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Research article

Evaluation of pre- and postoperative clinical signs, endoscopic findings and histomorphology of caudal soft palates in French bulldogs with brachycephalic obstructive airway syndrome that underwent elongated soft palate surgery

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Abstract

Brachycephalic obstructive airway syndrome (BOAS) is frequently seen in brachycephalic dog breeds including Bulldogs, French Bulldogs, and Pugs. Dogs suffering from this syndrome have respiratory difficulties manifested as snoring, inspiratory discomfort, exercise intolerance, panting in most cases and syncope in some. These clinical signs may be a result of several structural abnormalities of the nasal canal, oropharynx, nasopharynx, and trachea such as stenotic nares, elongated soft palate, and everted laryngeal saccules. We studied 12 French bulldogs presenting with BOAS and assessed the abnormalities by physical examination and endoscopy before surgical treatment. Palatoplasty or staphylectomy was performed in all dogs, in addition to the correction of nostril opening and resection of laryngeal saccules for some. Histo-morphology of the excised palates was also investigated. Correlations between the pre-and postoperative clinical signs, endoscopic severity and histo-morphology were evaluated. The surgical outcomes were successful in terms of postoperative signs ($P < 0.05$). Furthermore, we establish a positive correlation between the edematous epithelium with lamina propria and clinical severity ($P = 0.015$) and endoscopic presentations ($P = 0.037$). Based on our results, we propose a thick caudal soft palate with mucosal hyperplasia with its edematous stroma as an important etiologic condition in French bulldogs with BOAS. We highly recommend surgical intervention in dog breeds displaying BOAS. Endoscopic examination is also recommended but non-compulsory.

Keywords: : Brachycephalic syndrome, Elongated soft palate, Endoscopy, Histomorphology, Dog

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INTRODUCTION

Caudal palatoplasty is a common surgical procedure to accommodate an overlong soft palate in dogs suffering from upper respiratory discomfort, known as brachycephalic obstructive airway syndrome (BOAS). BOAS is predominantly seen in brachycephalic dog breeds including Bulldogs, French Bulldogs, Pugs, and Pekingese. Clinical signs include snoring, wheezing, dyspnea and exercise intolerance, as well as gastrointestinal signs like salivation, regurgitation or vomiting in some cases (Monet, 2003). These signs are a result of structural abnormalities in the upper airway passage such as stenotic nares, thick and overlong soft palate, caudal protrusion of nasal conchae, abnormal movement of the larynx, and abnormally short structure of the skull (Monet, 2003; Ginn et al., 2008 and Caccamo, 2014). Palatine muscle myopathy has been reported in brachycephalic dogs but not in mesocephalic dogs (Arai et al., 2016; Arrighi et al., 2011; Pichetto et al., 2011). Arai et al., 2016 suggested that the evidence of both atrophic and hypertrophic muscles coincide with fiber type grouping and increasing type I and II myofibers are a consequence of the denervation from peripheral nerve damage and subsequent re-innervation of palatinus (Arai et al., 2016b). The appearance of muscle hyalinization, sarcoplasm fragmentation, activation of satellite cells and endomysium and perimysium fibrosis were also extensively found in the soft palate of brachycephalic dogs with BOAS (Crosse et al., 2015; Pichetto et al., 2011). These muscular changes were believed as one factor that causes soft palate malfunction and initiation of upper respiratory obstruction (Arai et al., 2016a). In addition, the thickening of soft palate mucosa from epithelial hyperplasia and intracellular edema, as well as connective tissue swelling in lamina propria, have been observed in BOAS. Furthermore, several lobules of the hyperplastic salivary gland with static mucin in dilated glandular lumen and duct have also been usually found within lamina propria. The edematous epithelium and hyperplastic palatine gland have been extensively investigated at the oral side of affected soft palate than in nasopharyngeal side. Moreover, the glandular tissues of the oral side are mostly composed of mucous acini containing sulfated acidic glycoconjugates which produce thick and viscous mucin. The high viscosity of secretion inhibits mucus flow and causes mucin stasis in dilated lumen and duct (Pichetto et al., 2011). Crosse et al. (2015) suggested that thick soft palate due to an abundant connective tissue and salivary gland in lamina propria could be the cause of description of BOAS rather than an overlong soft palate (Crosse et al., 2015). Importantly, all abnormalities of mucosal epithelium, stromal connective tissue, palatine salivary glands and muscular fibers found in BOAS as mentioned previously do not exist in the soft palate of neonate brachycephalic dogs. The alterations of these tissues have been suspected to occur after receiving prolonged vibration and trauma from depressive respiration (Pichetto et al., 2015).

