Morphological Aspects of Bronchitis and Bronchiolitis in the Horse

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Exposure of the airways to the external environment is never er entirely without hazard. Although the airways have a remarkably effective cleansing mechanism i.e. the mucociliary system, it is not always sufficient to remove all inhaled particles. Breakdown of this protective mechanism will result in inflammation. Inflammation of the bronchial tree usually involves concurrently, but with different degrees of severity, both large and small bronchi and bronchioles. Inflammation of the bronchial tree concerns three interrelated processes: alteration, exudation and proliferation.

Alteration

When a noxious agent infiltrates the airways the epithelium may be damaged to varying degrees. The first and least damaging reaction is the loss of cilia from the epithelium. More aggressive noxious agents cause degeneration of the epithelial cells with detachment of both ciliated and goblet cells (Fig. 1). When the epithelium is attacked severely, necrosis of the epithelium and sometimes of the underlying stroma will occur.

Exudation

Alteration of the epithelium usually is accompanied by exudation. The exudate is made up of fluid and cellular components (Table 1). The fluid component is formed by leakage of plasma from the peribronchial and peribronchiolar capillaries into the interstitium and into the lumen of the bronchial tree. Another part is formed by the extrusion of

fluid exudate	cellular exudate
plasma (water, electrolytes, albumin, fibrinogen etc.) mucus	granulocytes · neutrophilic eosinophilic (basophilic) mononuclear phagocytes (lymphocytes)

Table 1: Composition of exudate in inflammation of the airways.

mucus from the goblet cells and bronchial glands into the lumen of the airways. The consistency of the fluid exudate will depend on the quality of mucus and the permeability of the capillary endothelium. The latter determines the protein content of the exudate. In severe acute vascular injury even fibrinogen may pass into the lumen of the airways resulting in a fibrinous exudate.

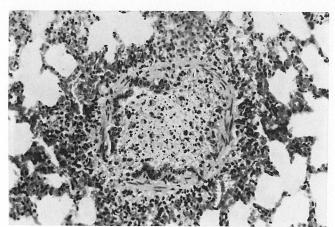


Fig. 1: Bronchiole with epithelial degeneration (cell swelling and karyopycnosis) and detachment of epithelial cells.

The cellular exudate also is derived from the capillaries, and penetrates through the epithelium into the lumen of the airways. It consists mainly of granulocytes and mononuclear phagocytes. The granulocytes may be neutrophilic and eosinophilic granulocytes. Neutrophils are present in most, if not all, inflammatory exudates, but their numbers may vary depending on the agent and the host's reaction to the agent. Eosinophils generally react to stimuli similar to those for neutrophils. However specific chemotactic factors for eosinophils have been established as well. Mast cells and more specifically mast cell degranulation are associated with eosinophils. Mast cell degranulation is most prominent in invasive and migratory phases of parasitic infection and some allergic diseases. Exudation is especially severe in acute inflammation of the airways.

Acute bronchitis and bronchiolitis in the horse are histologically characterized by a relatively normal epithelium. Bronchial and bronchiolar plugging with exudate is most obvious (Fig. 2). Often the exudate extends into the related acini. The exudate consists of neutrophilic granulocytes and mucus (Fig. 3). As much mucus is often found in the bronchioles and alveoli, aspiration of exudate to the lower airways and alveoli is a common occurrence. A varying peri-

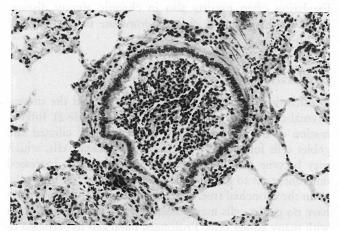


Fig. 2: Acute bronchiolitis. Bronchiole with normal epithelium and plugging with exudate. HE 200x

