The abscess was thin walled and apparently mature. After sedation and routine preparation for surgery, the lymph node was first punctured using a 19 gauge x 3.81 cm needle and, after retrieval of purulent exudate, lanced using the needle as guidance. The node was flushed once daily with diluted povidone iodine solution (Betadine)[1]. Despite surgical drainage, rectal temperature continued to fluctuate and the animal showed only mild clinical improvement. After 8 days, appetite was still decreased and the pony remained dull. Rectal temperature was 37.8–39.5ºC and, on Day 9, severe neurological signs developed, including head pressing, compulsive circling, depression and inconsistent ataxia. Because of the severity of signs, the pony was subjected to euthanasia and necropsy.

Case 2: In the 4-year-old Shetland pony an attempt was made to drain the abscess of the right retropharyngeal lymph node, using the same technique as in *Case 1*. Unfortunately, the attempt failed and resulted in a haematoma. Six days later the abscess was lanced successfully and subsequently flushed daily with diluted povidone iodine solution. Rectal temperature fluctuated between 38–39.9ºC. Five days after successful drainage of the abscess, the pony appeared dull, anorectic, showed compulsive circling to the right and a head tilt towards the right. Depression interchanged with excitation when manipulated and hyperaesthesia, inconsistent unilateral blindness, neck rigidity and an ataxic gait were noted. Haematology revealed mild anaemia, marked leucocytosis, with mature neutrophilia and a moderately elevated β and γ fraction. Biochemical details are shown in Table 1. The pony was treated with procaine penicillin G (Depocilline)[2] (22,000 iu/kg b.i.d.) i.m. for 4 days.

Neurological signs did not improve and the pony was, therefore, premedicated with acepromazine (Vetranquil)[3] and methadone (Methadon HCl)[4], anaesthesia induced by guaiphenesin (Myorelax)[5] and thiopentone (Nesdonal)[6] and maintained with halothane[7] in oxygen. The pony was positioned in right lateral recumbency and MR imaging performed using an open 0.2 Tesla Magnet (Magnetom Open)[8]. Contiguous 7 mm transverse and 10 mm dorsal slices were generated with T1-weighted (TR 560 msec, TE 15 msec), T2-weighted (TR 3020 msec, TE 112 msec) and PD-weighted (TR 3020 msec, TE 25 msec) spin-echo pulse sequences (Thomson *et al.* 1993; Mayrhofer *et al.* 1995; Chaffin *et al.* 1997; Arencibia *et al.* 2000). A 30 cm field-of-view was used and a large space-occupying lesion in the rostral part of the right hemisphere identified (Fig 1). The structure consisted of 2 spherical, rostrodorsal and caudoventral parts. The process was hypointense to isointense on T1-weighted images (not shown) and

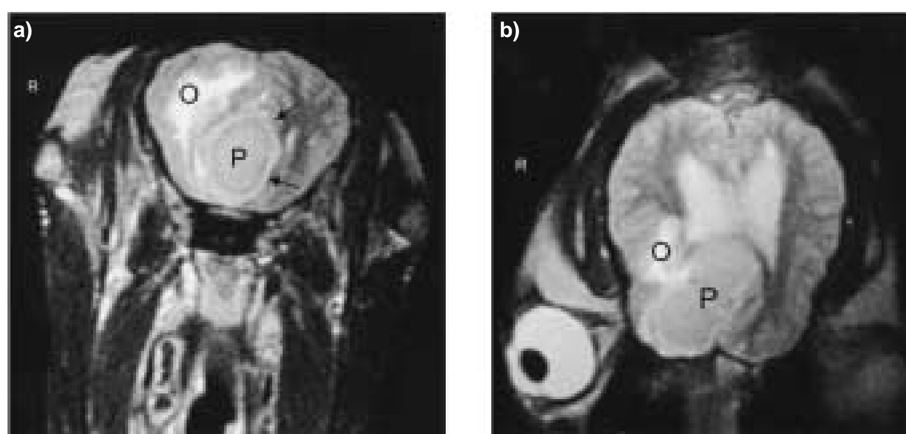

Fig 1: a) Transversal, (b) dorsal, (a,b) T2-weighted MR images of the brain in Case 2 *demonstrate a large space-occupying mass in the rostral part of the right (R) hemisphere. The images clearly demonstrate a hyperintense rim (black arrows) around the abscess (P) and surrounding oedema (O).*

