Case Study

Histopathological Characterization of Soft Palate in Brachycephalic Syndrome in Pugs: A Review of 12 Cases

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A B S T R A C T

The present study was conducted to evaluate the histological features of elongated soft palate in dogs affected with brachycephalic syndrome. Soft palate samples were collected from dogs which underwent surgical correction of elongated soft palate. Histological observations of brachycephalic soft palate in this study were edema of the lamina propria, superficial epithelial hyperplasia, interfibrillar matrix swelling, hyperplasia of mucous gland and structural degeneration of muscle fibers. Histological study indicate that the increased thickness of the elongated soft palate in dogs with brachycephalic airway syndrome was not due to muscle hypertrophy or fat deposition but is instead due to significantly increased stroma within the lamina propria, edema and increased proportion of salivary tissue. Hence multifactorial changes are responsible for elongation and thickening of soft palate in brachycephalic dogs.

Keywords: Brachycephalic dogs, Histopathology of soft palate, Diode laser staphylectomy, CO\textsubscript{2} laser staphylectomy

Introduction

Brachycephalic airway syndrome is defined as partial obstruction of upper airway of dogs and cats having shortened head due to selective breeding. Obstruction is due to anatomical abnormalities of stenotic nares, elongated soft palate, everted laryngeal saccules and hypoplastic trachea (Poncet \textit{et al.}, 2005). Surgical correction of the elongated soft palate has been reported since 1942 (Farquharson 1942). Clinical signs include dyspnea, snoring, stridor, exercise intolerance, regurgitation, vomiting, cyanosis, syncope and collapse (Gruenheid \textit{et al.}, 2018) 80% of dogs with elongated soft palate were brachycephalic and was considered as one of the most common abnormality found in brachycephalic airway syndrome.
The free border of soft palate extend beyond the glottal opening and tip of the epiglottis (Davidson et al. 2001). In brachycephalic dogs due to shortening of viscerocranium the soft palate extends 1-3 mm beyond the tip of epiglottis and is considered as elongated soft palate (Lodato and Hedlund 2012).

The proximal portions of the respiratory (nasopharynx) and digestive systems (Oropharynx) are separated by soft palate (velum palatinum). Soft palate overlaps epiglottis as it continues caudally to a varying degrees and synchronized functioning of the soft palate and epiglottis is important for both swallowing and respiration. An elongated and thickened soft palate causes obstruction to respiration. Obstructive sleep apnea syndrome is a similar condition in humans associated with the abnormal functioning of soft palate along with other factors (Hamans et al., 2000; Berger et al., 2002).

The increasing popularity of brachycephalic breeds as pets has led to rise in the occurrence of brachycephalic obstructive air way syndrome. Different surgical techniques have been done to address the different components of the air way obstruction. Resection of the soft palate (staphylectomy) is done to correct the elongated soft palate. Studies on the histopathology of the elongated soft palate are few and so far to the best knowledge the author there are no publications in this countries till date. In the present study emphasis was given to understand the histological changes of the soft palate in relation to its physiological function and its pathological changes in relation to respiration.

**Materials and Methods**

Samples collected from the dogs presented with brachycephalic syndrome treatment to Madras Veterinary College Teaching hospital. All dogs had a complete clinical examination of an upper airway including radiography and Computed tomography under general anesthesia. Soft palate samples collected from brachycephalic dogs in 10 % neutral formalin after surgical resection using carbon dioxide laser and conventional techniques. After fixing in formalin the soft palate sample was divided into two equal parts and dehydrated in ethanol and embedded in paraffin.

4-6µm thick sections are used for histological examination after staining with hematoxylin and eosin technique (H&E) and examined under microscope (Arrighi 2011). Computer-assisted image acquisition was done with Olympus digital camera with DP software for series of sections.

**Results and Discussion**

All the specimens in the present study showed hyperplasia of the superficial epithelia, extensive swelling of interfibrillar matrix and edema of the lamina propria, mucous gland hyperplasia, and degenerated muscle fibers histologically.

