

# RESPIRATORY DISEASE IN THE OLDER FOAL AND YEARLING

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## INTRODUCTION

Respiratory diseases are common in young horses and in one US study were the primary cause of disease and death in one to six month old individuals. Older foals, weanlings and yearlings are susceptible to a wide range of respiratory disease. This may be a consequence of a number of factors including the waning of maternally derived immunity, the gradual development of their own innate immunity and environmental and management impositions. The respiratory diseases can be broadly categorised according to their aetiology; bacterial, viral, parasitic and other. Of these the most important group is the bacterial diseases.

## BACTERIAL RESPIRATORY DISEASE

### *Streptococcus equi*, "Strangles"

Typically infection with *S. equi* occurs in young animals from 3 months to 3 years of age. Infection is restricted to the upper respiratory tract but occasionally the organism may spread to other parts of the body, usually subcutaneously or to internal lymph nodes ("bastard strangles"). The organism enters the upper respiratory tract by inhalation or ingestion, passes through the respiratory mucosa and resides in the lymphatic tissue of the head (submandibular and retropharyngeal lymph nodes and guttural pouches). Incubation can be 2-21 days following infection and the organism can persist for prolonged periods both in the environment and in the guttural pouches of previously infected individuals.

*Clinical signs* include pyrexia, anorexia, nasal discharge (initially serous then purulent), lymph node enlargement and depression. A cough may be present and dyspnoea and dysphagia may develop.

*Diagnosis* is confirmed by culture of the organism from nasal discharge, abscessated lymph node or guttural pouch washing. PCR analysis will detect the presence of bacterial DNA and RNA specific to *S. equi*. This will provide evidence of prior exposure to the bacterium, but does not necessarily indicate a source of infection.

*Treatment* is not always necessary as many cases are self-resolving. Untreated abscessated

lymph nodes will rupture and drain with a rapid clinical improvement. The organism is sensitive to penicillin and treatment may be given in early cases before abscessation is present or in cases of bastard strangles.

*Prevention* relies on early identification and isolation of clinical cases. All new arrivals on a premises should be isolated and guttural pouch washings may be cultured from possible carrier animals. Immunity following infection or vaccination is short-lived and its persistence may require repeated exposure or vaccination.

### *Streptococcus zooepidemicus*

*S. zooepidemicus* is a ubiquitous organism found in the environment and upper respiratory tract of horses. It may be involved in both upper and lower respiratory tract disease in the younger horse. It is generally considered to be an opportunistic invader of compromised respiratory mucosa. In the upper tract this may follow damage to the mucosa by respiratory viruses, whilst in the lungs other stresses such as transport, weaning, overcrowding, poor nutritional or parasite status and concurrent disease may be involved.

### *Rhodococcus equi*

*R. equi* is potentially the most significant cause of bacterial respiratory disease on a stud farm. The bacterium survives well in the environment, is resistant to many disinfectants and can withstand drying. It has been shown to persist for at least 12 months in the soil. In addition, it can multiply not only in an infected foal, but also in soil contaminated with equine faeces and the rate of replication increases with environmental temperature. The organism has the potential to be present in large numbers on heavily stocked premises in warm climates, especially if there have been infected foals present in previous years. Infection is usually by inhalation and an increased risk has been associated with dry, dusty conditions when more of the organisms may be air-borne. Recent work has indicated that the time of infection is probably early in the foal's life and most likely within the first two weeks. In many cases infection results only in multiplication of the organism in the foal and shedding in the faeces with no apparent signs of illness. Individual factors, including the

dose and virulence of the organisms inhaled or ingested, are probably important in determining which foals succumb to the disease. Signs of illness are usually not seen until the foal is at least several weeks old. It is thought that maternal antibodies received by the foal in colostrum may be responsible for controlling the infection at this early stage, but that as these antibodies wane at two to three months of age clinical disease can occur.

*Clinical signs* in affected foals include fever, dullness and lethargy, poor growth, purulent nasal discharge, cough and tachypnoea or dyspnoea. If tissues other than the lungs are affected then other signs may be seen such as lameness and diarrhoea.