Apart from radiography, endoscopy can be used for diagnosing BOAS. Endoscopy, as it clearly reveals abnormal nasopharyngeal and laryngeal structures, can be advantageous before surgical intervention (Monet, 2003; Reiter and Holt, 2012; Oechtering et al., 2016a and b). Surgical treatment including enlargement of stenotic nares, resection of caudal soft palates (staphylectomy), and excision of everted laryngeal saccules can alleviate respiratory discom-

fort, with staphylectomy the main procedure (Monet, 2003; Poncet, 2006). We hypothesize that the abnormal endoscopic features of oropharyngeal organs and histo-morphological appearance of the soft palate are associated with the clinical signs of BOAS in brachycephalic dogs.

MATERIALS and METHODS

Study subjects

Twelve French bulldogs showing various clinical signs of BOAS were registered at the Kasetsart University Veterinary Teaching Hospital, Faculty of Veterinary Medicine, Kasetsart University, Bangkean Campus, Bangkok, Thailand. They underwent a thorough clinical examination including radiography and endoscopy prior to the surgery, which was eventually performed in all cases. This study was approved by the Kasetsart University committee on animal welfare for scientific study (ACKU61-VET–020).

Assessment of clinical signs

Clinical signs were recorded during the standard physical examination. Abnormal breathing was assessed by the severity of clinical appearances, and upper respiratory discomfort was classified as described by Crosse et al. (2015) and Poncet et al. (2005). Respiratory stridor, snoring, dyspnea or fainting were categorized as grade 3 (severe), respiratory discomfort and exercise intolerance as grade 2 (moderate), mild respiratory discomfort as grade 1 (mild), and absence of any symptoms as grade 0 (Table 1). Animals presented with ambiguous clinical signs, endoscopic appearances or disease causation, including disease of nasal turbinate, tumor or bleeding disorders, were not included in the study.

Endoscopic examination

Endoscopic examination (Olympus GIF XP150N, Olympus, Japan; Telescope 30^0 2.7 mm. 11 cm, STORZ, Germany) of the larynx, oropharynx, and upper airway was performed and the findings were classified as follows: a) grade 1 with overlong soft palate (caudal end was beyond epiglottis), b) grade 2 had overlong soft palate with very small laryngeal saccule, and c) grade 3 with thick, aberrant soft palate, overlong soft palate (extending beyond laryngeal opening), the presence of laryngeal saccule, abnormal narrow pharyngeal space and caudal extent of nasal conchae (Table 1). Endoscopy was performed under light to medium plane of anesthesia. Diazepam (Atlantic Laboratories, Thailand) (0.5 mg/kg, intravenously) and propofol (Troikaa Pharmaceuticals, India) (3 mg/kg, intravenously) were used as the anesthetic pre-medication and induction agents, respectively. Isoflurane (Piramal Critical Care, USA) 2% in 100% oxygen was used to maintain the animals under anesthesia via endotracheal intubation once the laryngeal organs were examined. The endotracheal tube was temporarily removed during the examination and was reinserted when the examination was completed.

Table 1 The criteria for categorizing severity in each group of examination.