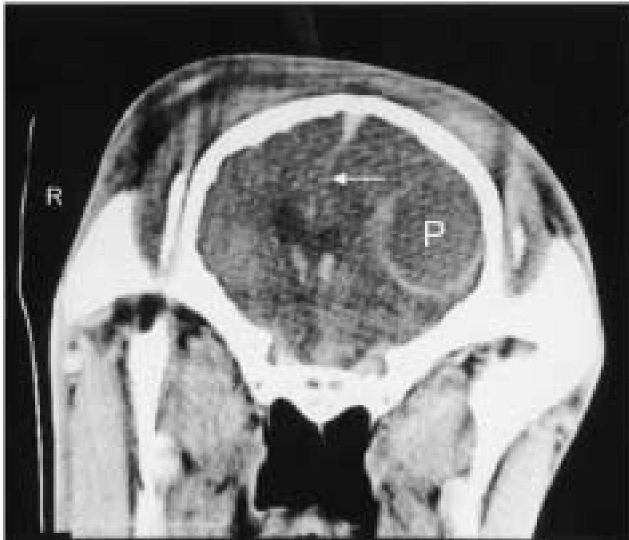

*Fig 2: Transversal CT image of the brain in a 4-month-old Arabian filly (*Case 4*) after injection of i.v. contrast medium reveals a large space-occupying lesion (P) in the left hemisphere (R = right) with displacement of the* falx cerebri *to the right (black arrows).*

hyperintense on T2-weighted images. T2-weighted images also showed an even more hyperintense rim around the process and strong hyperintensity of the remaining rostral parts of the right hemisphere (Fig 1). Additional observations were enlarged retropharyngeal lymph nodes on the transverse images and some hyperintense areas in the left maxillary and frontal sinuses on the transverse and dorsal images. The presumptive diagnosis was abscesses in the right hemisphere, surrounded by oedema. Because of the very poor prognosis the pony was subjected to euthanasia.

Case 3: A 2-month-old Arabian colt was presented with a rectal temperature of 40ºC. The foal was lethargic and held its head in an extended position. Mucous membranes were pale. Mandibular and retropharyngeal lymph nodes were mildly enlarged on palpation, but not painful. Haematology revealed leucocytosis with mature neutrophilia and mild anaemia with an elevated α-globulin fraction. Biochemical details are shown in Table 1. The initial therapy consisted of i.v. fluid support (lactated Ringers solution), hyperimmune serum (Hyperimmune FPT plasma)[9], dexamethasone sodium phosphate (Dexadreson)[10] (0.2 mg/kg, i.v.) and trimethoprim sulphadoxine (Borgal)[11] (30 mg/kg i.v. bid). During the following 24 h, rectal temperature dropped to within normal limits. However, neurological signs worsened. In addition to the persisting extension of the neck and lethargy, circling behaviour developed. Dimethylsulphoxide (DMSO)[12] (1000 mg/kg/l saline) was administered i.v. with dexamethasone sodiumphosphate[10] (0.2 mg/kg/500 ul). However, further progression of neurological signs, including the development of opisthotonus within the following 12 h, led to the decision to subject the foal to euthanasia.

Case 4: A 4-month-old Arabian filly was recumbent at presentation and showed periodic seizures. General examination did not reveal any abnormalities of respiratory nor circulatory systems. Rectal temperature was normal. On palpation, retropharyngeal lymph nodes were enlarged, mandibular lymph nodes were of normal size. The pupillary light reflex and menace response of the right eye were absent and anisocoria was present. There was a head tilt to the right. Spinal reflexes were normal. The foal showed a horizontal nystagmus, opisthotonus and had uncontrolled front limb movements (paddling). It was able to walk with assistance, but showed a right-sided hypermetria and leaned extremely to the right. The foal was sedated with diazepam (Diazepam)[13] (20 mg dosages, i.v.) to effect but, when it recovered from sedation, seizure activity reappeared and further anticonvulsive therapy was necessary. Phenobarbital (Fenobarbital 10%) [14] (12 mg/kg, i.v.) was administered over a 30 min period. Medically, initial treatment included i.v. fluid support (Ringer glucose 5%) at maintenance rates (2 ml/kg/h), gentamicin sulphate (Gentamicine 5% Injectie)[15] (6.6 mg/ kg i.v., s.i.d.), benzylpenicillin natrium (Benzylpenicilline Natrium)[16] (22,000 iu/kg i.v., q.i.d.), dexamethasone sodiumphosphate[10] (16 mg, i.v., s.i.d.) and thiamine HCl (Thiamine HCl)[4] (400 mg, i.v., s.i.d.).

