Congestive heart failure in horses: 14 cases (1984–2001)

Jennifer L. Davis, DVM; Sarah Y. Gardner, DVM, PhD, DACVIM; Brooke Schwabenton, BS; Babetta A. Breuhaus, DVM, PhD

Objectives—To identify clinical signs, underlying cardiac conditions, echocardiographic findings, and prognosis for horses with congestive heart failure.

Design—Retrospective study.

Animals—14 horses.

Procedure—Signalment; history; clinical signs; clinicopathologic, echocardiographic, and radiographic findings; treatment; and outcome were determined by reviewing medical records.

Results—All 14 horses were examined because of a heart murmur; tachycardia was identified in all 14. Twelve horses had echocardiographic evidence of enlargement of 1 or more chambers of the heart. Other common clinical findings included jugular distention or pulsation, crackles, cough, tachypnea, and ventral edema. Nine horses had signs consistent with heart failure for >6 days. Underlying causes for heart failure included congenital defects, traumatic vascular rupture, pericarditis, pulmonary hypertension secondary to heaves, and valvular dysplasia. Seven horses were euthanatized after diagnosis of heart failure; 5 were discharged but were euthanatized or died of complications of heart disease within 1 year after discharge. The remaining 2 horses were discharged but lost to follow-up.

Conclusions and Clinical Relevance—Results suggest that congestive heart failure is rare in horses. A loud heart murmur accompanied by either jugular distention or pulsation, tachycardia, respiratory abnormalities (crackles, cough, tachypnea), and ventral edema were the most common clinical signs. Echocardiography was useful in determining the underlying cause in affected horses. The long-term prognosis for horses with congestive heart failure was grave. (J Am Vet Med Assoc 2002;220:1512–1515)

The term congestive heart failure defines a group of clinical signs that occur secondary to cardiac decompensation. The condition appears to be rare in horses, and there is little information in the veterinary literature pertaining to clinical signs or diagnosis of congestive heart failure in horses or to the prognosis of affected horses.1,4 Clinical findings would be expected to be similar to those in other species and include heart murmurs, tachycardia, jugular distention and pulsation, cough, ventral or pulmonary edema, decreased appetite accompanied by weight loss, and some degree of exercise intolerance. The purpose of the study reported here was to identify clinical signs, underlying cardiac conditions, echocardiographic findings, and prognosis for horses with congestive heart failure.

Criteria for Selection of Cases

Medical records of all horses examined at the Veterinary Teaching Hospital at North Carolina State University because of congestive heart failure, regardless of cause, were reviewed. Horses were included in the study if the final diagnosis included congestive heart failure, regardless of the underlying cause. Data recorded included age; breed; sex; history; duration of clinical signs; physical examination findings; classification of the heart murmur; results of arterial blood gas analyses; results of hematologic and serum biochemical analyses, including measurement of serum activities of the cardiac isoenzymes of creatine kinase (CK) and lactate dehydrogenase (LDH); echocardiographic, electrocardiographic, and radiographic findings; treatment; outcome; and necropsy findings.

Results

Signalment—Fourteen horses met the criteria for inclusion in the study. This represented 10.4% of the horses brought to the veterinary teaching hospital for cardiac evaluation during the study period. Median age of horses included in the study was 8.5 years, but horses ranged from 1 month to 18 years of age (mean ± SD, 8.6 ± 6.4 years). Seven horses were female, 4 were geldings, and 3 were stallions. There were 3 Thoroughbreds, 3 Quarter Horses, 2 American Paints, 2 Tennessee Walking Horses, 1 American Saddle Horse, 1 warmblood, 1 Arabian, and 1 Arabian-cross.

History and clinical abnormalities—Five horses had signs of acute congestive heart failure; the remaining 9 horses had had clinical signs of congestive heart failure for 6 days to 8 months (median, 1 month; mean ± SD, 63 ± 75 days) prior to examination at the teaching hospital. Rectal temperature was normal in 11 horses; high in 2, and low in 1. Respiratory rates for the 2 horses <1 year of age were 48 and 56 breaths/min. Median respiratory rate for the 12 horses >1 year of age was 28 breaths/min (range, 20 to 84 breaths/min; mean ± SD, 32 ± 20 breaths/min). Abnormal lung sounds (crackles) were detected in 10 horses, and 9 horses had a cough. All 14 horses had tachycardia. Heart rates of the 2 horses <1 year of age were 128 and 108 beats/min. Median heart rate for the 12 horses >1 year of age was 62 beats/min (range, 48 to 90 beats/min; mean ± SD, 64 ± 13 beats/min). Twelve horses had jugular distention or pulsation, and 7 had ventral...
edema. Mucous membranes were classified as grossly normal (7 horses), pale (3), injected (2), icteric (1), or cyanotic (1). Capillary refill time was < 2 seconds in 9 horses and prolonged in 5. Six horses were considered to have an ideal body condition, 5 were underweight, and 1 was obese. Murmurs could be ausculted in all horses and were grade III/VI or higher. Murmurs were heard on both sides of the thorax in 7 horses, the left side only in 5, and the right side only in 2. The point of maximal intensity of the murmurs was not consistently recorded. One horse had muffled heart sounds secondary to pericardial effusion. Thirteen horses had systolic murmurs; the remaining horse had systolic and diastolic murmurs.

