Tracheal collapse in a Holstein heifer

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A four-week-old, 44 kg, Holstein heifer was presented to the University of Illinois Veterinary Medicine Teaching Hospital with a two-week history of stertorous breathing, dyspnea while drinking from a bottle, and regurgitation. Because of regurgitation, the heifer had been fed whole milk via an esophageal feeder for ten days prior to admission. Ceftiofur (Ceftifur, Upjohn, Kalamazoo, Michigan, USA) and gentamicin (Gentocin, Schering, Kenilworth, New Jersey, USA) had been administered over the previous ten days for suspected pneumonia.

The calf was born after an uncomplicated pregnancy, labor, and vaginal delivery. Sire and dam were unrelated. The heifer was housed in an individual calf hutch. Eight other calves of similar age and housing were unaffected. The affected heifer was vaccinated at birth for enteric *Escherichia coli* (Bovine Ecolizer, Grand Laboratories, Larchwood, Iowa, USA), rotavirus, and coronavirus (Calf-Guard, Norden Laboratories, Lincoln, Nebraska, USA). An intramuscular vitamin E and selenium supplement (BO-SE, Schering) had also been administered.

Initial examination at presentation revealed a rectal temperature of 39.6°C, heart rate of 160/min, and respirations of 60/min. The trachea was normal upon palpation. However, palpation of the larynx and proximal trachea induced a "honking" noise. The arytenoid cartilages appeared inflamed on oral examination. Upon auscultation of the thorax, referred upper airway sounds were heard during inspiration. Crackles were heard in all lung fields on expiration, with greatest intensity over the left thorax.

Dorsoventral and right and left lateral recumbent radiographs revealed evidence of lobar alveolar disease of the lung in the cranial and ventral lobes. The left middle lung lobe was the most severely diseased. Fluoroscopic examination of the cervical and intrathoracic trachea was performed and multiple spot films made. A definitive narrowing of the cervical trachea was associated with both inspiration and the "honking" noise. The overall lumen size was reduced more than 75% from that of a normal calf. Tracheal diameter was 6 mm at maximum inspiration and 1.6 cm during maximum expiration. Due to the severity of the tracheal collapse and the poor prognosis even with surgical intervention, the calf was euthanized.

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At necropsy, the trachea was moderately dilated throughout its entire length. Also, the tracheal cartilages did not curve inward to close over the lumen, resulting in a "D-shaped," enlarged lumen. The dorsal one-third of all rings of cartilage bent inward into the lumen at sharp angles. Often the dorsal portions of the rings were fragmented and small thin spicules of cartilage were free within the dorsal tracheal membrane. The dorsal tracheal membrane was thickened (0.5 cm) and widened (2.0 cm) and contained numerous prominent longitudinal mucosal folds (Figure 1). Trachealis muscle within the dorsal membrane was markedly thickened and bulged from the cut surface. An age-matched control calf had a dorsal tracheal membrane 0.2 cm in thickness and 0.2 cm in width. Tracheal cartilage of the affected calf had a thickness of 2.5 mm, whereas the age-matched control was 2.0 mm.

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Histologically, the tracheal epithelium was diffusely thickened circumferentially, and had fewer goblet cells than normal. Low numbers of neutrophils were scattered diffusely within the epithelial layer, and small aggregates of 3–10 neutrophils were also observed. The propria-submucosa was diffusely infiltrated by moderate numbers of neutrophils, macrophages, lymphocytes, and plasma cells, especially beneath the epithelial basement membrane. Within the dorsal membrane, the propria-submucosa was substantially thickened. The trachealis muscle was thickened almost threefold, due to substantially increased numbers of smooth muscle fibers and a mildly edematous intramuscular fibrous stroma. This stroma was also infiltrated by low numbers of neutrophils. Adventitial tissue dorsal to the trachealis muscle was edematous and infiltrated by low numbers of lymphocytes, plasma cells, and macrophages. The tracheal cartilage, despite its abnormal gross appearance, was histologically normal. There was no qualitative reduction in thickness or mass of cartilage when compared to an age-matched control. Toluidine blue stain did not indicate