Haematology revealed leucocytosis with mature neutrophilia and slightly elevated α-globulin fraction (Table 1). Because the foal was suspected of having a cerebral abscess, a CT (Philips Tomoscan CX/S)[17] examination was performed under general anaesthesia. The animal was positioned in right lateral recumbency with the head in maximal extension. Transverse contiguous 10 mm slices with 4.5 sec scanning time (120 kV, 220 mA) were made of the brain before and after i.v. injection of a

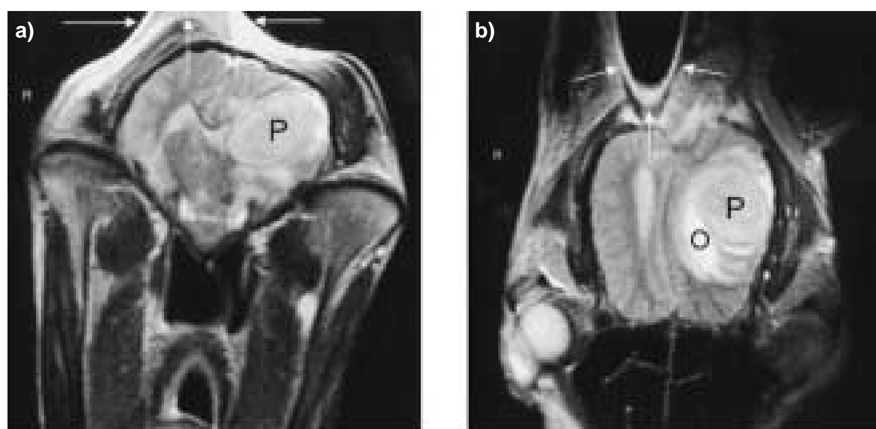

Fig 3: a) Transversal, (b) dorsal, (a, b) T2-weighted MR images of the brain in Case 4 *demonstrate the extent of the abscess. The images clearly differentiate between the abscess (P) and surrounding oedema (O). A large susceptibility artefact (white arrows) was noted in both images (R = right).*

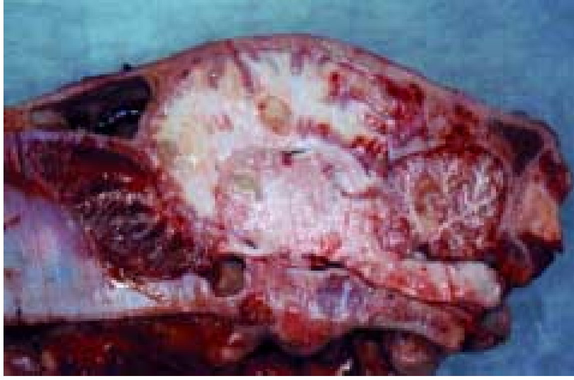

*Fig 4: Sagittal section of the head (*Case 1*) with several small abscesses of approximately 1 cm diameter in hemisphere and cerebellum.*

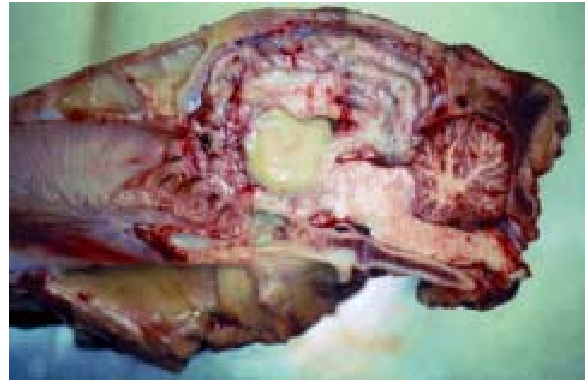

*Fig 5: Sagittal section of the head (*Case 2*) shows an inflamed frontal sinus and an abscess in the right hemisphere near the pituitary gland and brainstem.*

bolus of 360 ml of iodinated contrast medium (Telebrix 350)[19]. A large, space-occupying lesion in the left hemisphere, that had displaced the falx cerebri to the right, was noted. Visibility of the borders of the lesion was enhanced after the injection of contrast medium (Fig 2). The presumptive diagnosis was cerebral abscess.