Clinicopathologic findings—Results of a CBC and serum biochemical profile were available for 13 horses, but there were no consistent abnormalities. Arterial blood gas analyses were performed in 7 horses, and all 7 had hypoxemia (median PaO$_2$, 53 mm Hg; range, 38 to 103 mm Hg; mean ± SD, 65 ± 26 mm Hg). Serum activities of the cardiac isoenzymes of CK and LDH (ie, CK-MB and LDHi$_2$&56$, respectively) were measured in 4 horses. The cardiac isoenzyme fraction of CK was normal in 3 horses and high in 1 (12.5%; reference range, < 5%). The cardiac isoenzyme fraction of LDH was normal in 3 horses and high in 1 (49%; reference range, < 40%). Pleural fluid analysis was performed in 2 of 4 horses with pleural effusion. Results of cytologic evaluation were unremarkable; however, the total protein concentration was high (40 g/dl) in 1 of the 2. Abdominocentesis and abdominal fluid analysis were performed in 4 horses; results were normal in all 4. Pericardial effusion was detected in 1 horse. Evaluation of pericardial fluid obtained at necropsy revealed a flocculent effusion that yielded a light growth of *Streptococcus bovis*. Blood samples from 3 horses were submitted for bacterial culture; none of the samples yielded any bacterial growth. Transtracheal washes were performed in 3 horses that had abnormal lung sounds and radiographic evidence of pneumonia or pleuropneumonia. Bacterial culture of wash samples from 1 horse yielded *Escherichia coli* and *Klebsiella* spp; bacterial culture of wash samples from a second horse yielded *Flavobacterium* spp and *Staphylococcus epidemidis*, both of which were considered to be contaminants; and bacterial culture of wash samples from the third horse did not yield any growth.

Electrocardiographic findings—Electrocardiography was performed on 6 horses with arrhythmias. Four had electrocardiographic evidence of atrial fibrillation, 1 had second-degree atrioventricular block, and 1 had occasional junctional premature complexes. All 6 horses had tachycardia.

Radiographic findings—Thoracic radiography was performed on 10 horses. Seven horses, all of which were suspected to have left-sided heart failure or biventricular heart failure, had a diffuse interstitial to broncho-interstitial pattern, consistent with pulmonary edema. Seven horses had radiographic evidence of cardiomegaly. One horse had an increased vascular pattern, and 3 horses had evidence of pleural effusion; all 4 of these horses were suspected to have right-sided heart failure or biventricular heart failure.

Echocardiographic findings—Echocardiography was performed in 13 horses. Examinations were made from the right side in the third and fourth intercostal spaces with a 3.5 MHz transducer. Twelve horses had evidence of enlargement of at least 1 chamber of the heart; the remaining horse had pericardial effusion with tamponade. Five horses had generalized cardiomegaly, 3 had enlargement of the left atrium, 3 had enlargement of both the left atrium and the left ventricle, and 1 had enlargement of the right atrium and right ventricle. Seven horses had evidence of thickened mitral valves with moderate to severe regurgitation. Eight horses had evidence of a thickened tricuspid valve with mild to moderate regurgitation. Six horses had both mitral and tricuspid regurgitation. Four horses had a pulmonary artery diameter greater than the aortic diameter, indicating enlargement of the pulmonary artery. One horse had evidence of an aorto-cardiac fistula. Three horses had evidence of congenital defects, including a foal with a ventricular septal defect, an atrial septal defect, and an overriding aorta with left-to-right shunting; a yearling with a ventricular septal defect and overriding aorta with left-to-right shunting, a hypoplastic right ventricle, tricuspid atresia, and an atrial septal defect; and an adult horse with a ventricular septal defect and severe tricuspid regurgitation.

Treatment and outcome—Four horses were euthanatized immediately after the diagnosis of congestive heart failure was made; the remaining 10 were treated. Three horses were treated for several days in the hospital but were euthanatized without being discharged. The remaining 7 were discharged from the hospital, but 3 were euthanatized (4 horses) or found dead (1) within 1 year after discharge. One horse was reportedly doing well after 1 month of treatment but was lost to further follow-up; the other horse was lost to follow-up after discharge. A necropsy was performed on 9 horses, 8 of which had undergone echocardiography. In all 8, the echocardiographic diagnosis was confirmed.

Discussion

Previous reports have described heart failure in horses with a variety of abnormalities, including valvular lesions, pericarditis, neoplasia, congenital defects, aorto-cardiac fistulas, myocarditis, and endocarditis. Similarly, congestive heart failure among horses in the present report was attributed to a variety of underlying causes. Mitral regurgitation appeared to be the most common underlying cause, which is not surprising considering it is the most common valvular insufficiency detected clinically in horses. No specific underlying cause for the valvular insufficiency was found in any of these horses, and valvular insufficiency was assumed to be a result of aging and fibrosis. Four horses had evidence of atrial fibrillation on auscultation that was confirmed electrocardiographically; all of these horses had left atrial enlargement. Atrial fibrillation in combination with a rapid ventricular response...
Cardiac troponin I has been used as a marker for myocardial damage experimentally and may be a more sensitive tool for use in the future.14

One horse in this study had cor pulmonale and right-sided heart failure secondary to heaves. The diagnosis was made on the basis of enlargement of the pulmonary artery, right atrium, and right ventricle, along with a history of heaves. Another horse had endocarditis, which was diagnosed at necropsy.

