Nasopharyngeal Disease in Cats: 2. Specific conditions and their management
Nicki Reed and Danièle Gunn-Moore
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What is This?
NASOPHARYNGEAL DISEASE IN CATS

2. Specific conditions and their management

Nicki Reed and Danièle Gunn-Moore

Acute rhinitis (cat ‘flu)

A number of different respiratory tract infections may give rise to the clinical signs of acute rhinitis (cat ‘flu). Primary infection may be viral (feline herpesvirus-1 [FHV-1] and calicivirus [FCV]) or bacterial (Chlamydophila felis, Mycoplasma species, Bordetella bronchiseptica). In addition, secondary bacterial infection by, for example, Pasteurella species may complicate viral infections (see Table 1, Part 1). Clinical signs vary with the causal agent, but generally include sneezing, nasal discharge, anorexia, pyrexia, conjunctivitis, ocular discharge, and oral and/or ocular ulceration. The severity of signs is dependent on the pathogenicity of the causal organism, and on the immune response of the cat. Nasal discharge is usually bilateral, and may initially be serous, progressing to mucopurulent in nature (Figure 1). Thick, tenacious nasal discharge may cause upper airway obstruction, resulting in a prolonged inspiratory phase and noticeable upper respiratory noise. The presence of nasal discharge may lead to ulceration of the nares with chronicity.

Identification of a causal agent may help with management.

Antibacterial therapy

Broad spectrum antibacterials are generally prescribed to prevent establishment of bacterial infection secondary to viral damage (Table 1). If Mycoplasma, Chlamydophila or Bordetella species are identified, tetracyclines (in particular doxycycline) are considered the treatment of choice. Good responses are also seen with fluoroquinolones or azithro-

Figure 1 Mucopurulent nasal discharge in a cat with FHV-1 infection
Table 1: Medications used in the management of nasopharyngeal disease in cats

