Tracheal collapse in American Miniature Horses: 13 cases (1985–2007)

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EQUINE

Objective—To describe the clinical and laboratory findings, diagnostic features, and outcome of tracheal collapse in American Miniature Horses at a referral institution.

Design—Retrospective case series.

Animals—13 American Miniature Horses with tracheal collapse.

Procedures—Medical records of American Miniature Horses with tracheal collapse at a referral hospital were reviewed. Data extracted included signalment, history, clinical signs, laboratory data, diagnostic procedures, outcome, and histologic findings.

Results—Tracheal collapse was documented in 5.6% of American Miniature Horses admitted to this referral hospital. Median age at onset of clinical signs was 11 years with a range of 2 to 15 years. Common complaints and clinical signs included respiratory distress, tachypnea, inspiratory honking noises, and increased abdominal expiratory effort, which were exacerbated by stressful events, pregnancy, exercise, a dusty environment, and eating. Tracheal collapse was confirmed by use of radiography, endoscopy, fluoroscopy, or postmortem examination. Dorsoventral flattening of the extra- or intrathoracic trachea, or both, was more common than lateral collapse. Tracheal chondromalacia was identified histologically in 4 cases, and mortality rate for affected horses was 10 of 13.

Conclusions and Clinical Relevance—Tracheal collapse was relatively common in this study of American Miniature Horses, and outcome was poor. The etiopathogenesis of the disease remains unknown. (*J Am Vet Med Assoc* 2008;233:1302–1306)

Tracheal diseases in horses include acquired tracheal stenosis from intraluminal and extraluminal causes,[1] collapse secondary to pulmonary infection,[2] trauma with open and closed wounds,[3,4] mass-occupying lesions (abscess, neoplasia, and hematoma),[1,5–7] foreign body,[1] and granulomatous tracheitis.[8] Tracheal collapse not associated with trauma or compression by extraluminal causes in equine species is an uncommon disorder, as evidenced by few reports[2,9–15] in the literature. Tracheal collapse has been reported in horses, ponies, miniature horses, donkeys, and mules;[1,15,16] however, pony and miniature breeds appear to be overrepresented in the veterinary literature.[1,9,10,12,14,17]

Proposed causes for tracheal collapse include congenital and degenerative abnormalities in younger and older animals, respectively. Congenital disorders of the trachea such as tracheal chondrodysplasia are rare in horses.[9] In contrast, tracheal collapse caused by weakening of the cartilage support of large airways is a common disorder of small breed dogs; Yorkshire Terrier, Toy and Miniature Poodle, Pomeranian, and Chihuahua breeds are overrepresented.[18] Histochemical and ultrastructural studies of tracheal cartilage from affected dogs reveal fewer chondrocytes than normal and damaged chondrocytes; matrix dissolution; decreased mucopolysaccharide, chondroitin sulfate, and glycosaminoglycan content; and transformation of normal hyaline cartilage into fibrocartilage.[19,20] These alterations suggest a form of chondromalacia.[21] The etiology of tracheal collapse in small breed dogs remains uncertain but likely includes multifactorial causes such as obesity, concurrent disease, and genetic predisposition.[18] With the exception of a few case reports, studies on tracheal collapse in horses are lacking. The purpose of the study reported here was to report the history, clinical signs, laboratory findings, diagnostic modalities, outcome, and histopathologic features in American Miniature Horses with tracheal collapse at a referral veterinary hospital.

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Materials and Methods

Case selection criteria—The computerized database at the University of California, Davis, William R. Pritchard Veterinary Medical Teaching Hospital was searched during the years of 1985 to 2007 for American Miniature Horses with a clinical diagnosis of tracheal collapse. Cases were excluded if a diagnosis was not confirmed by use of endoscopy, fluoroscopy, radiography, or postmortem examination or if collapse was associated with trauma or extraluminal compression caused by masses or inflammation.

Procedures—Medical records were searched under different fields (clinical diagnosis, ancillary diagnostic tests [radiology, endoscopy, fluoroscopy, and ultrasonography], and pathologic diagnosis) by use of the following words: tracheal collapse, tracheal flattening, and tracheal narrowing in American Miniature Horses.