*Diagnosis:* non-specific changes are usually found on blood samples and infection can only be proven by culturing and isolating the organism from affected tissues. This may be a sample of airway discharge, synovial fluid or faeces. Characteristic signs of lung consolidation and abscessation may be seen with radiography and ultrasonography. At present there is no reliable serological (antibody) test to identify clinically affected individuals nor is there a reliable means of identifying carriers of the disease.

The intestinal form of the disease is less common than the respiratory form. It arises from colonisation of the intestinal tract following ingestion of *R. equi* from the environment. In these cases the organism is able to invade the local lymph nodes in the abdomen, where it leads to the formation of abscesses. It is thought that up to 50% of the foals with *Rhodococcal* pneumonia may also have intestinal disease. Clinical signs of the intestinal disease include weight loss, depression, fever and colic or diarrhoea. The presence of multiple abscesses in abdominal lymph nodes, means the prognosis for recovery from the intestinal form of the disease is poor.

*R. equi* can also be spread in the bloodstream. Most frequently this results in the organism being able to infect bones and joints. Such cases show marked lameness with pain, heat and swelling around the involved structures and they often require specific surgical treatment as well as antibiotic therapy. Rarely the disease is seen in multiple sites and this carries a poor prognosis.

*Treatment:* The most commonly used is a combination of erythromycin and rifampin. (erythromycin 25mg/kg orally q8 hours and rifampin 5 mg/kg orally q12 hours). These have a synergistic effect when used together and not only enter the diseased tissues of the lung, but also are able to penetrate the white blood cells where the organism is multiplying. Some isolates of *R. equi* have been found to be resistant to this combination and hence culture and sensitivity testing is advised. Different formulations of

erythromycin are available and these have different levels of absorption so it is important to ascertain which product is to be administered and design an appropriate treatment regime. More recently other antibiotics have been used in the treatment of *Rhodococcal* pneumonia. These include azithromycin (10 mg/kg orally q24 hours for 5 days and then q48 hours) and increasingly clarithromycin (7.5 mg/kg orally q12 hours). These drugs have enhanced absorption and increased penetration of white blood cells compared to erythromycin. In addition there is greater compliance with these dosing regimes by stud personnel.

*Prevention:* Whilst antibiotics and other therapeutic agents coupled with ancillary support and nursing can effect spectacular recoveries in some foals, the ultimate goal for this disease is avoidance. The disease can affect any foal but a stud farm with a large number of mares and foals and dry, dusty conditions with heavy faecal contamination of the pastures and foaling pens will increase the odds in favour of the disease. This can result in a build up of the organism on the premises with time and then an apparent outbreak of disease in foals in one year. Management changes can be very effective in reducing the foal's exposure to the organism. Good ventilation and avoidance of overstocking are important as are the elimination or avoidance of dry, dusty paddocks for the foals. Pasture rotation, selective grazing and grassland irrigation may all help to achieve this. Infected foals are a major source of the organism and should be isolated.

Where there is evidence of a high risk of the disease on a premises then specific prophylactic therapy may be used. Currently the most effective strategy involves the administration of plasma containing *R. equi* antibodies. It is important that this protection is present before the foal is exposed to the organism and hence it is often administered on the second day of life. This protection may also be boosted by a further infusion at three to four weeks of age. It should be remembered that this strategy is most effective when combined with environmental changes and even then, whilst reducing the incidence of the disease, it should not be expected to eliminate it. Recently blanket antimicrobial therapy for all foals during their first two weeks of life has been proposed as a control measure on endemic farms.

Early recognition of the disease is important to successful therapy. Where foals may be at a high risk of the disease then careful daily observations (body temperature and resting respiratory rate) coupled with regular veterinary checks will allow affected individuals to be identified early. This approach is often very cost-effective as early recognition allows prompt treatment and a reduced duration, and therefore cost, of

therapy. Ultimately vaccination against *R. equi* may become the best method of prevention and control of this disease.

