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A retrospective study of non-specific rhinitis in 22 cats and the value of nasal cytology and histopathology

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Case records from 40 cats subjected to rhinoscopic examination for investigation of chronic nasal disease were reviewed. Cases in which no specific underlying cause (eg neoplasia) was detected were further selected for detailed retrospective study. In these 22 cats (55% of the initial population), a final diagnosis of non-specific chronic nasal disease was made. The radiographic, rhinoscopic, cytological and histopathological findings were reviewed.

Mucosal biopsy specimens were obtained in 20 cases. Despite clinical signs of more than 4 weeks duration, histopathology indicated acute inflammation in four cases. Two cases had chronic lymphoplasmacytic inflammation and 14 had mixed (lymphoplasmacytic and neutrophilic) inflammation. Specimens for cytology were obtained from 17 cases by brush sampling. Three of these samples were not diagnostic due to the poor quality of the slides; one showed normal cytology. Acute inflammation was diagnosed by cytology (n=11) more commonly than chronic (n=1) or mixed inflammation (n=1). Concurrent samples, of quality suitable for both histopathological and cytological interpretation, were collected from 12 cases only. Cytological results were in agreement with the histological results in 25% of these cases, the main discrepancy being the nature of the dominant inflammatory cell type. Therefore cytology does not appear to be a reliable means for detection of chronic inflammation.

Further studies are needed in order to investigate the correlation between the nature of mucosal inflammation as defined by both histological and cytological evaluation, and the relationship of these test results to prognosis and therapy.

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Introduction

Feline chronic rhinitis is a diagnostic and therapeutic challenge. History and clinical signs include nasal discharge, sneezing and stertorous breathing. Despite a complete diagnostic investigation, aetiology is not often defined (Hawkins 1988, Cape 1992, Van Pelt and Lappin 1994, Allen et al 1999). Treatment is rarely curative, particularly in cats with chronic, non-specific disease (Hawkins 1988, Van Pelt and Lappin 1994). Moreover, treatment regimes are often empirical and are generally not based on the cytological and histopathological assessment of the lesions.

Determination of the type and severity of mucosal inflammation in non-specific chronic nasal disease would help to better characterise the condition, and could also be used to monitor the progression of the disease or the response to therapy in prospective studies. The aim of the present study was therefore to review the clinical, radiographic and rhinoscopic findings in a series of cats with chronic rhinitis for which no underlying cause could be found and to analyse the cytological and histological results in these cases. The agreement between cytology and histopathology was also assessed.

Materials and methods

Medical records of cats subjected to rhinoscopic evaluation between January 1997 and June 1999 at

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the Department of Small Animal Clinical Sciences of the University of Liège, were reviewed. Cats with clinical signs compatible with a nasal and/or frontal sinus disease of a minimum 4 week duration were initially selected, and a subgroup of these cats was further selected as having non-specific disease when no underlying aetiology was identified by the diagnostic investigations described below.

In each case, historical information including breed, age, sex, bodyweight, lifestyle (indoor/outdoor) and duration of illness was reviewed. Clinical examination of these cats revealed a range of signs including fever, lethargy, anorexia, nasal discharge (uni- or bilateral; serous, mucopurulent or sanguinous), sneezing, stridor, dyspnoea, cough, ocular discharge (uni- or bilateral), nasal airflow changes (decreased or increased, as measured by placing a thin strand of cotton wool in front of each nostril), changes in the planum nasale (hyperkeratosis, depigmentation, ulceration), facial or planum nasale deformation and neurological abnormalities.

Complete blood count was performed in 17 cats and a serum biochemistry profile in seven cats. Serology for feline leukaemia and feline immunodeficiency viruses (Speed duo FeLV–FIV, Immunochromatograpy, Bio Veto Test) was performed in 17 cats. Serology for feline infectious peritonitis (Speed PIF, Immunochromatograpy, Bio Veto Test or Immunocomb PIF, Immunoassay, Biogal Galed) was tested in six cats. Feline calicivirus (FCV) and herpesvirus (FHV) status was not assessed, and testing for *Chlamydophilia felis* (speed Chlam, Immunochromatograpy, Bio Veto Test) was performed in four cases.