Clinical signs of congestive heart failure are often divided into those associated with left-sided heart failure (eg, heart failure secondary to mitral regurgitation or patent ductus arteriosus), right-sided heart failure (eg, heart failure secondary to tricuspid insufficiency or pulmonic stenosis), and biventricular heart failure (eg, heart failure associated with cardiomypathy, and mitral and tricuspid regurgitation). Signs of left-sided congestive heart failure generally include those associated with pulmonary edema, such as cough and dyspnea, whereas signs of right-sided congestive heart failure include jugular venous distention and pulsation, hepatosplenomegaly, ventral edema, ascites, and, occasionally, pleural or pericardial effusion.15 Signs of biventricular congestive heart failure vary, but most affected horses would be expected to have some combination of signs of right- and left-sided heart failure. Regardless of the type of congestive signs (ie, right-sided, left-sided, or biventricular), signs of low cardiac output (ie, exercise intolerance, collapse, syncope, azotemia, weak pulses, and poor tissue perfusion) may also be seen,15 although signs of low cardiac output may be seen in the absence of signs of congestive heart failure.13 Tachycardia is a frequent but nonspecific finding in animals with heart failure.

For 5 horses in the present study, the predominant clinical signs were suggestive of left-sided congestive heart failure; all of these horses had mitral regurgitation. Signs of right-sided congestive heart failure predominated in 3 horses, including 1 with multiple congenital defects, 1 with tricuspid regurgitation and a ventricular septal defect, and 1 with cor pulmonale. Signs of biventricular congenital heart failure were seen in the remaining 6 horses. Two of these horses had mitral and tricuspid regurgitation, 1 had viral myocarditis, 1 had multiple congenital defects, 1 had septic pericarditis with pulmonary edema, and 1 had an aorto-cardiac fistula.

One horse in this study had pulmonary edema in association with septic pericarditis. Pulmonary edema is an atypical finding in animals with pericardial disease but could have resulted from restriction of left atrial or ventricular inflow associated with the pericarditis. Alternatively, it might have been a reflection of noncardiogenic pulmonary edema. Signs of biventricular congestive heart failure in the horse with the aorto-cardiac fistula may have been a result of shunting of blood into the right ventricular outflow tract, effectively bypassing the right ventricle and resulting in a volume overload for the left ventricle.

Four horses in the present study with left-sided congestive heart failure were reported to have jugular distention or pulsation. Most likely, this was a result of over-interpretation of a normal jugular pulse or misidentification of a visible carotid pulse. Alternatively, this may have reflected the effects of right ventricular pressure overload secondary to left-sided heart failure. One horse suspected of having right-sided congestive heart failure did not have evidence of jugular distention or pulsation. However, this horse had severe anemia and was recumbent, making evaluation difficult.

All of the horses in the present study had clinically important heart murmurs (grade III/VI or higher) and tachycardia. Other common clinical signs included jugular distention or pulsation (12 horses), crackles (10), cough (9), tachypnea (8), ventral edema (7), and arrhythmias (6). Cyanotic mucous membranes were seen in 1 horse that had evidence of right-to-left shunting secondary to overriding of the aorta.

Twelve horses had echocardiographic evidence of enlargement of 1 or more cardiac chambers. Nine horses had a history of clinical signs consistent with heart failure for > 6 days. Exercise intolerance was not a common complaint in these horses, most likely because they were not performance horses. Results of hematologic and serum biochemical analyses performed on these horses were inconsistent. Arterial blood gas analyses consistently revealed hypoxemia most likely attributable to ventilation-perfusion inequalities, with or without diffusion impairment secondary to pulmonary edema. Others have reported evidence of stress, azotemia, and hyponatremia in horses with heart failure,3 but these were not consistently seen in the present study.3 A diffuse, unstructured bronchointerstitial pattern was a consistent finding on thoracic radiographs of horses with left-sided or biventricular heart failure, suggesting that these horses had pulmonary edema. Cardiomegaly was detected in 7 of 10 horses.

Treatment of heart failure in horses in the present study was similar to that suggested for other...
species. Treatments centered on increasing contractility and slowing the heart rate (digoxin), decreasing volume overload (furosemide), reducing afterload (hydralazine), inhibiting angiotensin-converting enzyme activity (enalapril), and correcting arrhythmias (quinidine sulfate). Other treatments centered on nonspecific treatment of clinical signs and specific treatment of underlying causes. There was an insufficient number of horses in this study to determine the efficacy of treatment.

Results for horses in this study suggest that the prognosis for horses with congestive heart failure is grave. This agrees with other reports in the literature. All of the horses in this study for which follow-up information was available were euthanatized or had died within 1 year after diagnosis of heart failure.

References