GRADE	CLINICAL SIGN
0	Normal appearance
I	Mild respiratory discomfort and exercise intolerance (mild)
II	Respiratory discomfort, stridor and exercise intolerance (moderate)
III	Respiratory stridor, snoring, dyspnea or fainting (severe)
	ENDOSCOPIC FINDING
0	Normal appearance
I	Overlong soft palate (the caudal end was beyond epiglottis)
II	Overlong soft palate with the presence of laryngeal saccule
III	Thick, aberrant soft palate, overlong soft palate, an abnormal narrow pharyngeal space and caudal extension of nasal conchae
	HISTOPATHOLOGICAL FINDING
	Mucosal hyperplasia and edema
0	Normal appearance of epithelium and stroma
I	Mild epithelial hyperplasia and stromal edema. Small clusters of edematous epithelium were found (cluster length ≤ 100 μm)
II	Moderate epithelial hyperplasia and stromal edema. Length of edematous epithelium was >100 μm but ≤200 μm
III	Severe epithelial thickening and stromal edema. Length of edematous epithelium was >200 μm
	Glandular alterations
0	Normal appearance of secretory unit and duct
I	Averaged ≤ 10 lobules of gland per 4x microscopic field with mild glandular hyperplasia and mucin stasis
II	Averaged > 10 lobules of gland but ≤ 20 lobules per 4x microscopic field with moderate glandular hyperplasia and mucin stasis in dilated lumens and ducts
III	Averaged > 20 lobules per 4x microscopic field with severe glandular hyperplasia. Mucin stasis in dilated lumens and ducts was obviously found with exfoliated glandular cells
	Palatine muscle modifications
0	Normal appearance but some dogs possessed a large or moderate amount of muscle fibers
I	Muscular disproportion in some fascicles. Slight degeneration and fragmentation or none
II	Disproportion of muscle was mostly found. Degeneration and fragmentation were observed in some fascicles
III	Obvious disproportion of muscle in all fascicles (normal sized fibers were absent). Degeneration and fragmentation were clearly observed

Surgical procedure

Surgery was prescribed once endoscopic examination was completed. Surgery was performed by only one surgeon. Caudal palatoplasty or staphylectomy was performed in all cases while the animals were stable in stage 3 plane 2 of anesthesia. Curve shape excision was done between tonsillar crypts once soft palate tip was stay-sutured. The simple continuous pattern was performed using Vicryl 4–0 (Johnson & Johnson, USA). Only large everted laryngeal saccules were excised without suture during surgery, while hemostasis was completed by slight pressure on the excision site. Surgical correction of stenotic nares was performed by vertical wedge technique as described by Monet (2003). Simple continuous pattern with Vicryl 4–0 (Johnson & Johnson, USA) was used to close the wound.

Histopathological study and evaluation

The excised caudal part of soft palates was collected and fixed in 10% neutral buffered formalin to perform paraffin section by standard protocols (Luna, 1968REF). All samples were cut at 5 μm thickness and stained with hematoxylin and eosin (H&E). The anatomical study of the soft palate in brachycephalic dogs showed clinical signs of BOAS (Pichetto et al., 2011). The histopathological morphology found in this particular case included mucosal (epithelium and lamina propria) hyperplasia and edema, palatine salivary gland hyperplasia and duct dilatation, mucus stasis with exfoliated glandular cells, muscular hypotrophy associated with compensatory hypertrophy, and hyaline degeneration along with fragmentation of muscle fibers. Therefore, we categorized the histopathological findings into three criteria: (1) mucosal hyperplasia with edematous epithelium and connective tissue (2) glandular hyperplasia with mucus retaining in dilated lumen and duct and (3) muscular disproportion and fragmentation of muscles. These tissue samples were evaluated at three levels from grade I to grade III depending on the degree of alterations (Table 1). The level shown as grade 0 was indicated no abnormality.

Statistical analysis

Correlation analysis of the different grades (variables) of clinical signs, endoscopic findings and histo-morphological appearance of soft palate was conducted using the SAS statistical program (University edition, 2018). A paired t-test for clinical signs between pre- and post-operation was also conducted (SAS, University edition, 2018). A p-value ≤ 0.05 was considered significant.

RESULTS

Animals

Among the twelve French bulldogs, the ration between male and female was equal. The age at the time of surgery ranged from 1.4 year to 5.1 years. Of them, six were of age less than 2 years, four were between 2 and 3 years, and two were of 4 and 5 years.