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Indications</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin–clavulanate</td>
<td>12.5–20 mg/kg q8–12h PO, IV, SC</td>
<td>Gram +ve aerobes, Gram –ve aerobes, Obligate anaerobes</td>
<td>Not effective against Mycoplasma; variable efficacy against Pseudomonas and Chlamyphila</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>10 mg/kg q24h PO</td>
<td>Chlamyphila, Mycoplasma, Bordetella</td>
<td>Can cause oesophagitis – give with food or follow with 5 ml water; can cause tooth discoloration in young animals; do not give to pregnant animals</td>
</tr>
<tr>
<td>Marbofloxacin</td>
<td>2 mg/kg q24h PO, IV</td>
<td>Mycoplasma, Chlamyphila, Bordetella, Pseudomonas, Pasteurella, Staphylococcus, Escherichia coli</td>
<td>Give following culture results; cartilage abnormalities in young animals; risk of retinal blindness; do not give to pregnant or lactating animals or those with epilepsy</td>
</tr>
<tr>
<td>Pradofloxacin</td>
<td>3–5 mg/kg q24h PO</td>
<td>Mycoplasma, Chlamyphila, Bordetella, Pseudomonas, Pasteurella, Staphylococcus, Escherichia coli</td>
<td>As for marbofloxacin except that retinal blindness has not been reported. Doses as high as 10 mg/kg have been used without evidence of side effects1</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>5.5–11 mg/kg q12h PO, IV</td>
<td>Gram +ve aerobes, Anaerobes Mycoplasma</td>
<td>Can cause oesophagitis – give with food or follow with 5 ml water</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>10–20 mg/kg q8–12h PO</td>
<td>Gram +ve cocci, Pasteurella, Mycoplasma, (Chlamyphila)</td>
<td>Gastrointestinal upset common side effect Variable efficacy against Chlamyphila</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>5 mg/kg q48h PO</td>
<td>Gram +ve cocci and bacilli, Pasteurella, (Chlamyphila), Mycoplasma, Bordetella</td>
<td>Pseudomonas usually resistant Variable efficacy against Chlamyphila</td>
</tr>
<tr>
<td>Famciclovir</td>
<td>5–20 mg/kg q8h PO, 90 mg/kg q8h PO</td>
<td>FHV-1 – reduction of clinical signs and viral shedding</td>
<td>Optimal dose yet to be decided</td>
</tr>
<tr>
<td>L-lysine</td>
<td>250 mg/kitten q12h PO</td>
<td>FCV</td>
<td>Efficacy not proven; products should not contain propylene glycol as preservative</td>
</tr>
<tr>
<td>Interferon α</td>
<td>1 MU/kg SC q24–48h 50,000–100,000 U PO q24h</td>
<td>FHV-1 – reduction of clinical signs and viral shedding</td>
<td>No controlled studies; can be used alongside L-lysine</td>
</tr>
<tr>
<td>Interferon ω</td>
<td>5–35 U q24h SC – high dose PO – low dose</td>
<td>FCV</td>
<td>No controlled studies; can be used alongside L-lysine; SC administration can lead to antibody formation</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>50 mg/cat q12–24h</td>
<td>Cryptococcus species; mycotic infections involving the central nervous system (CNS)</td>
<td>Several weeks/months of treatment may be required; do not administer to pregnant animals; dose reduction in patients with renal disease. Treat until LCAT (latex cryptococcal antigen agglutination test) titre is zero</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>5–20 mg/kg q24h PO</td>
<td>Cryptococcus species, Aspergillus species</td>
<td>Several weeks/months of treatment may be required; do not administer to pregnant animals; many drug interactions. Monitor liver enzymes – may cause hepatotoxicity</td>
</tr>
<tr>
<td>Voraconazole</td>
<td>10 mg/kg q24h PO</td>
<td>Cryptococcus species</td>
<td>Several weeks/months of treatment may be required; do not administer to pregnant animals; neurological abnormalities reported</td>
</tr>
<tr>
<td>Ketoconazole</td>
<td>5–10 mg/kg q12h PO</td>
<td>Cryptococcus species</td>
<td>Several weeks/months of treatment may be required; do not administer to pregnant animals; neurological abnormalities reported</td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>0.5 mg/kg in 350 ml 0.45% saline and 2.5% dextrose SC one to three times weekly4</td>
<td>Cryptococcus species – including CNS infection; Aspergillus species</td>
<td>Nephrotic – toxicity reduced by use of liposome-encapsulated or lipid-complex formulations. Infuse in saline or DW5, as incompatible with lactated Ringer’s. Monitor renal function</td>
</tr>
<tr>
<td>Fluycytosine</td>
<td>30–75 mg/kg PO q6–12h</td>
<td>Cryptococcus species – including CNS infection; Aspergillus species</td>
<td>Synergistic with amphotericin B; resistance develops if used alone</td>
</tr>
<tr>
<td>Clotrimazole</td>
<td>Topical infusion (~20 ml)</td>
<td>Aspergillus species</td>
<td>Hour long infusion5,6</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>0.1 mg/kg PO q24h</td>
<td>Pain/inflammation associated with chronic rhinitis Adenocarcinoma?</td>
<td>Risk of renal, hepatic and gastrointestinal toxicity</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>0.3 mg/kg PO q48–72h</td>
<td>Adenocarcinoma?</td>
<td>Risk of renal, hepatic and gastrointestinal toxicity</td>
</tr>
<tr>
<td>Ciclosporin</td>
<td>5–10 mg/kg q24h</td>
<td>Allergic rhinitis? Chronic rhinitis?</td>
<td></td>
</tr>
<tr>
<td>Zafirlukast</td>
<td>0.5–1 mg/kg PO q12–24h</td>
<td>Allergic rhinitis? Chronic rhinitis?</td>
<td></td>
</tr>
<tr>
<td>Montelukast</td>
<td>0.25–0.5 mg/kg PO q24h</td>
<td>Allergic rhinitis? Chronic rhinitis?</td>
<td></td>
</tr>
</tbody>
</table>

PO = orally, IV = intravenously, SC = subcutaneously, DW5 = 5% dextrose in water
While every effort has been made to ensure correct doses are given, the authors advise independent verification of doses prescribed
Many drugs listed do not have a veterinary product licence
mycin. However, as these infections often occur in young kittens where the side effects of these antibacterials may be undesirable alternative choices may be needed. A good alternative for the treatment of *Chlamydia* species infection is amoxicillin–clavulanate.

*Mycoplasma* species do not have a cell wall, rendering β-lactam antibiotics ineffective. For the treatment of these infections macrolides such as erythromycin and azithromycin may be sensible alternatives; however, resistance to these drugs is rapidly emerging with human *Mycoplasma pneumoniae* infections. Although not licensed for veterinary use, azithromycin has the advantage that it comes in a liquid formulation, which may be easier to administer and enables accurate dosing, particularly for small kittens. No veterinary studies have been performed to test the efficacy of azithromycin against *Bordetella bronchiseptica*, although human strains of *Bordetella pertussis* are susceptible. Although azithromycin alleviated clinical signs of *Chlamydia* species in one feline study, it did not result in clearance of the infection, and in a second study there was no improvement in respiratory signs in shelter cats treated with azithromycin compared with amoxicillin.