Tracheal collapse was retrospectively graded on the basis of endoscopic or pathologic examination, or both, by use of a method well established for use in dogs[18,22] as follows: grade

1 = minor protrusion of dorsal tracheal membrane into airway lumen with < 25% reduction of airway diameter; grade 2 = mild elongation and flattening of tracheal rings with 50% reduction of airway diameter; grade 3 = marked flattening of tracheal rings and lengthening of dorsal tracheal membrane with 75% reduction of airway diameter; and grade 4 = severe flattening of tracheal rings with dorsal deviation of ventral tracheal surface and < 10% airway diameter.

Results

Review of medical records identified 13 American Miniature Horses with tracheal collapse not associated with trauma, infection, or mass lesions. During the study period, 231 American Miniature Horses were examined at our institution, yielding tracheal collapse in 5.6% of American Miniature Horses examined at our hospital. Of those 13 horses, sex distribution was 8 females, 3 sexually intact males, and 2 geldings. Three of the 8 mares were evaluated in the last months of pregnancy, and 1 of the mares had dystocia. Of the remaining 5 mares, 2 had recently foaled, 1 had aborted a 5-month-gestation fetus, and 2 were not pregnant. Age of affected horses ranged from 3 to 17 years with a median of 12 years. Clinical signs had been present from 1 to 5 years in 9 of 13 horses, and acute signs were reported in the remaining 4 horses. Nutritional status was recorded in 11 of 13 horses; 8 had a good nutritional status (weight ranged from 104.5 to 119.5 kg [229.9 to 262.9 lb], 2 were obese, and 1 was emaciated.

The most common complaints included respiratory noises described by the owners as honking, squeaking, or wheezing in all horses; severe respiratory distress and tachypnea in 7 horses; exercise intolerance in 5; and trouble eating in 4. Other complaints included gasping for air, collapse, lethargy, anorexia, and recumbency. One mare with respiratory distress died while being transported to our hospital. Owners reported that horses did not appear ill and physical activity was not compromised at the onset of respiratory honking noises. Some of the described complaints were initially intermittent and many times associated with stressful events, exercise, dusty environment, and eating. All owners complained about the progression of signs, and signs were more prominent during pregnancy in mares. One stallion with a 1-year history of respiratory noises associated with exercise collapsed while mounting a mare. One horse was referred as a patient with esophageal obstruction, and a second horse had recurrent airway obstruction that had failed to respond to systemic corticosteroids and bronchodilators. None of the 13 horses had a history of tracheal trauma, tracheotomy, or endotracheal intubation.

Physical examination findings included severe respiratory distress with flaring of the nostrils, severe exercise intolerance, loud respiratory stridor or honking noise during inspiration, and increased abdominal expiratory effort in all horses. Tracheal collapse was identified in 2 horses by palpation of a flattened trachea with lateral protrusion of cartilage rings. Heart rate ranged from 60 to 130 beats/min (reference range, 28 to 40 beats/min), respiratory rate from 11 to 50 breaths/min (reference range, 8 to 16 breaths/min), and rectal temperature from 38.2° to 40.0°C (100.8° to 104°F; reference range, 37.0° to 38.2°C [98.6° to 100.8°F]).

Concurrent problems included dental problems (n = 2 horses), aspiration pneumonia (1), dorsal displacement of the soft palate (1), left rostral and caudal maxillary sinusitis (1), and dystocia (1). Two horses were suspected of having pituitary pars intermedia dysfunction on the basis of phenotype.

