Purpura Hemorrhagica

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ABSTRACT: Purpura hemorrhagica is a noncontagious, immune-mediated vasculitis of horses that is characterized by subcutaneous edema of the head, ventral abdomen, and limbs and by petechial hemorrhages of the mucous membranes. Purpura hemorrhagica most often occurs as a rare complication of Streptococcus equi subsp equi infection but can also develop after infection with other bacterial and viral organisms, particularly those that cause formation of purulent or necrotic foci. Purpura hemorrhagica has also reportedly occurred after vaccination or drug administration as well as idiomatically. Most of the clinical signs of the disease result from deposition of antigen–antibody complexes in the small blood vessels of the skin. Treatment includes the use of immunosuppressive drugs as well as removal of the underlying antigenic stimulus.

Cutaneous vasculitis, the hallmark of purpura hemorrhagica, results from inflammation and injury to the small blood vessels of the skin caused by deposition of antigen–antibody complexes within the blood vessels. The consequence of this injury is leakage of large proteins, electrolytes, and fluid from the vessels. In addition, extravasation of erythrocytes through vessel walls causes nonthrombocytopenic petechiations (i.e., purpura), which are common on the mucous membranes of affected horses. The former names for purpura hemorrhagica—morbus maculosus or speckled sickness—refer to these characteristic lesions.1 Edema of the lower limbs and ventral midline is another consistent and distinctive clinical sign of the vasculitis. This article discusses the pathophysiology of purpura hemorrhagica and its systemic effects and treatment options.

PATHOGENESIS

In humans, numerous types of vasculitis have been recognized, many of which are immune-mediated. Mechanisms that have been identified in these syndromes include immune-complex formation and deposition, autoantibody production, and pathogenic T-lymphocyte activity. In veterinary medicine, although the causes and types of vasculitis are less clearly defined and categorized, several types appear to have an immune-mediated cause as well. Immune-mediated vasculitis is often called secondary vasculitis to distinguish it from vasculitis that is not immune-mediated and that results directly from inflammation induced by infectious disease, trauma, or photosensitization.2

The pathogenesis of purpura hemorrhagica (in horses) has primarily been investigated in connection with Streptococcus equi subsp equi

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infection (from here on referred to as *S. equi* or strains). Purpura hemorrhagica secondary to strangles reportedly occurs in 1% to 17% of cases, and clinical signs typically appear 1 to 2 weeks after presumed recovery from a strangles episode. There seem to be two prerequisites for the development of purpura hemorrhagica: a large amount of antigenic material and an exaggerated immune response.

Draining abscesses probably often serve as the source of this material because purpura hemorrhagica has been reported as a sequel to a variety of bacterial and viral organisms associated with formation of purulent foci (see box on this page). The antigen within these foci is M-like protein, the major streptococcal antigen in cases of purpura hemorrhagica caused by strangles infection.

Recovery following a bout of strangles is normally mediated by production of both mucosal and humoral antibodies against M-like protein. These antibodies, which consist primarily of IgA and a variety of IgG subtypes, bind to circulating M-like protein to form limited quantities of large antigen–antibody complexes. Because of their size, these complexes are easily removed by the reticuloendothelial system and do not cause problems. However, when the quantity of antigen greatly exceeds that of circulating antibody, such as when draining abscesses are present, much smaller immune complexes tend to form. These small aggregates are not removed by the reticuloendothelial system and travel through the general circulation until they are eventually deposited in the endothelial basement membranes of capillaries and other small blood vessels of the skin.

Once deposited, these antigen–antibody complexes activate complement, including the neutrophil chemoattractant C5a. As neutrophils penetrate the vessel walls and phagocytose the antigen–antibody complexes, they release lysosomal enzymes and radical oxygen species into their immediate environment. These chemicals compromise the integrity of the vessel walls and allow fluid and erythrocytes to leak into the extracellular space. C5a is an anaphylatoxin that causes increased vascular permeability and induces mast cell and basophil activity.

In purpura hemorrhagica, the characteristic histologic finding of a leukocytoclastic vasculitis (i.e., vasculitis characterized by fragmented neutrophils) is caused by these events and leads to the resulting edema and petechial hemorrhages that create the clinical appearance of purpura hemorrhagica.

