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Asymptomatic Congenital Lobar Emphysema in a Pekinese Dog

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Abstract: Asymptomatic lung lobar emphysema was incidentally found in a four-year-old male Pekinese dog in routine health check. No clinical signs associated with lobar emphysema have been observed at the presentation. Diagnostic imaging and pathological studies revealed Congenital Lung lobar Emphysema (CLE). This is the first report of asymptomatic CLE in dogs.

Key words: Canine, emphysema, lobar, congenital, CLE

INTRODUCTION

Emphysema is a disease where there is permanent destruction of the walls of the air sacs (alveoli) and the small airways (bronchioles), primarily by congenital malformation in bronchiolar cartilage and secondarily by chronic obstructive pulmonary diseases. Congenital Lobar Emphysema (CLE) is a rare malformation of one or multiple lung lobes and has been rarely reported in dogs (Amis *et al*., 1987; Herrtage and Clarke, 1985; Hoover, 1992; Tennant and Heywood, 1987; Voorhout *et al*., 1986). CLE presents with overexpansion of a pulmonary lobe with resultant compression of the remaining ipsilateral lung. Mediastinal shift away from the increased volume lung can also compress the contralateral lung. In CLE, due to congenital bronchial narrowing (by weakened or absent bronchial cartilage), there is inspiratory air entry but collapse of the narrow bronchial lumen during expiration causing lobar air trapping. Although the exact mechanism of lobar air trapping in CLE has not been clarified, malformations in bronchial cartilage have been associated with CLE in dogs (Amis *et al*., 1987; Hoover, 1992; Tennant and Heywood, 1987; Voorhout *et al*., 1986).

Clinical signs associated with CLE are apparent at between 6 weeks and 5 months of age (Herrtage and Clarke, 1985; Hoover, 1992; Tennant and Heywood, 1987; Voorhout *et al*., 1986). Presentation with the condition after six months of age is uncommon (Karnak *et al*., 1989). It is rarely reported in adult dogs (Amis *et al*., 1987). Right middle lung lobe is predominantly affected in literature (Herrtage and Clarke, 1985; Voorhout *et al*., 1986) although multilobar involvement has also been reported (Hoover, 1992; Tennant and Heywood, 1987). In humans, CLE is usually unilateral (predominantly left upper lobe) and bilateral and lower lobe involvement are rare (Karapurkar *et al*., 1993; Karnak *et al*., 1999). Main clinical signs associated with CLE are progressive dyspnea, followed by coughing and exercise intolerance. Dyspnea, tachypnea, cyanosis, increased lung sounds and decreased lung sounds are also reported (Billet and Sharpe, 2002). In human, CLE is classified into CLE in infancy, symptomatic CLE in older children and asymptomatic CLE (discovered accidentally). However the asymptomatic form of CLE has not yet been described in animals. This case report described left sided lung lobe emphysema found accidentally in routine examination in a dog.

CASE REPORT

A 4-year-old intact male Pekinese (4.5 kg), was presented at the Veterinary Teaching Hospital, Kangwon National University for dextrocardia. According to referring veterinarian, abnormally right-side deviated heart was incidentally found in routine health check. No clinical signs associated with right deviation of heart were observed, except excessive panting in excitement and after exercise. In cardiac auscultation, heart sound was heard best at the right apex and was muffled at the left apex. However no signs related to right axis deviation and right cardiac enlargement were observed in the 12-lead electrocardiography. No significant abnormalities were observed in routine hematology and blood chemistry.

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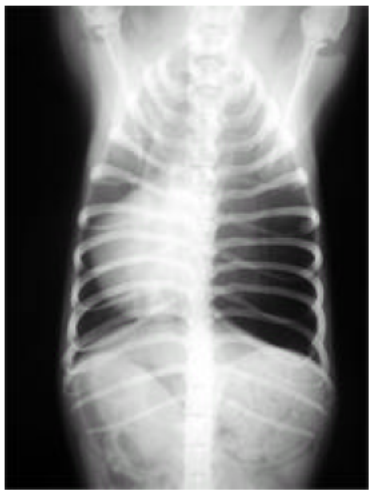