A CBC was performed in 7 horses, and results of 4 CBCs were within reference values. One horse had hyperproteinemia (9.4 g/dL; reference range, 5.7 to 7.5 g/dL) and hyperfibrinogenemia (500 mg/dL; reference range, 100 to 400 mg/dL). The second horse had mild leukopenia (WBC, 3,680 cells/µL; reference range, 5,000 to 11,600 cells/µL), neutropenia (2,009 cells/µL; reference range, 2,600 to 6,800 cells/µL), lymphopenia (983 cells/µL; reference range, 1,600 to 5,800 cells/µL), hyperproteinemia (8.4 g/dL), and hyperfibrinogemia (800 mg/dL). A third horse had mild lymphopenia (1,148 cells/µL). A serum biochemical panel was performed in 3 horses, and results were within reference ranges in 1 horse. One horse had mild azotemia (creatinine, 3.4 mg/dL; reference range, 0.9 to 2.0 mg/dL; BUN, 47 mg/dL; reference range, 12 to 27 mg/dL) and hypokalemia (2.3 mmol/L; reference range, 3.0 to 5.6 mmol/L); and 1 horse had mild hyperglycemia (184 mg/dL; reference range, 60 to 107 mg/dL) and mildly increased activity of sorbitol dehydrogenase (12 U/L; reference range, 0 to 8 U/L).

Tracheal collapse was confirmed by use of radiography (9/9 horses), endoscopy (7/7 horses), fluoroscopy (1/1 horse), and postmortem examination (10/10 horses). Radiographs of the trachea and carina were taken in 9 horses, and all revealed evidence of dorsoventral tracheal collapse. Collapse of the cervical portion of the trachea was observed in 3 horses, and 6 horses had collapse from the cervical trachea to the carina **(Figure 1)**. Other radiographic findings included evidence of aspiration pneumonia (n = 1), diffuse bronchointerstitial pneumonia (1), maxillary sinusitis (1), and tracheal ring calcification (1). Fluoroscopy was performed in 1 mare and revealed dynamic tracheal collapse from the cervical trachea to the carina (grade 4).

Endoscopy was performed in 7 horses and confirmed dorsoventral tracheal collapse in 6 of 7 horses and lateral collapse in the remaining horse **(Figure 2)**. One horse was walked on the treadmill during endoscopy but developed severe exercise intolerance, and the examination was inter-

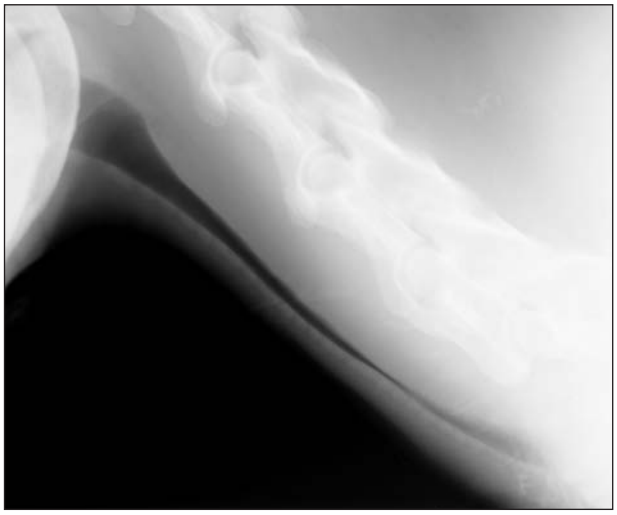

Figure 1—Lateral cervical radiographic view of a horse with extrathoracic tracheal collapse (grade 4). Notice severe narrowing of the tracheal lumen.

rupted. In 6 horses, collapse was so severe that it precluded advancement of the endoscope beyond the affected region. In 1 horse, grade 3 collapse extended from the origin of the trachea to 4 cm cranial to the carina. In all horses, edema of the pharynx and larynx was evident. Additional findings included intermittent dorsal displacement of the soft palate and prominent arytenoids and epiglottis.

An emergency tracheotomy was performed in 5 horses but provided minimal relief until an endotracheal tube was passed to open the collapsed trachea; however, 1 horse died while inserting the endotracheal tube. Because of severe flattening of the trachea, a tracheotomy resulted in laceration of the carotid artery and esophagus in 1 horse each. The horse with the lacerated carotid artery was immediately euthanatized. Surgical repair of the esophagus and trachea was attempted 4 days later in the second horse but was unsuccessful because of cellulitis and hemorrhage. This horse received intensive care for 4 additional days until the owner elected euthanasia.

