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Case Report

An Unusual Case of Acquired Nasopharyngeal Stenosis Associated with Chronic Displaced Skull Fractures in a Cat

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Simple Summary

Acquired nasopharyngeal stenosis in cats is usually caused by membranous or fibrotic tissue, while bony obstruction is more commonly congenital. This case describes an unusual presentation of acquired nasopharyngeal stenosis associated with chronic osseous remodelling following presumed facial trauma in a cat. Balloon dilation resulted in only limited luminal enlargement, although the factors contributing to the poor treatment response could not be definitively established.

Abstract

A 3-year-old neutered male domestic shorthair cat was referred for bilateral mucopurulent nasal discharge, marked stertor, sneezing, and reverse sneezing that, according to the owner, developed immediately after a road traffic accident about one year earlier. Clinical signs had shown mild, transient improvement during repeated courses of antimicrobial and anti-inflammatory treatment. Computed tomography revealed multiple chronic comminuted skull fractures with smooth, sclerotic margins. Displaced fracture fragments were associated with marked narrowing of the rostral nasopharynx, with a minimum luminal diameter of 1.5 mm. Retroflexed nasopharyngoscopy identified a fixed, concentric stenosis that could not be traversed with a 3.5 mm endoscope. A deflated 10 mm balloon catheter was advanced antegrade across the lesion; seven inflations produced minimal luminal enlargement and no clinically meaningful improvement. Brush cytology showed predominantly neutrophilic inflammation, and bacterial culture was negative. Stertor persisted despite oral prednisolone, while nasal discharge recurred after treatment. At reassessment 1 month later, the clinical condition was unchanged; repeat imaging and endoscopy were declined.

Keywords: nasopharyngeal stenosis; craniofacial trauma; osseous remodelling; balloon dilation; computed tomography; rhinoscopy

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1. Introduction

Nasopharyngeal stenosis (NPS) is an uncommon fixed narrowing of the airway located below the choanae. It may be congenital, but acquired disease is more commonly attributed to inflammation, aspiration, previous surgery, or, less often, trauma [1]. Typical signs include stertor, reduced nasal airflow, nasal discharge, and sneezing [1]. Contemporary feline CT series predominantly describe NPS as a focal, concentric or web-like

soft-tissue narrowing in the caudal nasopharynx, often accompanied by rhinitis but not destructive nasal disease [2,3]. Although lateral radiography can be a useful screening test, CT and retroflexed nasopharyngoscopy provide complementary lesion characterisation and treatment planning [2–4]. Balloon dilation and extended palatoplasty can both produce favourable outcomes in selected cats [5]. Recurrence remains a major concern, with one feline study showing that planned serial balloon dilations were the only factor associated with longer recurrence-free outcomes [6]. Acute traumatic narrowing caused by fracture displacement and nasopharyngeal wall collapse has been successfully treated in two dogs with three balloon dilation procedures, initiated 9 or 18 days after injury [7]. Chronic acquired post-traumatic nasopharyngeal stenosis associated with mature osseous remodelling represents an unusual clinical presentation in cats. This report describes the CT and endoscopic findings, as well as the response to balloon dilation one year after the reported facial trauma.

2. Case Description

A 3-year-old neutered male domestic shorthair cat with a body weight of 3.5 kg (body condition score 6/9), was referred for continuous bilateral mucopurulent nasal discharge, marked stertor audible at a distance, sneezing, and reverse sneezing. According to the owner, the cat had been involved in a road traffic accident approximately one year before presentation. No respiratory signs had been observed before the reported accident, whereas persistent upper respiratory signs developed shortly thereafter. Antimicrobial therapy and meloxicam were administered immediately after the reported trauma. Contemporaneous clinical records and diagnostic imaging were unavailable; therefore, a direct causal relationship between the reported trauma and the subsequent anatomical abnormalities could not be established. Subsequently, repeated 10-day courses of amoxicillin, doxycycline, or cefalexin were administered at least monthly. These treatments produced only mild, transient improvement, most evident for the nasal discharge, whereas the stertor remained essentially unchanged.

