

PURPURA HEMORRHAGICA

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OVERVIEW

Purpura Hemorrhagica is a type III hypersensitivity reaction, which occurs as a sequelae to pathogen re-exposure in equids. Pathogens known to cause this reaction are Equine influenza Virus, *Corynebacterium pseudotuberculosis*, *Rhodococcus equi*, Equine Herpes Virus, and most commonly *Streptococcus equi*. When the horse experiences its first encounter with *S. equi*, the disease that may result is known as Strangles. Not all horses will develop Strangles, however when they do lymphadenomegaly is often seen accompanied by upper respiratory signs such as bilateral purulent nasal discharge. A horse with Strangles that receives antibiotic treatment has a good prognosis, however a sensitized horse may develop Purpura Hemorrhagica after re-exposure to the same antigen later in life. Important to note is another potential complication of *S. equi* infection, "Bastard Strangles," which is the development of abscesses within organs due to *S. equi*. Bastard Strangles can result in mortality and is a different complication of *S. equi* than Purpura Hemorrhagica.

EPIDEMIOLOGY

Development of Purpura Hemorrhagica in horses with two exposures to *S. equi* M-like protein occurs in 6.5% of the population. M-like protein can be encountered by either direct exposure to the pathogen or by vaccination against strangles. Horses who travel to and from crowded areas (such as shows) are at a much higher risk of coming into contact with *S. equi* than horses that remain within a closed environment. It can be helpful for horse owners to be aware of their horse's exposure/vaccination history however it can be difficult to know whether or not a horse has been exposed in the past because some horses may not develop clinical signs following their exposure. Due to this, development of Purpura Hemorrhagica can seem idiopathic without testing. Preventative measures can be taken for Strangles such as vaccination of horses 2 weeks prior to their first time traveling. This will ensure sufficient vaccination induced immunity prior to first natural exposure and will reduce the risk of strangles infection. Biosecurity measures such as not sharing buckets, hoses, feed bags, brushes, and feed pans also reduce the risk of transmission of *S. equi* from horse to horse.



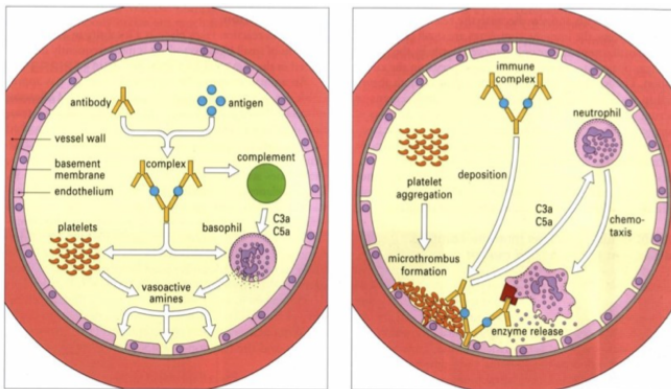
[HTTPS://WWW.MERCKVETMANUAL.COM/MULTIMEDIA/IMAGE/PURPURA-HEMORRHAGICA-HORSE](https://www.merckvetmanual.com/multimedia/image/purpura-hemorrhagica-horse)

CLINICAL SIGNS

- Swelling in the head, legs, and ventral abdomen
- Petechiae of mucous membranes
- Skin ulceration/sloughing on distal limbs
- Epaxial & gluteal muscle atrophy
- Dyspnea
- Tachypnea
- Ascites
- Pyrexia
- Inappetence
- Stiff gait
- Lethargy

PATHOPHYSIOLOGY

After the second encounter of the antigen, the horse's body undergoes a type III hypersensitivity reaction. The antibodies that were formed during the previous infection bind to the antigen and form Ag-Ab complexes, which would normally bind red blood cells and travel to the spleen to be broken down. In the case of purpura hemorrhagica, the complexes are not bound to red blood cells. They activate complement, which induces granulocytes, such as neutrophils and basophils, to release vasoactive amines. These amines lead to increased vascular permeability via retraction of endothelial cells. This causes the immune complexes to be deposited in the wall of the blood vessel. This deposition activates platelet aggregation, which then form microthrombi on the exposed collagen of the basement membrane. Neutrophils have also been attracted to the site by complement, so they release their lysosomal chemicals since they cannot phagocytose the large complexes. These chemicals cause even more damage to the vessel wall, allowing fluid to leak out, resulting in the edema that can be seen in the legs, muzzle, and ventral abdomen and chest. In severe cases, the edema can actually start to leak out of the skin, and necrosis from all the pressure, especially in the legs, will cause ulceration and eventually sloughing of skin.



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DIFFERENTIAL DIAGNOSES

- Equine infectious anemia
- Equine viral arteritis
- Anaplasmosis
- Trauma
- Lymphangitis
- Cellulitis
- Lymphedema
- Right-sided heart failure
- Equine Herpesvirus 1-4
- Leptospirosis
- Babesiosis
- Pemphigus foliaceus
- Eosinophilic dermatitis

TREATMENT & PROGNOSIS

Corticosteroids suppress the immune system to help reduce the clinical signs seen in purpura hemorrhagica. Antibiotics are given to inhibit other infections from occurring while the horse is immunosuppressed. NSAIDs can be given to help reduce inflammation and reduce pain. The prognosis is very good if the condition is recognized and steroids are started early. Without steroid intervention, the Ag-Ab complexes will continue to cause vasculitis and damage to the muscle, lungs, and kidneys, which will eventually result in death.

PREVENTION

If you know your horse has contracted strangles in the past, you would want to be more cautious about taking them to locations with horses from different areas or allowing them to have direct contact with new, unfamiliar horses. Additionally, strangles vaccines are not recommended for horses with previous occurrences of Purpura Hemorrhagica as Strep equi is a causative agent. Horses with high levels of antibodies to strangles are more at risk for developing purpura hemorrhagica.

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