Infarctive purpura hemorrhagica in five horses

Heather J. Kaese, DVM, MS; Stephanie J. Valberg, DVM, PhD, DACVIM;
David W. Hayden, DVM, PhD, DACVP; Julia H. Wilson, DVM, DACVIM;
Patricia Charlton, DVM, MS; Trevor R. Ames, DVM, MS, DACVIM; Ghanem M. Al-Ghamdi, DVM, MS

- Classic signs of purpura hemorrhagica in horses include well-demarcated subcutaneous edema of all 4 limbs and petechiation; other signs may include fever, lethargy, anorexia, weight loss, reluctance to move, and, occasionally, colic.
- In rare instances, some horses may develop a severe form of purpura hemorrhagica characterized by leukocytoclastic vasculitis in numerous tissues that progresses to infarction.
- Signs of infarctive purpura hemorrhagica include muscle swelling, abdominal discomfort, neutrophilia with a left shift, hypoalbuminemia, and high serum creatine kinase activity.

A 10-year-old American Quarter Horse mare (horse 1) was referred to the **University of Minnesota Veterinary Medical Center (UMN-VMC)** for evaluation of muscle stiffness and severe ventral edema. The horse had been vaccinated against eastern and western equine encephalitis, tetanus, West Nile virus infection, and rabies and had been dewormed with ivermectin every 2 months. It was kept in a pasture with 3 other horses and had no direct contact with horses on neighboring farms. Five years previously, the horse had recovered without complications from an episode of suspected streptococcal lymphadenitis (strangles). One month prior to examination at the UMN-VMC, the horse had developed a severe upper respiratory tract infection characterized by copious nasal discharge and was treated successfully with a 7-day course of trimethoprim-sulfamethoxazole. Eighteen hours prior to examination, the horse was found to be unwilling to walk. Ventral abdominal swelling and swelling of the right pectoral muscles and muscles of the proximal portions of the hind limbs were evident. A colt on the property reportedly had a serous nasal discharge and cough but no palpable lymphadenopathy. The horse was treated by the referring veterinarian with procaine penicillin (22,000 U/kg [10,000 U/lb], IM), flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IV), and dexamethasone (0.1 mg/kg [0.045 mg/lb], IV), but the following morning, the muscular stiffness and swelling were worse. The horse was treated with lactated Ringer's solution (10 L, IV) and flunixin meglumine (1.1 mg/kg, IV) prior to referral.

On initial examination at the UMN-VMC, the horse had signs of mild depression, but heart rate, respiratory rate, and rectal temperature were within reference ranges. Two 0.5-cm areas of ecchymotic hemorrhage were noticed on the gingival mucous membranes. Auscultation of the heart and lungs with a rebreathing bag revealed no abnormalities. Normal borborygmi were present in all quadrants. The horse walked with a short-strided, wide-based, stiff hind limb gait. There was no subcutaneous edema involving the distal portions of the limbs or ventral aspect of the abdomen. Rather, there were marked, asymmetric, firm swellings of the ventral abdominal (**Figure 1**), hind limb adductor, semimembranosus, and pectoral muscles. The epaxial musculature was grossly normal. Ultrasonographic examination of the musculature revealed hypoechoic pockets of edema within the swollen areas of skeletal muscle and fascial planes. Musculature adjacent to these swollen areas appeared normal ultrasonographically, and no subcutaneous edema was evident. Results of a CBC were unremarkable other than leukocytosis and band neutrophilia (WBC count, 24.7 × 10^3 cells/µL [reference range, 4.1 to 11.3 × 10^3 cells/µL]; band neutrophils, 0.49 × 10^3 cells/µL). Serum biochemical abnormalities included slightly high fibrinogen concentration (0.5 g/dL; reference range, 0.1 to 0.4 g/dL), high **creatine kinase (CK**; 64,015 U/L; reference range, 92 to 388 U/L) and **aspartate transferase (AST**; 3,872 U/L; reference range, 115 to 379 U/L) activities, low albumin concentration (2.5 g/dL; reference range, 3 to 4 g/dL), high glucose concentration (254 mg/dL; reference range, 75 to 116 mg/dL), high total bilirubin concentration (4.1 mg/dL; reference range, 0.2 to 2.4 mg/dL), and low phosphorus concentration (1.1 mg/dL; reference range, 1.4 to 5.1 mg/dL). Serum titer of antibodies against the M protein of *Streptococcus equi* was high (1:25,600), which was considered suggestive of **purpura hemorrhagica (PH)** or possibly disseminated (bastard) strangles. Biopsy specimens were obtained from 3 areas of the semimembranosus muscle, and frozen and formalin-fixed sections were examined. Two of the biopsy specimens from the semimembranosus muscle appeared to be histologically normal with no evidence of necrosis or inflammatory infiltrates. However, a specimen taken directly from a swollen area of the semitendinosus muscle had histologic evidence of marked acute myofibrillar degeneration and necrosis of entire muscle fascicles without evidence of inflammatory cells.