**Antiviral therapy**

If FHV-1 is identified then famciclovir has been demonstrated to reduce clinical disease. The use of L-lysine in cases of FHV-1 is controversial. It may be of benefit in reducing clinical signs and shedding of the virus, but administration of tablets is more beneficial than administering it in food, and as such the stress of pilling has to be weighed up against the benefit. Although feline interferon omega was associated with decreased viral replication in vitro, no significant differences were found when this drug was used in the management of FHV-1 infected cats in vivo. Similarly, there are no published studies demonstrating benefit of interferon (human or feline recombinant) in the management of rhinitis associated with FCV.

**Supportive treatment**

Supportive treatment with good nursing care is required for these patients, which are anorectic and dehydrated. Fluid therapy may be indicated, and syringe feeding or tube feeding may be required as the inability to smell food will affect appetite. Nasal discharges should be removed by bathing – nebulisation with saline may facilitate clearance of nasal discharge. Analgesia may be indicated where sinus pain is present, and some authors recommend decongestants.

**Neoplasia is the most common cause of chronic nasopharyngeal disease in cats, with lymphoma being the most frequently identified tumour.**

**Chronic rhinosinusitis/chronic nasopharyngeal disease**

**Nasal neoplasia**

Neoplasia is the most common cause of nasopharyngeal disease in cats. Lymphoma is the most common tumour affecting the nasal cavity (29–70% of neoplasms), followed by adenocarcinoma (13–15%). Carcinomas (squamous cell, undifferentiated) and sarcomas (fibrosarcoma, osteosarcoma, chondrosarcoma) are less commonly reported, along with various other neoplasms. Stertor, nasal discharge (including epistaxis) and facial deformity are typical presenting signs. Neurological signs, including seizures, may be seen with extension of the disease through the cribiform plate.

**Lymphoma**

Nasal lymphoma typically affects middle-aged to older cats, with mean ages of 8.9 and 11.4 years, and a range of 3–17 years, reported. Male cats may be at increased risk compared with female cats, comprising up to 79% of the study population. Siamese cats are potentially overrepresented, comprising up to 14% of cases, although the reported prevalence was not compared with the reference population. It is, however, in keeping with previous studies, which have suggested that Siamese cats are predisposed to lymphoma in general.

Nasal lymphomas show B cell predominance, with 68–100% of tumours reported as B cell immunotype, although epitheliotropic nasal lymphomas have also been reported. Most (90%) feline nasal lymphomas are
classified as high grade and, although traditionally thought of as being localised to the nasal cavity, one study identified that in 67% of cats in which a post-mortem examination was carried out, extension of the disease was present. Numerous locations, including lymph nodes, intestine, spleen, liver and kidney, are reported, but local extension was also identified in this and another study. Diagnosis is based on histopathology, although cytology from impression smears can be supportive of the diagnosis (Figure 2).

Treatment options comprise radiation therapy or chemotherapy (COP or CHOP protocols). Debulking of the mass (eg, by forced flush) may improve clinical signs where these therapies are declined. The prognosis without treatment or with prednisolone alone is generally poor, with a reported median survival of only 22 days. Chemotherapy with the COP protocol has had variable reported success. Henderson et al described a median survival time of only 98 days, whereas in a study by Teske et al, 75% of cats were still alive at 1 year. The reported response to radiation therapy appears to be better, with a median survival of 40.8 months based on seven cats. The combination of radiation therapy and chemotherapy did not appear to improve the outcome, with a median survival for 19 cats of 955 days. A second, retrospective study also failed to identify significant differences between treatment with chemotherapy, radiation or chemotherapy and radiation, with a median survival time, regardless of treatment modality, of 536 days. This is perhaps surprising in the light of the frequency of systemic involvement previously reported, as radiation therapy alone would not treat disseminated disease.

Cats in which the cribiform plate was destroyed had a poorer prognosis in the study by Sfiligoi et al, but not in another. The presence of anaemia has also been identified as a poor prognostic factor. Dichotomous populations are identified, with cats that fail therapy early and survive less than 6 months, and those that experience remission and go on to have a prolonged survival. Further work is required to determine factors that predict a good prognosis and the best form of therapy.