Rhinoscopy was performed by a single investigator (CC). For anterior rhinoscopy, a rigid rhinoscope (Storz K., 64018A optique hopkins 0°-67065CC gain) was used. Posterior rhinoscopy was performed using a videoendoscope with an outer diameter of 4.8 mm (Fujinon EB-4105, Onys s.a., Brussels, Belgium). All cats were anaesthetised after premedication with acepromazine (Combistress® 0.03 mg/kg, IM) and methadone (Mephenon® 0.5 mg/kg, IM). Anaesthesia was induced and maintained with intravenous administration of propofol (Diprivan® up to 6 mg/kg) and gas inhalation; all cats were intubated and a sterile sponge was placed behind the soft palate. Skull (nasal cavity and frontal sinus, ventro-dorsal open mouth view, lateral and rostral–caudal views) and chest radiographs were performed in eight cases each, prior to rhinoscopy. Rhinoscopy was performed with the cat positioned in sternal recumbency. The oral cavity, nasopharynx and larynx were thoroughly examined, then retrograde and direct rhinoscopy were performed, after gentle aspiration of mucopurulent discharge.

When present, intranasal mucopurulent material was collected from deep inside the nasal cavity, using sterile swabs for aerobic bacterial and fungal culture. Antibiotic sensitivity testing was performed routinely on each aerobic isolate.

Specimens for brush cytology were obtained during direct rhinoscopy by use of an endoscopic brush (TW IV/1a-2.0 mm). The brush was introduced either through the endoscope protector sheath or introduced alone under rhinoscopic vision and was rubbed vigorously over the site of the lesion. Areas were sampled when the tissue appeared to be macroscopically abnormal. Collected material was then gently smeared over glass slides that were air-dried and stained with May–Grünwald–Giemsa and haematoxylin and eosin. The slides were retrospectively and independently reviewed by two investigators (MJD and PH). Normal cytology was diagnosed when smears were lightly cellular and contained little mucus. A normal cellular population consisted of a mixed population of a few erythrocytes (sample collection artefact), occasional leukocytes (including neutrophils, lymphocytes, mast cells or eosinophils), occasionally, squamous epithelial cells (keratinised and non-keratinised) and occasionally a mixed bacterial population. Acute rhinitis was diagnosed when there were large numbers of neutrophils, or neutrophils and macrophages. Chronic rhinitis was diagnosed when there were large numbers of mononuclear cells, chiefly lymphocytes and plasma cells. Mixed inflammation had neutrophilic, histiocytic and lymphoplasmacytic components. When smears were of insufficient quality to allow a correct evaluation, the sample was not interpreted (non-diagnostic sample).

Specimens for histopathology were obtained by use of perendoscopic biopsy forceps (MTW 061402111 or BM-BFN 23/180) or by using the 'core technique' as described by Withrow (1985). Biopsies were taken in areas where the tissue was macroscopically abnormal. When the tissue was the same throughout both nasal cavities, one to three biopsies were taken, depending on the size of the biopsies. When several areas with abnormal tissue were present, a biopsy was taken from each abnormal area. If there was one cavity without gross abnormality, no biopsy was taken from

this side. All biopsy specimens were fixed in 10% neutral-buffered formalin, processed and embedded in paraffin wax. Sections were stained with haematoxylin and eosin and retrospectively reviewed by one investigator (MJD). Tissue biopsies were characterised as normal, acute inflammatory, chronic inflammatory or mixed inflammatory using the same criteria as described for cytology.

The final diagnosis in each case was based on the review of history, physical examination, radiography, rhinoscopic findings, microbiological analysis of the nasal discharge and cytological and histopathological findings. Cats in which rhinitis was associated with a specific underlying cause were excluded from the study. The diagnosis of neoplasia was always supported by the presence of a mass and/or abnormal tissue on rhinoscopy. Moreover, cytological samples were not considered to be diagnostic of neoplasia without histological confirmation. When no underlying cause was detected, a diagnosis of non-specific chronic nasal disease was made. The subpopulation of cats with non-specific chronic nasal disease formed the basis of the present study.

Results

Forty cats with clinical signs of chronic upper respiratory disease, that underwent rhinoscopy, were initially considered. Fifteen of these cats were excluded as underlying causes were identified. Specific causes included congenital abnormalities (soft palate defect, two cases), oronasal fistula (one case), foreign body (one case), intranasal mycosis (one case), nasopharyngeal polyp (one case), nasopharyngeal stenosis (one case) and neoplasia of nose and frontal sinus (eight cats including five with lymphoma). Three more cats were excluded because there were discrepancies between the recorded history, clinical findings, rhinoscopic findings and cytological and histopathological findings. Twenty-two cats with non-specific chronic nasal disease were selected for further detailed retrospective study.

