CASE REPORT

Esophageal Ectasia in a Quarterhorse Colt

S. M. BARBER, B. G. McLAUGHLIN AND P. B. FRETZ

Department of Veterinary Clinical Studies (Barber and Fretz) and Department of Veterinary Pathology (McLaughlin), Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0

Summary

A one month old Quarterhorse colt was presented after a week history of bilateral nasal discharge and respiratory difficulty. The cervical esophagus was greatly dilated, tortuous and filled with diluted milk. A nasogastric tube could not be passed beyond the base of the heart. An aspiration pneumonia was found at postmortem examination and the esophageal segment from the pharynx to the base of the heart was dilated, thin-walled, had degenerative muscular changes, and a reduction in size and number of ganglion cells of the myenteric plexus. Muscular hypertrophy of the terminal esophagus had reduced its lumen size. Some similarities and disparities of this condition to achalasia of man and megaesophagus of dogs are discussed.

Résumé

Ectasie de l'oesophage, chez un poulain Quarterhorse

Un poulain Quarterhorse, âgé d'un mois, présentait, depuis une semaine, un écoulement nasal bilatéral et des difficultés respiratoires. La partie cervicale de son oesophage était très dilatée, tortueuse et remplie de lait dilué. Il s'avéra impossible de faire progresser un tube oesophagien au delà de la base du coeur. La nécropsie permit de constater la présence d'une pneumonie par aspiration; l'histopathologie révéla que le segment de l'oesophage qui s'étend du pharynx à la base du coeur était dilaté, aminci et qu'il présentait de la dégénérescence musculaire, ainsi qu'une réduction des dimensions et du nombre de cellules ganglionnaires du plexus myentérique. L'hypertrophie musculaire de la partie terminale de l'oesophage en avait réduit le diamètre de sa lumière. Les auteurs commentent certaines ressemblances et différences entre cette condition et l'achalasie de l'oesophage, chez l'homme.

Introduction

Ectasia (dilatation) is a sign of esophageal dysfunction rather than a specific disease and has been associated with a number of etiological factors (1-4). It has been reported commonly in people with achalasia (5-8) and in dogs with neuromuscular dysfunctions called achalasia and megaesophagus (9-24).

Achalasia of the esophagus in man is a specific smooth muscle disorder characterized by aperistalsis of the lower two-thirds of the esophagus and failure of the lower esophageal sphincter to relax after swallowing (5-8). It can be differentiated by specific diagnostic tests and has characteristic gross and histological changes (5-8).

Megaesophagus in dogs is thought to represent some developmental immaturity of the innervation and/or musculature (12,20,21). Improvement with age is seen clinically and on radiographic and manometric studies (12,21). The term achalasia has often been used to refer to these cases. This is a misnomer as experimental studies have shown that achalasia is not present in these dogs with esophageal dysfunction (20,21).

Achalasia of mature dogs has been identified and radiographically, manometrically, and clinically resembles the condition in man (23).

Although obstruction of the equine esophagus is common (3,25), ectasia is not a common finding. It has been seen in foals with esophageal dysfunction and has similarities to both achalasia of man and megaesophagus of dogs (2,26,27).

History

A one month old Quarterhorse colt was presented to the Western College of Veterinary Medicine with a one week history of continuous nasal discharge and respiratory difficulty that did not respond to the owner's antibiotic therapy. The owner believed the foal was normal at birth but did not observe him again until the mare was brought in from the pasture to be bred.

Clinical Findings

The foal was depressed, in poor bodily condition, had a dry hair coat, and an elevated respiratory rate (48/minute) and rectal temperature (38.9°C). There was a continuous, copious, bilateral nasal discharge of a whitish liquid. On auscultation the tracheal sounds were harsh and marked fluid sounds were detected. Very harsh and moist rales were heard over both lung fields, particularly in the anterior ventral lobes. Some of these moist rales at the hilar region were believed to be referred from the trachea.

The esophagus, displaced to the left jugular groove, was visibly enlarged to a diameter of 5 cm along the entire cervical area. It was tortuous in its course and had fluctuating contents.