Non-lymphoid neoplasia
Non-lymphoid neoplasia of the nasal cavity is far less common than lymphoid neoplasia. Epithelial tumours (carcinomas) are more common than non-epithelial tumours (sarcomas). Although a large number of different types of tumour have been reported, the most commonly encountered are adenocarcinoma, undifferentiated carcinoma, squamous cell carcinoma and fibrosarcoma. Adenocarcinomas may be subdivided into four types – acinous, cystic, mucinous and papillary (Figure 3).

Radiotherapy is considered the treatment of choice, with median survival times of 382 days reported. Treatment of adenocarcinoma with piroxicam and chemoembolisation has also been reported. The theoretical benefit of piroxicam is based on the presence of COX-2 expression in tumours. One study to assess the presence of COX-2 in various feline neoplasms did not identify expression in nasal tumours. However, all the nasal tumours in this study were lymphomas; therefore, the expression of COX-2 in epithelial nasal tumours has yet to be investigated.

Chronic rhinosinusitis
The majority of cases of chronic upper respiratory tract disease are defined as chronic post-viral rhinitis/sinusitis. The initial viral infection causes damage to the nasal mucosa, which allows secondary infection with oropharyngeal bacteria, and hence the establishment of chronic osteomyelitis of the turbinate bones. However, FHV-1 and FCV are very prevalent in the general feline population, and latent infection is relatively common; as it may not be possible to detect FHV-1 or FCV at this late stage of disease, the condition should more correctly be termed chronic rhinosinusitis.

Chronic rhinosinusitis is rarely curable so the emphasis is on management to improve the patient’s quality of life (see box on page 321).

Allergic rhinitis
Some cases of chronic nasal discharge may be suspected to be allergic in origin on the basis of an eosinophilic or lymphoplasmacytic infiltrate being identified on nasal biopsy, or concurrent asthma-like signs. However, as the underlying allergic trigger is rarely identified, as with chronic rhinosinusitis, therapeutic strategies aim for management rather than cure. Glucocorticoids may be indicated if allergic rhinitis or lymphoplasmacytic rhinitis is present. Inhaled forms may be preferable to systemic administration, due to the reduced risk of systemic side effects. Topical steroid nasal drops are rarely well tolerated by cats. Ciclosporin or antileukotriene medications (zafirlukast, montelukast) may be considered if the systemic side effects of corticosteroids...
Nasopharyngeal disease: causes and management

Fungal rhinitis

Fungal rhinitis in cats is primarily attributed to cryptococcal (Cryptococcus neoformans and Cryptococcus gattii) infection. Cryptococcosis is seen worldwide; however, it is rare in the UK, and only comprised 4% of cases in a study conducted in the USA. Other causes of fungal rhinitis include Aspergillus species and Penicillium species, with occasional case reports of hyalohyphomycosis (Scedosporium apiospermum or Fusarium species infection), trichosporonosis (Trichosporon loubieri infection), and Metarhizium anisopliae and Alternaria species infections.

Clinical signs typically consist of nasal discharge (± epistaxis), sneezing, stertorous respiration and/or facial swelling. Turbinate lysis is frequently identified on computed tomography. The pathophysiology, diagnosis and treatment of feline cryptococcosis have been reviewed recently. A number of drugs have been employed in the treatment of cryptococcosis (Table 1), but fluconazole is an appropriate first choice due to minimal side effects and good penetration into the brain in cases with local extension. Treatment efficacy may be assessed by monitoring serum titres for cryptococcal capsular antigen until the titre reaches zero. Treatment of other causes of fungal rhinitis has comprised intranasal clotrimazole infusion, oral itraconazole, surgical debridement with topical (enilconazole) and systemic (itraconazole) therapy and oral voriconazole.

Fungal rhinitis warrants a guarded prognosis, particularly if there is local extension of the disease into the CNS. These cases may benefit from combination fluconazole and subcutaneous amphotericin B therapy. While 60% of cases of cryptococcosis in one study appeared to be cured, some cases that initially responded to therapy subsequently relapsed. Owners, therefore, need to be warned of the potential for recurrence of clinical disease (Figure 4).
Nasopharyngeal polyps
Nasopharyngeal polyps are benign, inflammatory growths arising from the lining of the middle ear or the Eustachian tube. They are comprised of fibrovascular connective tissue covered by stratified squamous or ciliated columnar epithelium. The polyps may extend into the pharyngeal area (nasopharyngeal polyps) and typically be accompanied by respiratory signs, or through the tympanic membrane into the external ear canal (aural polyps) and typically be accompanied by signs of otitis externa. Nasopharyngeal polyps usually occur in young cats, although an age range of 3 months to 15 years is recorded. In studies of nasopharyngeal disease the frequency of polyps has ranged from 1.3–28%. However, these studies were based on referral populations, which may not reflect the true prevalence of this disease in the general population. The authors’ clinical experience would suggest this condition is rare.

