

## Cerebellar peduncle abscess secondary to disseminated strangles in a six-week-old miniature foal

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### Abstract

During a strangles outbreak within a herd of miniature horses, a six week old foal developed acute onset clinical signs of sepsis and neurological deficits. The foal was euthanized and submitted for post-mortem at the Animal Health Laboratories, Guelph Ontario. Gross *post-mortem* examination noted severe bronchopneumonia, hypopyon of the right eye and a singular cerebellar peduncle abscess. Culture of the lungs and cerebellum produced a pure growth of *Streptococcus equi* ssp. *equi*. *Streptococcus equi* ssp. *equi*, the causative agent of equine strangles, produces an acute pyrexia, purulent lymphadenopathy of submandibular and retropharyngeal lymph nodes. Commonly, lymph node abscesses rupture and resolve without complication. Rarely, complications may include: dissemination of the bacteria with diffuse abscess formation, immune mediated disease (purpura haemorrhagica), rarely abscess formation within the central nervous system (CNS) can occur. These can be managed medically with appropriate antibiotics and drugs to reduce intra-cranial pressure, however surgical drainage and debulking of the abscess has been attempted successfully in a few cases.

### Herd history

In the spring of 2011, 8 horses were introduced to a herd of 20 miniature horses. Neither group of animals had been immunized with core vaccines and no quarantine protocol was carried out by the owner. Initial clinical signs of mucopurulent nasal discharge, swollen submandibular lymph nodes and pyrexia were displayed by the imported horses; approximately one month later, the original herd displayed similar signs of disease. One 24-hour-old foal died suddenly over night and two 4-6-week-old foals were euthanized when they became recumbent and showed clinical signs of sepsis.

Naso-pharyngeal swabs were submitted for culture, produced a growth of *Streptococcus equi* ssp., *zooepidemicus*. Repeat naso-pharyngeal and abscess swabs submitted for PCR

were taken approximately 1 week later. These yielded a positive result for *Streptococcus equi* ssp. *equi*.

### Six-week-old foal presentation

A 6-week-old foal born on the farm was seen as an emergency. Physical examination found the foal in lateral recumbency, unable to maintain a sternal position. Pupillary light reflex was absent and horizontal nystagmus was noted in both eyes. Hypopyon of the right eye was observed. The foal had a temperature of 38.4°C and a heart rate of 60 bpm. Mucous membranes were pink but tacky. The foal was struggling to breathe and crackles were auscultated throughout the right lung, while reduced lung sounds were auscultated on the left. No signs of colic were observed.

Prior to euthanasia blood samples were collected for hematology and biochemistry, the results of which showed a marked mature neutrophilia with evidence of mild-moderate toxic change. Increased serum haptoglobin and serum amyloid A were consistent with significant ongoing tissue inflammation/infection. Concurrent elevations in acute phase proteins and a mature neutrophilia suggested chronicity of disease.

Based on a poor prognosis for recovery, the foal was euthanized by intravenous injection of pentobarbital and submitted to the Animal Health Laboratory, Guelph Ontario, for *post-mortem* and histopathology.

### Post-mortem

Gross *post-mortem* results found the trachea and bronchi to contain viscous purulent exudates and a purulent bronchopneumonia of the antero-ventral half of the left caudal lobe. Multiple large abscesses (2-3 cm in diameter) were evident in all affected areas. The stomach was empty, while the small intestine was grossly distended with green mucoid contents. Examination of the cranium revealed a 1 cm abscess of the cerebellar peduncular area.

Culture of the lung and brain abscesses produced a pure culture of *Streptococcus equi* ssp. *equi*.

### Histopathology

Histopathology of the lung found a marked neutrophilic reaction within the lumens of some bronchioles. There was a marked generalized pulmonary congestion and oedema and erosion of the bronchiolar mucosa in affected lung lobes. Multiple abscesses, varying in size, were present. The lumen of these abscesses was filled with cellular debris, neutrophils and cocci. All of the abscesses were surrounded by macrophages, lymphocytes and plasma cells (Figure 1). Moderate lymphoid depletion and congestion of the red pulp was found within the spleen.

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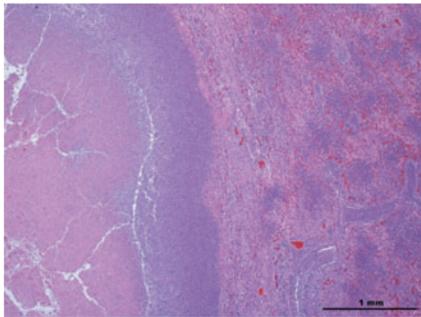
The large abscess of the cerebellar peduncle area was examined closely. Marked peri-vascular cuffing primarily by neutrophils was evident. Hypertrophy of endothelial cells, leukocytosis and fibrin thrombi were also present in some capillaries (Figure 2).