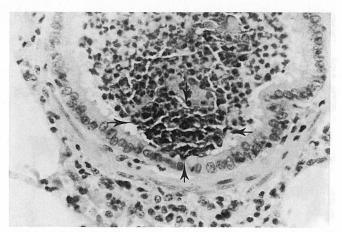


Fig. 3: Acute bronchiolitis. Bronchiole with normal epithelium; the exudate consists of neutrophils and mucus (arrows). Alcian blue 400x

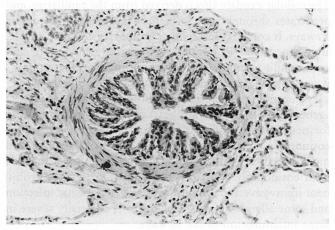
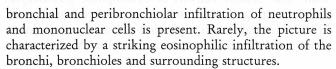


Fig. 4: Normal bronchiole with a simple columnar epithelium without goblet cells. Alcian blue 200x



Exudation often persists also in chronic airway disease. However, proliferative changes dominate in this stage of the disease.

Proliferation

Proliferation concerns both the epithelial and the mesenchymal component of the bronchial tree (Table 2). Inflammation of the bronchial tree leads to loss of ciliated and goblet cells followed by regeneration of basal cells, which may become manifest as basal cell hyperplasia. Increased differentiation to goblet cells leads to goblet cell hyperplasia in the bronchial tree. Even bronchioles which normally have no goblet cells may possess a hyperplastic epithelium with many goblet cells. Prolonged basal cell hyperplasia finally causes stratified squamous epithelium to develop. Proliferation of the mesenchymal component causes an

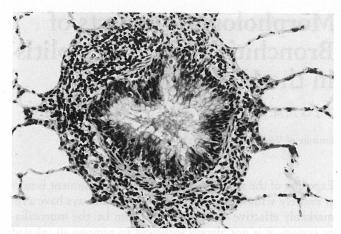


Fig. 5: Chronic bronchiolitis. Bronchiole with hyperplastic epithelium and many goblet cells; marked mononuclear infiltration of the submucosa and adventitia. HE 200x

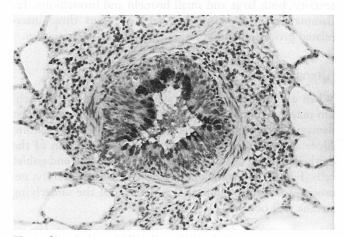


Fig 6: Chronic bronchiolitis. Bronchiole with hyperplastic epithelium and many goblet cells (dark); marked mononuclear infiltration of the submucosa and adventitia. Alcian blue 200x

increase of fibrous tissue and inflammatory mononuclear cells. The increase of fibrous tissue occurs around the bronchial tree and often radiates into the adjoining alveolar septa. These septa may show secondary epithelization. The inflammatory mononuclear infiltrate consists mainly of lymphocytes and plasma cells although sometimes lymphoid aggregates, even with germinal center formation, may be seen.

Chronic bronchitis and bronchiolitis in the horse are mainly characterized by proliferation. Exudation is also usually

epithelium	mesenchyme
basal cell hyperplasia goblet cell hyperplasia squamous metaplasia	— inflammatory mononuclear cells · lymphocytes · plasma cells · mast cells
	— fibrosis

Table 2: Proliferative changes in epithelium and mesenchyme in chronic inflammation of the airways.

present and increases greatly during acute exacerbations. The bronchi and bronchioles contain a varying quantity of exudate. The epithelium, particularly of the small airways, shows goblet cell hyperplasia. Also, bronchioles which normally have a simple columnar epithelium without goblet cells (Fig. 4) show a hyperplastic epithelium with many goblet cells (Fig. 5 and 6). The bronchial and bronchiolar submucosa and adventitia show a moderate to marked mononuclear cell infiltrate and fibrosis (Fig. 5 and 6). The fibrosis often extends into the adjoining alveolar septa. Also

epithelization of these alveoli may be seen. The inflammatory mononuclear infiltrate consists of lymphocytes, plasma cells, histiocytes and mast cells. Also neutrophils and a solitary eosinophilic granulocyte may be seen.

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