A tentative diagnosis of PH was made, and treatment was initiated. Lactated Ringer's solution was

From the Department of Veterinary Population Medicine, College of Veterinary Medicine, University of Minnesota, Saint Paul, MN 55108. Dr. Kaese's present address is Eye Care for Animals, 372 S Milwaukee Ave, Wheeling, IL 60090. Dr. Charlton's present address is Department of Comparative Physiology and Medicine, Pfizer Global Research and Development, Eastern Point Rd, Groten, CT 06340.

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Address correspondence to Dr. Kaese.

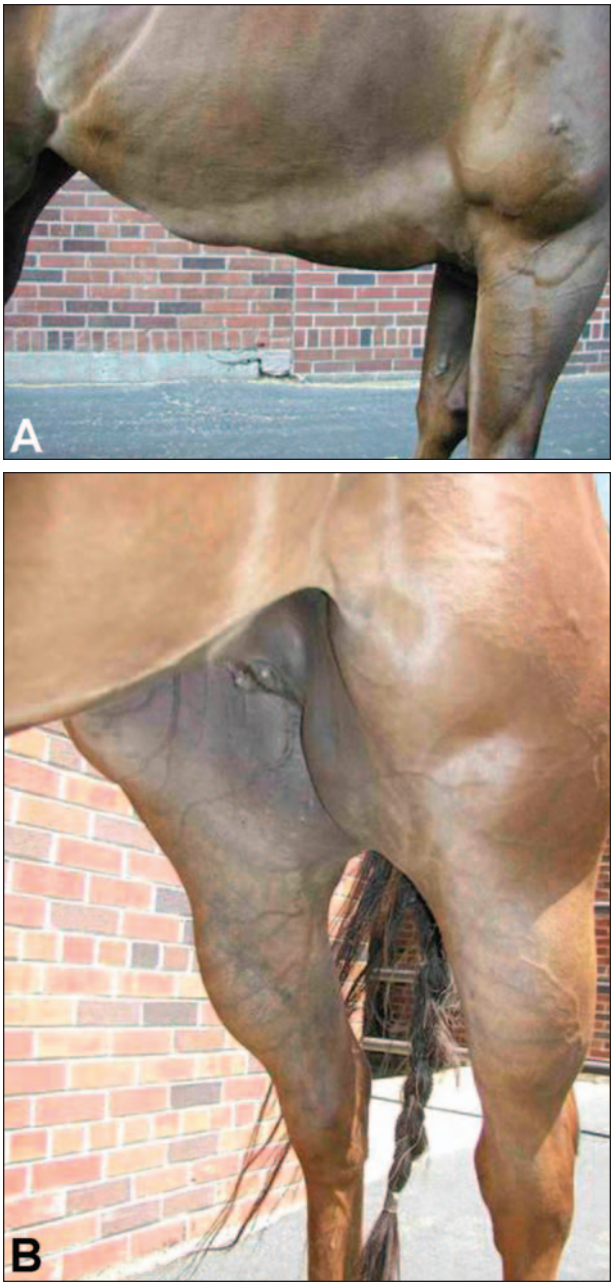

Figure 1—Photographs of a horse with infarctive purpura hemorrhagica (PH). Notice the asymmetric firm swelling extending from the axilla to the caudal third of the abdomen (A) and the extensive swelling of the gastrocnemius and adductor muscles of the left hind limb (B).

