

Short Communication

Nasopharyngeal stenosis secondary to soft palate dysgenesis in a cat

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Abstract

This report is the first description of soft palate dysgenesis inducing nasopharyngeal stenosis in a cat. The symptomatology included continuous stertorous respiration and changes in phonation. Open-mouth breathing, seromucous nasal discharge and sneezing were intermittently present. On physical examination the cat showed a respiratory pattern characteristic of upper airway disease, with stridor, increased inspiratory effort and severe bilateral obstruction of nasal passages to the airflow. Pharyngo-laryngoscopic evaluation showed an abnormal conformation of the soft palate that appeared attached to the caudal and lateral pharyngeal wall and stretched the whole length of the pharynx. A strip of soft tissue emanated from the caudal part of the hard palate to the right aspect of the base of the tongue. Caudal to this strip of tissue, the intrapharyngeal opening was stenosed, therefore preventing normal airflow. Surgical reconstruction was not recommended. The cat was clinically stable with mild respiratory symptoms 17 months after the diagnosis.

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The nasopharynx extends from the choanae to the intrapharyngeal arches and connects the nasal cavity with the larynx by means of the intrapharyngeal opening. Suspicion of nasopharyngeal disease is based on the presence of clinical signs that may include snuffing, sneezing, wheezing, gagging, nasal discharge, and varying grades of obstruction to the airflow. Upper airway obstruction is detected as stertor and/or stridor and intermittent or persistent open-mouth breathing, accompanied with varying degrees of obstructive respiratory pattern (Novo and Kramek, 1999; Holt, 2004; Billen et al., 2006). The most frequently reported obstructive processes affecting to the feline nasopharynx are neoplasia (mainly lymphosarcoma), inflammatory polyps and acquired nasopharyngeal stenosis (Allen et al., 1999; Willard and Radlinsky, 1999; Holt, 2004). However, congenital obstructive diseases are not commonly reported in small animals. This report describes

nasopharyngeal stenosis secondary to a soft palate dysgenesis in a cat.

A 1.5 year old female domestic shorthair cat weighing 4 kg was presented to the Cardiorespiratory Service of the Veterinary Teaching Hospital of the University of Murcia for evaluation of a chronic upper respiratory problem. The owner stated that the cat had a persistent breathing problem characterized by continuous stertorous respiration and phonation changes ('hoarseness') that had lasted since the cat was 8 weeks old. Open-mouth breathing, seromucous nasal discharge and sneezing were intermittently present. Several combinations of antibiotics and anti-inflammatories had been used leading to a positive clinical response but without achieving complete resolution.

On physical examination the cat was found to be in good bodily condition (body condition score 3/5) and appeared alert and playful. The animal's mucous membranes were normal and the capillary refill time was <2 s. Respiratory pattern was characteristic of upper airway disease, showing stridor (most prominent on inspiration) and

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increased inspiratory effort. Respiratory rate was 35 breaths/min. No external deformities, swelling, softening or painful areas were found during detailed inspection and palpation of the nasal and frontal bones. Normal nostrils with mild seromucous nasal discharge were observed. Severe bilateral obstruction of nasal passages to the airflow was suspected when the flow of air was assessed by holding a small strand of cotton placed over each nostril while the opposite nostril was closed with a finger. Respiratory auscultation revealed accentuated laryngeal and tracheobronchial sounds. Cardiac auscultation was normal with a heart rate of 155 beats/min. The remainder of the physical examination was within normal limits.

Mild lymphopenia (lymphocyte count, $1.06 \times 10^3/\mu\text{L}$; reference range, $1.4\text{--}7 \times 10^3/\mu\text{L}$) was the only abnormality on the complete blood count. Mild uremia (51.9 mg/dL; reference range, 20–50 mg/dL) with normal serum creatinine concentrations (0.96 mg/dL; reference range, 0.5–1.5 mg/dL) and urine specific gravity (1030; reference range, 1025–1035) and mild hyperglycemia (145 mg/dL; reference range, 70–110 mg/dL) were found in serum biochemical analysis and urinalysis.