Owners of 3 of the remaining live horses (n = 9) declined hospitalization for further stabilization, and horses were lost to follow-up. Two other horses were hospitalized for treatment of concurrent problems (aspiration pneumonia and sinusitis). The horse with aspiration pneumonia developed colitis caused by *Salmonella enterica* serovar Typhimurium 9 days after evaluation, and its condition rapidly deteriorated despite aggressive supportive therapy; the owner elected euthanasia. The horse with sinusitis had an endotracheal tube placed through a tracheotomy. This tube had to be placed up to or close to the carina to result in complete relief of the respiratory distress. Attempts to remove the tube for cleaning resulted in respiratory distress and collapse. The owner elected euthanasia. Surgical repair of the tracheal collapse (implantation of an intraluminal or extraluminal stent) was offered for the remaining horses (n = 4) but was declined because of financial constraints, humane reasons, and poor prognosis. These horses were euthanatized, resulting in a mortality rate of 10 of 13.

On the basis of gross postmortem examination, tracheal collapse was categorized as grade 3 in 1 horse and grade 4 in 9 horses (**Figure 3**). In 4 horses, histo-

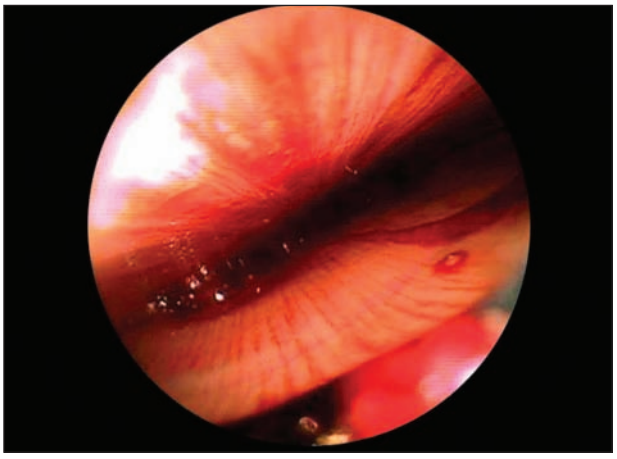

Figure 2—Endoscopic cervical view of the horse in Figure 1. Notice severe intraluminal narrowing and the striated appearance of the trachea.

Figure 3—Photographic appearance of cross sections of the trachea of a horse with extra- and intrathoracic tracheal collapse (grade 4). Notice dorsoventral flattening (grades 3 to 4) at different levels and elongation of the dorsal membrane (left side of the tracheal sections). Scale = 1 cm.