When a nasogastric tube was passed into the cervical esophagus approximately 500 mL of dilute, whitish, mucoid liquid immediately refluxed from the tube and the esophagus collapsed. Repeated attempts, even while the foal was sedated with xylazine,[1] to

[1]Rompun, Haver-Lockhart, Mississauga, Ontario.

pass small and large nasogastic tubes into the stomach all resulted in obstruction at the base of the heart. After drainage of the fluid from the esophagus the harsh, moist tracheal and pneumonic sounds were reduced.

In view of these serious problems and an infected umbilicus, the owners requested euthanasia.

Postmortem Findings

Gross Findings — The esophagus was uniformly dilated, to approximately 6 cm diameter, from the pharyngeal opening to the base of the heart. At this point it narrowed abruptly to approximately 1.5 cm diameter and continued uniformly to the stomach (Figure 1). The wall of the dilated proximal segment was decreased in thickness, with a thinned and bluish appearing mucosa. The lumen of the distal thickened segment was decreased in diameter and the esophageal wall was thickened and inflexible.

The lungs were voluminous, firm and mottled dark red and grey in appearance with a patchy distribution. Purulent exudate was in bronchioles in the ventral areas of the lungs. The left lung appeared more severely affected than the right.

Histological Findings — The bronchioles and terminal airways were filled with polymorphonuclear cells (PMN) and necrotic debris. The alveoli often contained PMN, debris, proteinaceous exudate, and the alveolar septa were thickened by cellular proliferation and distended capillaries.

Measurements of the proximal esophageal segment, the thickened distal esophageal segment and a normal esophagus of a foal of similar age are presented in Table I. The thin proximal segment was less than one-half the thickness of the normal esophagus when measured at comparable sites. There was a reduction in thickness of the mucosa, inner muscular layer (circular layer) and outer muscular layer (longitudinal layer). The mucosal rugae were flattened and the muscularis mucosae layer greatly thinned. The inner muscular layer contained numerous, scattered skeletal muscle bundles that were homogenous, brightly eosinophilic, surrounded by a clear space (Figure 2), and had loss of normal cross-striations.

Myenteric plexuses were evident

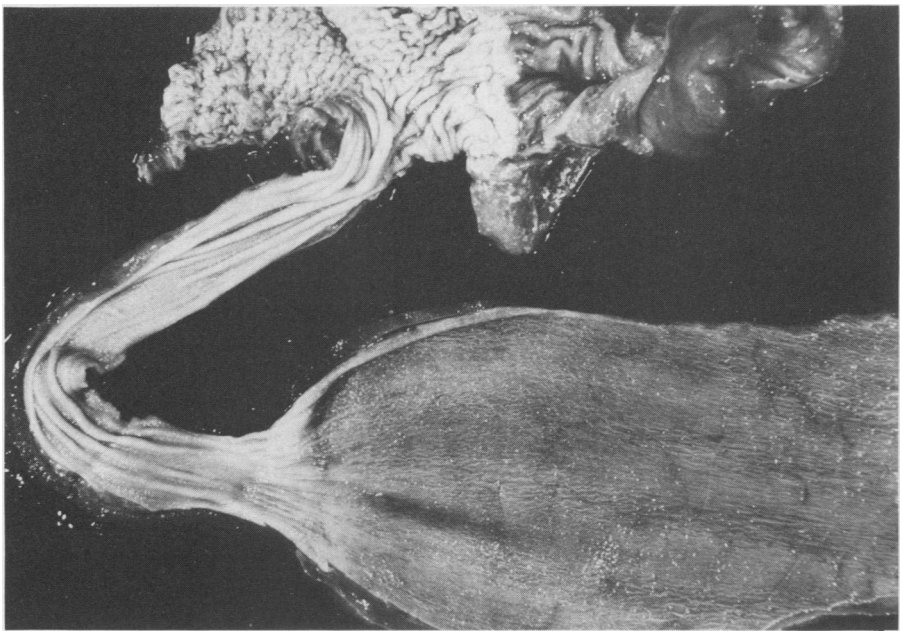

FIGURE 1. Abnormal esophagus of a foal. Note greatly dilated proximal segment and thick walled distal segment.