### Discussion

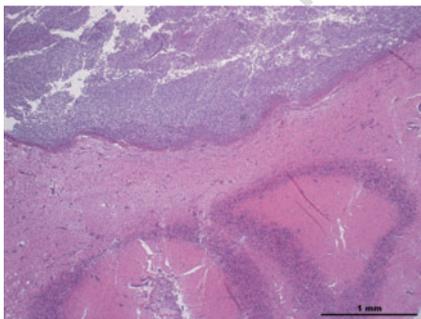
The causative agent of equine strangles is *Streptococcus equi* ssp. *equi*, which causes an acute pyrexia and purulent lymphadenopathy of the submandibular and retropharyngeal lymph nodes.<sup>1-3</sup> *Streptococcus equi* ssp. *equi* is a highly host adapted Gram-positive, beta-hemolytic coccus bacteria that is transmitted via the oro-nasal route in nasal secretions or via fomites.<sup>1</sup> The bacterium gains entry via the cryptal lymphoepithelium cells of the nasopharyngeal tonsil, which are continuous with the lymphoid tissue directly beneath.<sup>3</sup> Small numbers of bacteria are able to gain access to the tonsillar tissue, at which point they slowly replicate until they disseminate throughout the tonsils and regional lymph nodes.<sup>3</sup> Typically, abscesses rupture, drain and resolve

with few complications. However, complications that may occur include: i) abscessation of retropharyngeal lymph nodes that can lead to guttural pouch empyema, chondroid formation and chronic shedding of the bacterium;<sup>1</sup> ii) immune mediated conditions such as purpura haemorrhagica, myopathies and myocarditis;<sup>4</sup> iii) dissemination of bacteria leading to a condition colloquially known as *Bastard Strangles*, which may involve abscessation sites in the lung, mesentery, liver, spleen, kidneys and brain.<sup>5</sup> While there have been reports of metastatic abscesses in the central nervous system (CNS), these are considered to be a rare sequelae of this disease.

Factors involved in the likelihood of metastatic spread include: i) infection dose; ii) variation in strain virulence; and iii) variations in host susceptibility. The immunological naivete of this foal likely played the largest role in the rapid dissemination of bacteria. Mechanisms of dissemination include lymphatic migration, haematogenous spread<sup>2</sup> or *per continuitatem*; when infected structures are in close or connected proximity to other vulnerable structures<sup>5</sup> (i.e. retropharyngeal



**Figure 1. Severe neutrophil and macrophage infiltration of the lung parenchyma consistent with abscess formation.**



**Figure 2. Cerebellar peduncle abscess.**

lymph node spread to the guttural pouches or spread via the cranial nerves). Haematogenous spread or local extension is the most likely mechanism of dissemination in this case, as lymphatic migration does not reach the central nervous system.<sup>5</sup>

In contrast to human patients, bacterial meningitis/abscessation in horses and ruminants is a typical sequelae to systemic disease. Alternative theories of local extension have been proposed, whereby upper respiratory infections, dental disease, periocular lesions and traumatic head injuries could offer a primary septic foci from which local extension to the CNS could occur.<sup>5,6</sup>

The presence of multiple CNS abscesses and purulent meningitis alongside pneumonia and abdominal abscess formation suggest hematogenous spread.<sup>5</sup> Furthermore, hypopyon is a confirmed indicator of haematogenous spread.<sup>7</sup> In this case, a single cerebellar peduncle abscess was found alongside diffuse, severe bronchopneumonia and hypopyon of the right eye. Based on the biochemical/haematological markers of severe inflammation and advanced sepsis, haematogenous spread is the most likely pathway. However, the close anatomical association between the cerebellar peduncle and optic chiasm prevents local extension from being excluded as a possible pathogenesis of the single cerebellar peduncle abscess.

Medical management of bacterial meningoencephalitis consists of concurrent administration of corticosteroids, osmotic agents and antibiotics<sup>8</sup> to restore blood-brain barrier permeability, reduce intra-cranial pressure and control the inciting infection. A combination of anti-inflammatories (dexamethasone), mannitol and specifically bactericidal antibiotics are recommended. Dimethylsulfoxide (DMSO) has been used to reduce intracranial pressure (ICP); however, a severe rebound increase in ICP has been noted.<sup>8</sup>

In addition, mannitol has a greater efficacy when used in properly hydrated animals. A surgical approach to intracranial abscesses has been described in the literature previously.<sup>8</sup> Surgical drainage and debulking has been attempted to relieve intracranial pressure and remove the infective foci, with minimal success. Prognosis can be increased through the use of CT or MRI to map the lesion prior to surgery.<sup>8,9</sup> The primary goals of surgical debridement should be: i) clinical diagnosis/biopsy; ii) intracranial mass debulking, resection and decompression of neural tissue.<sup>8</sup>

Metastatic spread of *Streptococcus equi* ssp. *equi* to the CNS is a rare occurrence. Route of

entry can be haematogenous, local extension or invasion via the cranial nerves.<sup>6</sup> Multi-focal abscess formation is a strong indicator of haematogenous spread. Local extension of infectious foci has been highlighted as a possible route, specifically when discussing the closely related structures of the equine head.<sup>6</sup> In this case, the singular cerebellar peduncle abscess could have formed from either haematogenous spread or local extension via the optic nerve.

The significant lesion identified in this foal highlights the need for meticulous preventative medicine and biosecurity protocols when introducing new horses to a herd.

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