Fig. 1: Thoracic radiography of this case (dorso-ventral view). The radio-density of left lung lobes was decreased due to trapped air. The heart and mediastinum are severely shifted to the right side due to over-distended left lung lobes

In the dorso-ventral view of thoracic radiography, the heart was abnormally displaced to the right side without the evidence of right atrial and ventricular enlargement. Mediastunum was also shifted to the right side. The radio-density of right lung lobes were increased and partially consolidated, while that of left lung lobes were decreased, especially from the caudal part of cranial lobe to the whole caudal lobe (Fig. 1). In this region, the vascular marking was almost absent. Those findings strongly suggest the entrapment of free gas in that region. In the lateral view, the sternal contact to cardiac silhouette was lost. Barium contrast study could not found abnormal deviation of gastric axis. Furthermore no abnormalities were observed in a two-dimensional echocardiography. Diagnostic thoracoscopy was performed at the fouth left intercostals region using electronic scope (EB-1570K video bronchoscope, Pentax, Japan) and revealed abnormally distended and air-filled left lung (Fig. 2).

Based on the diagnostic findings, the case was diagnosed as congenital lobar emphysema. Because the dog had refractory epilepsy and the owner requested euthanasia for this dog, we performed necropsy with the owner's consent.

In the necropsy, there is no large lung bulla in the left thoracic cavity. In stead, the whole left lung lobes were emphysematous (Fig. 3). The heart was displaced to the right side due to abnormally expanded left lung lobes. No other abnormalities were observed in necropsy. On histological examination of the affected lobe, marked enlargement and hyperinflation of alveoli was identified with loss and displacement of the alveolar walls (Fig. 4).

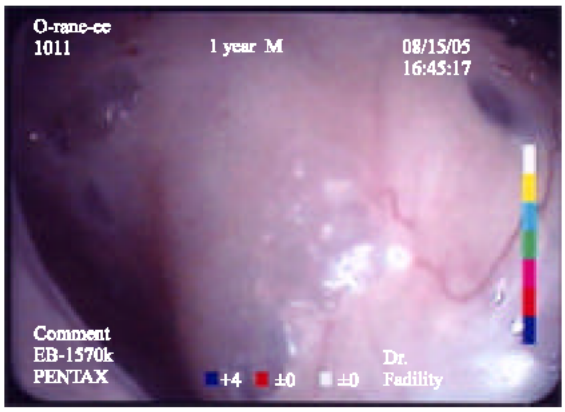

Fig. 2: Thoracoscopy of this case. The left lung lobes were abnormally distended and air-filled even in expiration

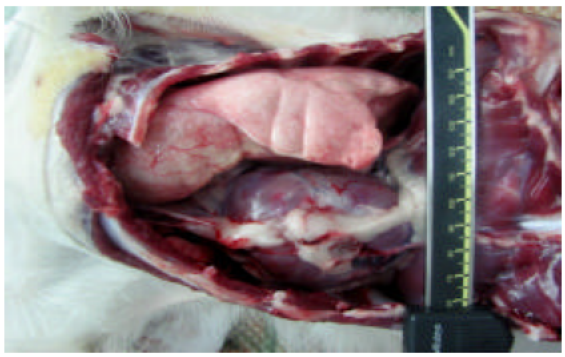

Fig. 3: Gross finding of this case. Instead of large lung bulla, the whole left lung lobes were evenly emphysematous. Marks of sternal contact of lung lobes were clearly seen. Heart is severely displace to the right side

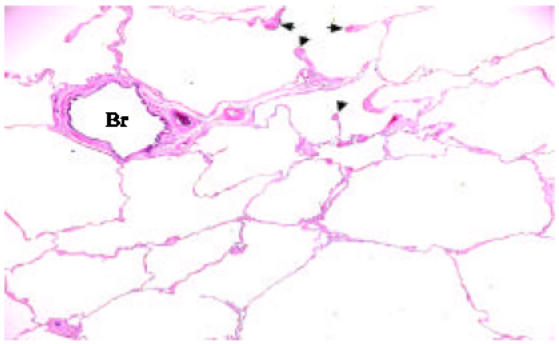

Fig. 4: Histopathological finding of this case. Lung section showed the thinness of the terminal bronchioles (arrows) trapped by air. The marked enlargement and hyperinflation of alveoli with loss and displacement of the alveolar walls are identified. H-E stain, ×40, Br: Bronchiole

The bronchus was lined with low number of cuboidal and attenuated epithelial cells with a lack of bronchial cartilage and was replaced into smooth muscles and connective tissues, indicating bronchiolar hypoplasia. No inflammatory cellular infiltration and evidence of pneumonia were seen in this case.