Because of the severity of the clinical signs and poor prognosis, the foal was subjected to euthanasia. After this, MR imaging was performed. Contiguous 8 mm transverse and dorsal slices were generated with T1-weighted, T2-weighted, PD-weighted and IR-weighted (TIRM, TR 6000 msec, TI 3540 msec, TE 93 msec) spin-echo pulse sequences with a 30 cm field-of-view (Thomson *et al.* 1993; Mayrhofer *et al.* 1995; Chaffin *et al.* 1997; Arencibia *et al.* 2000). The transversal and dorsal images demonstrated the large space-occupying lesion in the left hemisphere well (Figs 3a, b). The process was isointense on T1-weighted (not shown) and hyperintense on T2-weighted images. T2-weighted images also demonstrated a hyperintense rim of oedema surrounding the lesion. Furthermore, a large so-called susceptibility artifact (Farahani *et al.* 1990) was noticed at the right dorsal side of the images. After the MRI examination, the abscess was punctured transcranially and material aspirated. Cytology of a direct smear showed degenerated neutrophils with many chain-forming cocci.

Findings at necropsy

Case 1: Gross lesions were restricted to the lymphoid and nervous systems. No abnormalites were found in the thoracic and abdominal cavities. There was a fistula that drained the right retropharyngeal lymph node at the angle of the right mandible, which was surgical in origin. Both mandibular lymph nodes contained abscesses. Empyema of the left guttural pouch was present and the right guttural pouch showed no abnormalities. On sagittal section of the head, several small abscesses of approximately 1 cm diameter were visible in both hemispheres and in the cerebellum (Fig 4). Microscopically, there was a focal meningitis with infiltration of mononuclear cells and neutrophils. *Streptococcus equi* subspecies *equi* was cultured from the abscesses. 'Bastard' strangles, characterised by a purulent meningoencephalitis with abscess formation, was diagnosed.

Case 2: A surgical drainage canal, 4 cm ventral to the right wing of the atlas, was present in the right retropharyngeal lymph node. The left retropharyngeal lymph node was enlarged. Both contained thick, purulent secretion and the surrounding area was fibrotic. The mandibular lymph nodes were normal. Both frontal sinuses were inflamed without indication of tooth root abscesses. In the diaphragmatic lobe of the left lung, a small (1 cm diameter) focus of chronic inflammation was found. No abnormalities were noted in the abdominal cavity. On sagittal section of the head, an abscess was found in the right hemisphere in the proximity of the pituitary gland and brainstem (Fig 5). The abscess was 5 cm in diameter and surrounded by a thick capsule. Microscopically, this capsule consisted of fibro-angioblastic tissue. *Streptococcus equi* subspecies *equi* was cultured from the abscess. No culture was performed from the lung lesion. 'Bastard' strangles was diagnosed with a cerebral abscess and a chronic, circumscribed pneumonia.

Case 3: Multiple cerebral abscesses and a purulent meningitis were diagnosed macroscopically and microscopically. Further, early stages of pneumonia and hepatitis were found together with the presence of large amounts of *Parascaris equorum* in the small intestine. Cultures of abscesses, pneumonia and hepatitis were positive for *Streptococcus equi* subspecies *equi*. 'Bastard' strangles was diagnosed with a purulent meningoencephalitis.

Case 4: There was subcutaneous oedema at the right side of the pharynx. Also on the right side, near the ear base, a subcutaneous haematoma was present. Severe oedema was noted in the walls of both guttural pouches. The right guttural pouch showed empyema. Microscopically, the right retropharyngeal lymph node was fibrotic and oedematously enlarged. The left retropharyngeal lymph node was hyperplastic and oedematous. Abdominal lymph nodes and the mesenteric root showed no abnormalities. In the caudal part of the cerebrum an abscess 4 x 6 cm with a thin, hyperaemic capsule was found left to the midline. Microscopically, the capsule of the abscess appeared to be infiltrated by neutrophils, macrophages, lymphocytes and plasma cells. Also, depositions of collagen were seen. Surrounding brain tissue was oedematous with swollen astrocytes and perivascular cuffing. There were some necrotic and calcified neurons. *Streptococcus equi* subspecies *equi* was cultured from the cerebral abscess. The diagnosis was 'bastard' strangles, characterised by an extended lymphoblastic meningitis with cerebral abscess.