The aetiology of nasopharyngeal polyps is incompletely understood. Their predominance in young cats has led to one theory that they may be a congenital abnormality of the first pharyngeal pouch, from which the Eustachian tube and the middle ear cavity derive. An alternative theory is that they arise as a result of chronic inflammation of the middle ear, either due to respiratory tract infection or to otitis externa attributed to, for example, infestation with Otodectes cynotis. Although some cats have a history of upper respiratory tract infection or ear mite infestation prior to diagnosis, not all cases do. A recent study failed to demonstrate a consistent association between infectious agents (FCV, FHV-1, Mycoplasma species or Chlamydo phila species) and the presence of nasopharyngeal polyps. The presence of inflammation is proposed to affect Eustachian tube function, resulting in inadequate middle ear ventilation and decreased middle ear pressure. The combination of mucociliary dysfunction and hypersecretion of mucus contribute to effusion within the middle ear, which may or may not lead to bacterial contamination and the development of inflammatory granulation tissue.

Clinical signs associated with nasopharyngeal polyps include stertor, nasal discharge and dysphagia. The nasal discharge is usually serous in nature, unless secondary bacterial infection is present. Dysphagia may lead to weight loss or failure to thrive in young kittens. Initial presenting signs may be attributed to respiratory tract infection, although sneezing and coughing are infrequently reported. Altered phonation is occasionally described. The presence of a mass within the middle ear may lead to signs of head shaking or Horner’s syndrome. If the polyp progresses into the inner ear, vestibular signs such as nystagmus, head tilt and ataxia may be present. Signs may have an insidious onset and be present for some time before veterinary attention is sought. Ventral deviation of the soft palate may be noted on intraoral examination (Figure 5a), or a mass lesion may be detected on otoscopic examination. Radiography may demonstrate soft tissue masses within the nasopharynx, dorsal to the soft palate, or opacification of the tympanic bulla (Figure 5b).

Treatment options are discussed in the box on page 323.

Inflammatory polyps of the nasal turbinates
Inflammatory polyps of the nasal turbinates were previously considered to be a rarer manifestation of nasopharyngeal polyps. However, more recent work suggests that these should be considered separately, as they arise from the nasal turbinates, rather than the Eustachian tube. In addition, their histological appearance is consistent with that of mesenchymal nasal hamartoma, rather than being comprised of fibrovascular tissue with a stratified squamous or ciliated columnar epithelium. The condition appears to be more common in Italy, which may relate to genetic
**Treatment options for nasopharyngeal polyps**

**Traction/avulsion**

Traction/avulsion is generally the simplest form of treatment, requiring minimal equipment. With the cat in dorsal recumbency, the polyp is grasped with Allis forceps and traction applied until the polyp detaches at its stalk (Figure 5c). Minor haemorrhage can be expected following this procedure, but it either stops spontaneously or can generally be managed with pressure application to the palate area. Access to the polyp may be facilitated by application of pressure to the soft palate, displacing the mass caudally, or by retracting the palate rostrally with a spey hook or placement of stay sutures. Alternatively, more direct access over the mass may be achieved via a midline incision in the soft palate (avoiding the distal 5 mm), retracting the edges of the incision with stay sutures. The polyp is again removed by traction and the soft palate is repaired with an absorbable suture. The main complications following this procedure include recurrence and development of Horner’s syndrome (Figure 5d). The latter has been reported in 43% of cases treated by traction, with signs usually resolving within 1 month.

Recurrence rates of up to 41% have been reported following removal by traction alone, although recurrence was more frequent with aural polyps than nasopharyngeal polyps. Recurrence is more likely in cases that have radiographic evidence of bulla involvement; that said, 30% of cases with bulla disease were treated successfully by traction alone. Anti-inflammatory doses of prednisolone administered for 4 weeks postoperatively significantly improved outcome. Polyp recurrence has been reported to occur from 19 days to 9 months postoperatively.