Clinical signs

Eleven dogs showed respiratory discomfort, wheezing and exercise intolerance. One had normal respiration but exercise intolerance was seen and it was assigned to grade I. Six dogs were assigned to grade II due to respiration difficulty and exercise intolerance, and another five had clear respiratory difficulty and wheezing with occasional dyspnea and syncope, and were classified as grade III (Table 2).

Table 2 The number of categorized patients in each group of examination.

GRADE	CLINICAL SIGN (pre/post-operative patients)	ENDOSCOPIC FINDING	HISTOPATHOLOGICAL FINDING		
			Mucosal hyperplasia and edema	Glandular alterations	Palatine muscle modifications
0	NA	NA	2	2	4
I	1/10	3	6	5	4
II	6/2	5	1	1	1
III	5/0	4	3	4	3

Endoscopic findings

The evidence of caudal soft palate over the epiglottis was seen in all cases. Four dogs also showed thick and aberrant soft palates with enlarged tonsils and everted laryngeal saccules, and were classified as grade III of severity. One case in the group presented a caudal extension of nasal turbinate bone in the nasopharynx. Another five dogs were categorized as grade II as they had an elongated soft palate and small everted laryngeal saccules. Only three dogs with grade II showed overlong soft palates without having a saccule (Figure 1, Table 2). Laryngeal paralysis (various types of abnormal movement of arytenoid cartilages) was not observed in any of the dogs.

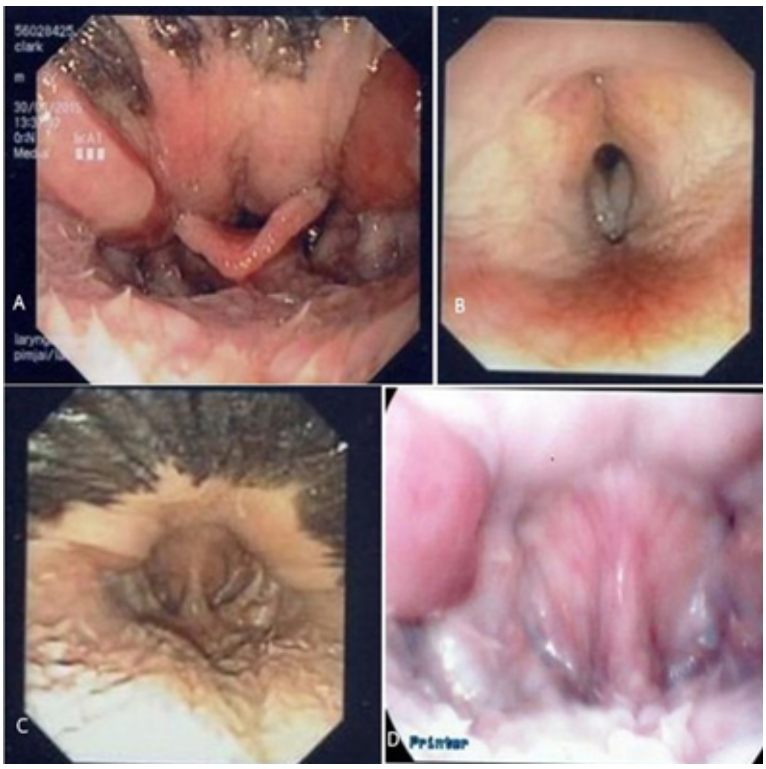

Figure 1 Endoscopic findings. An over-long and aberrant soft palate is shown in Figure 1A. Large tonsils are also illustrated in Figure 1A and 1D. The small opening of the larynx and everted laryngeal saccules is shown in 2B. Figure 2C shows a mild grade of endoscopic grading, which is shown only by a long soft palate in the narrow oropharynx.

Surgical outcomes

Staphylectomy was performed in all cases (Figure 2). Two dogs also had corrective surgery of stenotic nares. The resection of everted laryngeal saccule was performed in one case, while large tonsil was not excised in any. Improvement in respiratory discomfort was seen in 11 out of the 12 cases within two months post-operation (Table 2).