Discussion

The prevalence of 'bastard' strangles was very high in both groups of animals that are described in this study. Of a total of 25 affected

animals, 7 (28%) developed this form of the disease. Of these, 5 were subjected to euthanasia, 4 of them with neurological signs. This resulted in a strangles specific death rate of 20%. Sweeney *et al.* (1987c) reported a much lower strangles specific death rate of 2.7% (n = 74) which corresponded with other reports (Ford and Lokai 1980).

The high incidence may have been influenced by infection dose or differences in virulence of strains, which are known to affect the complication rate (Bazeley 1943; Timoney 1993; Chanter *et al*. 2000). Alternatively, differences in host susceptibility and possibly other unidentified underlying factors may be involved. It has been stated that antimicrobial treatment following the development of an abscess might contribute to metastasis and increase complication rate (Swerczek 1984), based on the theory that antimicrobial treatment alters the streptococcus organism and causes an inadequate immune response (Bryans and Moore 1972). However, Sweeney *et al*. (1987b) reported that no experimental or clinical data exist to support the theory that treatment of strangles with penicillin might increase the prevalence of 'bastard' strangles. Whether this is true or not, such a mechanism cannot have played a role in this study because antibiotics were not used before complications were seen. Only when clinical signs in *Cases 2*, *3* and *4* worsened and neurological signs were seen was antibiotic treatment started. *Cases 2* and *4* were treated with penicillins[2,16]. In *Case 3* trimethoprim sulphadoxine[11] was used because of good penetration into the brain. *Cases 3* and *4* were also treated with dexamethasone sodium phosphate[10] and dimethylsulphoxide[12] to control increase of intracranial pressure and reduce neurological sequelae (Mampalam and Rosenblum 1988; Cornelisse *et al.* 2001).

Several migratory routes can play a role in the development of 'bastard' strangles. Bacteraemia may be a sequel to an infection with *Streptococcus equi* (Evers 1968), but spread of bacteria may occur via lymphatics as well (Timoney 1993). In our cases, the lymphatic migration route is improbable because this route does not provide an entry to the CNS. Further, infection can occur *per continuitatem* when a structure is in close connection to a septic focus and connecting structures (eg. cranial nerves) may function as a transport mechanism for the infection.

In this study, the diffuse spread of abscesses in the brain in *Case 1* is suggestive of a haematogenous infection. Necropsy showed an infected left guttural pouch but no other abnormalities that might indicate another route. In *Case 2*, the single, confined abscess argues against a haematogenous route, although an embolic thrombus may have been involved. The frontal sinuses were infected in this case, but there was no pathological evidence that supported a *per continuitatem* route.

In 2 of the cases, migration may have been facilitated by surgical intervention because of neurological signs in *Cases 1* and *2*. Signs appeared after surgical intervention, 11 days after initial and 6 days after successful intervention, respectively. However, in *Case 2* the *post mortem* findings showed that pathological changes were substantially older, and not, therefore, probably related to the surgical procedure, although in *Case 1* the possibility remains.

Case 3 had multiple cerebral abscesses with a purulent meningitis. These changes, in the CNS, together with pneumonia and hepatitis, are again strongly suggestive of a haematogenous spread of *Streptococcus equi* subsp. *equi*. *Case 4* featured a single confined abscess but had several other changes at necropsy that might indicate different routes. The diffuse oedematous changes in the surrounding brain tissue and perivascular cuffing can be caused both by bacteraemia and lymphatic migration. However, here too an embolic thrombus cannot be excluded.

In the 4 cases described, neurological signs can be explained by the pathological findings. In *Cases 2* and *4*, there were clinical signs of unilateral central vestibular disease (ataxia, circling and head tilt) and of diffuse cerebral disease (depression, head pressing, hyperaesthesia and inconsistent unilateral blindness). In *Case 2,* compulsive movements were towards the side of the lesion, as would be expected (DeLahunta 1977). In *Case 4*, the abscess was localised contralateral to the side of the neurological signs. This could theoretically be explained by the paradoxic vestibular syndrome in which head tilt and ataxia may be directed to the side opposite the lesion (Oliver *et al.* 1987). The paradoxic vestibular syndrome has been reported in dogs (Palmer *et al.* 1974) and a horse (Raphel 1982). However, as in *Case 4*, the abscess was not located near the caudal cerebellar peduncles. A better explanation is that extensive meningitis and other diffuse changes may have been responsible for the signs. Impaired vision, as noted in *Cases 2* and *4*, has been reported consistently in association with equine brain abscesses with cerebral involvement (DeLahunta and Cummings 1967; DeLahunta 1977; Raphel 1982; Mayhew 1989).