These 22 cats were aged 6 months to 12 years (mean±SD=4.3±4.1 years) when referred. There were six females (four neutered) and 17 males (seven neutered). Breeds represented included 11 domestic short hair cats (50%), five Persians, two Siamese, two Chartreux and two Oriental cats. The mean bodyweight±SD was 4.3±1.0 kg. Duration of clinical signs ranged from 4 weeks to 12 years with a mean of 15.6±31.2 months.

The most commonly encountered clinical signs were sneezing (20 cats) and nasal discharge (18 cats; unilateral in four cats and bilateral in 14). Nasal airway flow was increased (uni- or bilaterally) in five cases and decreased in 12 others. Stertor was observed in 14 cats, ocular discharge in eight and depressed mentation was present in four cases.

A complete blood count was available in 17 cases. Abnormalities were recorded in five cats and included anaemia, neutrophilia, eosinopenia, lymphopenia or neutropenia. A serum biochemistry profile was performed on seven cats and abnormal results were seen in three, including elevated total protein, urea, creatinine, and/or liver enzymes. Seventeen cats were tested for infection with FeLV and FIV, only a single cat was seropositive for FeLV. Six cats were tested for infection with feline infectious peritonitis virus (FCoV) and all were negative.

On rhinoscopic examination, almost all cats had oedema and/or congestion of the nasal mucosa. Mucopurulent exudate was present in the nasal cavity of 21 cats (95%), and this was found bilaterally in 17 cases and unilaterally in four cases. Other findings included turbinate destruction in 18 cases (82%), abnormal turbinate architecture in 12 cases (52%) and the presence of abnormal tissue compatible with either neoplastic infiltration or inflammatory tissue in 10 cases (45%).

Nasal cavities radiographic findings performed in eight cats included absence of abnormalities (two cases), increased opacity within the nasal cavity (six cases), loss of trabecular pattern (five cases) and vomer deviation (two cases). Lesions were unilateral in three cases and bilateral in three others. Frontal sinus was radiographically normal in five cases, and showed unilateral increased opacity in two cases and unilateral slight bone destruction in one case.

Thoracic radiographs showed no abnormalities in three cases, bronchial pattern in two cases, bronchoinsterstitial pattern in two cases and cardiomegaly in one case.

Fifteen of 21 aerobic bacterial cultures performed on nasal discharge were positive. More than one type of bacteria was isolated in seven of these 15 cases (47%). Bacteria isolated included *Escherichia coli* (40%), *Pseudomonas* spp (53%), *Streptococcus* spp (20%), *Staphylococcus* spp (13%), *Pasteurella* spp (13%), *Serratia* spp, *Klebsiella* spp and *Proteus* spp. Based on sensitivity testing, the fluoroquinolone antibiotics were most likely to be efficacious, followed by ceftazidime and gentamicin.

Specimens for histopathology were obtained in 20 cases and all were suitable for interpretation (Table 1). Acute, chronic and mixed inflammation were diagnosed by histology in four, two and 14 cases, respectively. Specimens for cytology were obtained in 17 cases by the brush technique. Three samples could not be interpreted due to poor quality of the slides and one showed normal cytology. Acute, chronic and mixed inflammation were diagnosed by cytology in 11, one and one of the remaining cases, respectively. Results for both histology and cytology were available in 12 cases, and in 25% of these cases cytological evaluation was similar to the histopathological diagnosis. Where there was a discrepancy between cytology and histopathology, the nature of the dominant type of inflammatory cell was the main difference.

Discussion

Possible primary conditions associated with chronic nasal discharge in cats include viral infections, bacterial infections, mycotic infections, nasal parasites, neoplasia, congenital defects, dental disease, nasal foreign body, nasopharyngeal polyps, allergic rhinitis and nasal trauma (Van Pelt and Lappin 1994). In the present study, despite a thorough diagnostic investigation, an underlying cause was found in only 15 of 40 cats, including eight cats with intranasal tumours. Twenty-two cats in which no primary cause was identified were selected for further detailed retrospective study.