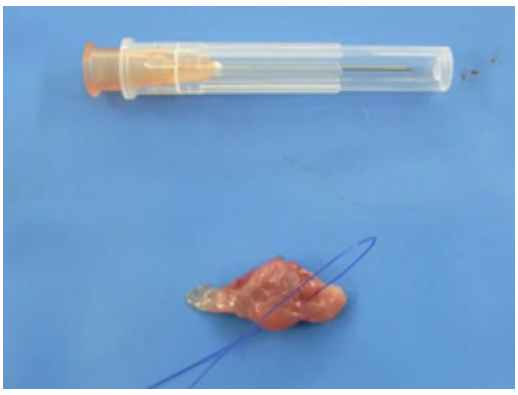

Figure 2 Excised caudal soft palate. Caudal soft palate excised from one of the moderate surgical patients along with 1.5 inches hypodermic needle for comparison.

Histopathological findings

The tissue sections of soft palate revealed vascularized stroma with lobules of palatine salivary glands consisting primarily mucous acini and very few serous acini. The fascicles of differently oriented palatine muscle fibers were found. Peripheral nerve fibers innervated all components within the connective tissue. The mucosa of soft palate was covered by non-keratinized stratified squamous epithelium. Among twelve dogs with BOAS, two dogs showed the normal appearance of covering epithelium and connective tissue in lamina propria. For other ten, distinct levels of epithelial hyperplasia and edema, as well as edematous stroma, were seen. Six of them had mild mucosal hyperplasia, where small clusters of ≤ 100 μm edematous epithelium showed a multifocal distribution, thus the cases were evaluated as grade I. Three dogs exhibited severe mucosal hyperplasia with obvious edema of epithelium (length > 200 μm) along the mucosa (Figure 3A). Moreover, the detachment of epithelium from underneath connective tissue was found in some tissue samples indicating that severe stromal edema had occurred. This group was referred as grade III. Another dog, placed in grade II, revealed a moderate level of mucosal hyperplasia, whose length of edematous epithelium was between that in grade I and III (Figure 3B and Table 2).