**Ventral bulla osteotomy**

Ventral bulla osteotomy has been proposed by some authors to be the treatment of choice due to the high incidence of middle ear involvement. With the cat in dorsal recumbency, access to the ventral bulla is obtained with a Steinmann pin and rongeurs, allowing the contents of the bulla to be sampled and the inflammatory tissue to be removed with a curette (Figure 6). Polyp recurrence rates are considered to be lower than for traction/avulsion, but the technique carries greater risks. Care must be taken not to damage the lingual artery, hypoglossal nerve or tympanic plexus. Postoperative complications include a higher risk of development of Horner’s syndrome (57%) and the potential to develop vestibular disease or facial nerve paralysis.

**Total ear canal ablation/lateral bulla osteotomy/myringotomy/lateral wall resection**

These procedures are not generally required for nasopharyngeal polyps, but may be required for aural polyps. They are expensive and carry a higher risk of morbidity. Surgical textbooks should be consulted or referral to a specialist surgeon considered if these procedures are felt to be warranted.

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**Figure 6** Ventral bulla osteotomy. (a) Removal of inflammatory material from the bulla by use of a curette (white arrow). (b) Appearance of the bulla after removal of inflammatory material, showing the larger ventromedial compartment (stippled arrow) and the smaller dorsolateral compartment (black arrow). Courtesy of A J Tattersall

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**Nasopharyngeal stenosis**

Nasopharyngeal stenosis is a relatively uncommon condition, accounting for 5/77 cases of nasal disease in one study, and 0/75 cases in a second. In this condition, the nasopharynx is occluded by a membrane, which is thought to form secondarily to a process that triggers scar formation (Figure 7). Examples include trauma, infectious diseases or vomiting with aspiration of gastric contents into the nasopharynx. A recent case report described the finding of hiatal hernia and megaoesophagus secondary to the presence of nasopharyngeal stenosis.

The stenotic membrane may be seen radiographically as a thin soft tissue opacity within the nasopharynx, dorsal to the soft palate (Figure 7a,b), which is sometimes accompanied by dorsal deviation of the soft palate at the site of attachment. Retroflex endoscopy
administration of a course of systemic corticosteroids may reduce the risk of recurrence. Alternative options for management of nasopharyngeal stenosis include forceps dilatation, mucosal advancement flap surgery, or placement of a stent. Despite the potential for recurrence, the prognosis is generally good.

Foreign bodies
Foreign bodies are reported to be the third most common cause of nasal disease in cats. Plant material (grass blades, awns, seeds) is most commonly documented; surprisingly this may sometimes be detected radiographically, providing the material does not abut the soft palate (Figure 8). Radiopaque material such as stones, needles and air-gun pellets may also be identified, and a recent report described a nasopharyngeal trichobezoar with the radiographic appearance of a soft tissue mass. Foreign material may be inhaled, or vomited/coughed up into the nasopharynx.

Cats with more rostral foreign bodies will typically present with nasal discharge, whereas foreign bodies in the nasopharynx typically produce retching, gagging and stertorous respiration. Purulent nasal discharge and halitosis may develop, particularly with chronicity. Removal usually necessitates visualising the foreign body and grasping it with forceps. Barbs on grass blades or plant awns can make this problematic, and flushing of the nasal passages may assist with removal. Larger foreign bodies may be removed using forced flushing or may be pushed into the pharynx following the insertion of a stiff urinary catheter into the nasopharynx from the nares.

Figure 7 Nasopharyngeal stenosis. (a) Lateral radiograph of a cat showing a membrane of soft tissue dorsal to the soft palate. (b) Highlighted region of nasopharyngeal stenosis. (c) Retroflexed endoscopic appearance of the stenotic nasopharynx. (d) Retroflexed endoscopic appearance of the nasopharynx following balloon dilatation. (e) Balloon catheter with manometer to monitor pressure exerted on the balloon

Figure 8 Nasopharyngeal foreign body. A radiopaque line can be seen dorsal and parallel to the soft palate. (inset) The foreign body was a blade of grass

KEY POINTS

- Chronic rhinosinusitis and neoplasia are the two most common causes of nasopharyngeal disease in cats.
- The underlying aetiology in cats with chronic rhinosinusitis is rarely identified, but the condition is speculated to be associated with previous viral damage.
- Chronic rhinosinusitis can be frustrating to manage.
- Neoplastic disease is more frequently identified in middle-aged to older cats, and carries a varied prognosis.
- Foreign bodies, nasopharyngeal polyps and nasopharyngeal stenosis can be rewarding to treat, with a cure effected in the majority of cases.
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References


24 Teske E, van Straten G, van Noort R and Rutteman GR. Chemotherapy with cyclophosphamide, vincristine and prednisolone (COP) in cats with...