administered IV at a maintenance rate, and flunixin meglumine (1.1 mg/kg, IV, q 12 h), dexamethasone (0.1 mg/kg, IV, q 24 h), 10% dimethyl sulfoxide (5 L, IV, q 24 h), potassium penicillin (44,000 U/kg [20,000 U/lb], IV, q 6 h), and vitamin E (5,000 U, PO, q 24 h) were administered. After 5 days, administration of potassium penicillin was discontinued and administration of procaine penicillin (22,000 U/kg, IM, q 12 h) was begun because of the owner's concerns about the cost of treatment. After an additional 5 days, administration of penicillin was discontinued and trimethoprim-sulfamethoxazole (24 mg/kg [11 mg/lb], PO, q 12 h) was administered for 2 weeks. Flunixin meglumine (1.1 mg/kg, PO) was administered twice daily for 3 days then once daily for 2 days. Within 48 hours after admission, gross swelling of the musculature had decreased and the ecchymotic gingival hemorrhages had resolved. Serum CK (10,200 U/L) and AST (3,230 U/L) activities had decreased by day 5. The dosage of dexamethasone was decreased by a total of 5 mg every other day until a maintenance dosage of 40 mg/d was achieved on day 5. The horse was discharged from the hospital 7 days after admission, at which time all signs of muscular swelling had resolved.

Treatment following discharge consisted of administration of dexamethasone (35 mg, IM, q 24 h for 7 days, then 30 mg, IM, q 24 h for 5 days). Digital pulses were mildly increased in all 4 limbs; however, the horse did not show any other signs of laminitis during treatment with dexamethasone. Serum CK and AST activities were measured before the dosage of dexamethasone was decreased and were found to be normal (336 and 1,428 U/L, respectively), indicating that muscle necrosis had ceased. The horse was clinically normal at this time, so the dexamethasone dosage was decreased to 20 mg, IM, every 24 hours. Administration of prednisolone (2 mg/kg [0.9 mg/lb], PO, q 12 h) was begun, and after 2 days, administration of dexamethasone was discontinued. Prednisolone was administered at this dosage for 14 days, and the horse was continued on stall rest with a gradual increase in activity, including twice-daily hand walking and small paddock turnout. Seven weeks after prednisolone treatment was initiated, serum CK (924 U/L) and AST (181 U/L) activities were measured and serum CK activity was again found to be high. Thus, prednisolone treatment was continued at the same dosage until the CK activity was within reference limits 2 weeks later. Thereafter, the prednisolone dosage was decreased by 100 mg/dose weekly. Serum CK and AST activities were within reference limits when measured 2 and 7 months after discharge. The horse resumed training for barrel racing 3 months after discharge.

An 11-year-old American Quarter Horse gelding (horse 2) was referred to the UMN-VMC for evaluation of colic and muscle stiffness. The horse had been treated for *S equi* infection of the auditory tube diverticula (guttural pouches) 3 weeks earlier. On the evening of referral, the horse had experienced an episode of colic that responded only transiently to treatment with flunixin meglumine.

On examination, respiratory rate and rectal temperature were within reference ranges, but heart rate was slightly high (52 beats/min) and borborygmi were decreased in all quadrants. Mucous membrane color was unremarkable, but petechiae were present in the buccal and nasal mucosae. Pitting edema of the limbs was not identified, but multiple firm swellings were noticed in the pectoral region and ventral aspect of the abdomen. Aspiration of these areas produced hemorrhagic fluid.

A CBC was performed. The WBC count was within reference range (7.9×10^3 cells/mL), but band neutrophils (0.2×10^3 cells/μL) and mild toxic changes (1+) were seen. Serum biochemical abnormalities included low albumin concentration (2.9 g/dL) and high CK (47,360

U/L) and AST (964 U/L) activities. Results of analysis of peritoneal fluid obtained by means of abdominocentesis were unremarkable. Activated clotting time was prolonged (> 260 seconds; reference range, 120 to 180 seconds), and fibrinogen split product concentration was high (> 40 µg/mL; reference range, < 10 µg/mL). Results of an ELISA for serum antibodies against the M protein of *S equi* were positive (1.92; positive control sample, 1.55; negative control sample, 0.32), suggesting that the horse had PH or possibly bastard strangles.