Rare episodes of epistaxis were also reported. Appetite, activity and general condition remained normal. No open-mouth breathing, clinically apparent dyspnoea, exercise intolerance, cyanosis, dysphagia or gagging was reported. On presentation, the cat was bright and responsive. Marked stertor was audible without auscultation, and bilateral nasal airflow was reduced. Continuous bilateral mucopurulent discharge and intermittent sneezing were present. No external facial or nasal deformity or nasal pain was detected. Rectal temperature, respiratory rate, mucous membrane colour and peripheral oxygen saturation were unremarkable. No abnormalities were detected on complete blood count or serum biochemical profile performed as part of the preanaesthetic assessment before CT and rhinoscopy. Feline immunodeficiency virus antibody and feline leukaemia virus antigen tests were negative. CT followed by endoscopic evaluation was performed during the same general anaesthetic episode. CT examination of the skull revealed multiple chronic comminuted fractures with smooth, sclerotic margins (Figure 1a–d). The fractures involved the horizontal, perpendicular and sphenoethmoidal plates of the palatine bones; the orbital and perpendicular plates of the ethmoid bone; the vomer; the presphenoid bone; the pterygoid processes of the basisphenoid bone; and the pterygoid bones. Displacement of the fracture fragments and associated chronic osseous remodelling were observed in close anatomical association with deformation of the nasopharyngeal wall and marked narrowing of the rostral nasopharynx (Figure 1a), with a minimum luminal diameter of 1.5 mm at the level of the pterygoid processes of the basisphenoid bone (Figure 1c). Soft-tissue-attenuating material was also present along the deformed nasopharyngeal wall, potentially representing displaced normal soft tissues, fibrous tissue, or a combination of both.

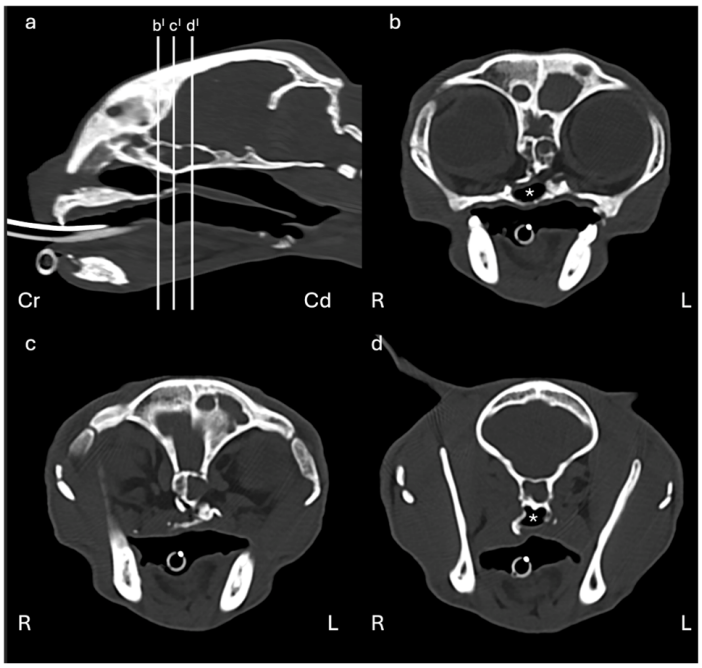


Figure 1. Precontrast CT images of a cat with acquired nasopharyngeal stenosis associated with chronic skull fractures. (**a**) Sagittal reformatted image showing marked narrowing of the rostral nasopharynx in association with deformation of the skull base and horizontal plates of the palatine bones. White lines b′, c′ and d′ indicate the levels of the transverse images shown in panels (**b**–**d**), respectively. (**b**) Transverse image at the level of line b′ showing multiple comminuted fractures involving the horizontal plates of the palatine bones, vomer, and orbital plates of the ethmoid bone, together with displacement of osseous fragments, deformation of the nasopharyngeal wall, and marked nasopharyngeal stenosis (white asterisk). Soft-tissue-attenuating material is also visible along the deformed nasopharyngeal wall. The caudal ethmoidal cavities are partially collapsed and contain soft-tissue-attenuating material. (**c**) Transverse image at the level of line c′ showing near-complete nasopharyngeal stenosis. (**d**) Transverse image at the level of line d′ showing progressive enlargement of the nasopharyngeal lumen caudal to the site of maximal narrowing.