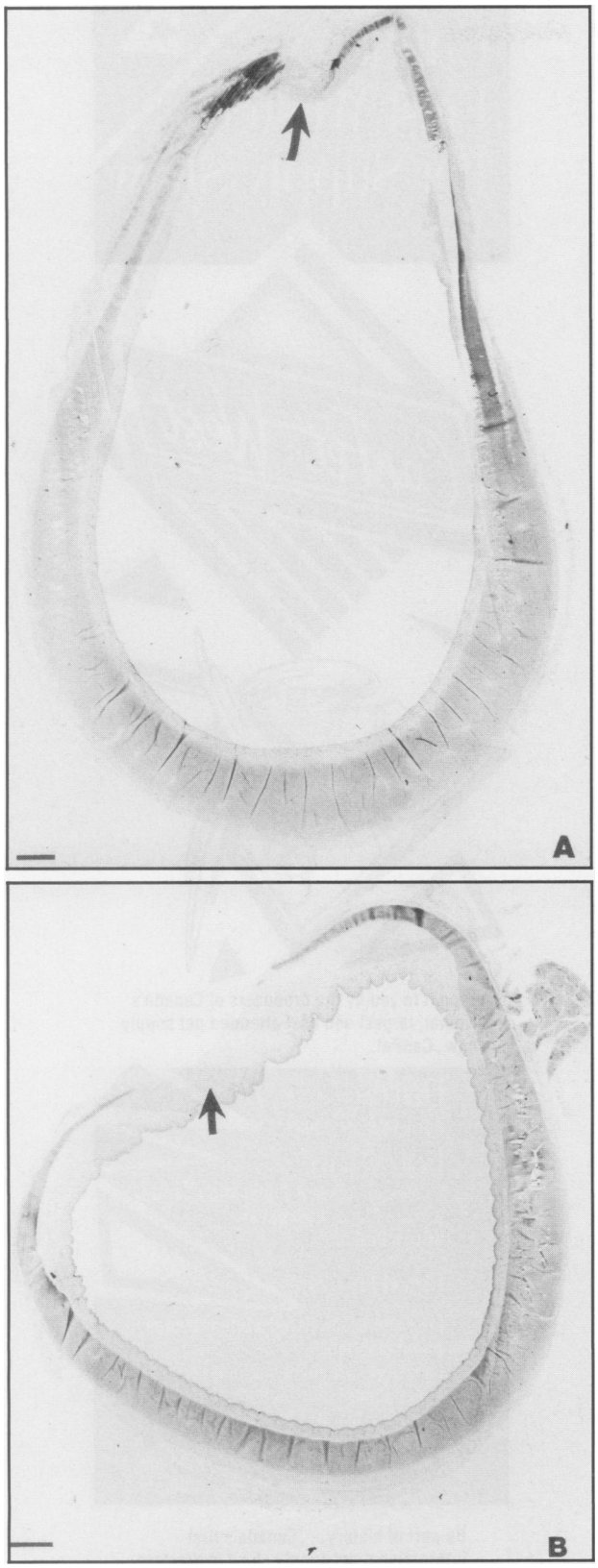

Figure 1. A. Trachea from a normal age-matched calf. The long axis of the tracheal lumen is oriented dorsoventrally. Note the short, thin dorsal tracheal membrane (arrow). B. Trachea from the calf with tracheal collapse. Although cartilage appears normal, the shape of the ring is abnormal, resulting in a lumen with its long axis oriented transversely. The dorsal tracheal membrane is elongated and thickened (arrow) due to secondary hyperplasia of the trachealis muscle and inflammation of the tracheal mucosa. (Hematoxylin and eosin; bars = 2 cm).

any qualitative difference in staining affinity of tracheal cartilage of the affected calf in comparison to tracheal cartilage in the age-matched normal calf.

Chronic suppurative laryngitis and severe chronic suppurative pyogranulomatous bronchopneumonia were also present. *Actinomyces pyogenes* was isolated from the pneumonia, which affected 40–70% of the cranial and middle lobes of both lungs. Numerous fibrous adhesions were present between the visceral and parietal pleurae covering the left middle lung lobe. There was lymphoid hyperplasia in bronchial lymph nodes.

Tracheal collapse has been reported in calves (1,2), a dog (3), horses (4), and man (5–7). It is uncommon in calves. The most common cause of tracheal collapse in calves is periparturient cranial thoracic trauma (2,8). In seven of nine reported cases, the calves had abnormalities of the cranial ribs consistent with healing fractures and redundant callus, indicating damage associated with parturition. The mean age at presentation to the referral center in these reports was 9.5 weeks, and the mean age at onset of clinical signs was 2.1 weeks. In our case, however, no rib lesions suggestive of periparturient trauma were noted radiographically or on postmortem examination.