Treatment included administration of lactated Ringer's solution IV at a maintenance rate, potassium penicillin (22,000 U/kg, IV, q 6 h), phenylbutazone (2.2 mg/kg [1 mg/lb], IV, q 12 h), and dexamethasone (0.07 mg/kg [0.032 mg/lb], IV, q 24 h). On the second day of hospitalization, the horse appeared brighter and more comfortable, but petechiae were still present on the nasal mucosa, and the serum CK activity had increased (88,222 U/L). Treatment was continued as on the day of admission. By the third day of hospitalization, the horse's condition had again improved slightly and the owners requested to take the horse home. Serum CK activity had decreased (56,880 U/L), but AST activity had increased (2,912 U/L). Intravenous administration of penicillin was discontinued, and oral administration of trimethoprim-sulfamethoxazole (24 mg/kg) was begun. Administration of dexamethasone was continued at the same dosage (40 mg, IV, q 24 h). Immediately prior to discharge, the horse had an episode of severe colic. Serum CK and AST activities had increased (84,400 U/L and 3,030 U/L, respectively), and palpation per rectum revealed distended large intestine. Abdominocentesis yielded a serosanguineous fluid that was characterized as a nonseptic exudate. The horse's condition deteriorated rapidly, and because of uncontrollable pain, the horse was euthanatized.

A 13-year-old American Quarter Horse mare (horse 3) was referred to the UMN-VMC with a 12-hour history of pain, as evidenced by sweating and trembling, progressive limb edema, and intractable bilateral epistaxis. One week earlier, the horse had been treated with procaine penicillin, flunixin meglumine, and dexamethasone because of fever, neck stiffness, and nasal discharge. An outbreak of strangles was occurring on the horse's farm at the time of examination.

On initial examination, the horse was sweating and trembling and appeared painful. Severe (grade IV/V) lameness of the left hind limb developed during the trailer ride to the UMN-VMC, and the horse had a firm swelling involving the musculature just distal to the tuber coxae. Signs of pain were evident on palpation of this swelling. The muscles of the gluteal and gaskin regions were firm on palpation. Edema was present distally in all 4 limbs and along the ventral aspect of the abdomen. The horse had tachycardia (80 beats/min), but rectal temperature was normal. Respiratory rate was normal; however, respiratory effort was increased. Multiple areas of petechiation were present in the buccal mucous membranes.

A CBC was performed. The RBC and platelet counts were within reference ranges, but the WBC count was high (14.7×10^3 cells/µL) and band neutrophils (0.51×10^3 cells/µL) and mild (1+) toxic changes were seen. Analysis of peritoneal fluid obtained by means of abdominocentesis revealed high nucleated cell (12,650 cells/µL) and RBC (0.7×10^6 cells/µL) counts and high total protein concentration (5.2 mg/dL). Examination of a gram-stained smear of the peritoneal fluid revealed chains of gram-positive cocci. Because the university's diagnostic laboratory was closed at the time the horse was admitted, a serum biochemical profile was not performed.

Treatment included administration of butorphanol and xylazine hydrochloride for management of pain and IV administration of hypertonic saline (7.2% NaCl) solution and lactated Ringer's solution. The hind limb swelling and stiffness increased over the next 2 hours. The horse then spontaneously refluxed gastrointestinal tract contents from its nares and collapsed. The musculature distal to the stifle joints was extremely firm on palpation, and the distal portions of the extremities were cold. The horse was euthanized because of its rapidly deteriorating condition and failure to respond to treatment.

A 10-year-old American Quarter Horse gelding (horse 4) and a 5-year-old Morgan gelding (horse 5) were referred to the UMN-VMC with histories of acute, mild colic. Both had been treated for strangles in the preceding month.

On examination, both horses had signs of extreme pain. Rectal temperature and respiratory rate were within reference ranges, but heart rate was high. Subcutaneous edema was present in the distal portions of the limbs and along the ventral aspect of the abdomen. Borborygmi were absent from all quadrants, and results of rectal palpation were unremarkable. No gastrointestinal tract reflux was obtained following passage of a nasogastric tube. During the examination, both horses passed dark-colored urine.