Because purpura hemorrhagica involves production of antigen–antibody complexes under a specific set of conditions, it is classified as a Type III hypersensitivity reaction. This is an allergic response to antigen belonging to the same category of immune-mediated diseases as serum sickness and systemic lupus erythematosus. However, purpura hemorrhagica is somewhat unusual in that IgA appears to be the primary immunoglobulin

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### Historical Findings in Horses That Developed Purpura Hemorrhagica

- *Corynebacterium pseudotuberculosis* infection
- Cranial sinus empyema
- Drug administration (unspecified)
- Equine herpesvirus infection
- Equine influenza
- Equine viral arteritis
- *Burkholderia mallei* infection (glanders)
- Respiratory infection (unidentified organism)
- *Rhodococcus equi* infection
- *S. equi* subsp *equi* infection
- *S. equi* subsp *zooepidemicus* infection
- M–like protein vaccine (intramuscular)
- Attenuated strangles vaccination (intranasal)
- Wounds

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involved, whereas in most Type III reactions, IgG predominates. In fact, IgG is absent in serum during the acute phase of purpura hemorrhagica, although its levels rise significantly during the recovery phase. Some authors have speculated that its initial absence is due to temporary suppression of IgG production or to incorporation of IgG into immune complexes, causing its depletion in serum.

However, production of large amounts of IgA is probably related to the other requirement for the development of purpura hemorrhagica—a greatly magnified immune response. Reasons for this are not well understood. One possibility is that horses with purpura hemorrhagica are innately predisposed to having increased immune system activity. This idea is supported by the finding that the serum of affected horses contains higher-than-normal plasma concentrations of the complement component C3, an activator of both the classic and alternate complement pathways, a stimulus for phagocytosis, and both a direct and indirect target of M-protein activity. Other explanations for elevated IgA levels include the inability of the liver to clear IgA from the circulation and unrestrained growth of IgA-specific B-cell populations. However, liver disease is rarely reported in purpura hemorrhagica and, thus far, no data support the latter hypothesis. Because 10% to 20% of all cases of purpura hemorrhagica occur in the absence of a history of previous illness or antigenic exposure and the disease is very difficult to reproduce experimentally, additional systemic immune processes that have not been identified are probably involved.

Although the capillaries, arterioles, and venules of the skin are most commonly affected in purpura hemorrhagica, antigen–antibody complexes can also settle in a variety of organs (e.g., the lungs, kidneys, and liver), the gastrointestinal (GI) tract, or skeletal muscle and can cause vascular inflammation and hemorrhage. Although there is only one definitive report of purpura hemorrhagica–induced glomerulonephritis, many purpuric horses present with azotemia, hematuria, and other signs of renal injury. Unfortunately, because there is no way to determine where the immune complexes may be deposited, the course of the disease is difficult to predict. Likewise, its often very wide-ranging clinical appearance, representing the involvement of any number of organ systems, can make diagnosis challenging.

**CLINICAL PRESENTATION**

Purpura hemorrhagica most commonly affects young adult horses, although the disease has been identified in yearlings as well as geriatric horses. There appears to be no breed or sex predilection. Affected horses most often initially present with depression and anorexia followed by urticaria and swelling around the nares—the earliest signs of vasculitis. Localized swelling quickly progresses to severe subcutaneous edema of the head, ventral abdomen, and limbs. The edema, which can cause significant discomfort to the horse, causes pitting, is nonpruritic, and can be either warm or cool. It is also usually sharply demarcated and can develop into large plaques, especially on the abdomen (Figure 1). In mild cases, edema may be more localized to the legs. Severe edema of the head often leads to respiratory stridor due to occlusion of the upper airway. Dyspnea can result from swelling around the upper or lower respiratory tract or from pulmonary edema. Dysphagia is another complica-

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**Figure 1.** Plaque-like ventral edema in a horse with purpura hemorrhagica. The horse came from a farm that had a strangles outbreak 1 month earlier.
tion. On the proximal and distal limbs, edema can lead to skin necrosis, which initially appears as ulcerative lesions that leak serum, crust over, and ultimately slough. The necrosis can cause horses to become unwilling to move and extremely footsore, especially if ulceration occurs around the coronary bands. Although edema is almost always a consistent finding in purpura hemorrhagica, there is one report of a miniature horse with leukocytoclastic vasculitis confirmed via skin biopsy, a history of exposure to *S. equi*, and petechiated mucous membranes without subcutaneous edema. The authors of the report speculate that very rapid development of ischemia and damage to end-arteries caused this highly unusual presentation of purpura hemorrhagica.

