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Diagnosis of Aorto-Iliac Thrombosis in a Quarter Horse Foal Using Doppler Ultrasound and Nuclear Scintigraphy

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A 5-day-old 52-kg Quarter Horse filly presented to the Boren Veterinary Medical Teaching Hospital, Oklahoma State University College of Veterinary Medicine, with a 1-day history of diarrhea, lethargy, and weakness. The foal was recumbent and depressed with tacky, injected mucous membranes; a capillary refill time of 4 seconds; rectal temperature of 101.3°F; tachycardia (180 bpm); and tachypnea (80 bpm). The extremities were cold with poor peripheral pulse quality, and dehydration was estimated to be 10–12%. The foal had malodorous diarrhea. During the initial examination, the foal made attempts to rise. With assistance, it could stand on its forelimbs and briefly support itself on the left hind limb, but the right hind limb was flaccid and non–weight bearing. No site of trauma, infectious synovitis, or osteomyelitis was obvious, and although reaction to stimulation was reduced in both hind limbs, deep pain perception was present in all limbs. Evaluation of initial laboratory testing identified an IgG concentration >800 mg/dL; mild azotemia (creatinine, 1.9 mg/dL); hypoalbuminemia (1.6 g/dL); neutrophilia (3,312/μL); and lymphocytosis (5,244/μL). Fibrinogen concentration was normal. Attempts to obtain an arterial blood sample for blood gas analysis using the left lateral metatarsal artery were unsuccessful because of poor peripheral perfusion. Venous blood gas analysis identified metabolic acidosis. Colitis and septicemia were suspected based on clinical signs, and a blood culture was submitted. A sample of feces was submitted for aerobic and anaerobic culture, including culture for *Campylobacter jejuni* and *Salmonella* spp, ELISA for *Clostridium difficile* toxins A and B, and electron microscopy for rotavirus and coronavirus.

A presumptive diagnosis of colitis and septic shock accompanied by decreased hind limb perfusion was made. Spinal or pelvic trauma also was considered as a diagnostic possibility for the hind limb dysfunction at this time. Initial treatment consisted of IV replacement fluid therapy and sodium bicarbonate (4.5 mEq/kg over 12 hours); broad-spectrum antibiotic therapy (ceftiofur sodium, 10 mg/kg IV q 8h and metronidazole, 15 mg/kg PO q 8h); and ketoprofen (2.2 mg/kg IV once) for shock and possible trauma. Omeprazole (4 mg/kg PO q 24h) was administered for ulcer prophylaxis. To facilitate resolution of the diarrhea, milk was withheld for the first 12 hours, and nutrition was supplemented with IV dextrose at 8 $mg \cdot kg^{-1} \cdot min^{-1}$. Serum glucose concentration was monitored and maintained within the normal range by adjusting the dextrose infusion rate. Because spinal cord trauma still remained a differential diagnosis, 100 mg methylprednisolone sodium succinate was administered IV.

The foal responded to treatment, became bright and alert, and would drink milk from a bowl. No further diarrhea was detected, but the foal would not stand and nurse unassisted. The hind limbs became progressively colder from the pelvis distally, and no pulse could be palpated in the femoral arteries, suggesting that the foal had a thrombus impeding arterial blood flow to the pelvic limbs. In an attempt to improve oxygen delivery to the pelvic limbs, 250 mL of polymerized hemoglobin (Oxyglobin[a]) was administered IV, and intranasal oxygen therapy was initiated. A coagulation profile identified prolonged prothrombin and partial thromboplastin times and increased concentration of fibrin degradation products. One liter of heparinized plasma (100 units/kg) was administered in an attempt to provide clotting factors and activated antithrombin III.