Hyaline degeneration, atrophy, and hypertrophy of myofibers in palatine muscle specimens was more severe in dogs effected with more severe clinical manifestations.

Separation of epidermal layer from connective tissue layer with vacuolations was a common feature. Mild increased thickness of epithelial layers indicative of edema which also cause collagen fibers to be irregularly disposed and distanced in the lamina propria.

Excretory ducts showed hyperplasia with increased dilatation and congestion. Hemorrhages were present in the palatine musculature. All blood vessels showed congestion. Hyperplasia, dilatation, basophilic
fluid were found in the lumen of palatine salivary gland. Degeneration, necrosis of muscle fibers and fragmentation of levator muscle fiber was also a common finding in all the specimens. Excretory ducts were dilated with basophilic fluid.

**Fig. 1** Increased collagen connective tissue in the lamina propria and congestion of blood vessels

**Fig. 2** Mucosal epithelium thickened due to hyperplasia

**Fig. 3** Interglandular mononuclear cell infiltration
**Fig.4** Levator palatine revealed degeneration and necrosis with edema, Congestion, Intestinal fibrosis

**Fig.5** Multiple excretory duct revealed dilatation with mucus secretion

**Fig.6** One excretory duct showed marked dilatation with thickening and other ducts showed dilatation
**Fig. 7** Hyperplasia of palatine salivary gland

**Fig. 8** Moderate to severe mucosal hyperplasia resulting in thickening of mucosal epithelial lining

**Fig. 9** Degeneration and necrosis of muscle fibers

**Fig. 10** Central nuclei in the muscle fibers

**Fig. 11** Hyaline degeneration of muscle fibers
The histological study indicated that the increased thickness of the soft palate in dogs with brachycephalic airway syndrome was not due to muscle hypertrophy or fat deposition but is instead due to significantly increased stroma within the lamina propria, edema and increased proportion of salivary tissue.

In this study histologically extensive degenerative lesions involving the majority of muscle fibers of the palatine musculature were found. The findings in the present study suggest that there is continuous injury to the palatine muscles which is not seen in normocephalic dogs (Arrighi et al., 2011).

Moderate to severe mucosal hyperplasia causes thickening of the mucosal epithelial lining of the soft palate (Pichetto et al., 2015).

Irregular dispersion of collagen fibers of the lamina propria was due to edema, sub epithelial dense connective tissue in the soft palate of brachycephalic dogs (Pichetto et al., 2015).

Extensive degenerative lesions observed in palatine musculature due to persistent traumatic injury of the palatine muscles due to brachycephalic syndrome in dogs with elongated soft palate and this condition is rarely seen in normocephalic dogs which coincides with the observations of Arrighi et al., 2011.

Many dogs were under corticosteroid treatment for brachycephalic syndrome which causes weakness in muscle fiber which coincides with the observations of Khaleeli et al., 1983.

Increased amount of intercellular space with thick epithelium is considered to be the expression of edema which was also observed in obstructive sleep apnea in humans which correlates with the findings of Hamans et al., 2000.

Hyperplasia of the palatine salivary glands was seen in all the samples for histopathological examination of soft palate.
Airway turbulence and friction of soft palate during inspiration and snoring could represent reasonable causes for palatine gland hyperplasia to occur as defensive mechanism (Woodson et al., 1991).

The function of palatine glands is to ensure constant humidity and lubrication of the palatine mucosa, to prevent friction damage during food (Kuehn and Moon, 2005). In brachycephalic dogs, palatine gland hyperplasia together with luminal mucin accumulation compounds thickening of the soft palate.

In conclusion the histological features of the brachycephalic palate were associated with muscular degeneration and atrophy of palatine muscles, combined with increased proportion of stroma and diffuse edema. The mucosal epithelial lining was thickened due to moderate to severe mucosal hyperplasia. Due to these changes in the histology of soft palate, changes in the elastic resistance coupled with progressive increase in volume of the soft palate results in the upper airway obstruction accompanied with typical clinical symptoms.

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References


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