Although petechial or ecchymotic hemorrhages of the mucosal, nasal, and conjunctival membranes are common, they can occasionally be entirely absent in horses with mild forms of the disease. Epistaxis is a less commonly reported clinical sign and is probably due to submucosal hemorrhage in the nasal cavity. Tachycardia occurs frequently and is probably the result of decreasing circulating fluid volume as well as discomfort. Many horses demonstrate concurrent signs of strangles, such as lymphadenopathy, draining abscesses, coughing, and nasal discharge. Pyrexia may or may not be present.

Signs of specific organ disease can occur if the immune complexes are deposited in sites other than skin. Hematuria associated with glomerulonephritis has been reported, as have severe colic and diarrhea resulting from ulcerative necrosis of the GI mucosa. In addition, lameness due to polyarthritis, synovitis, or joint infections is possible.
Immune-mediated myopathies, including a particularly severe and usually fatal form called infarctive purpura hemorrhagica, have also been described. Affected horses presented with muscle swelling, stiffness, or colic and, at necropsy, were found to have widespread muscle infarctions as well as multifocal sites of pulmonary hemorrhage. Thrombophlebitis, cellulitis, and pneumonia are further complications of purpura hemorrhagica.

CLINICAL PATHOLOGY

The results of complete blood cell counts and serum clinical chemistry tests are often nonspecific. Hematologic changes include mild to moderate anemia and leukocytosis with pronounced neutrophilia and a mild left shift. In general, horses with purpura hemorrhagica have normal clotting profiles and platelet counts. Thrombocytopenia is extremely rare in patients with purpura hemorrhagica, and its absence in the presence of petechial and ecchymotic lesions should arouse suspicion of purpura hemorrhagica. Common biochemical abnormalities include hyperproteinemia, hyperfibrinogenemia, and hyperglobulinemia. Elevated muscle enzymes, including aspartate aminotransferase and creatine kinase, can occur in association with S. equi–induced myopathy.

DIAGNOSIS

Diagnosis of purpura hemorrhagica is typically based on the results of skin biopsy evaluation, a history of recent respiratory infection or vaccination, relevant clinical signs, and the absence of other causes of vasculitis. Full-thickness, 6-mm skin biopsy samples should be taken using a punch biopsy tool within 10 hours of the appearance of urticaria or localized edema and before drug treatment, particularly steroid use. After this period of time, skin biopsy is often inconclusive. Similarly, ulcerated lesions are rarely useful diagnostically.

To obtain the sample, a small bleb of lidocaine should be injected subcutaneously, but the skin should not be scrubbed or clipped beforehand. Samples should be submitted in formalin for histopathology. Immunofluorescence testing for equine globulins requires that samples be placed in solution, an ammonium sulfate transport medium, rather than formalin.

The characteristic histopathologic finding is necrotizing leukocytoclastic vasculitis distinguished by fragmented neutrophils and nuclear debris surrounding small blood vessels (Figure 2). Edematous blood vessel walls, dermal and subcutaneous hemorrhage, inflammation, and thrombi may be visible as well.

Because treatment of the disease requires removal of any antigenic stimulus, it is essential for clinicians to look for an underlying cause of the disease. Careful history taking, with special emphasis on possible exposure to other animals, recent vaccination, and current medications, is important. Purpura hemorrhagica generally develops within 2 weeks after a bout of strangles, but delays as long as 3 months before the onset of clinical signs have been reported. Although the incidence and timing of purpura hemorrhagica following vaccination have been poorly studied, in one study using a commercially available intramuscular M-like protein vaccine, two of 1,300 foals developed purpura hemorrhagica within 6 weeks after vaccination, and purpura hemorrhagica–like signs have been reported following use of the intranasal vaccine as well.

Diagnosis of S. equi infection has been discussed in detail elsewhere, but a summary of diagnostic methods includes culture of draining abscesses and visible wounds, endoscopy of the guttural pouches with accompanying bacterial culture and polymerase chain reaction (PCR) testing, and bacterial cultures and PCR testing of nasal swabs or washes. The specific finding of S. equi

Figure 2. Necrotizing leukocytoclastic vasculitis showing fragmented neutrophils and extravasated erythrocytes. (Hematoxylin–eosin, original magnification ×200; courtesy of Perry Habecker, VMD, Department of Pathobiology, School of Veterinary Medicine, University of Pennsylvania)
(streptococcal spp are commensals in horses’ upper airways) in a horse with ventral edema and petechiated mucous membranes is strongly suggestive of purpura hemorrhagica. Testing via ELISA for antibodies to S. equi M-like protein in serum is also helpful. However, some animals may not produce a significant antibody response and, as discussed earlier, acute-stage patients with purpura hemorrhagica may not have measurable serum IgG levels.