Pelvic and thoracic spinal radiographs disclosed no abnormalities. Doppler ultrasound of the femoral artery and vein was performed with the foal in lateral recumbency. The femoral artery was difficult to discern from surrounding soft tissue, but the right and left femoral veins were visualized and both contained echogenic luminal material. A vascular waveform indicating venous flow was detected using pulse wave Doppler evaluation. The venous blood appeared stagnant in real time, and a small oscillating character of to and fro movement was noted within the lumen of the femoral vein. The terminal abdominal aorta was evaluated via transabdominal approach. No pulsed Doppler waveform or color Doppler signal could be obtained from the aorta caudal to the left kidney. Nuclear angiography was performed for further evaluation. This procedure was carried out with the foal sedated with diazepam (5 mg IV to effect) and briefly restrained in dorsal recumbence. Forty-five millicuries of sodium ^{99m}Tc-pertechnetate was administered via a right jugular catheter for first-pass radionuclide angiography. Dynamic images were acquired at a rate of 3 seconds per frame for 45 seconds beginning at the start of injection. Radioisotope uptake within the soft tissues cranial to the kidney and within both kidneys was readily apparent, but uptake caudal to either kidney was not observed (Fig 1). These findings confirmed the diagnosis of aorto-iliac thrombosis.

Therapy was initiated immediately to dissolve the thrombus. A 2-mg dose of tissue plasminogen activator[b] was administered IV. To reduce the potential consequences of reperfusion injury, 1 L of lactated ringers solution with 10% dimethyl sulfoxide also was administered at a maintenance rate. The foal developed a bleeding diathesis. Due to severe coagulopathy, clinical deterioration, and failure to detect

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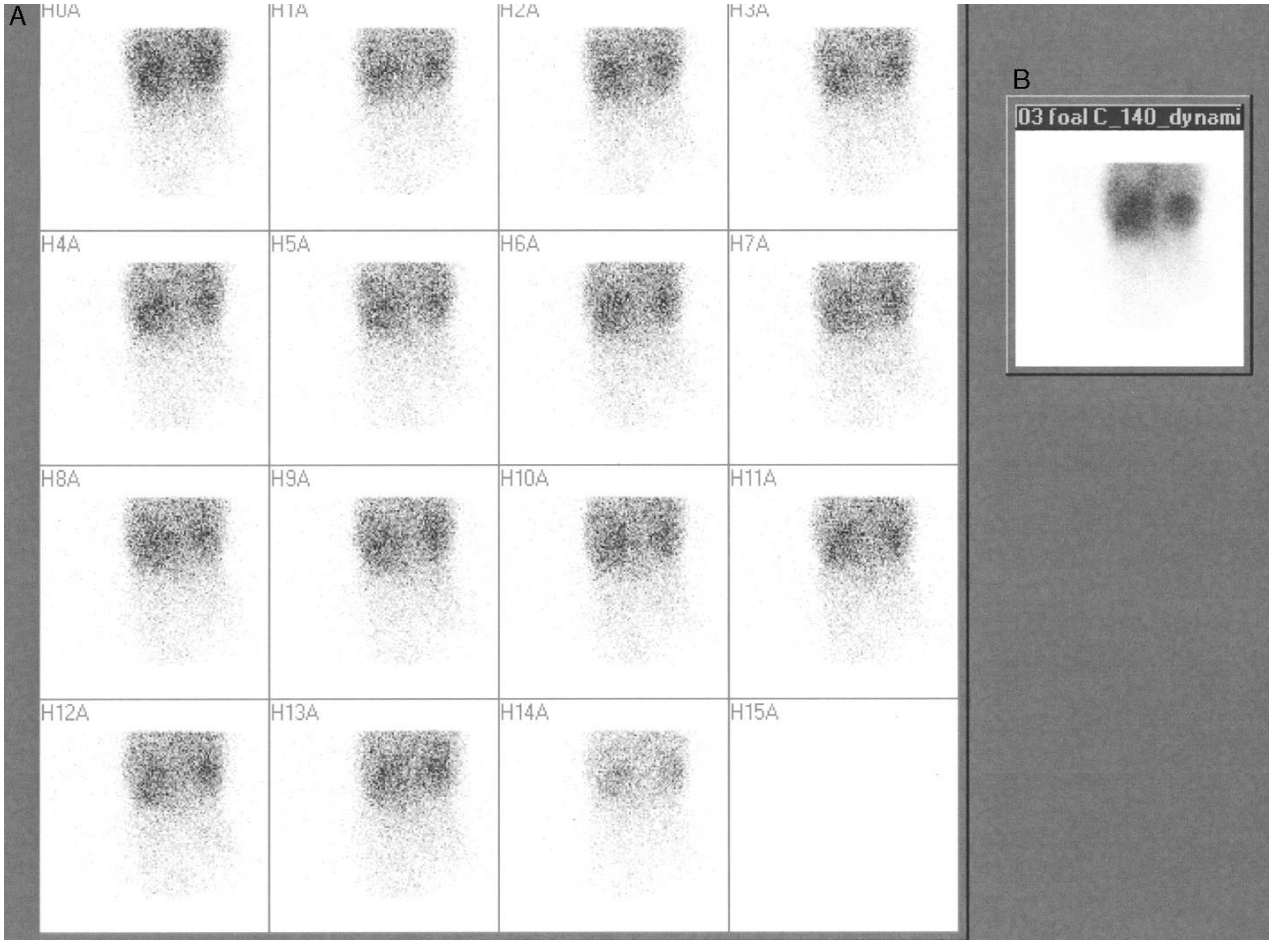