Complete blood counts were performed and revealed leukocytosis (horse 4, 19.9×10^3 cells/µL; horse 5, 16.2×10^3 cells/µL) characterized by band neutrophilia (horse 4, 4.18×10^3 cells/µL; horse 5, 2.27×10^3 cells/µL) and toxic changes (horse 4, 2+; horse 5, 1+). Platelet counts were normal. Remarkable serum biochemical abnormalities included hyperproteinemia in horse 4 (9.2 g/dL; reference range, 6.1 to 7.9 g/dL) and high CK (horse 4, 280,000 U/L; horse 5, 156,840 U/L) and AST (horse 4, 6,960 U/L; horse 5, 6,080 U/L) activities. A coagulation profile was performed on horse 5. Activated clotting time (250 seconds; reference range, 120 to 180 seconds), partial thrombin time (51.5 seconds; reference range, 25.7 to 47.3 seconds), and thromboplastin time (37.7 seconds; reference range, 15.4 to 22.6 seconds) were prolonged, and fibrinogen split product concentration (> 40 µg/mL; reference range, < 10 µg/mL) was high. Histologic examination of biopsy specimens from the gluteal muscle of horse 4 revealed severe vasculitis with focal areas of coagulative necrosis, and myosin ATPase staining of frozen sections revealed areas of type 2 muscle fiber atrophy.

Treatment for both horses included IV administration of lactated Ringer's solution at a maintenance rate and repeated doses of analgesics. However, the pain was not responsive to analgesic treatment. Because of

the unrelenting pain, horse 4 was euthanatized several hours after initial examination. Exploratory celiotomy was performed in horse 5 and revealed multiple infarcts in the cecum, small intestine, and mesentery. Because the lesions were inoperable, the horse was euthanatized.

Necropsy of horses 2, 3, 4, and 5 revealed dark, red-black, multifocal coalescing hemorrhages in the skeletal muscles of the limbs, thorax, and abdomen (**Figure 2**). The lungs had multifocal areas of hemorrhage and consolidation scattered throughout several lobes. Retropharyngeal lymph nodes in horses 2, 3, and 4 had signs of abscess formation; submandibular abscesses were found in horse 5. Petechial hemorrhages of the oral mucosa were found in all horses. In horse 2, right dorsal displacement of the colon with 360° torsion was present. Multifocal irregular and circular reddened areas from 1 to 5 cm in diameter involving the serosal and mucosal surfaces were found in the large colon (horses 2 and 5), cecum (horses 2 and 5), small intestine (horses 2, 3, and 5), and stomach (horses 3 and 5). The intestinal tract appeared grossly normal in horse 4. Pure growth of *S equi* was obtained from abscesses in the 4 horses. In horse 3, pure growth of *S equi* was also obtained from the liver and lungs.

Histologic examination of necropsy specimens from horses 2, 3, 4, and 5 revealed marked, multifocal, coalescing areas of coagulative necrosis of skeletal muscle (**Figure 3**) and fibrinoid degeneration of small blood vessels. Affected skeletal muscle fibers had variable swelling, cytoplasmic eosinophilia, vacuolation, mineralization, loss of cross-striations, fragmentation, and nuclear pyknosis or karyolysis. Extensive hemorrhage was present in areas of necrosis. Inflammatory cells in the areas of necrosis consisted primarily of degenerate neutrophils, lymphocytes, plasma cells, and macrophages. The inflammatory cells appeared perivascular, and proliferation of satellite cells was prominent in some areas. In horses 2, 3, and 5, sections of the small and large intestine had evidence of leukocytoclastic vasculitis. There were areas of edema, hemorrhage, and inflammation in the submucosa, tunica muscularis, and serosa. Some areas included fibrinoid degeneration of small blood vessels and infiltration of vessel walls with degenerative leukocytes (**Figure 4**). Pulmonary sections from all 4 horses were characterized by multifocal areas of hemorrhage and inflammation frequently oriented around pulmonary blood vessels (**Figure 5**). The inflammation was mixed but consisted of predominantly degenerative neutrophils, with neutrophils extending into vessel walls. Fibrin exudation and necrosis of alveolar walls were prominent in these areas. In horses 2 and 5, the dermis had multifocal coalescing areas of mixed inflammation that were frequently oriented around or near blood vessels. In some areas, fibrinoid vascular necrosis and thrombosis or infiltration of the vessel wall with degenerative leukocytes was surrounded by edematous and hemor-