Other causes of vasculitis should be ruled out. Several diseases, including equine granulocytic anaplasmosis (Anaplasma phagocytophilum infection; formerly Ehrlichia equi infection), babesiosis, equine infectious anemia, equine viral arteritis (EVA), equine herpesvirus infection, and immune-mediated thrombocytopenia, can produce vasculitic syndromes, but affected animals are usually thrombocytopenic. However, because in rare cases purpuric horses present with low platelet counts, testing for these diseases is still important because they can serve as the source of the antigenic stimuli rather than as a primary cause of vasculitis. An agar gel immunodiffusion test (Coggins test) or ELISA can identify equine infectious anemia. Equine granulocytic anaplasmosis and babesiosis can be identified in blood smears of leukocytes or by serology. EVA and equine herpesvirus infection can be ruled out by serologic testing using paired serum samples or by virus isolation. Antinuclear antibody testing and the Coombs’ test do not appear to be clinically useful in diagnosing purpura hemorrhagica, although some horses may test weakly positive for antinuclear antibody.

Unfortunately, no underlying disease or exposure to antigenic stimulus is found in 10% to 20% of all cases. In these situations, purpura hemorrhagica becomes a diagnosis of exclusion based on clinical signs and biopsy results. Connective tissue diseases, neoplasia, and congenital deficiencies in complement have been recognized as endogenous sources of antigen in immune-mediated vasculitis in humans. These have not been identified in conjunction with purpura hemorrhagica.

**TREATMENT**

Treatment of purpura hemorrhagica is threefold:
- Removal of the antigenic stimulus (if identified)
- Reduction of the immune response
- Supportive care
Eradication of antigenic stimuli requires drainage of abscesses and exploration of wounds. As mentioned earlier, endoscopy of the guttural pouches is also important, and flushing should be repeated if needed.

Most animals presenting with purpura hemorrhagica are treated with intravenous broad-spectrum systemic antibiotics. Potassium or sodium penicillin at an initial dosage of 22,000 to 44,000 IU/kg IV q6h is recommended because of the efficacy of this class of drugs against streptococcal organisms. Additional antibiotics for gram-negative organisms should be added if warranted. Intramuscular injections should be avoided because inflamed muscle tissue may be further aggraved. Because thrombophlebitis is a complication of purpura hemorrhagica, careful attention to aseptic placement of intravenous catheters and daily examination of the catheter site are important. Switching to oral antibiotics such as trimethoprim sulfadiazine (15 to 30 mg/kg PO q6h) to avoid complications of long-term catheter use or for financial reasons is also an option. Antibiotic use should continue throughout treatment of the disease.

If no bacterial cause is identified, antibiotic use is debatable. Antibiotic use can induce immune-mediated reactions, including cutaneous vasculitis. Thus if a horse develops purpura hemorrhagica while receiving antibiotic treatment, the drug should be discontinued immediatly and a different class of antimicrobials should be used. If cellulitis, pneumonia, or septic thrombophlebitis is present, antibiotics are clearly indicated. However, even when no definitive infectious organism is identified, the high probability of an underlying infection representing the antigenic stimulus generally warrants antibiotic use.

Suppressing the immune response by using glucocorticoids is the second essential component of treatment. Current recommendations include the use of dexamethasone (0.05 to 0.2 mg/kg IV q24h initially). Prednisolone is thought to be less effective during the acute stage of purpura hemorrhagica. As clinical signs improve, the dose of dexamethasone should be slowly decreased over a 2- to 3-week period. When the dose of dexamethasone is 0.04 mg/kg or lower, it can be replaced by oral prednisolone at 10 times the final dose of dexamethasone (i.e., if the final dose of dexamethasone is 0.04 mg/kg, switch to prednisolone at 0.4 mg/kg). As the horse recovers, the dose of prednisolone should be gradually decreased to alternate-day dosing to allow recovery of the hypothalamo–pituitary–adrenal axis.

Although prolonged steroid use can lead to concerns about laminitis and reduced immune system activity when simultaneously treating bacterial infection, early discontinuation of steroid therapy has been associated with relapses. Similarly, switching from dexamethasone to prednisolone too early in the course of the disease can cause treatment failure. Most horses with purpura hemorrhagica receive dexamethasone for at least 10 days and glucocorticoids for 3 to 4 weeks. In our experience, the development of laminitis secondary to administration of aqueous forms of corticosteroids is extremely rare. Because this drug class is essential in treating purpura hemorrhagica, the benefit of its use far outweighs the potential risk for complications.