No additional sites of stenosis were identified within the imaged upper airway. Immediately after CT, with the cat maintained under general anaesthesia, retroflexed nasopharyngoscopy was performed with the cat in sternal recumbency using a Five-S flexible videoendoscope (0° direction of view, 3.5 mm outer diameter, 65 cm working length and 1.2 mm working-channel diameter; Karl Storz SE & Co KG, Tuttlingen, Germany). Bilateral antegrade rhinoscopy was subsequently performed using a 30° forward-oblique HOPKINS rigid telescope (Ø 2.7 mm, length 18 cm) from the same manufacturer. Antegrade rhinoscopy of the right nasal cavity revealed severe disruption of the normal intranasal architecture, with complete loss of the turbinate structures and formation of a markedly enlarged common nasal passage extending caudally towards the choanal region (Figure 2b). A free osseous fragment was identified within the right nasal cavity and subsequently retrieved using endoscopic instrumentation. The retrieved osseous fragment was not submitted for microbiological culture or histopathological examination. In contrast, the left nasal cavity showed preserved intransal architecture, although the mucosa appeared erythematous, with increased surface reflectivity and a moderate amount of seromucous discharge.

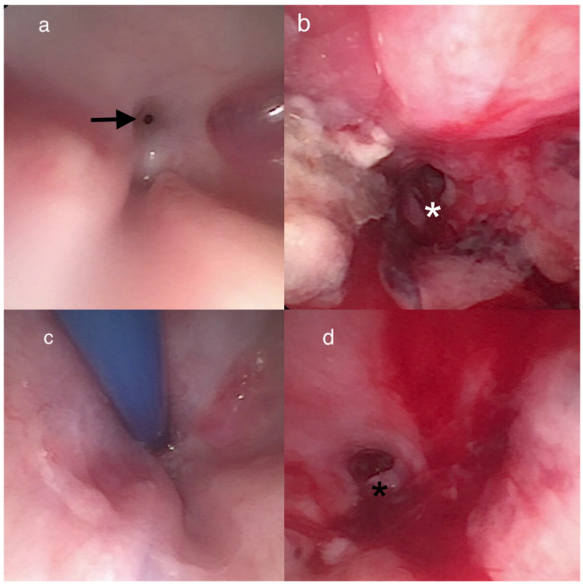


Figure 2. Endoscopic views of the nasopharyngeal stenosis before and after balloon dilation. (**a**) Retroflexed endoscopic view of the nasopharynx showing a severe concentric stenosis with a markedly reduced residual lumen (black arrow); the choanae are not visible. (**b**) Endoscopic view of the right nasal cavity showing severe disruption of the normal intranasal architecture, with complete loss of the turbinate structures and formation of a markedly enlarged common nasal passage extending caudally towards the choanal region; a free osseous fragment is visible along the left side of the image and was subsequently retrieved endoscopically. The maximum luminal enlargement achieved following balloon dilation is visible from the antegrade endoscopic perspective (white asterisk). (**c**) Trans-stenotic passage of a 10 mm balloon dilation catheter in its deflated state, confirming successful catheter advancement across the stenotic segment. (**d**) Retroflexed endoscopic view of the stenotic segment following balloon dilation, showing only limited widening of the residual stenotic opening. The maximum luminal enlargement achieved following balloon dilation is visible from the retroflexed endoscopic perspective (black asterisk).

The stenotic segment could not be traversed with the flexible endoscope. A deflated 10 mm diameter, 40 mm length high-pressure balloon dilation catheter (BARD X-FORCE, reference 997004; BARD) with a nominal inflation pressure of 10 atm and a rated burst pressure of 30 atm, was therefore advanced across the stenosis via an antegrade approach (Figure 2c). This balloon was selected because its deflated profile permitted passage through the severely narrowed nasopharyngeal lumen. Inflation was performed using a pressure-monitored inflation device equipped with a locking mechanism. At a maximum recorder inflation pressure of approximately 2.4 atm, the portion of balloon visible outside the stenotic segment appeared to reach its nominal diameter of 10 mm, based on qualitative endoscopic assessment rather than objective measurement. Seven consecutive balloon inflations were performed; the first five were maintained for approximately 3 min each and the final two for approximately 6 min each. However, only minimal enlargement of the stenotic nasopharyngeal lumen was observed. The combination of limited luminal enlargement at the stenotic site and the inability to increase inflation pressure beyond 2.4 atm, which remained below the manufacturer's nominal pressure of 10 atm, raised the possibility that the balloon may not have achieved its nominal diameter within the stenotic segment. Given that the manufacturer's nominal inflation pressure had not been reached, further dilation using a larger diameter balloon was not attempted.