Clinical signs, radiographic and rhinoscopic findings in the cats of this series were comparable to those commonly described in upper respiratory diseases (Norris and Laing 1985, Hawkins 1988, Venker-van-Haagen et al 1990, O'Brien et al 1996). As expected, results of complete blood count and serum chemistry were not specific. However, these procedures are indicated in order to exclude underlying systemic disease (Hawkins 1988).

Feline herpesvirus-1 (FHV 1) and FCV are known to be the major pathogens of the upper respiratory tract in cats (Ford 1997a). Both viruses may cause damage to the mucosal epithelium and osteolysis of the nasal turbinates (Hawkins 1988, Ford 1997b). Such lesions, more severe in young kittens, may predispose to persistent or recurrent bacterial rhinitis and sinusitis. Although we did not attempt to prove FHV 1 and FCV involvement in our study, infection with these viruses possibly accounts for most of our selected cases. Indeed, in a previous study, feline idiopathic chronic rhinitis was considered likely to be virus induced (Cape 1992). *C felis* is another well known cause of feline upper respiratory tract disorders (Gaskell 1993, Sykes 1999). In the present study, testing for *Chlamydophilia* was performed in four cats with concurrent ocular signs but was not positive in any of these cases.

FeLV or FIV infection is reported to be a common concomitant or predisposing infection in cats infected by a range of micro-organisms. In the present study, all cats tested for FIV infection were negative, and only one was positive for FeLV. This low prevalence correlates well with data presented by Cape (1992) and Norsworthy (1993) on the association between retroviral infection and chronic nasal disease.

Bacteria are traditionally considered as secondary invaders to viral infection. The normal microbial flora of the feline upper respiratory tract includes many potential pathogens (Ford 1993). This flora may become responsible for clinical signs such as sneezing or nasal discharge, in the presence of a coexisting pathological process, which decreases the respiratory defence mechanism (Ford 1997b). Another class of secondary upper respiratory pathogen would be the mycoplasmas, but their precise role in feline chronic rhinitis is poorly defined (Sykes 2001). The presence of these agents was not examined in the cats of this series.

Bordetella bronchiseptica has been reported to act as a primary pathogen in cats with upper respiratory tract disease, particularly in young kittens and in cats living in stressful environments (Speakman et al 1999). *B bronchiseptica* was not identified in our study, however, it is more difficult to isolate this species in comparison with other commensal oronasal flora (Speakman et al 1999).

Rhinoscopy allows access to the nasal cavity for examination, diagnostic sample collection and in some cases, therapeutic procedures, therefore, rhinoscopy is one of the most useful diagnostic procedures for evaluation of a patient with chronic nasal disease (McCarthy and McDermaid 1990). Direct exploration of the oral cavity, oropharynx and nasopharynx during the procedure is mandatory since it permits detection of a primary cause. In the present study the primary cause was detected in 15% of the cases. In a study in dogs, rhinoscopic examination detected a primary cause in 8% (Forbes Lent and Hawkins 1992) of the cases. Retrograde rhinoscopy allows visualisation of the nasopharyngeal area, up to

Table 1. Rhinoscopic, cytological and histopathological findings in 22 cats with rhinitis