Use of NSAIDs may also be beneficial because of their ability to alter leukocyte chemotaxis and prevent leukocyte adhesion to the already-damaged endothelium, which may avert further damage to vessel walls. Although flunixin meglumine (1.1 mg/kg IV q12h) or phenylbutazone (2.2 mg/kg IV or PO q12h) is indicated to provide analgesia to patients with colic, foot pain, or discomfort from edema, these drugs also exacerbate the risk for GI ulceration and renal injury, particularly in animals also receiving high doses of glucocorticoids. Therefore, clinicians should consider administering antiulcer medications (omeprazole [1 to 4 mg/kg PO q24h] or ranitidine [6.6 mg/kg PO q6h]) to horses being treated for purpura hemorrhagica and should be alert to clinical signs of ulceration.

Creatinine and blood urea nitrogen levels as well as urine specific gravity should be carefully monitored for evidence of dehydration and azotemia. Prerenal azotemia, which is common because of continuing third-space fluid loss, can be exacerbated by NSAIDs and glucocorticoids. Use of NSAIDs should be reconsidered if prerenal azotemia develops. For horses with renal involvement, renal protection (via the use of anti-inflammatory drugs and/or fluid therapy) is critical to prevent permanent renal damage.
losses, should be treated with appropriate amounts of intravenous isotonic fluids. Hypoalbuminemic animals with edema may benefit from judicious use of colloids, such as plasma or hetastarch (10 ml/kg). Changes in urine specific gravity or the presence of pigmenturia or urine casts should alert clinicians to the possibility of purpura hemorrhagica–induced glomerulonephritis or renal compromise stemming from NSAID administration. Ancillary diagnostic testing (e.g., sequential urinalysis, measurement of urine output, fractional clearance of electrolytes, renal ultrasonography and biopsy) and blood pressure monitoring are recommended if renal disease is suspected. Unfortunately, the prognosis probably worsens for animals with purpura hemorrhagica–induced glomerulonephritis.

After therapy has been initiated, the effects of glucocorticoids (including hyperglycemia, neutrophilia, and lymphopenia) on blood work may complicate interpretation of complete blood cell counts. Resolution of edema and petechiation as well as improvement in attitude and appetite are probably more important clinical clues of recovery than are alterations in clinical chemistry and hematologic values. Supportive therapy includes hydrotherapy, thick bedding, counter-pressure leg wraps, and protective dressings for weeping skin lesions. Some clinicians advocate the use of furosemide (0.3 mg/kg IV q6–8h) to decrease edema, particularly pulmonary edema. However, in animals that are already azotemic, furosemide use may cause further renal compromise. Light exercise for horses that are not laminic or too sick to move can also help dissipate edema. Animals with severe head edema may require tracheostomy. Supplemental oxygen is beneficial in horses with pulmonary edema. Arterial blood gases should be used to assess oxygenation status in horses that are dyspneic or have severe respiratory stridor. The nutritional needs of dysphagic horses must be addressed as well. Some horses may be able to lap gruel. Partial parenteral nutrition should be considered in horses able to ingest small amounts of food but not enough to meet metabolic requirements. An indwelling nasogastric tube can be used to assist enteral feeding if the severity of nasal and pharyngeal edema does not preclude passage of a tube. Esophagostomy can be considered as long as neck edema is not severe. Total parenteral nutrition is an extremely expensive and time-
consumption option in adult horses but may be the only alternative for some patients.

In humans, Schönlein-Henoch purpura, an immune-mediated disease in children that bears a remarkable similarity to purpura hemorrhagica and is characterized by IgA deposition in small vessels, is treated with dapsone, azathioprine, cyclophosphamide, and plasmapheresis in addition to corticosteroids.\(^3\) Cyclophosphamide and corticosteroids were also used to treat three shar-pees presenting with acute febrile neutrophilic vasculitis resembling purpura hemorrhagica.\(^4\) None of these additional therapies has been reported in horses with vasculitis.