DISCUSSION

Two major forms of pulmonary emphysema are generally recognized. Alveolar emphysema is abnormal permanent enlargement of air spaces distal to the terminal bronchiole and destruction of alveolar septal walls without apparent fibrosis. Interstitial emphysema is the presence of air within the supporting connective tissue stroma of the lung (interlobular, subpleural, mediastinal, subcutaneous).

Two interesting findings have been recognized in this case. Firstly, the dog was asymptomatic till we recognized abnormality in the physical examination, although the whole left lung lobes were emphysematous. Three reports described in veterinary literature had only single affected lung lobe with marked clinical signs related to respiratory distress (Amis *et al*., 1987; Billet and Sharpe, 2002; Herrtage and Clarke, 1985). However this case had more severely affected, no clinical signs were observed except mild panting in excitement. However, asymptomatic congenital lobar emphysema has been recognized in human medical literature (Laberge *et al*., 2005).

The 2nd interesting finding was that we could rarely identify bronchi and bronchioles in the multi sectioned lung tissues. Bronchi and bronchioles existed had poor development of cartilage and replaced into smooth muscle and connective tissues. Even though no large bulla existed, the diffuse alveolar emphysema was seen in the whole left lung lobes. Most CLEs of dogs described in veterinary literature had a large bulla which could be surgically managed (Billet and Sharpe, 2002; Tennant and Heywood, 1987). However, this case showed diffuse alveolar emphysema which has never been reported in veterinary literature and could not be easily managed by surgical methods. Karnak *et al.* (1999) suggested the close relation between lobar emphysema and bronchial cartilage abnormality. In this case, the main cause of lobar emphysema might be the congenital bronchial cartilage hypoplasia as seen in histopathological examination.

CONCLUSION

In conclusion, the case described in this case report is the first case of asymptomatic congenital lung lobe emphysema by bronchial cartilage aplasia in a dog.

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REFERENCES

Amis, T.C., D. Hager, D.L. Dungworth and W. Hornof, 1987. Congenital bronchial cartilage hypoplasia with lobar hyperinflation (congenital lobar emphysema) in an adult Pekingese. J. Am. Anim. Hosp. Assoc., pp: 321-329.

Billet, J.P.H.G. and A. Sharpe, 2002. Surgical treatment of congenital lobar emphysema in a puppy. J. Small Anim. Pract., pp: 84-87.

Herrtage, M.E. and D.D. Clarke, 1985. Congenital lobar emphysema in two dogs. J. Small Anim. Pract., pp: 453-464.

Hoover, J.P., G.A. Henry and R.J. Panciera, 1992. Bronchial cartilage dysplasia with multifocal lobar bullous emphysema and lung torsions in a pup. J. Am. Vet. Med. Assoc., pp: 599-602.

Karapurkar, S.A., J.D. Borkar and B.J. Birmole, 1993. Malformation of lung in neonates (lobectomy for congenital lobar emphysema and lung cyst). Postgrad. Med. J., pp: 224-227.

Karnak, I., M.E. Senocak, A.O. Ciftci and N. Buyukpamukcu, 1999. Congenital lobar emphysema: Diagnostic and therapeutic considerations. J. Pediatr. Surg., pp: 1347-1351.

Laberge, J.M., P. Puligandla and H. Flageole, 2005. Asymptomatic congenital lung malformations. Semin. Pediatr. Surg., pp: 16-33.

Tennant, B.J. and S. Haywood, 1987. Congenital bullous emphysema in a dog: A case report. J. Small Anim. Pract., pp: 109-116.

Voorhout, G., S.A. Goedegebuure and R.C. Nap, 1986. Congenital lobar emphysema caused by aplasia of bronchial cartilage in a Pekingese puppy. Vet. Pathol., pp: 83-84.