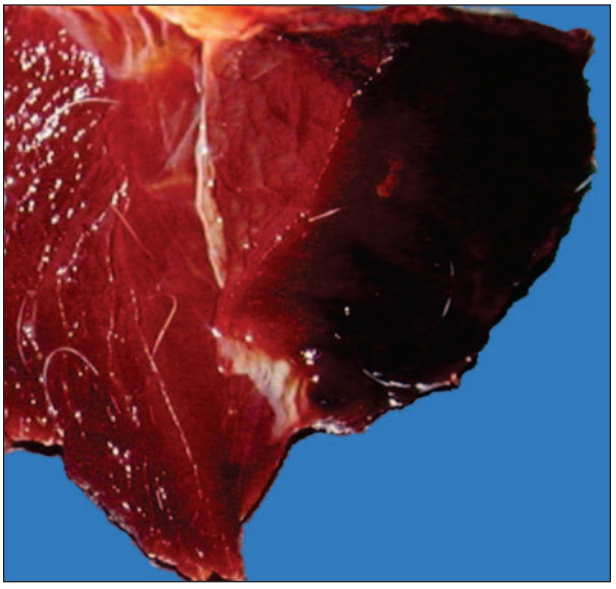

Figure 2—Photograph of a section of skeletal muscle from a horse with infarctive PH. A well-demarcated hemorrhagic area can be seen.

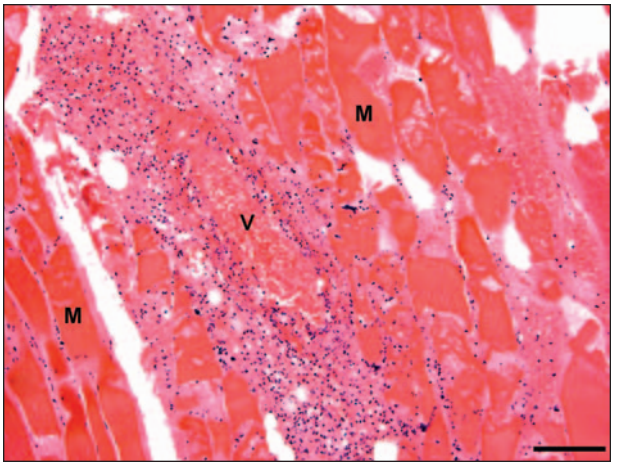

Figure 3—Photomicrograph of a longitudinal section of infarcted skeletal muscle from the horse in Figure 2. Necrotic muscle fibers (M) are swollen and hypereosinophilic and separated by hemorrhage. Necrotizing neutrophilic vasculitis and perivasculitis involves the wall of an interstitial vein (V). H&E stain; bar = 100 μm.

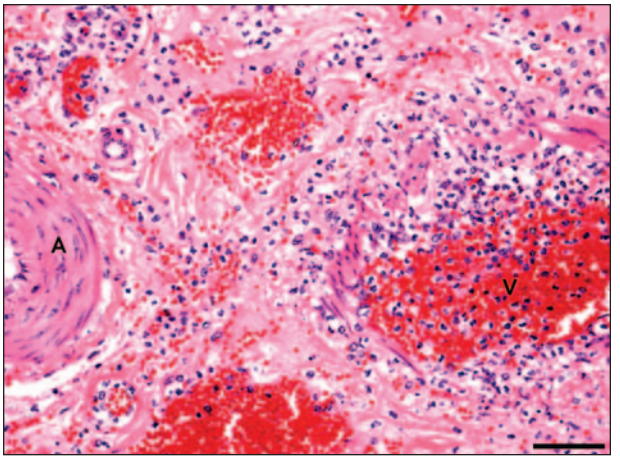

Figure 4—Photomicrograph of a section of small intestine from the horse in Figure 2. The wall of a submucosal vein (V) is disrupted by infiltration of neutrophils. The accompanying artery (A) is histologically normal. Neutrophils and focal hemorrhages are seen in the stroma between the 2 blood vessels. H&E stain; bar = 50 μm.

