Journal of Pulmonary & Respiratory Medicine

Adamama-Moraitou, J Pulmonar Respirat Med 2012, 2:2
DOI: 10.4172/2161-105X.1000e117

Editorial Open Access

Tracheobronchomalacia: Does it Share the Same Aetiology in Men and Dogs?

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Tracheomalacia (TM), bronchomalacia (BM) or tracheobronchomalacia (TBM) occur in humans and dogs and they are characterized by structural and subsequently, dynamic defect of tracheal and/or bronchial cartilages, resulting in flattening of airway lumen [1-3]. Although all these conditions are well recognized in human patients, BM has been only recently described as a sole clinical entity in dogs [3,4] and like in men, it is considered to be less common than TM or the combined pathology of TBM [1,5-7]. The most commonly reported distribution type in human patients with bronchoscopically confirmed TBM is the diffuse type, involving the trachea and bronchi. Isolated tracheal involvement is less frequently reported, while the opposite is true in dogs. The prevalence of TBM in human patients relies on aetiology and depending on the study population, may vary from 4% to 23-26%. Many classification subsystems have been created in order, a common language for evaluating airway malacia to be established. According to the location of the disease, three subtypes have been described in people. Moreover, three subtypes have been recognized in children with TBM, which incorporate clinical, histopathological and bronchoscopical findings. However, only one subsystem has been reported in dogs with TM, which takes into account the severity of airway collapse and subdivides the disease into four stages. But does airway softening share the same or even similar aetiopathogenesis in both species?

Human TBM is classified as congenital (primary) and acquired (secondary). The primary form can be idiopathic, associated with prematurity, congenital cartilaginous weakness, congenital syndromes, and other congenital anomalies such as tracheoesophageal fistula. It is believed that it is a consequence of a faulty division of the foregut during embryonic development, where trachea receives more tissue at embryonic separation. Trauma, genetic defects, external compression by various tissues [6], neurologic impairment, idiopathic causes, inflammation, infectious tracheobronchitis and relapsing polychondritis have been proposed as possible causes of the acquired form [1,2]. It is of interest, that although the aetiology of acquired TBM in adults is not developmental, the histopathologic findings are similar to those found in children.

On the other hand, the aetiology of canine airway malacia remains enigmatic, although it seems that the same classification with the human disease is met. Especially, for canine TM, it is considered to occur secondary to metabolic defects in the tracheal cartilages. Generally, softening of the cartilages may be related to congenital disease, external compression, chronic inflammation or changes in elastic fibers [3]. The congenital form may be the underlying abnormality, while various acquired factors may contribute in becoming clinically evident, perpetuating and deteriorating [8]. Pathological changes reported in soft canine tracheas include hypocellularity of the cartilages, loss of hyaline cartilage, which is replaced by fibrocartilage or fibrous tissue, and deficiency of chondroitin sulfate, calcium and glucosaminoglycans, as well as damaged chondrocytes between areas of normal cartilaginous tissue [9]. In dogs and people, TM, BM and their combination may demonstrate the same histological changes [7].

Infection and/or inflammation have been suggested as possible contributing factors in canine TM [7,8,10]. Similarly, infection has been proposed as a possible cause of weakening of the airway wall in man [11], but predisposition to secondary inflammation or infection, perhaps due to increased accumulation of secretions and decreased mucociliary clearance, may also be suspected [1,11]. It has been proposed that primary BM leads to decreased local immunity that permits the colonization and eventually the infection of the lower airways [3,12]. Chronic cough irritation was incriminated in dogs with TM and small airway pathology [7]. Contrary, it has been suggested that chronic cough might have been the vehicle by which colonization by microorganisms and eventually infection of the lower airways occurs [13]. Moreover, differences in resistance, effort, flow and pressures have been proposed to contribute to BM in dogs with brachycephalic airway syndrome [14].

Information concerning canine airway malacia is still limited. Based on these data, it is concluded that in both human and canine diseases the aetiopathogenesis is multifactorial. Although we should hypothesize that some aetiologies are common between men and dogs, any interrelation is still hazardous. Currently, research projects are focused on genetics aiming on understanding the fundamental disease mechanisms in both species.

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Received March 28, 2012; **Accepted** March 30, 2012; **Published** April 02, 2012

Citation: Adamama-Moraitou KK (2012) Tracheobronchomalacia: Does it Share the Same Aetiology in Men and Dogs?. J Pulmonar Respirat Med 2:e117. doi:10.4172/2161-105X.1000e117

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