Based on the history and physical examination an obstruction of the upper respiratory tract was suspected. The list of potential causes included laryngeal dysfunction, laryngeal edema, elongated soft palate, nasopharyngeal polyps, nasopharyngeal stenosis, choanal atresia and obstructive nasal disease. Rhinoscopy and pharyngo-laryngoscopy were considered useful procedures to investigate the origin of the obstruction.

The cat was tranquilised using a combination of IM acepromazine (0.02 mg/kg) and butorphanol (0.3 mg/kg). Anaesthesia was induced and maintained with IV propofol (5 mg/kg). The cat was positioned in sternal recumbency and the upper airway was examined. Visual inspection was first performed with the aid of a light source. Laryngeal movements were evaluated in a light plane of anaesthesia and found to be normal. Prepharyngeal structures, including gingiva, teeth, tongue, hard palate, palatoglossal arches and tonsils were also considered normal. However an abnormal conformation of the soft palate was identified. The free caudal margin of the soft palate appeared to be attached to the caudal pharyngeal wall, so the soft palate stretched the whole length of the pharynx. A strip of soft tissue emanated from the caudal part of the hard palate to the right aspect of the base of the tongue. Caudally a small oval slit was seen and was the only communication between this single laryngo-pharyngeal cavity and the nasopharynx. The communication constituted a stenosed intrapharyngeal opening (Fig. 1). No abnormalities of the nasopharyngeal area were felt during digital palpation through the abnormal soft palate tissue.

The exploration was carried out using a flexible video-endoscope (Fujinon EB-4105, Onys). The pharyngeal mucosa appeared congested and with abundant seromucous secretion. Since the soft palate was adhered to the pharyngeal wall, the larynx was easily seen at the end of

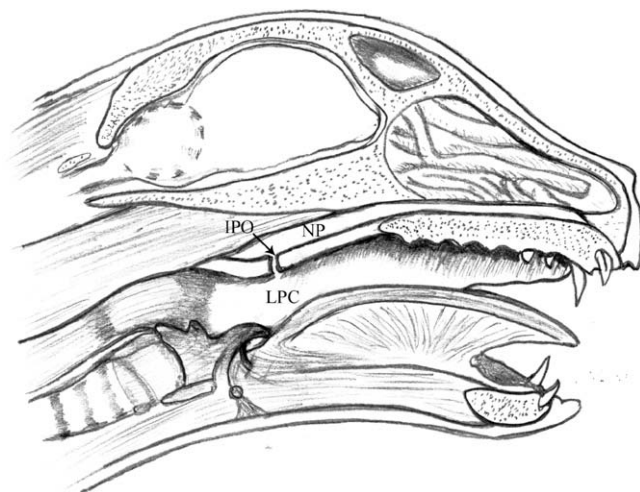


Fig. 1. Schematic drawing showing the soft palate abnormality of the cat. The free caudal margin of the soft palate is adhered to the pharyngeal wall. A stenotic intrapharyngeal opening (IPO) is the only communication between the single laryngo-pharyngeal cavity (LPC) and the nasopharynx (NP), inducing nasopharyngeal stenosis.

the laryngo-pharyngeal cavity. The laryngeal mucosa appeared severely oedematous with abundant clear secretions. Dorsal to the larynx, a normal esophageal vestibulum was seen (Fig. 2). The intrapharyngeal opening was too narrow to introduce the endoscope and visualisation of the nasopharyngeal area was not possible. In order to evaluate the patency of the internal nasal orifices (choanas), a soft 5 Fr catheter was introduced into each nostril



Fig. 2. Endoscopic view of the laryngeal opening of the cat. On initial examination the laryngeal mucosa was severely oedematous with abundant clear secretions. Note the abnormal laryngeal shape secondary to the laryngeal oedema. Dorsal to the larynx, a normal oesophageal vestibulum (EV) can be seen.

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