Magnetic resonance imaging is a relatively new imaging modality, especially in veterinary medicine (Chaffin *et al.* 1997). Its use in equine medicine has been limited until recently, mostly because of logistic problems associated with the acquisition of MR images in large animals (Arencibia *et al.* 2000). With the open magnet design, the technique has become more readily available for equine imaging. To the authors' knowledge, there are no reports about the use of the technique for the visualisation of space-occupying processes in the equine central nervous system. In the normal foal, MR imaging is superior to CT for imaging the complex soft tissue structures in the brain (Chaffin *et al.* 1997). Used on the 2 cases in this study, MR imaging proved to be an excellent tool with a high correlation with findings at necropsy. In *Case 2* there was a hypointense and isointense lesion with a hyperintense rim. These findings were suggestive of an abscess with oedema, which was subsequently confirmed at *post mortem*. In *Case 4*, MR imaging showed clear advantages over CT imaging. With the use of CT, so-called beam hardening artifacts often obscure small pathological changes. Beam hardening artifacts are streak-like artifacts of low density which are caused by the absorption of the lower energy photons in the x-ray beam by large radio-dense structures like the skull base (Hathcock and Stickle 1993). MR imaging provided superior spatial resolution and soft tissue contrast with a better delineation of the abscess from the surrounding oedema. Furthermore, the acquisition of MR images can be accomplished in any plane, regardless of the orientation of the patient between the magnet, providing excellent anatomic insight. In *Case 4*, a large magnetic susceptibility artifact appeared, which originates when materials with large differences in ability of becoming magnetised in an external magnetic field are imaged. In spin-echo imaging these artifacts appear as a geometrical distortion of the boundary interface (Farahani *et al.* 1990). In our case, the haematoma at the right ear base caused a distortion of the delineation of the skull and brain in almost all images. However, this distortion did not influence the interpretation of the images.

The case series described in this study represents a rare, large number of neurological cases due to metastatic brain abscesses as a complication of a *Streptococcus equi* subspecies *equi* infection. The high prevalence may be coincidental, or may be a sign of

increasing severity of this age-old affliction of equids. In *Cases 1* and *2* it might even highlight the possible role of surgical drainage as an important factor in provoking metastasis. No reported data exist to support this theory.

The use of the MR imaging technique in 2 of the 4 cases provided high quality images of the involved soft tissue of the central nervous system, which correlated well with the *post mortem* findings. MR imaging, therefore, seems to be an excellent diagnostic tool that may be used for the assessment of the severity of the condition. However, the true worth of MR imaging will probably be in identifying smaller, more discrete lesions associated with much milder and subtle clinical presentations. The technique also may have value in developing and assessing treatment possibilities for these lesions, as has been described earlier for CT (Allen *et al.* 1987; Cornelisse *et al.* 2001).

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Manufacturers' addresses

[1]Asta Medica, Diemen, The Netherlands.
[2]Mycofarm Nederland B.V., De Bilt, The Netherlands.
[3]Sanofi Sante Animal Benelux B.V., Maassluis, The Netherlands.
[4]Veterinary Farmacy, Faculty Veterinary Medicine, Utrecht, The Netherlands.
[5]Eurovet, Bladel, The Netherlands.
[6]Rhône Mérieux bv, Amstelveen, The Netherlands.
[7]Sanofi Sante Animal Benelux B.V., Maassluis, The Netherlands.
[8]Siemens AG, Germany.
[9]Veterinary Immunogenics Ltd, Penrith, England.
[10]Intervet, Boxmeer, The Netherlands.
[11]Hoechst, Roussel Vet. Brussels, Belgium.
[12]Merck, Darmstadt, Germany.
[13]Centrafarm, Etten-Leur, The Netherlands.
[14]Pharmachemie B.V., Haarlem, The Netherlands.
[15]Eurovet, Bladel, The Netherlands.
[16]A.U.V./ Eurovet, Cuyk, The Netherlands.
[17]Philips, Eindhoven, The Netherlands.
[18]Laboratoire Guerbet, France.