Segmental tracheal stenosis may also be associated with impaired airflow through the trachea. This condition may be congenital or acquired, and has been reported in children (10). The congenital form is associated with pulmonary a genesis and usually involves the distal trachea. Acquired injuries leading to scarring and stenosis of the upper airway in children usually involve the subglottic larynx rather than the trachea, while stenosis secondary to irritation from the tip of an endotracheal or tracheostomy tube usually occurs in the trachea just proximal to the carina. The animal in this case was not intubated intratracheally, and no evidence of preexisting or ongoing tracheal trauma was observed. Furthermore, the trachea was collapsed and flattened throughout its entire length, not segmentally as would occur if segmental agenesis, trauma, or local irritation were the inciting factors.

In dogs, congenital tracheal collapse is associated with subnormal amounts of cartilage in the rings, which leads to an abnormal shape (flattening) of the rings and narrowing of the tracheal lumen (10). In acquired cases, there is no decrease is size or thickness of the tracheal rings but a loss of ring rigidity due to loss of organic matrix, mainly glycoprotein and glycosaminoglycans (11). The cause of this resorption is unknown. Toluidine blue staining of the tracheal rings from our calf and from an age-matched normal calf revealed no observable qualitative differences in cartilage matrix between the two. Although the tracheal cartilage in this calf did not have histologically evident dysplasia, a quantitative defect in cartilage matrix still may have been present, resulting in readily deformable tracheal rings. In this calf also, dynamic airflow changes associated with labored breathing secondary to chronic pneumonia could have contributed to collapse of the weakened cartilage rings, with secondary hypertrophy and hyperplasia of the trachealis muscle and severe inflammation of the dorsal tracheal membrane. Mucosal inflammation and hyperplasia

of the trachealis are common sequelae of tracheal collapse (12).

The exact cause of tracheal collapse in the case reported herein is undetermined. However, a heritable chondrodysplasia must be considered. In mice, the chondrodysplasia gene, when homozygous, alters chondrocyte function and results in flattened and less rigid tracheal cartilage. This results in decreased deposition of organic cartilage matrix in the rings. The uniform involvement of the trachea throughout its entire length would suggest that an underlying biochemical defect, common to the entire trachea, was responsible for the tracheal collapse.

Surgical procedures for repair of tracheal collapse have been described (2). Although there is a predisposition for occurrence of tracheal collapse in miniature breeds of dogs, the heritability of the condition is unknown. CVJ

References

1. Vestweber JG, Leipold AW. Tracheal collapse in calves. J Am Vet Med Assoc 1984; 184: 735–736.
2. Fingland RB, Rings DM. Etiology and surgical management of tracheal collapse in calves. Vet Surg 1990; 19: 64.
3. Done SH. Canine tracheal collapse-aetiology, pathology, diagnosis, and treatment. Vet Annu 1978; 18: 255–260.
4. Hanselka DV. Tracheal collapse and laryngeal hemiplegia in the horse. Vet Med Small Anim Clinician 1973; 68: 859–862.
5. Loeff DS, Miller RM, Gorenstein A, *et al.* A new intratracheal stent for tracheobronchial reconstruction: experimental and clinical studies. J Pediatric Surg 1988; 23: 1173–1177.
6. Grillo HC, Mathisen DJ. Surgical management of tracheal strictures. Surg Clin North Am 1988; 68: 511–524.
7. Schuller DE, Parrish RT. Reconstruction of the larynx and trachea. Arch Otolaryngol Head Neck Surg 1988; 114: 278–286.
8. Kasari TR. Weakness in calves associated with dystocia. Agri-Practice 1989; 10: 19–25.
9. Seegmiller R, Ferguson CC, Sheldon H. Studies in cartilage. VI. A genetically determined defect in tracheal cartilage. J Ultrastruct Res 1972; 38: 288–301.
10. Healy GB, Jonas RA, Schuster SR, McGill TJ. Correction of segmental tracheal stenosis in children. Ann Otol Rhinol Laryngol 1988; 97: 444–448.
11. Ettinger SJ, Ticer JW. Diseases of trachea. In: Ettinger SJ, ed. Textbook of Veterinary Internal Medicine. 3rd ed. Philadelphia: W.B. Saunders, 1989: 795–815.
12. Dungworth DL. The respiratory system. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals. 3rd ed. vol. 2. Academic Press, 1985: 433–436.