Fig 1. Dynamic **(A)** and summed dynamic dorsal plane images **(B)** of the nuclear angiogram. Note the lack of radiopharmaceutical uptake (arrow) within the soft tissues caudal to the kidneys. Cranial is to the top, caudal is to the bottom.

clinical evidence of reperfusion of the pelvic limbs, the foal was euthanized 17 hours after presentation.

Blood culture was negative. The only finding from the fecal cultures was a large growth of a potential pathogen, *Bacteroides fragilis.* Postmortem examination identified a saddle thrombus at the junction of the terminal aorta and the origin of the iliac arteries (Fig 2). The thrombus was tan to red, firm, lamellated, and was nearly completely covered by a thin layer of dark red (postmortem) clotted blood. Other clinically relevant findings included a grossly evident, severe fibrino-suppurative colitis; hemorrhage and necrosis of the adrenal glands; and pulmonary hemorrhages and edema. Microscopically, microthrombi were disseminated throughout the pulmonary vasculature. Bacterial culture results of material obtained from tissues collected at necropsy were negative.

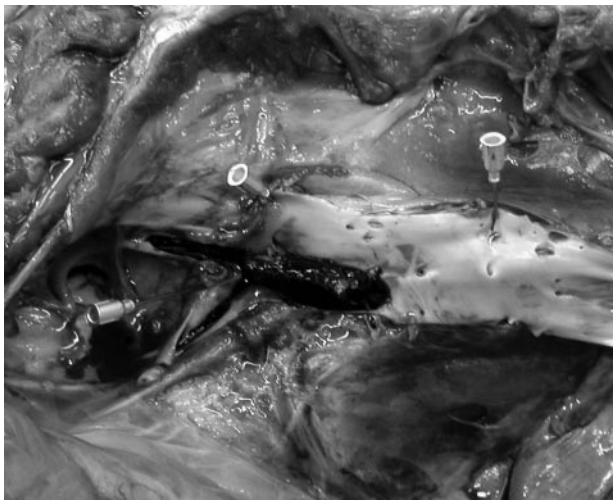

Fig 2. Saddle thrombus at the aorto-iliac junction of a foal.