The immediate response to balloon dilation was assessed qualitatively by endoscopic examination, which revealed only minimal luminal enlargement (Figure 2d), with the flexible endoscope remaining unable to traverse the stenotic segment. No quantitative

postprocedural luminal measurements were obtained. No intraoperative or immediate postprocedural complications occurred. Following recovery from anaesthesia, the stertor remained unchanged.

Cytological samples obtained by endoscopic brushing of the stenotic region revealed predominantly neutrophilic inflammation, including both non-degenerate and degenerate neutrophils, without evident cytological atypia. Aerobic and anaerobic bacterial cultures of material collected from the same site yielded no growth. Fungal investigations were not performed. Antimicrobial therapy had been discontinued three weeks before sampling. Nevertheless, the negative culture result does not definitively exclude an infectious component, particularly in the presence of chronic mucopurulent nasal discharge and extensive disruption of the nasal architecture. Endoscopic forceps biopsy was attempted, but despite successful grasping of the mucosal surface, tissue could not be detached, and no specimen suitable for histopathological examination was obtained. Consequently, histopathology was not performed. Following the procedure, oral prednisolone was administered at 1 mg/kg q24h for 10 days, followed by 0.5 mg/kg q24h for a further 10 days. Sneezing transiently decreased, whereas the stertor remained unchanged. Following discontinuation of prednisolone, bilateral mucopurulent nasal discharge recurred and progressively worsened. At re-examination 1 month after the procedure, stertor and nasal discharge persisted, with no meaningful overall improvement from baseline. Repeat CT and endoscopic examinations were recommended to reassess the stenosis and determine the feasibility of further intervention, including repeat balloon dilation, surgical correction, or stent placement. The owner declined further diagnostic evaluation and, consequently, no additional intervention was undertaken.

3. Discussion

The temporal association between the onset of respiratory signs and the owner-reported facial trauma, together with the absence of previously recognised respiratory disease and the close anatomical relationship between chronic skull fractures, osseous remodelling, and nasopharyngeal stenosis, is consistent with a presumed post-traumatic presentation. However, the absence of contemporaneous clinical records and diagnostic imaging prevents definitive confirmation of a causal relationship. Accordingly, the findings should be interpreted as an association between chronic craniofacial abnormalities and acquired nasopharyngeal stenosis rather than direct evidence of trauma-induced obstruction.

This presentation differs from the focal soft-tissue bands or soft-palate adhesions described in most feline CT series [2,3]. Previous reports of balloon dilation in cats have predominantly involved thin or fibromembranous stenoses, with resolution of clinical signs, although use of a 15 mm balloon and/or additional procedures was sometimes required [8–10]. A thin membranous stenosis was also successfully treated using 12 mm balloon dilation combined with membrane excision and two topical applications of mitomycin C [11], whereas balloon dilation alone resulted in resolution of clinical signs in one cat with stenosis considered consistent with a stricture or choanal atresia [12]. In the present case, only minimal luminal enlargement was achieved following seven inflations with a 10 mm balloon catheter. Although chronic osseous remodelling may have contributed to reduced tissue compliance, the limited response cannot be attributed exclusively to intrinsic lesion rigidity. In particular, the relatively low recorded inflation pressure, the absence of confirmation that the balloon reached its nominal diameter, and the lack of escalation to a larger balloon represent relevant procedural limitations. Therefore, the possibility that incomplete balloon expansion contributed to the limited therapeutic response cannot be excluded. These findings do not establish that balloon dilation is ineffective for all chronic traumatic stenoses. In a previous feline case, a 10 mm balloon achieved only slight enlarge-

