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# MANAGEMENT AND COMPLICATIONS OF A STRANGLES OUTBREAK

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***Abstract***

*The present study is a report of a strangles outbreak in a horse breeding farm. The clinical signs began to appear in the first week of July, and included coughing, purulent nasal discharge, lymph node abscesses and fever. Bacteriological tests confirmed Streptococcus equi ssp. equi infection. The horses were treated with Penicillin 12 mg/kg, from the onset of the clinical signs: 38 of the 42 affected horses showed improved clinical status after 5-10 days of therapy. The other 4 foals were diagnosed Rhodococcus equi induced pneumonia, and they were placed under rifampicin (7.5 mg/kg) and claritromicin (7.5 mg/kg) treatment. Of the 38 horses treated successfully with penicillin, 6 horses aged 3 to 6 months, were later dignosed with Streptococcus equi ssp. zooepidemicus induced pneumonia, and were also treated with rifampicin and clarithromicin. By the end of September, none of the horses present on the farm showed any more signs of respiratory infections. Daily monitoring of the herd, active bacteriological surveillance and early onset of antibiotic therapy were key factors in avoiding severe complications of strangles and limiting mortality.*

***Key words****: Equine strangles, horses, respiratory infection, Streptococcus equi, Streptococcus equi ssp. Zooepidemicus.*

## INTRODUCTION

Strangles is one of the most frequently diagnosed and important infectious diseases of horses worldwide. The disease is highly contagious, affects mainly young horses, but mature, naive horses may also be susceptible (Piche, 1984). The etiological agent of strangles is *Streptococcus equi* subspecies *equi* (*S. equi*), a β-hemolytic Lancefield group C bacterium, first identified in 1888 (Schutz, 1888). After an incubation period of 3 to 14 days, the acute form of the disease is characterized by fever, abscessed lymph nodes of the head and neck, dyspnea and anorexia. Following the onset of fever, in the first 24 hours, the affected animals develop bilateral serous or mucoid nasal discharge, that later becomes mucopurulent (Neamat-Allah and Damaty, 2016). Initially, submandibular and retropharyngeal lymph nodes are enlarged and firm, and 7 to 10 days later they begin to fluctuate and may rupture and drain into the guttural pouches and via the nasopharynx, or directly through the skin, into the environment. In susceptible populations, the infection can reach a morbidity of up to 100%, but a low mortality rate (10%), if the appropriate treatment is implemented. Most of the affected horses fully recover within an average period of 3 weeks (Roberts, 2014). Following infection, if horses recover without antibiotic treatment, 75% of them develop a protective immune response that lasts up to 5 years. There is evidence that treatment with penicillin during the acute stage of strangles can impair the persistence of antibodies against *S. equi*, decreasing the duration of the serological response (Pringle et al., 2020). However, during an emerging outbreak, the early start of antibiotic therapy increases the chances of complete recovery and can prevent complications (Boyle et al., 2018). In approximately 20% of *S. equi* infections, severe complications can occur, such as metastatic infections (“bastard strangles”) (Berlin et al., 2013), suppurative necrotic bronchopneumonia, guttural pouch empyema and chondroids, purpura hemorrhagica, muscle infarction and rhabdomyolysis (Constable et

al., 2017). Also, there is evidence that on extremely rare occasion, *S. equi* infections can become zoonotic, causing infections of the central nervous system in humans (Kerstens et al., 2021).

## MATERIALS AND METHODS

The outbreak of strangles emerged in a horse breeding farm, housing at the time 142 horses: 8 yearlings, nine 2 year old horses, 31 foals aged 1 to 6 months, 4 stallions, 70 mares used for reproduction and 20 mares used as surrogates in the reproduction process. The dynamics of the size and structure of the population were due to the newborn foals during that period (31 foals were born between January and July). Occasionally, some of the horses traveled abroad for competitions. None of the horses had left the farm in the months before the outbreak; however, 4 new, clinically healthy mares were introduced to the population in June, approximately 4 weeks before the outbreak began in the first week of July. The first horses to exhibit symptoms of strangles were 4 of the yearlings and 5 foals aged 3-6 months: the horses presented with inflamed submadibular lymph nodes, cough, purulent nasal discharge and slightly elevated rectal temperatures, ranging between 38.8°C and 39.5°C. Nasal swab samples were collected for bacteriological investigation and antibiotic susceptibility tests. The affected animals were placed under treatment with penicillin, 12 mg/ kg bodyweight, administered intramuscularly, for 5-7 days. The infection spread rapidly throughout the stable, and within 3 days, 11 other foals, aged 1 to 4 months, showed similar symptoms and were placed under therapy. Due to the large number of recent births and the characteristics of the farm (limited capacity of the stables, limited staff), an adequate quarantine area, in order to limit the spread of the infection was not set. By the end of July, all of the foals, yearlings, one of the 2 year-old horses and 2 adult mares had shown symptoms of strangles, with various degrees of severity, and were treated accordingly. The affected horses were examined daily, rectal temperatures, abnormal respiratory noises and the evolution of the clinical signs being recorded. Non steroidal anti-inflammatory medication (flunixin 1.1 mg/kg bodyweight) was administered intravenously in cases where temperature exceeded 39°C. Horses with persistent cough and abnormal respiratory sounds (crackles and wheezing) received, in addition to antibiotic, bronchodilator and mucolytic medication (Venti Plus) as an orally administered product containing clenbuterol hydrochloride (0.8 g/kg bodyweight) and dembrexin hydrochloride (0.3 mg/kg bodyweight). There were 42 cases of *S. equi* infections confirmed on the farm in July. Clinical signs observed throughout the breakout included:

- submandibular and retropharyngeal lymph node inflammation - lymph nodes enlaged, firm, and painful to palpation; this was the first symptom noticed in all the affected animals, regardless of age. After 4-5 days, the lymph nodes became fluctuant, ruptured and drained, mainly via the nasopharinx;
- bilteral mucopurulent nasal discharge - present in all the affected animals;
- coughing, initially dry, later became productive - very frequent in all young animals; the infected mares presented only dry cough in the first 2-3 days after the clinical onset;
- fever - the majority of the horses registered slightly elevated body temperature (38.8-39.5°C) at onset of the disease, that returned to normal after 1 – 3 days of antibiotic therapy;
- dyspnea and abnormal respiratory sounds (wheezing and crackling) - present in some of the more severe cases of strangles in 1 to 6 month old foals. Approximately 50% of the affected foals presented these symptoms, along with persistent productive cough, requiring up to 10 days of antibiotic therapy;
- guttural pouch empyema - one of the more severely affected foals, a 3 month old filly, developed guttural pouch empyema.

Of the 42 affected horses, 4 foals aged 2-3 months did not respond to the penicillin treatment, presenting with elevated temperature (39.3-39.6°C), lethargy, cough and dyspnea, 7 days into the antibiotic therapy. Based on the history of the farm, *Rhodococcus equi* pneumonia was suspected. Pulmonary ultrasounds and bacterial examinations of nasal

swab samples were performed in order to confirm the diagnosis. The foals and their mothers were separated from the herd and were treated with rifampicin 7.5 mg/kg and clarithromicin 7.5 mg/kg, orally, twice daily for 8 weeks, orally administered probiotics, mucolytic medication, flunixin 1.1 mg/kg, when necessary to decrease high temperature, and vitamin C, 1 g/animal/day, for 5 days, intravenously. The foals were closely monitored daily, and pulmonary ultrasounds were performed weekly in order to assess the evolution of the pulmonary lesions. One of the 4 foals was unresponsive to the treatment and died 15 days after the *R. equi* infection was confirmed. Necropsy and bacteriological examinations were performed in order to establish the cause of death.

Of the 38 horses affected by *S. equi* infection and treated successfully with penicillin, 6 horses aged 3-6 months reported recurrence of respiratory distress, coughing and purulent nasal discharge 2-3 weeks after the remission of the initial symptoms. The clinical signs indicated towards a relapse of strangles, and nasal swab samples were collected for bacteriological examination. One 4 month old filly presented more severe symptoms such as peracute respiratory distress, complete loss of appetite, high fever (40.1°C) and died the day after the symptoms were first observed. Necropsy was performed and samples of lung tissue were collected for bacteriological examination. Nasal swab and lung tissue cultures identified *Streptococcus equi* ssp. *zooepidemicus*, and the 5 remaining foals were placed under treatment with rifampicin 7.5 mg/kg and clarithromicin 7.5 mg/kg, per os, twice daily, according with results of the antibiotic susceptibility test. They were also given probiotics and mucolytic drugs during the treatment, and vitamin C 1 g/animal/day, for the first 5 days, intravenously.

For the bacteriological examinations, Columbia agar with 5% sheep blood was used for seeding and isolation of the bacterial strains. The identification of the isolates was performed based on cultural characteristics, morphological aspects examined microscopically using the Gram stain method and biochemical characteristics, determined using API strips (Biomerieux): API 20 Strep for *S. equi* and *S. equi* ssp. *zoopeidemicus*, and API Coryne for *R. equi*. The interpretation of the API test results was performed according to the producer's instructions, using the provided software. The *R. equi* isolate was also seeded on selective culture media: MacConkey agar and Lowenstein-Jensen agar. For the antibiotic susceptibility tests, performed by disc diffusion method, Liofilchem antimicrobial discs were used, and the results were interpreted according to Liofilchem and EUCAST standards.