### ***Mycoplasma spp***

*Mycoplasma spp.* and *M. felis* in particular have been implicated as causes of lower respiratory tract inflammation and poor performance in young racehorses in training. However, they do not appear important pathogens in lower respiratory tract disease in foals.

### **Pleuropneumonia**

This condition occurs when infection or inflammation of the lung extends to the pleural surface and subsequently the pleural cavity. A pleural effusion is frequently seen. It is uncommon in foals but may occur in association with a *R. equi* pneumonia. In yearlings it is typically seen following transport (“shipping fever”) and in particular when individuals are kept stalled with a continuously raised head and neck posture for long periods.

*Clinical signs* of pyrexia, anorexia, tachycardia and tachypnoea may be accompanied by chest pain and a cough. Thoracic auscultation may be unremarkable but pleural friction rubs are usually detectable. Judicious use of a rebreathing bag may be required. Absence of lung sounds ventrally and a change in percussion may demonstrate a fluid line. Nasal discharge is a variable feature.

*Diagnosis* is based on clinical signs and confirmed by ultrasonography and thoracocentesis.

*Treatment* requires antimicrobial and anti-inflammatory agents. Thoracocentesis of a pleural effusion is indicated to allow culture and sensitivity testing. Significant volumes of pleural fluid should be drained.

### **Secondary bacterial infections**

Although *S. zooepidemicus* is the most common, other bacteria may be involved in secondary invasion of a compromised respiratory tract. These include *S. pneumoniae*, *Pasteurella spp.*, *Pseudomonas spp.*, *Staphylococcus spp.*, *Actinobacillus equuli*, *Bacteroides spp.* and *Clostridium spp.* These may be present singly or in combination and often will become of more clinical significance than the original stressor. Infection of the upper and lower respiratory tract may occur.

*Clinical signs* are related to the site of secondary infection and the nature of the primary stressor. Bacterial pneumonia (other than *R. equi*) may occur as a primary event. Foals present with typical pneumonic signs on auscultation and with marked leucocyte changes and raised fibrinogen. Culture of a transtracheal wash

shows bacteria other than *R. equi* and sensitivity testing is essential. Thoracic radiography is non-specific unless a pleural effusion is present.

Treatment, when required, is with appropriate antimicrobial agents. This often necessitates sample collection, culture and antimicrobial sensitivity testing.

## **VIRAL RESPIRATORY DISEASE**

### **Equine herpes virus (rhinopneumonitis)**

This is a common infection of youngstock and is often endemic within breeding stock. EHV-4 appears more prevalent than EHV-1. Infection is usually by inhalation and the virus replicates within the respiratory epithelium. Almost invariably all animals will eventually encounter the virus and the severity of clinical signs tends to decrease with increasing age. Secondary bacterial invasion of the compromised respiratory mucosa may occur a few days after initial infection (see earlier). Infection occurs most commonly in weanlings in the autumn, probably as a consequence of increased stress at this time and the opportunity for exposure to older animals, which may be shedding the virus asymptotically. The potential for EHV-1 infection to be present in youngstock should be considered whenever there is potential contact with in-foal mares. There is also the sporadic occurrence of neurological disease related to EHV exposure.

*Clinical signs* include pyrexia, dullness, serous nasal discharge, conjunctivitis, anorexia and lymphadenopathy. Coughing is variable and may only be seen following secondary bacterial invasion.

*Diagnosis* is based on clinical signs. Viral isolation can be performed on nasopharyngeal swabs and the buffy coat layer of blood samples collected during the initial viraemia (usually up to 12 days after infection). Paired serology may demonstrate an increasing antibody titre, although levels can be already high in the acute sample. Recently PCR techniques have become available to allow rapid detection of the virus in nasopharyngeal swabs or EDTA blood samples.

*Treatment* is rarely necessary, with most cases resolving without treatment. Antimicrobial therapy should be reserved for those cases with significant secondary bacterial involvement.