TABLE I
COMPARISON OF THE THICKNESS OF THE ESOPHAGUS OF A NORMAL FOAL WITH THE ONE WITH ESOPHAGEAL ECTASIA

Measurement	Abnormal Esophagus		
(cm)	Proximal Segment	Distal Segment	Normal Esophagus
Total thickness	2.13	6.25	4.51
Mucosa	0.38	1.25	1.75
Entire muscular layer	1.75	5.00	2.76
Inner muscular layer	1.50	3.75	2.38
Outer muscular layer	0.25	1.25	0.38

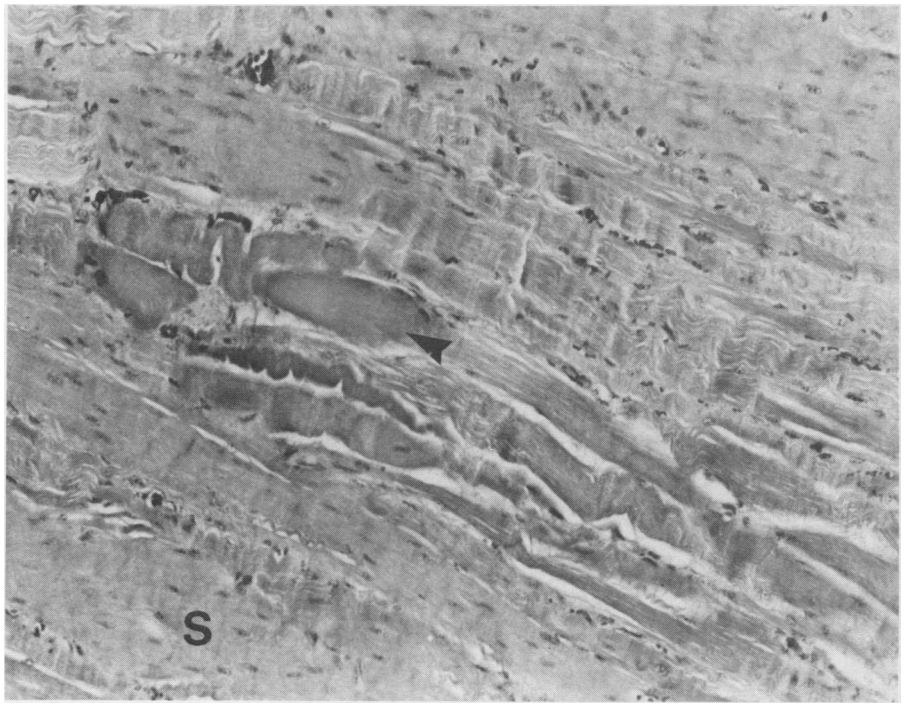

FIGURE 2. Swollen skeletal muscle bundles in thin proximal segment (marker), and smooth muscle bundle(s).

between the inner and outer muscular layers of the thinned proximal esophageal segment, but were fewer and smaller than those of a normal foal.

The distal, thickened esophageal segment had slight thinning of the mucosa, and great thickening of the inner and outer muscular layers. The myenteric plexuses were judged to be normal.

The dilated and the thickened segments contained numerous scattered muscle bundles with multiple internal nuclei (Figure 2). Reticulin stains revealed a fine internal meshwork of reticular fibres creating a honeycomb appearance with a nucleus inside each compartment. These muscle bundles have been previously described and considered abnormal, however, it is our opinion that they are normal individual bundles of smooth muscle.

Discussion

There is an age similarity between this foal, the other foals with esophageal dysfunction, and dogs with mesaesophagus (11,12,14,17,20,21,23, 26,27). No ethnic, sexual, nor familial relationship is present in achalasia of man (5,7) but is present in megaesophagus (4,9,17). Although both previous reports were in Thoroughbred foals, and all three were colts, insufficient numbers have been identified to suggest any breed or sex relationship (26,27).