References

Al-Ghandi, G.M., Kapur, V., Ames, T.R., Timoney, J.F., Love, D.N. and Mellencamp, M.A. (2000) Use of repetitive sequence-based polymerase chain reaction for molecular epidemiologic analysis of *Streptococcus equi* subspecies *equi*. *Am. J. vet. Res*. **61**, 699-705.

Allen, J.R., Barbee, D.D., Boulton, C.R., Major, M.D., Crisman, M.V. and Murnane, R.D. (1987) Brain abscess in a horse: Diagnosis by computed tomography and successful surgical treatment. *Equine vet. J.* **19**, 552-555.

Arencibia, A., Vázquez, J.M., Jaber, R., Gil, F., Ramírez, J.A., Rivero, M., González, N. and Wisner, E.R. (2000) Magnetic resonance imaging and cross sectional anatomy of the normal equine sinuses and nasal passages. *Vet. Radiol. Ultrasound* **41**, 313-319.

Bazeley, P.L. (1943) Studies with equine streptococci. V. Some relations between virulence of *Streptococcus equi* and immune response in the host. *Austr. vet. J.* **19**, 62-85.

Bazeley, P.L. and Battle, J. (1940) Studies with streptococci. I. A survey of beta-hemolytic streptococci in equine infections. *Austr. vet. J.* **16**, 140-146.

Bergsten, G. and Persson, S. (1966) Studies on the ECG in horses with acute strangles. In: *Proceedings of the 1st International Conference Equine Infectious Diseases,* Ed: J.T. Bryans, University of Kentucky, Lexington, USA. pp 76-81.

Biggers, J.D. and Ingram, P.L. (1948) Studies on equine purpura haemorrhagica. *Br. vet. J.* **104**, 214-220.

Bryans, J.T. and Moore, B.O. (1972) In: *Streptococci and Streptococcal Diseases: Recognition, Understanding and Management*, Eds: L.E. Wannamaker and J.M. Matson, 1st edn., Academic Press, New York. pp 327-338.

Chaffin, M.K., Walker, M.A., McArthur, N.H., Perris, E.E. and Matthews, N.S. (1997) Magnetic resonance imaging of the brain of normal neonatal foals. *Vet. Radiol. Ultrasound* **38**, 102-111.

Chanter, N., Talbot, N.C., Newton, J.R., Hewson, D. and Verheyen, K. (2000) *Streptococcus equi* with truncated M-proteins isolated from outwardly healthy horses. *Microbiol.* **146**, 1361-1369.

Cornelisse, C.J., Schott II, H.C., Lowrie, C.T. and Rosenstein, D.S. (2001) Successful treatment of intracranial abscesses in 2 horses. *J. vet. intern. Med.* **15**, 494-500.

Dalgleish, R., Love, S., Pirie, H.M., Pirie, M., Taylor, D.J. and Wright, N.G. (1993) An outbreak of strangles in young ponies. *Vet. Rec.* **132**, 528-531.

DeLahunta, A.A. (1977) *Veterinary Neuroanatomy and Clinical Neurology*, W.B. Saunders Co., Philadelphia. pp 25-32.

DeLahunta, A.A. and Cummings, J.F. (1967) Neuro-ophthalmologic lesions as a cause of visual deficit in dogs and horses. *J. Am. vet. med. Ass.* **150**, 994-1011.

Evers, W.D. (1968) Effect of furaltadone on strangles in horses. *J. Am. vet. med. Ass.* **152**, 1394.

Farahani, K., Sinha, U., Sinha, S., Chiu, L.C.L. and Lufkin, R.B. (1990) Effect of field strength on susceptibility artifacts in magnetic resonance imaging. *Comp. Med. Imag. Graph.* **14**, 409-413.

Ford, J. and Lokai, M.D. (1980) Complications of *Streptococcus equi* infection. *Equine Pract*. **2**, 41-44.

Foreman, J.H. (1999) Equine respiratory pharmacology. *Vet. Clin. N. Am.: Equine Pract.* **15**, 665-666.

Hatchcock, J.T. and Stickle, R.L. (1993) Principles and concepts of computed tomography. *Vet. Clin. N. Am.: Small Anim. Pract.* **23**, 399-415.