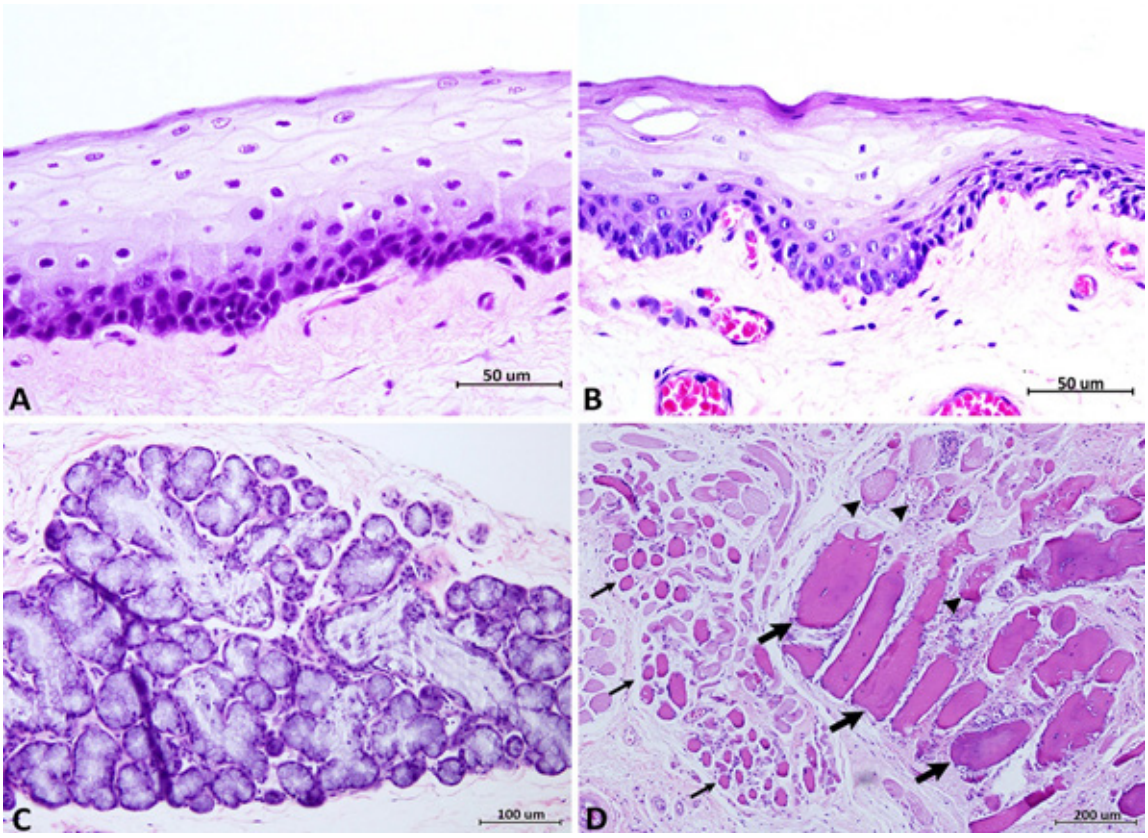

Figure 3 Histopathological findings of the excised soft palate. (A) Severely edematous epithelium at the oral surface of soft palate indicated as grade III mucosal hyperplasia. (B) Moderate epithelial edema evaluated as grade II mucosal hyperplasia. (C) Glandular hyperplasia with exfoliated glandular cells and retained mucus in the dilated lumen and duct evaluated as grade III glandular hyperplasia. (D) The disproportion of muscle showing fascicles of muscular hypotrophy (thin arrow) compensated by hypertrophic fibers (thick arrow). Sarcoplasm fragmentation of muscle fibers was also observed (arrowhead).

The examination of glandular alterations demonstrated that four cases of BOAS had a high density of hyperplastic glands for more than 20 lobules per 4x microscopic ocular field. Each lobule exhibited dilated lumen of the secretory units and ducts with retained mucin and exfoliated glandular cells. Such degree of alterations was indicated as grade III glandular hyperplasia (Figure 3C). Meanwhile, one dog revealed the hyperplastic appearance of glandular tissue less than 20 lobules per field with moderate mucin stasis in dilated lumens and ducts, referred as grade II. Five dogs that were evaluated as grade I presented approximately 10 lobules per field or less, with mild glandular hyperplasia and mucin statis. A normal appearance of secretory units and duct was found in the other two dogs (Table 2).

The levels of palatine muscle modifications were also investigated in all cases. Four of them possessed normal muscular morphology, size, and arrangement, though some dogs had a large or moderate amount of muscular tissue. The rest showed different scales of muscular disproportion. The hypotrophic and hypertrophic muscle fibers were either noticed within separated fascicles or in the same fascicle. Additionally, most fibers lost their striation and some fibers underwent hyaline degeneration and sarcoplasm fragmentation (Figure 3D). The grading of muscular modifications depended on the frequency of muscular disproportion, degeneration, and fragmentation found in muscle fascicles. Regarding these criteria, the number of cases categorized in grade I, II and III of muscular modifications were four, one and three dogs, respectively (Table 2).

Statistical Analysis

Post-operative clinical signs were significantly improved compared to the pre-surgical signs in all dogs, as per the signed rank test ($P = 0.002$). Moreover, we did find a statistical correlation (Spearman's Rank Correlation) between mucosal (epithelium and lamina propria) indices and clinical signs ($P = 0.067$) or endoscopic findings ($P = 0.037$) (Table 3). We did not observe any positive correlation between endoscopic findings and clinical signs. Other parameters of histological patterns of both palatine muscle arrangement and glandular pattern did not show any correlation with others

Table 3 Spearman's correlation coefficients.