Because both intramuscular and intranasal vaccination against strangles may increase the risk for developing purpura hemorrhagica if a horse concurrently has a high \(S.\ equi\) antibody titer, use of these vaccines on horses recovering from purpura hemorrhagica is contraindicated.\(^5\) However, it is unclear whether horses that have survived purpura hemorrhagica should ever be vaccinated against \(S.\ equi\). Vaccination against strangles using intramuscular M-like protein vaccines has not been shown to be particularly efficacious or long-lasting, which is probably a function of these vaccines’ failure to induce a mucosal antibody response.\(^6\) However, the attenuated intranasal vaccine is associated with side effects such as nasal discharge, enlarged lymph nodes, and lymph node abscessation. Thus the risks of using these vaccines, including induction of purpura hemorrhagica, have to be weighed against the need for protection.

**PROGNOSIS**

Unfortunately, the course of purpura hemorrhagica is difficult to predict. Little information is available on the number of horses that survive the disease and return to work. There is one report\(^7\) of a horse acquiring hindlimb fibrotic myopathy after recovering from purpura hemorrhagica. The literature cited in this article reports survival rates of 63% to 92%. Poor prognostic indicators include respiratory distress, the appearance of pleural effusion or pulmonary edema, and sudden onset of diarrhea.\(^8\) Fever is also associated with a poor prognosis.\(^9\) The most common reasons for euthanasia or death include development of secondary complications such as renal disease, colic, rhabdomyolysis, and laminitis. Typical postmortem findings in these animals are significant hemorrhage and infarcts in the GI tract and the kidneys.\(^10\)

**SUMMARY**

Although cases of purpura hemorrhagica are uncommon, they require early recognition so that immunosuppressant and antimicrobial therapy can begin promptly. Diagnosis may be challenging because of the wide variety of clinical signs and because often no precipitating cause can be found. Drug administration, particularly involving corticosteroids and antibiotics, along with supportive care is usually required for extended periods. The prognosis for recovery is fair to good and generally depends on whether the horse sustains additional internal organ injury from the circulating antigen–antibody complexes.

**REFERENCES**

ARTICLE #1 CE TEST

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1. Which has not been implicated in the development of purpura hemorrhagica?
   a. EVA
   b. rhinopneumonitis
   c. Mycobacterium pseudotuberculosis
   d. S. equi subsp equi vaccine

2. In the antibody-antigen complexes identified after strangles-related purpura hemorrhagica, the most abundant immunoglobulin is
   a. IgM.
   b. IgA.
   c. IgG.
   d. IgE.

3. Which is a rare hematologic alteration in purpura hemorrhagica?
   a. neutrophilia
   b. leukocytosis
   c. thrombocytopenia
   d. mild to moderate anemia

4. Which statement regarding antimicrobial use in patients with purpura hemorrhagica is correct?
   a. Antimicrobial use is controversial if no source of infection is evident.
   b. Antimicrobials are an essential component in treating purpura hemorrhagica.
   c. Initiation of antibiotic therapy should be delayed until glucocorticoid therapy has been discontinued.
   d. Antibiotic use decreases the need for high levels of glucocorticoids.

5. Which statement regarding glucocorticoid use in treating patients with purpura hemorrhagica is correct?
   a. Prednisolone is recommended during the acute stage.
   b. Glucocorticoid use should be discontinued as soon as edema begins to abate because of the risk for gastric ulceration and laminitis.
   c. The combination of antimicrobials, glucocorticoids, and NSAIDs can be difficult to manage and can lead to gastric ulceration, decreased effectiveness of antibiotic therapy, and alterations in blood work.
   d. Glucocorticoids can be administered orally in mild cases of purpura hemorrhagica.

6. Which test is not helpful in diagnosing purpura hemorrhagica?
   a. skin biopsy
   b. guttural pouch endoscopy with accompanying serology and PCR testing
   c. paired serum samples for equine herpesvirus and EVA
   d. hepatic biopsy

7. The prognosis of horses with purpura hemorrhagica
   a. is better for older animals.
   b. is worse when the underlying cause is not S. equi infection.
   c. depends on the severity of the associated edema.
   d. is difficult to determine because the course of the disease is unpredictable.

8. Clinical findings in a horse with purpura hemorrhagica do not include
   a. diarrhea.
   b. pruritus.
   c. unwillingness to move.
   d. lymph node abscessation.

9. Which has not been reported as a complication of purpura hemorrhagica?
   a. rhabdomyolysis
   b. pneumonia
   c. laminitis
   d. cerebral edema

10. Which does not damage vessel walls in patients with purpura hemorrhagica?
    a. T-lymphocyte activity and granuloma formation
    b. neutrophilic infiltration and release of lysosomal enzymes in vessel walls
    c. complement-induced activation of mast cells and basophils
    d. deposition of IgA and M-like protein complexes in vessel walls