## RESULTS AND DISCUSSIONS

**Bacteriological examinations**. Cultural and morphological characteristics of *Streptococcus equi* ssp. *equi* and *Streptococcus equi* ssp. *zooepidemicu*s were identical: the colonies were small, smooth, translucent, shiny, β-hemolytic on Columbia blood agar after 20 hours incubation at 37°C; microscopic examination revealed Gram positive, chain forming cocci.

*R. equi* colonies were medium-sized, whitish, mucoid, confluent and presented weak alpha-hemolysis on Columbia blood agar, after 48 hours incubation at 37°C. On MacConkey agar, the colonies were transparent, shiny and lactose positive. On Lowenstein – Jensen agar, the culture developed slowly, becoming evident after 72 hours incubation at 37°C. The morphological examination of the cultures revealed Gram- positive polymorphic bacteria, with a tendency to form chains.

**Biochemical characteristics**. The catalase and oxidase tests were negative for the two *Streptococcus* stains. The *R. equi* isolate was catalase positive and oxidase negative. The biochemical characteristics of the three isolates, determined using API (Biomerieux) tests, are detailed in Tables 1, 2 and 3.

Table 1. Biochemical characteristics of the *Streptococcus equi* ssp. *equi* isolate

| VP | HIP | ESC | PYRA | AGAL | BGUR | BGAL | PAL | LAP | ADH | RIB | ARA | MAN | SOR | LAC | TRE | INU | RAF | AMD | GLYG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| - | - | - | - | - | + | - | + | + | + | - | - | - | - | - | - | - | - | + | + |

Legend: VP – Acetoin production (Voges Proskauer), HIP – Hydrolysis of hipuric acid, ESC – esculin, PYRA – PYRrolidonyl Arylamidase, AGAL – α-GALactosidase, BGUR – β-GIUcuRonidase, BGAL – β-GALactosidase, PAL – Alkaline Phosphatase, LAP – Leucine AminoPeptidase, ADH – Arginine DiHydrolase, RIB – Ribose, ARA – Arabinose, MAN – Mannitol, SOR – Sorbitol, LAC – Lactose, TRE – Trehalose, INU – Inulin, RAF – Raffinose, AMD – Amidon, GLYG – Glycogen, "+" - positive result, "-" – negative result.

Table 2. Biochemical characteristics of the *Streptococcus equi* ssp. *zooepidemicus* isolate

| VP | HIP | ESC | PYRA | AGAL | BGUR | BGAL | PAL | LAP | ADH | RIB | ARA | MAN | SOR | LAC | TRE | INU | RAF | AMD | GLYG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| + | - | - | - | - | + | - | + | + | + | + | - | - | + | + | - | - | - | + | + |

Legend: VP – Acetoin production (Voges Proskauer), HIP – Hydrolysis of hipuric acid, ESC – esculin, PYRA – PYRrolidonyl Arylamidase, AGAL – α-GALactosidase, BGUR – β-GIUcuRonidase, BGAL – β-GALactosidase, PAL – Alkaline Phosphatase, LAP – Leucine AminoPeptidase, ADH – Arginine DiHydrolase, RIB – Ribose, ARA – Arabinose, MAN – Mannitol, SOR – Sorbitol, LAC – Lactose, TRE – Trehalose, INU – Inulin, RAF – Raffinose, AMD – Amidon, GLYG – Glycogen, "+" - positive result, "-" – negative result.

Table 3. Biochemical characteristics of the *Rhodococcus equi* isolate

| NIT | PYZ | PYRA | PAL | βGUR | βGAL | αGLU | βNAG | ESC | URE | GEL | GLU | RIB | XYL | MAN | MAL | LAC | SAC | GLYG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| + | + | - | + | - | - | + | - | - | + | - | - | - | - | - | - | - | - | - |

Legend: NIT - Potassium nitrate, PYZ - Pyrazinamidase, PyrA - Pyrolidonylarylamidase, PAL - Alkaline phosphatase, βGUR - β-glucuronidase, βGAL - β-galactosidase, αGLU - α-glucosidase, βNAG - N-Acetyl-β-glucosaminidase, ESC - Esculin, URE - Urease, GEL - Gelatin, GLU - Glucose, RIB - Ribose, XYL - Xylose, MAN - Mannitol, MAL - Maltose, LAC - Lactose, SAC -Sacchrarose, GLYG - Glycogen, "+" - positive result, "-" – negative result.

**Antibiotic susceptibility test**. The *S. equi* ssp. *equi* strain was susceptible to multiple antimicrobials, including penicillin, resistant to gentamicin, and intermediately susceptible to enrofloxacin (Table 4), in accordance other researcher's findings (Erol et al., 2012). The *S. zooepidemicus* isolate was resistant to all tested antimicrobials, with the exception of rifampicin (Table 4).