	Spearman's correlation coefficients (P-value)			
	Endoscopic findings	**Glandular alterations**	**Palatine muscle modifications**	**Mucosal hyperplasia and edema**
Clinical signs	0.24 (0.44)	0.292 (0.35)	0.158 (062)	0.677 (0.015)
Endoscopic findings	-	–0.072 (0.82)	0.021 (0.50)	0.604 (0.037)

DISCUSSION

All clinical respiratory signs of BOAS including snoring and wheezing decreased significantly after surgical intervention in this study. The findings concord with the fact that surgical resection of the overlong part of the caudal soft palate can improve respiratory discomfort in most cases of BOAS (Monet, 2003) and the elongated soft palate is the most common observation in brachycephalic dog breeds (Monet, 2003). In addition, other gastrointestinal signs including salivation, vomiting, and nausea that are present in BOAS were also alleviated. The present study showed that the respiratory signs decreased significantly ($P = 0.002$) postoperatively, in accordance with a previous study (Poncet et al., 2006) which reported 90% improvement in BOAS symptoms.

Several concomitant gastrointestinal signs have been described in BOAS previously (Poncet et al., 2005). From all BOAS cases studied in this work, four possessed severe (grade III) and one showed moderate (grade II) grade of glandular alterations consisting of glandular hyperplasia and mucin stasis in the dilated lumens of secretory units and secretory ducts. A large number of secretory cells which were seen in these dogs indicated that excessive glandular secretion may be an etiological factor of the respiratory and gastrointestinal signs. The glandular hyperplasia was present either at nasopharyngeal and oral sides of the soft palate. There were more mucous acini in the oral stroma than those the in the nasopharyngeal side. In addition, the histochemical study of mucous cells in oral side demonstrated highly sulfated acidic glycoconjugates which caused high viscosity and thickness of mucous secretion (Pichetto et al., 2011). The excessive viscous mucus produced from hyperplastic glandular cells is mostly retained inside the lobules leading to luminal dilatation of mucous acini and ducts. The thick secretion that stuck on the oral mucosal surface of soft palate might induce nausea and vomiting, but hyper-salivation might be subsequently happened by other salivary glands. Importantly, the mucin stasis and dilated lobular lumens were possible to be the major causes of soft palate thickness that might affect respiratory obstruction. However, we did not see any positive correlation between the respiratory and gastrointestinal signs, since the latter was not a priority in our study. We found that even in some cases with mild clinical and endoscopic findings, grade III of glandular hyperplasia was observed. The individual differences of tolerance to respiratory distress might be the reason behind this phenomenon. The intestinal signs in BOAS patients could also be related to gastroduodenal tract lesions as described by Poncet et al. (2005).

Flexible endoscopy can be used to observe the nasal canal, nasopharynx and caudal part of the nasal turbinate bone in cases of BOAS. An enlarged nasal turbinate bone or caudal extension into nasopharyngeal space was not seen in our study. The endoscopic findings did not show any correlation with the clinical signs, but evidently correlated with mucosal indices of an aberrant epithelium and its lamina propia ($p= 0.037$). We categorized endoscopic features according to their appearances that might be more pronounced in case of visually abnormal texture. This might be the reason of concordance between mucosal architecture and endoscopic finding but not for clinical signs. As a result, endoscopy cannot be used for the prognosis of clinical signs, otherwise more delicate criteria have to be approved.

Using computed tomography imaging (CT), the obstruction of nasal canal due to abnormal rostral and caudal nasal turbinate in Pugs was observed (Oechtering et al., 2016a, and b); however, the only abnormal rostral nasal canal was less commonly seen in French bulldogs and English bulldogs. In the same study, endoscopy following CT was used, thus the lesions were easily seen by sagittal plain slices. We recommend the use of a standard laryngoscope to evaluate BOAS with the incorporation of endoscopy in case of ambiguous diagnosis. CT should also be performed if an aberrant turbinate is suspected in French bulldogs.

