What Is Your Diagnosis?

History

A 5-month-old 218-kg (480-lb) Appaloosa filly was evaluated because of fever, inappetence, abdominal distension, and labored breathing of 1 week's duration. The owner also reported that several horses on neighboring premises had had mucopurulent nasal discharge for a few days preceding onset of the foal's signs. The foal had been previously treated with gentamicin, phenylbutazone, and flunixin meglumine for 3 days, but no improvement in clinical signs was reported. On initial evaluation, the foal was quiet but alert and responsive. The foal had a rectal temperature of 39.4°C (102.9°F), heart rate of 60 beats/min, and respiratory rate of 56 breaths/min. No heart murmur was heard, but the heart sounds were muffled. On thoracic auscultation, harsh lung sounds were evident and a moderate amount of subcutaneous edema was noticed in the caudal aspect of the ventral portion of the abdomen. Results of a CBC revealed mild anemia and marked hyperfibrinogenemia. Serum biochemical analysis revealed mild azotemia, hyperphosphatemia, hyperglycemia, hypoproteinemia, and hypoalbuminemia. Thoracic radiographs were obtained to evaluate for thoracic disease as an underlying cause for the foal's dyspnea (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

Figure 1—Right lateral radiographic views of the cranioventral (A) and caudodorsal (B) aspects of the thorax of a 5-month-old Appaloosa filly with fever, inappetence, and labored breathing.
Diagnostic Imaging Findings and Interpretation

Marked elevation of the trachea is evident and indicative of cardiomegaly. There is border effacement between the cardiac silhouette, caudal vena cava, ventral aspect of the lung fields, and diaphragm (Figure 2). There are air bronchograms and loss of vascular margins in the peripheral regions as well as severe interstitial infiltration in the caudodorsal lung fields. Differential diagnoses for these findings include pericardial effusion, pleuropneumonia, and heart failure secondary to a congenital cardiac anomaly.

Echocardiography revealed a substantial volume of hypoechoic fluid within the pericardial space as well as a moderate amount of pleural effusion. The epicardium was extensively covered with thick fibrin, and several fibrinous adhesions extending from the epicardium to the pericardium were present. There was diastolic right atrial collapse, consistent with cardiac tamponade (Figure 3). Left ventricular systolic function was within reference limits.

Comments

Approximately 2.5 L of transparent, yellow fluid was removed via pericardiocentesis. Pericardial fluid cytologic evaluation revealed high nucleated cell count and protein concentration. Most cells were markedly degenerate neutrophils. Extracellular clusters of cocci and individualized rods were noted occasionally. Bacterial culture yielded Actinobacillus equuli. Pleural fluid was characterized by a total nucleated cell count, to
The radiographic findings of an enlarged cardiac silhouette and pleural effusion in a foal of this age made pericardial effusion or congenital cardiac defect likely differential diagnoses. Echocardiography was essential for establishing a definitive diagnosis and guiding treatment.

Pericarditis is uncommon in horses and can be effusive, fibrinous, or constrictive. Fibrinous pericarditis, as seen in this foal, is characterized by thick, shaggy layers of fibrin accumulation and can occur with or without fluid accumulation. Cardiac tamponade occurs when the effusion is of sufficient volume to result in an intrapericardial pressure that equals or exceeds the right atrial and right ventricular diastolic filling pressures.

Echocardiographically, this appears as right atrial and sometimes right ventricular collapse, resulting in decreased diastolic filling, stroke volume, and cardiac output. Signs of overt right-heart failure, including jugular distention and pulsation, pleural and peritoneal effusion, and ventral subcutaneous edema, develop if cardiac tamponade is chronic.

Treatment of pericarditis in horses should initially consist of broad-spectrum antimicrobials and supportive care until the results of the cytologic and bacterial evaluations are available. Pericardial fluid drainage relieves signs of cardiac compromise and allows removal of fibrin and inflammatory mediators.

Antimicrobial treatment was successful in resolving A. equuli pericarditis in this foal. Actinobacillus equuli is a small, nonmotile, gram-negative, facultative anaerobic rod. It is considered part of the normal flora of the oral cavity, respiratory tract, and gastrointestinal tract of adult horses but has been reported to cause respiratory tract disease, pericarditis, endocarditis, peritonitis, arthritis, orchitis, hemorrhagic diathesis, and abortion in adult horses. In neonates, A. equuli is a common cause of acute septicemia and enteritis, and foals frequently die of the disease within hours or days after infection despite aggressive treatment. The route of entry into the pericardium in the foal described in the present report remains uncertain. It has been suggested that Actinobacillus spp are pericardiotrophic in horses, and the foal of the present report had been exposed to other horses with signs of respiratory disease. The foal's respiratory signs and radiographic changes were most likely secondary to Actinobacillus pleuropneumonia; however, further diagnostic testing such as a transtracheal wash would be necessary to confirm this diagnosis.