Similar to other studies (Giguere, 2017; Lupu et al., 2021), it was determined that the R. equi isolate was susceptible to rifampicin and clarithromicin (Table 4).

Table 4. Antibiotic susceptibility test results for the bacterial isolates

| | |
|---|---|
| ***Streptococcus equi* ssp. *equi*** | **Susceptible:** ampicillin, amoxicillin + clavulanic acid, penicillin, trimethoprim + sulfomethoxazole, cloramphenicole, cefazoline, marbofloxacin, rifampicin.<br>**Intermediately susceptible:** enrofloxacin.<br>**Resistant:** amikacin, tetracycline, gentamicin, doxyciclin, kanamicin. |
| ***Streptococcus equi* ssp. *zooepidemicus*** | **Susceptible:** rifampicin.<br>**Intermediately susceptible:** cloramphenicole, doxyciclin, erythromycin, enrofloxacin, trimethoprim + sulfametoxazole, cefixime<br>**Resistant:** cephalotin, gentamicin, streptomycin, penicillin, norfloxacin |
| ***Rhodococcus equi*** | **Susceptible:** gentamicin, doxyciclin, rifampicin, norfloxacin, clarithromicin.<br>**Intermediately susceptible:** spectinomicin, streptomycin<br>**Resistant:** cefixime, vancomycin, oxacillin, trimethoprim + sulfomethoxazole, cephalotin, clindamycin. |

**Pulmonary ultrasounds** performed on the foals affected by *R. equi* infections, at the onset of the disease, showed the presence of abscesses of various sizes and inflammation of the pleura. Towards the end of the treatment, the abscesses were no longer visible, the surface of the lung appearing slightly irregular, possibly due to the presence of scar tissue.

**Post-mortem examinations**. In the case of *R. equi* induced pneumonia, necropsy findings included pleurisy and suppurative bronchopneumonia, with the presence of multiple abscesses measuring between 2 mm and 1.5 cm in the caudal lobes and foci of pulmonary atelectasis.

*R. equi* was isolated from purulent material collected from the abscesses in the lung tissue.

The pneumonic lesions of the foal that died of peracute respiratory distress were consistent with serohaemorrhagic pneumonia with focal pulmonary necrosis. *S. equi* ssp. *zooepidemicus*

was isolated in pure culture from samples collected from the lung tissue.

**Management and treatment**. The entire herd was monitored constantly during the outbreak, by the members of the medical team and the groomsman. The affected animals were examined daily, recording the rectal temperature, the presence or absence of nasal discharge, the characteristics of the lymph nodes and respiratory sounds.

The two adult mares, the yearlings and the 2 year-old that showed symptoms of strangles recovered after 5 days of treatment with penicillin, without any complications. Of the 31 foals aged 1 to 6 months, 50% had mild symptoms, such as inflamed lymph nodes and nasal discharge, and required 5-7 days of treatment for a complete recovery. 16 foals had more severe manifestations, such as persistent productive couch, fever, copious mucopurulent nasal discharge, pathological respiratory sounds and abscess formation in the submandibular lymph nodes. Penicillin administration was prolonged for up to 10 days, until the abscesses drained and the nasal discharge subsided. The bronchodilator and mucolytic medication aided in clearing the airways of secretions and relieving the cough.

Of the 4 foals with *R. equi* infection, one died 2 weeks after the onset of the symptoms, due to extensive lung damage. The other 3 foals responded well to the treatment: after the first week of therapy, the temperature and appetite returned to normal and they were no longer lethargic. After 4 weeks of treatment, the foals were no longer coughing, and imaging showed a decrease in the size of the pulmonary abscesses. The treatment was continued for another 4 weeks, until the abscesses were no longer visible.

The 5 foals treated for *S. zooepidemicus* pneumonia fully recovered after 5 weeks of treatment.

## CONCLUSIONS

The present study highlights the different clinical patterns of strangles and the efficacy of early onset of antibiotic treatment in preventing the development of severe complications and death.

Daily monitoring of the herd allowed the medical team to prescribe personalized treatment for each affected horse, depending on their clinical condition.

Active bacteriological surveillance was a key factor in discrimination between strangles and the other respiratory infections that showed similar clinical pattern.

The antibiotic susceptibility tests allowed for a targeted and safe therapeutic approach. Infection with *S. zooepidemicus* demonstrates the imperative of carrying out a complete bacteriological investigation, including the antibiophenotype of each isolated strain: the strain identified in this case was susceptible to rifampicin alone.

In the case of *R. equi* infected foals, pulmonary ultrasounds were a useful tool in assessing the remission of lesions, and in adapting the duration of the treatment.

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