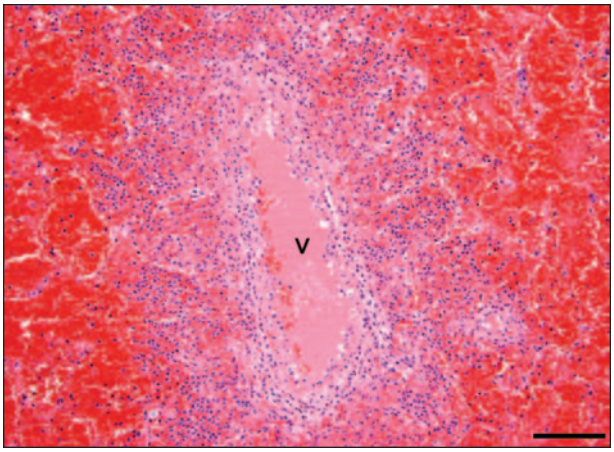

Figure 5—Photomicrograph of a section of lung from the horse in Figure 2. The wall of a pulmonary blood vessel (V) and the adjacent alveoli are heavily infiltrated by neutrophils. Other alveolar spaces are obliterated by hemorrhage. H&E stain; bar = 100 µm.

rhagic dermal tissue. Pancreatic sections were characterized by marked coalescing interstitial hemorrhages and moderate depletion of pancreatic zymogen granules. Adjacent to viable pancreatic parenchyma in horse 2 were lobules of eosinophilic ghost cells with pyknotic nuclei, fibrin exudation, and multifocal areas of perivascular inflammation and thrombosis similar to that found in the lungs.

Three *S equi* isolates were obtained from the 4 horses at necropsy. Isolates were grown on brain-heart infusion agar plates for 24 hours at 37°C in 5% CO_2, and DNA was obtained from each isolate as described.[1] A repetitive sequence-based polymerase chain reaction assay was performed as described,[1] and fingerprint patterns of the resulting gels were analyzed with a computer-assisted program. Results indicated that the 3 isolates consisted of 2 separate subtypes of *S equi*.

In horses, strangles has typically been associated with high morbidity and low mortality (2.6%) rates; however, complication rates as high as 20% have been reported in some outbreaks.[2] Complications include PH, guttural pouch empyema, upper respiratory tract obstruction, bastard strangles, pneumonia, pleuritis, agalactia, and periorbital abscess formation.[2] Classic signs of PH in horses include well-demarcated subcutaneous edema of all 4 limbs and petechiation. Other signs may include fever, lethargy, anorexia, weight loss, reluctance to move, and, occasionally, colic. In 1 study,[3] 92% of horses with PH recovered; treatment consisted of administration of corticosteroids and antimicrobials and limb bandaging. Three of the horses that died of PH had massive areas of hemorrhage in multiple tissues.

The 5 horses described in the present report had some of the common clinical signs and hematologic abnormalities associated with classic PH.[3] These include plaques of pitting edema involving the ventral aspect of the abdomen (2 horses) and lower portions of the limbs (2), petechial or ecchymotic hemorrhages on mucous membranes (5), signs of abdominal pain (4), neutrophilia (5), and high fibrinogen concentration (5). Mild increases in CK and AST activities are commonly reported with PH,[3,4] whereas in the horses described in the present report, CK and AST activities were markedly increased, and multifocal, firm areas of hemorrhage and neutrophilic inflammation were seen in skeletal muscle. In addition, 4 of the horses described in the present report had hemorrhagic areas in the lungs that resembled infarctions, and 3 had similar lesions involving the intestinal tract. Thus, horses described in the present report appear to have had a severe form of PH characterized by leukocytoclastic vasculitis in numerous tissues that progressed to infarction. In a previous report,[3] 3 of 53 horses with PH had similar necropsy findings, and another report[5] described a horse examined because of colic, leukocytosis, neutrophilia with a left shift, and a high total protein concentration that at necropsy had widespread leukocytoclastic vasculitis of the skeletal muscle and intestine in addition to chronic β-hemolytic streptococcal pulmonary infection. Another report[6] described 5 horses with PH that had hemorrhage and necrosis of skeletal muscles. Histologic examination showed various degrees of muscle fiber degeneration, hemorrhage, and neutrophilic inflammation with mononuclear inflammation in horses with more chronic signs; however, leukocytoclastic vasculitis and vasculitis of intestinal or pulmonary tissues were not reported. The vascular reaction in horses described in the present report did not appear to be a result of infection with a specific strain of *S equi*, as results of the polymerase chain reaction assay indicated that horses were infected with at least 2 subtypes of the organism.