*Prevention* is difficult as most individuals will eventually meet herpes virus. Early exposure and recovery may be preferred for animals that are subsequently to encounter the stresses of joining a training premises. To date vaccination has shown to be of limited value.

## Equine influenza

This represents a serious threat to youngstock, especially foals where lower respiratory tract involvement can be fatal. In the older animals infection is usually restricted to the upper respiratory tract. Secondary bacterial invasion of compromised respiratory mucosa can occur. The A/Equi 2 strain is more prevalent than the A/Equi 1 strain.

*Clinical signs* include pyrexia, dullness, a dry cough, anorexia, lethargy, muscle soreness and serous to mucopurulent nasal discharge. There may be rapid spread within susceptible populations. Fortunately the recent outbreaks of influenza in slightly older animals (predominately on training premises) has not been accompanied by spread to younger animals on stud farms.

*Diagnosis* is based on clinical signs together with viral isolation or identification (by ELISA) from nasopharyngeal swabs and serology.

*Treatment* is non-specific with rest in a dust-free environment. Antimicrobial agents may be required if significant secondary bacterial involvement occurs.

*Prevention* is based on isolation of confirmed cases and routine vaccination. Good but not complete protection is achieved with vaccination. Vaccinated youngstock may be protected or show mild or transient clinical signs when exposed to the virus. Vaccination protection is, however, short-lived.

## Others

Other viral agents may cause signs of upper respiratory tract disease. These include adenovirus, rhinovirus and picornavirus. Clinical signs are typical of upper respiratory tract infection, are usually mild and resolve spontaneously without treatment.

## PARASITIC RESPIRATORY DISEASE

### *Parascaris equorum*

Typical of ascarids, the equine *Parascaris equorum* migrates through the lungs as part of its lifecycle. This may result in clinical signs of lower respiratory tract disease, typically an afebrile pneumonia often with tachypnoea. The condition itself is self-limiting but indicates the need for adequate parasite control on the premises.

This may be responsible for the usually undiagnosed condition of "July blowers" seen in foals during the summer months.

### *Dictyocaulus arnfieldii*

This is usually a non-patent infection in the horse, unlike the donkey where the parasite lifecycle is completed and adult egg-laying worms are present. Hence there is usually a history of exposure to grazing shared with donkeys or mules.

*Clinical signs* include a chronic cough, increased respiratory rate and often increased respiratory effort.

*Diagnosis* is based on identification of increased eosinophils and *Dictyocaulus* larvae in discharge or washings from the lower airway. Faecal examination is not useful as the infection is non-patent.

*Treatment* is with anthelmintics, typically ivermectins.

*Prevention* relies on suitable pasture management

## OTHER

### Acute respiratory distress syndrome, interstitial pneumonia, *Pneumocystis pneumonia*

A syndrome of acute severe interstitial pneumonia has been described in foals 2-6 months of age. Individual affected cases and clusters have been reported. Many aetiologies have been suspected including viruses, bacteria, *Pneumocystis carinii*, heat stroke and toxins.

*Clinical signs* are characterised by sudden onset respiratory distress with tachypnoea and marked pyrexia. The presentation can be similar to acute Rhodococcal pneumonia. There is usually only a mild leucocytosis and elevation in fibrinogen (c.f. *Rhodococcal pneumonia*). The foals usually remain bright despite the respiratory distress.

*Diagnosis* is usually made after negative culture for *R. equi* and thoracic radiography showing a diffuse interstitial pattern with no nodules or abscessation. A poor response to symptomatic and antimicrobial therapy is seen and cases are often fatal.

*Treatment* requires corticosteroids (dexamethasone 0.4 - 0.8 mg/kg IV q12 hours for 48 hours and then tapered) provided that *R. equi* is believed unlikely. Intranasal oxygen may be required. Supportive treatment with antimicrobials, bronchodilators, and ulcer prophylaxis is usually given. Potentiated sulphonamides would be included if *Pneumocystis carinii* is suspected.

*Prognosis* is guarded but may be favourable with prompt corticosteroid therapy.