Jubb, K.V.F. and Kennedy, P.C. (1970) *Pathology of Domestic Animals*, 2nd edn., Vol. 1., Academic Press Inc., New York. pp 344-348.

Kaplan, N.A. and Moore, B.R. (1996) *Streptococcus equi* endocarditis, meningitis and panophthalmitis in a mature horse. *Equine vet. Educ*. **8**, 313-316.

Mampalam, T.J. and Rosenblum, M.L. (1988) Trends in the management of bacterial brain abscesses: A review of 102 cases over 17 years. *Neurosurg.* **23**, 451-458.

Mayrhofer, von E., Henninger, W., Hittmair, K. and Kneissl, S. (1995) Röntgenstrahlen, Ultraschall, Magnetresonanz–Möglichkeiten ihres Einsatzes in der Veterinärmedizin. *Wien. Tierärztl. Mschr.* **82**, 276-288.

Mayhew, I.G. (1989) *Large Animal Neurology*, 1st edn., Lea & Febiger, Philadelphia.

Meijer, M.C., van Weeren, P.R. and Rijkenhuizen, A.B.M. (2001) *Streptococcus equi* in the fetlock joint of a mature horse. *Equine vet. Educ.* **13**, 72-74.

Nation, P.N. (1978) Epistaxis of guttural pouch origin in horses. Pathology of three cases. *Can. vet. J.* **19**, 194-197.

Oliver, J.E., Hoerlein, B.F. and Mayhew, I.G. (1987) *Veterinary Neurology*, 1st edn., W.B. Saunders Co., Philadelphia. pp 40-42.

Palmer, A.C., Malinowski, W. and Barnett, K.C. (1974) Clinical signs including papilloedema associated with brain tumors in twenty-one dogs. *J. small anim. Pract.* **15**, 359.

Pillers, A.W.H. (1954) Remarks on the clinical aspects of contagious nasal catarrah in a horse. *Vet. Rec.* **14**, 1153-1160.

Raphel, C.F. (1982) Brain abscess in three horses. *J. Am. vet. med. Ass.* **180**, 874-877.

Rooney, J.R. (1979) Sequelae of strangles. *Mod. vet. Pract.* **60**, 463-464.

Rossdale, P.D. and Ricketts, S.W. (1974) *The Practice of Equine Stud Medicine,* Williams & Wilkins Co., Baltimore. pp 328-330.

Sweeney, C.R., Benson, C.E., Whitlock, R.H., Meirs, D.A., Barningham, S.O. and Whitehead, S.C. (1987a) *Streptococcus equi* infection in horses- part 1. *Comp. cont. Educ. pract. Vet.* **9**, 689-693.

Sweeney, C.R., Benson, C.E., Whitlock, R.H., Meirs, D.A., Barningham, S.O. and Whitehead, S.C. (1987b) *Streptococcus equi* infection in horses- part 2. *Comp. cont. Educ. pract. Vet.* **9**, 845-851.

Sweeney, C.R., Whitlock, R.H., Meirs, D.A., Whitehead, S.C. and Barningham, S.O. (1987c) Complications associated with *Streptococcus equi* infection on a horse farm. *J. Am. vet. med. Ass.* **191**, 1446-1448.

Swerczek, T.W. (1984) Pathogenesis, treatment and control of *Streptococcus equi* infection (strangles in horses). In: *Proceedings of Symposium on Trimethoprim/ Sulfadiazine*. Orlando, Florida. pp 26-30.

Takai, S., Anzai, T., Yashiro, H., Ishii, C., Tsubaki, S., Wada, R. and Timoney, J.F. (2000) Detection of DNA restriction fragment polymorphisms in *Streptococcus equi*. *Vet. Rec.* **146**, 159-161.

Thomson, C.E., Kornegay, J.N., Burn, R.A., Drayer, B.P., Hadley, D.M., Levesque, D.C., Gainsburg, L.A., Lane, S.B., Sharp, N.J.H. and Wheeler, S.J. (1993) Magnetic resonance imaging- a general overview of principles and examples in veterinary neurodiagnosis. *Vet. Radiol. Ultrasound* **34**, 2-17.

Timoney, J.F. (1993) Strangles. *Vet. Clin. N. Am.: Equine Pract*. **9**, 365-374.