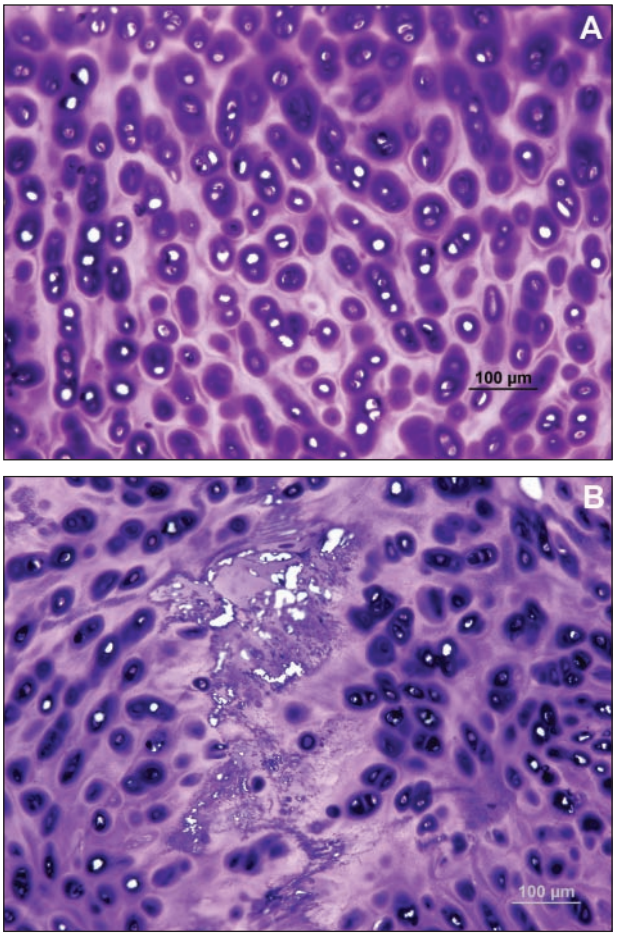

Figure 4—Photomicroscopic appearance of a section of the trachea of a horse with a normal tracheal ring (A) or tracheal collapse (B). In panel B, notice a focal area of chondromalacia and fewer chondrocytes, compared with panel A. H&E stain; bar = 100 µm.

logic examination was available and compatible with tracheal chondromalacia at stages that varied from mild multifocal hyalinization of the extracellular matrix in the central areas of the tracheal rings with no evidence of chondrocyte degeneration to marked multifocal to coalescing areas of chondromalacia characterized by complete degradation of the cartilaginous matrix and loss of chondrocytes (**Figure 4**). Scattered loss of cartilaginous matrix with hyalinization of the territorial matrix in the central regions of the rings was observed in all 4 horses. Mineralization of these areas was observed in 1 horse.

Discussion

In this study, almost 6% of the American Miniature Horses evaluated at a referral institution had a diagnosis of tracheal collapse. Affected horses in this study were middle-aged (median age, 11 years), which was similar to the situation in small breed dogs,[23,24] 2 ponies (12 and 13 years old),[10,12] and one 7-year-old American Miniature Horse.[17] The disorder has also been reported in 2 American Miniature Horse foals that were 2 and 7 months of age.[9,14] The 2-month-old foal had moderate to severe chondrodysplasia with disorganized fibrous connective tissue.[9] The fibrous joints were located bilaterally at the most lateral aspect of the tracheal rings, allowing the rings to collapse.[9] Chondrodysplasia was suspected but not confirmed in a 7-month-old foal on the basis of age, history, and clinical findings.[14] It is possible that tracheal collapse in the American Miniature Horse could have been present at an earlier age but not recognized because of mild or inapparent signs. Tracheal collapse has also been reported in young calves ranging from 2 weeks to 4 months of age.[25] In dogs, young or aged animals may have clinical signs referable to tracheal collapse.[18]

In the present study, most horses (8/11) with tracheal collapse in which body condition was recorded had a good body condition, and this differs from small breed dogs in which almost half are obese.[18] In previous reports[9,10,12,14,17] of tracheal collapse, body condition was not provided. Although there were more females than sexually intact and castrated males in our study, there was no obvious sex predilection, compared with the American Miniature Horse population at our hospital during the study period. However, 6 of 8 mares with tracheal collapse developed or had exacerbation of signs during the middle and latter months of pregnancy. This suggests that increased pressure on the thorax because of a space-occupying mass effect by the fetus, increased metabolic demands, or stress associated with pregnancy may have exacerbated or contributed to respiratory signs.

Clinical signs of tracheal collapse vary depending on the site and severity of airway collapse. With extrathoracic tracheal collapse, maximal narrowing will occur during inspiration because of the relative negative airway pressure, compared with the pressure around the trachea.[14] In intrathoracic tracheal collapse, maximal narrowing will occur during expiration because of the higher pressure surrounding the trachea, compared with intraluminal pressure.[14] In the present study, tracheal collapse was identified at the extrathoracic level in 2 horses, whereas both extra- and intrathoracic collapse was observed in 11 horses. The most common complaints and clinical signs included respiratory distress with honking noises on inspiration in combination with increased abdominal expiratory effort. These findings were compatible with severe collapse at both the cervical and intrathoracic levels of the trachea. In addition, tachycardia, tachypnea, and severe exercise intolerance were common in this study. Previous reports[9,12,14] in pony and miniature breeds reported comparable signs of honking noises, respiratory distress, nostril flare, and exercise intolerance. However, tracheal collapse was found incidentally at the time of necropsy in a research female Shetland pony.[10] Such a case raises the possibility that tracheal collapse may not be clinically apparent in an animal that is not exercised or placed under stress.