In a previous study, the palatine muscle in resected soft palate had signs of both atrophy and hypertrophy in brachycephalic dogs, but not in mesocephalic and dolichocephalic dogs (Arai et al., 2016; Arrighi et al., 2011; Pichetto et al., 2011). In our study, we found disproportion of palatine muscle in which hypotrophic and hypertrophic muscles were located either within the same fascicle or in separated fascicles. Furthermore, hyaline degeneration and sarcoplasm fragmentation of muscle fibers were also detected. These abnormalities might be due to peripheral nerve damage, eventually affecting proper moving and tone of the soft palate. This consequently resulted in compensatory hypertrophy. The microscopic modifications of palatine muscle as mentioned above have also been described by many researchers (Arai et al., 2016a; Arai et al., 2016b; Pichetto et al., 2011). These studies also included immunohistochemical staining for significant molecules involving muscular structure, regulation, and function (dystrophin, neurofilament and myosin ATPase, respectively) and morphometric analysis of muscle. The studies implied that all abnormalities were the consequences of the damage of peripheral nerve, which innervates palatine muscles of the soft palate in BOAS, and the subsequent compensation of destroyed muscular tissue (Arai et al., 2016a; Arai et al., 2016b). Therefore, we hypothesized that the microscopic modifications of skeletal muscle which manifested in this study might affect the clinical signs. However, we do not suggest any correlation between palatine muscle features and other parameters. Aberrant muscle features do not explain most of the BOAS cases in the present study. This might due to the complexity of disease etiology.

Crosses et al. (2015) considered the thickness of caudal soft palate, which could be a result of increased connective tissue stroma in the palate tissue, as the likely cause of breathing difficulties in brachycephalic dog breeds rather than an overlong soft palate (Crosses et al., 2015). In our study, in ten of the twelve dogs, various degrees of hyperplastic edematous epithelium coincided with connective tissue edema. Three of these showed grade III of mucosal hyperplasia and edema consistent with the presence of severe (grade III) clinical signs. Meanwhile, in these three cases, two dogs also showed severe (grade III) and one dog showed moderate (grade II) endoscopic grade. Consequently, an aberrant epithelium and connective tissue were positively correlated with clinical presenting ($P = 0.015$) and endoscopic findings ($P = 0.037$) (Table 3). A significant correlation between the connective tissue features and clinical signs also support the notion that a thick soft palate might be an important factor of the respiratory signs seen in BOAS. However, hyperplastic edematous epithelium and edema of its lamina propria may be due to abnormal movement of soft palate both from physical damage of turbulent airflow and

laryngopharyngeal reflux which also occur in human patients with obstructive with sleep apnea (Crosses et al. 2015). Further studies are warranted to elucidate the particular features of epithelium and submucosa, which can lead to etiology of BOAS.

The resection of the caudal soft palate (staphylectomy) was performed along with corrective surgery of stenotic nares in some cases and the resection of the everted laryngeal saccule was required in one case. All dogs showed improvement in respiratory signs, indicating the significance of an overlong soft palate in BOAS, and the utility of caudal soft palate resection. Some clinicians recommend the resection of everted laryngeal saccules (Harver, 1982a, b and Cantotore et al., 2012), although it does not lead to improved post-operative signs as reported by Poncet et al. (2006). Cantotore et al. (2012) observed that self-regression of the laryngeal membrane may not occur, even after the completion of staphylectomy. The greatest advantage of everted laryngeal saccule resection is that it reduces the resistant airflow through the larynx, although some may develop excessive fibrous tissue at the surgical site instead. We performed this resection in only one case which presented large saccules. We also did not perform tonsillectomy since this gland is important for controlling post-operative inflammation (Poncet et al., 2006). Caccamo et al. (2014) studied the extent of glottis opening and the skulls of brachycephalic dogs but failed to show a clear correlation between them. In addition, the short nose and skull in brachycephalic breed may or may not be associated with a narrow opening of the epiglottis.

Elongated soft palate was shown to be a major factor influencing clinical signs in BOAS in brachycephalic dogs. Other abnormalities in any other related organ can be evaluated by CT and endoscopic examination in case of the ambiguous diagnosis. The surgical resection of an aberrant thick or overlong soft palate part is highly recommended.

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