The underlying histologic lesion found in the dermis of horses with PH is leukocytoclastic vasculitis with necrosis of blood vessel walls.[7] Immune complexes are present in the sera of horses with PH and appear to be composed primarily of IgM or IgA and streptococcal M protein.[8] Deposition of complement near immune complexes in vessel walls may result in cell membrane destruction and cell death. Clinical signs and pathologic abnormalities in horses with PH are similar to those in people with Henoch-Schönlein purpura, an immune complex disease.[4,9] Henoch-Schönlein purpura is believed to be a sequela to streptococcal or other bacterial infection, viral infection, or a drug reaction and is also characterized by circulating IgA immune complexes and high serum concentrations of C3d.[10] Henoch-Schönlein purpura is most common in children (90% of patients are < 10 years old), with a more severe recurrent form seen occasionally in adults.[11] Clinical signs typically seen in adult patients with this more severe form include a rash on the legs and buttocks, with 75% of patients developing arthritis of the knees and ankles; 40% of patients developing nephritis; and 50% to 75% of patients developing abdominal pain, gastrointestinal tract bleeding, and, rarely, intussusception. Muscle involvement is rarely documented in these patients.[9,12] Patients with Henoch-Schönlein purpura, including patients with intestinal involvement, have been successfully treated with high doses of methylprednisolone IV, followed by administration of other immunosuppressive agents such as cyclophosphamide and azathioprine.[13,14]

The high fatality rate for horses described in the present report and in previous studies[3,5,7] indicates the

difficulty in successfully diagnosing and treating infarctive PH in horses. Three of the 5 horses in the present report were examined because of clinical signs of colic, and the diagnosis of PH was not immediately evident on physical examination. Two of the horses were examined because of lameness and muscle stiffness. Horses 3, 4, and 5, all of which were examined because of severe colic, had a short clinical course, and treatment was unsuccessful because of extensive gastrointestinal tract infarction. Treatment of horse 2 with dexamethasone at a moderate dosage was initially successful; however, the horse was euthanatized because of subsequent development of gastrointestinal tract infarctions and torsion.

Successful treatment of horse 1 required early intervention and prolonged administration of corticosteroids at high dosages, similar to the treatment for Henoch-Schönlein purpura. The aim of treatment was to remove the antigenic stimulus, reduce the immune response, reduce inflammation, and provide supportive care. Monitoring of serum CK activity appeared to be important in determining when to taper the dosage of corticosteroids.

Immune-mediated myopathies have previously been reported in association with *S equi* infection in horses,[15] with the primary clinical sign being rapid, severe atrophy of the epaxial muscles. This form of immune-mediated myositis occurs in Quarter Horses subsequent to respiratory tract infection or exposure to *S equi* and is typified by mild to moderate increases in serum CK and AST activities and a normal leukogram.[16] The primarily neutrophilic vasculitis and extensive myonecrosis of muscle fascicles found in skeletal muscle of horses described in the present report and in previous reports[3,5,6] of horses with PH do not resemble the mononuclear vasculitis and scattered mononuclear infiltration of myofibers found in Quarter Horses with immune-mediated myositis.

In conclusion, infarctive PH appears to be a rare and highly fatal form of PH that has some clinical and histologic similarities to Henoch-Schönlein purpura in humans. A distinctive feature of all 5 cases described in the present report was the presence of infarctions in skeletal muscle. One horse that had signs of muscle stiffness without evidence of abdominal pain responded well to aggressive treatment with antimicrobials and corticosteroids. Early recognition of the signs of muscle swelling, abdominal discomfort, neutrophilia with a left shift, hypoalbuminemia, and a marked increase in CK activity could enhance the likelihood of a successful outcome.

a. Equine Biodiagnostics, Gluck Center, Lexington, Ky.

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