Concurrent problems were present in several horses with tracheal collapse, including aspiration pneumonia, sinusitis, dental disease, dystocia, and presumed pituitary pars intermedia dysfunction. In dogs, airway infection or inflammation, trauma, cardiomegaly, or congestive heart failure may contribute to or exacerbate signs of airway collapse.[24,26] Dogs commonly have waxing and waning clinical signs of airway collapse that are worsened by heat, humidity, exercise, and eating or drinking.[18] Similarly, owners of affected horses in this study reported intermittent signs with exacerbation associated with stressful events, pregnancy, exercise, a dusty environment, and eating.

In the present study, tracheal collapse was identified via physical examination, fluoroscopy (n = 1), palpation of the trachea (2), endoscopy (7), and radiography (9). A common endoscopic finding was pharyngeal and laryngeal edema that was likely attributable to increased respiratory effort. An emergency tracheotomy in 5 horses did not relieve the respiratory distress because of the severity and extent of tracheal collapse. Furthermore, tracheotomy resulted in the laceration of the esophagus and carotid artery. These complications highlight the importance of being more cautious when performing a tracheotomy in horses with tracheal collapse because severe collapse may result in an anatomic shift of neighboring structures. An alternative treatment to consider might be nasotracheal intubation with a foal-size nasogastric tube. Bypassing the area of tracheal collapse and supplementing with exogenous oxygen might be considered for temporary stabilization of affected animals.

Dorsoventral flattening of the trachea was observed in 12 of 13 American Miniature Horses, whereas only 1 horse had lateral collapse. Similarly, lateral collapse in small breed dogs is uncommon and likely represents traumatic injury.[23] In the present study, most horses had the most severe form of tracheal collapse (grade 4 in 10/13 horses and grade 3 in 3/13 horses). Dogs that require surgical intervention for tracheal collapse most commonly have grade 4 (52%) or grade 3 (34%) collapse.[23] In the present group of horses, surgical intervention was offered but declined.[27]

Histologic examination was available in 4 horses with tracheal collapse, and various stages of tracheal

chondromalacia (tracheomalacia) were identified. Tracheomalacia refers to weakness of the trachea, frequently caused by reduction or atrophy of the longitudinal elastic fibers of the pars membranacea or impaired cartilage integrity such that the airway is softer and susceptible to collapse.[28] Tracheomalacia may involve one portion of the trachea or the entire trachea.[28] If the mainstem bronchi are also involved, the term tracheobronchomalacia is used.[28] In the present study, the histopathologic findings included mild degenerative alterations of the extracellular matrix, hyalinization of the tracheal rings, complete degradation of the cartilaginous matrix, loss of chondrocytes, and mineralization of the tracheal rings. These findings suggest a degenerative rather than a chondrodysplastic process. However, because chondrodysplasia has been reported as the cause of tracheal collapse in American Miniature Horses,[9] a detailed prospective morphologic and histochemical study of various sections of the tracheal rings in affected horses would be beneficial to determine whether various disease processes are involved in the development of tracheal collapse in American Miniature Horses.

Results from the present study suggest that tracheal collapse in American Miniature Horses is primary in nature and consistent with tracheomalacia. Prognosis for tracheal collapse in affected horses was poor and likely caused by advanced disease at the time of evaluation. Medical management would not likely result in successful management of tracheal collapse in these horses if a degenerative or chondrodysplastic process is involved. However, any stressful events should be avoided, and medical management of concurrent diseases must be addressed. Presently, the benefits and pitfalls of surgical intervention in the short- and long-term outcome for the management of tracheal collapse in horses are unknown but appear to have limited success on the basis of a few case reports.[11,12,14,17]

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