Hypertrophy of the distal esophagus, as seen in this foal, was not reported in the other foals (26,27), but is a characteristic finding in achalasia of man (5-8,28). The dilated thin-walled proximal esophagus was also seen in the other foals and is similar to the lesions seen in megaesophagus (14, 19,21,22,26,27) and may have resulted from chronic distention from accumulated ingesta. Similar lesions have been seen in dogs with esophageal obstruction caused by persistent right aortic arch. The decreased number of ganglionic cells in the myenteric plexuses of the proximal esophagus, seen in this foal and one other reported (27), is consistent with lesions seen in achalasia (5-8,28) in man. However there was not a decreased number of ganglionic cells in the thickened distal segment.

Lesions of the dorsal motor nucleus of the vagus, the vagal nerve fibers and ganglion cells of Auerbach's plexus are typical of achalasia in man (5-8,28) but have not been identified in dogs with esophageal dysfunction (19,22). A decreased number of ganglionic cells of the nucleus ambiguus has been seen in dogs (18). Because the dog has a striated neuromuscular defect, a neural lesion of the vagal motor nucleus ambiguus might be expected (18,22). None of the foals reported have been examined for lesions of the vagal nerve fibers or dorsal motor nucleus (26,27).

Hypersensitivity to the intramuscular injection of mecholyl produces a characteristic esophageal contraction in people with achalasia (5-8). This reaction is not present in dogs with megaesophagus (20) and has not been reported in horses with esophageal dysfunction (2,4,27).

Surgical treatment has been demonstrated to be detrimental in the treatment of megaesophagus (17), but useful for achalasia in dogs (23,24). Further support of achalasia should be demonstrated before any surgical treatment is recommended in horses.

To make a definitive diagnosis of achalasia, aperistalsis of the caudal esophagus and failure of the lower esophageal sphincter to relax following swallowing must be demonstrated manometrically or with fluoroscopy (6-9,17). The ectasia and esophageal dysfunction seen in these foals has similarities to both achalasia of man and megaesophagus in dogs. Additional fluoroscopic, electrophysiological, manometric pharmacological, and histological studies are necessary before this condition can be fully characterized and understood in the horse.

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BOOK REVIEW

Canine Hip Dysplasia and Other Orthopedic Problems. F.L. Lanting. Published by Alpine Publications, Colorado. 1981. 212 pages. Price US $12.95.

Should a veterinarian buy and read a book on canine hip dysplasia written by a layman? In this case, I believe the answer is yes. This book is a good review of the literature and research to date on canine hip dysplasia. It is relatively complete and accurate, well written in an easily read fashion, and provides the type of background information which clients expect their veterinarian to have and discuss with them.

The text is not completely free of bias. Although essentially every theory on hip dysplasia is presented, those theories which the author finds most attractive are often put forth in greater detail. Also, the paraphrasings of some reference articles are slightly inaccurate and sometimes misleading. Because of these deficiencies, I believe that this book should be viewed as a resource rather than a reference work; i.e. it should not be considered a definitive text on the subject. It is, however, certainly the most comprehensive book on canine hip dysplasia available at present.

There are some omissions from the reference list, in particular Dr. G.F. Hanlon's excellent booklet published by the University of Minnesota.[1] Where clinical evaluation of the hip joint is concerned, a noteworthy omission is the failure to emphasize the importance of a complete physical examination (gait analysis, joint palpation and radiography) regardless of the age of the dog. It is my belief that joint palpation is valuable in adult dogs as well as in puppies.

Although the book deals mostly with hip dysplasia, there are chapters on osteochondrosis, panosteitis, and hypertrophic osteodystrophy. Contributions by Drs. Sten-Erik Olsson and Wayne Riser are included, and the author also received input from many other veterinarians, both practitioners and academics.

In summary, despite the reservations expressed above, I believe that Mr. Lanting's book would be a valuable addition to the library of anyone concerned about canine hip dysplasia.

J.W. Pharr.

[1]G.F. Hanlon. Hip Dysplasia in the Dog: Questions and Answers. Biomedical Graphics/Marketing Services, University of Minnesota, Minneapolis, Minnesota 55455.

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