ment before successful dilation with a 15 mm balloon [8], and two cats in six-cat series required immediate escalation from 10 to 15 mm balloons [10]. Furthermore, treatment in the present cat was limited to a single anaesthetic episode. Planned serial procedures have been associated with improved recurrence-free outcomes in cats [6], and dogs with acute traumatic stenosis underwent three separate dilations using a 12 mm balloon [7]. In the latter cases, treatment was initiated 9 and 18 days after injury, respectively, before mature fracture remodelling would be expected, whereas the present cat was treated approximately 1 year after trauma. Chronic osseous remodelling may have contributed to the mechanical resistance encountered during balloon dilation; however, its specific role in the limited treatment response could not be established without histopathological confirmation. A concomitant fibrous component cannot be excluded, and lesion chronicity, balloon diameter and the absence of serial dilation procedures represent additional potential contributing factors. Alternative therapeutic approaches have yielded favourable outcomes, although complications have been reported. Temporary silicone stenting following dilation resulted in complete resolution in 14/15 cats and improvement in one [13]. Balloon-expandable metallic stents resulted in an immediate resolution of clinical signs in six animals, with normal breathing reported in 5/6 at 12–28 months [14]. In a larger series, the overall treatment success rate was 78%; however, balloon dilation alone was successful in 41% of patients, and stent-related complications occurred in 68% [15]. In another cat, placement of a permanent braided-wire stent was associated with obstructive granulation tissue and persistent intermittent nasal congestion [16]. Reported surgical approaches include mucosal advancement following membrane excision, with resolution maintained for 28 months in one cat [17], and extended palatoplasty, which improved all six cats in one series [18] and was also successfully used to treat an imperforate nasopharynx in another cat [19]. However, these techniques were used for soft-tissue or imperforate lesions, and their applicability to post-traumatic stenosis associated with remodelled bone remains uncertain. Congenital abnormalities are an important differential diagnosis for osseous nasopharyngeal obstruction. Reported feline conditions include stenosis associated with soft-palate dysgenesis [20], bilateral bony choanal atresia [21], and palatal dysgenesis resulting in a blind-ending nasopharynx [22]. In these cases, clinical signs developed during kittenhood and the anatomical abnormalities were centred on congenital palatal or choanal malformations. In contrast, the onset of respiratory signs in adulthood immediately following major trauma, together with the presence of extensive healed fractures, makes a congenital origin unlikely in the present cat. Several limitations should be acknowledged. Contemporaneous clinical records and diagnostic imaging from the time of the accident were unavailable, precluding direct confirmation of the relationship between the reported trauma and the subsequent anatomical changes. Histopathological examination was not available; therefore, the contribution of osseous remodelling to the stenosis was inferred from CT and endoscopic findings rather than confirmed histologically. No quantitative postprocedural luminal measurements were obtained, and follow-up CT or endoscopic examinations were not performed, precluding objective assessment of luminal changes over time. The negative bacterial culture does not exclude previously suppressed or localised infection. The marked disruption of the nasal architecture, including partial collapse of the ethmoidal cavities and loss of the right turbinates, may have contributed to the mucopurulent nasal discharge independently of the nasopharyngeal stenosis. Furthermore, prednisolone administration following balloon dilation may have influenced the transient improvement in sneezing, limiting the attribution of changes in nasal signs to the endoscopic intervention alone. However, stertor remained unchanged following the procedure and throughout medical treatment. Follow-up was limited to 1 month, and the owner's decision to decline further reassessment precluded evaluation of serial dilation or alternative thera-

peutic approaches. The extensive disruption of the nasal architecture and loss of turbinate structures, together with the predominantly neutrophilic inflammation, raise the possibility of concurrent chronic destructive rhinitis. Although these findings may be associated with the previously reported trauma and chronic anatomical abnormalities, an independent inflammatory or infectious process cannot be definitively excluded. Furthermore, the free osseous fragment identified during rhinoscopy may have acted as a local inflammatory stimulus or potentially contributed to secondary bacterial colonisation. The persistence of mucopurulent nasal discharge after its removal suggests that additional factors, including chronic mucosal inflammation and altered nasal anatomy, may have contributed to the clinical signs. However, the relative contribution of these mechanisms remains uncertain.

4. Conclusions

This case describes an unusual presentation of acquired nasopharyngeal stenosis associated with chronic displaced skull fractures and extensive osseous remodelling in a cat with a history of owner-reported facial trauma. A single balloon-dilation procedure resulted in only limited luminal enlargement and no clinically meaningful improvement. However, the relative contributions of anatomical remodelling, possible fibrous tissue, and procedural factors to the limited treatment response could not be established. Computed tomography combined with endoscopic examination was valuable for characterising the complex anatomical abnormalities and guiding therapeutic assessment. Although staged balloon dilation, surgical correction, and stent placement have been described for other forms of nasopharyngeal stenosis, their effectiveness in cases associated with chronic osseous remodelling remains uncertain. Further clinical experience is needed to establish appropriate therapeutic strategies for this uncommon presentation.

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Institutional Review Board Statement: The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed Consent Statement: Informed consent (written) was obtained from the owner of the animal described in this work (non-experimental) for all procedures undertaken in prospective or retrospective studies. For any animals or people individually identifiable within this publication, informed consent (written) for their use in the publication was obtained from the people involved.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

NPS Nasopharyngeal stenosis

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