Aorto-iliac thrombosis has been described previously in adult horses as an uncommon and idiopathic condition associated with weakness in the pelvic limbs, lameness, ataxia, collapse, and, less commonly, failure of ejaculation.[1] Another review described acute thrombosis in association with gram-negative bacteremia in neonatal foals and in association with inflammatory bowel disease, diarrhea, and toxemia in older foals and adults.[2] A hypercoagulable condition and poor perfusion to the distal extremities as a result of endotoxemia or septic shock may lead to thrombosis of limb arteries.[2] The stimulation of procoagulant activity by

endotoxin has been documented in horses with colic and in experimental models of equine endotoxemia.[3,4]

Aorto-iliac thrombosis has been described as a sequela to septicemia in a neonatal foal with clostridial diarrhea,[5] and other reports describe thrombosis of other arteries in septicemic foals.[5–8] Neonatal foals have lower fibrinogen and protein C antigen concentrations, and lower antithrombin III, plasminogen, alpha-2 antiplasmin, and tissue plasminogen activities during the first month postpartum,[9] perhaps explaining the increased incidence of coagulopathies (eg, arterial thrombosis) in septic foals. There were several similarities between the foal with aorto-iliac thrombosis in the previous report and the foal in this report. Both foals had severe colitis, and both foals also had evidence of adequate passive transfer of immunity from the dam, suggesting that septicemia, if present, was secondary to gastrointestinal disease. Enteric bacteria (eg, *Salmonella* spp, *E coli, B fragilis,* and *Clostridium* spp) produce exotoxins or enterotoxins that may damage the epithelium of the mucosa and lead to inflammation and subsequent absorption of endotoxins and bacteria leading to sepsis or endotoxemia. Lack of a positive culture from this foal at postmortem likely was due to the antemortem antibiotic therapy. Regardless, endotoxemia, with or without sepsis, can stimulate the systemic inflammatory response syndrome, and clinical and postmortem findings were supportive of endotoxemia. *B fragilis,* a bacterium not commonly associated with foal diarrhea, was cultured from the feces of this foal. A previous report described diarrhea associated with enterotoxigenic *B fragilis* in foals of a similar age to the foal in this report.[10]

Methylprednisolone sodium succinate has a demonstrated beneficial effect at high dosages (30 mg/kg) to treat spinal cord injury, provided it is administered within 8 hours of injury.[11] Trauma was likely to have been recent in this foal because deep pain sensation and some motor function still were present on presentation. Although the use of corticosteroids in the treatment of spinal cord trauma is widely accepted clinically in veterinary medicine, their use in the treatment of septic shock is more controversial.[12–14]

Suggested techniques for diagnosing aorto-iliac thrombosis in foals include ultrasonography, angiography, and nuclear scintigraphy. Scintigraphy has been used previously to diagnose aorto-iliac thrombosis in an adult horse.[15,16] Similar to what was observed in the foal in this report, scintigraphy identified marked reduction in blood flow through both external iliac arteries and absence of blood flow in the internal iliac arteries.[16]

Few reports describe the use of tissue plasminogen activator (t-PA) in the treatment of thrombosis in veterinary patients.[17–19] Tissue plasminogen activator potentiates the conversion of plasminogen to plasmin, which hydrolyzes fibrin and fibrinogen.[20] Administration of t-PA at the thrombus site was considered in this foal, but hind limb vascular access previously was unsuccessful for arterial blood gas determination. Due to financial reasons, the dose of t-PA administered to this foal was lower than that previously described in small animals.

This report describes the use of Doppler ultrasonography and nuclear scintigraphy in the diagnosis of aorto-iliac thrombosis in a foal. Aorto-iliac thrombosis in foals with septic or endotoxic shock has a poor prognosis. Early recognition of this clinical condition and administration of thrombolytic therapy may be beneficial in selected situations. Systemic thrombolytic therapy has been successful in the management of this syndrome in humans and in other veterinary species.[17–21]

Footnotes

[a] Oxyglobin® (hemoglobin glutamer-200 [bovine]), Biopure Corporation, Cambridge, MA

[b] Cathflo™ Activase® Alteplase, Genentech, Inc, South San Francisco, CA

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