Breed	Sex	Weight (Kg)	Age (years)	Durations of symptoms (months)	Rhinoscopy findings	Cytology results	Histology results
Persian	M	3.5	0.5	2	Turbinate destruction (B)	Acute rhinitis	Acute rhinitis
European	FS	4.5	11	12	Abnormal turbinate architecture (B), abnormal tissue (U)	Normal epithelium=NC	Acute rhinitis
European	M	3.6	0.75	5	Turbinate destruction (U)	Acute rhinitis	Acute rhinitis
Persian	M	2.2	2	2.5	Turbinate destruction (B)	ND	Acute rhinitis
Persian	M	5.7	12	144	Turbinate destruction (B), abnormal tissue (B), abnormal turbinate architecture (U)	Acute rhinitis	Chronic rhinitis
European	M	2	0.5	2	Turbinate destruction (B), abnormal turbinate architecture (U)	Acute rhinitis	ND
Chartreux	MC	4.5	4	12	Turbinate destruction (U), abnormal tissue (U)	ND	Chronic rhinitis
European	F	4	10	12	Abnormal tissue (U), abnormal turbinate architecture (U)	Chronic rhinitis	ND
Siamese	M	2.8	0.66	2	Turbinate destruction (U), abnormal tissue (B), abnormal turbinate architecture (U)	Acute rhinitis	Mixed rhinitis
Persian	M	4	1	10	Turbinate destruction (B), abnormal tissue (B), abnormal turbinate architecture (B)	Acute rhinitis	Mixed rhinitis
Persian	FS	2	1.75	3	Turbinate destruction (B), abnormal turbinate architecture (U)	ND	Mixed rhinitis
Chartreux	M	2.4	0.6	1	Abnormal turbinate architecture (U)	Acute rhinitis	Mixed rhinitis
European	F	3.4	2	NA	Turbinate destruction (B), abnormal tissue (B), abnormal turbinate architecture (B)	Acute rhinitis	Mixed rhinitis
European	FS	3.8	8	1.5	Turbinate destruction (U), abnormal turbinate architecture (U)	ND	Mixed rhinitis
European	MC	4.4	1.5	18	Turbinate destruction (B), abnormal tissue (B), abnormal turbinate architecture (B)	Acute rhinitis	Mixed rhinitis
European	MC	2.8	10	9	Purulent material (B), turbinate destruction (B), abnormal tissue (U)	Acute rhinitis	Mixed rhinitis
European	FS	3.9	3	12	Turbinate destruction (U), abnormal tissue (U)	NC	Mixed rhinitis
Oriental	M	3.2	1	4	Turbinate destruction (B)	Mixed rhinitis	Mixed rhinitis
Oriental	MC	4.5	1	7	Turbinate destruction (U)	NC	Mixed rhinitis
European	MC	4.2	9	2	Abnormal turbinate architecture (U)	Acute rhinitis	Mixed rhinitis
Siamese	MC	NA	8	18	Turbinate destruction (U)	ND	Mixed rhinitis
European	MC	4	7	48	Turbinate destruction (B)	NC	Mixed rhinitis
Mean		3.6	4.3	15.6			

NC, non-diagnostic sample; ND, non-determined; B, bilateral; U, unilateral.

the level of the choanae and is required to detect choanal atresia or stenosis, nasopharyngeal stenosis, nasopharyngeal foreign body or polyp, which are all possible primary causes of chronic rhinitis. Frontal sinus involvement was detected by skull radiography in x cases because rhinoscopy was unable to visualise this area.

In the present study, all tissue specimens were of suitable quality for histopathological interpretation to be made. In a previous study in dogs (Forbes Lent and Hawkins 1992), 23% of tissue specimens, obtained by use of a perendoscopic biopsy forceps, could not be assessed. By contrast, the proportion of diagnostic cytological samples was lower (13 of 17 cases, or 76%). The cytological diagnosis of nasal inflammation is always made in recognition of the fact that the sample obtained is derived from the superficial mucosa, and does not rule out the possibility of deeper underlying pathology such as neoplasia or mycotic infection. Also it cannot be excluded that a false diagnosis of chronic rhinitis has possibly been made in neoplastic or fungal diseases because biopsy sampling missed the specific lesions.

In the 22 cats with non-specific chronic nasal disease selected in the present study, acute rhinitis was the predominant finding on histopathology in four cases. In two further cases, histopathology identified lymphoplasmacytic infiltration typical of chronic rhinitis. In the remaining 14 cases, concurrent lymphoplasmacytic and neutrophilic infiltrations were seen. The four cases defined histopathologically as having acute rhinitis are of note, as in each of these cats the clinical disease had been present for several weeks. This finding might simply reflect the random choice of site for mucosal biopsies, but could also indicate that even in cats which exhibit clinical signs for more than 4 weeks, chronic histopathological modifications of the nasal mucosa are not yet present. Another explanation would be that the presence of a deep mucosal chronic infiltrate could be hidden by a superficial neutrophilic inflammatory process (Caniatti et al 1998). Further studies should determine whether such cases of acute inflammation are more responsive to treatment than others.

Cytology was in agreement with histology in only 25% of the cases (three of 12) where concurrent diagnostic samples were collected. Cytology reflected an acute inflammatory process in 11 of 14 cases. As already mentioned, this can be explained by the fact that specimens for cytological evaluation are more superficial in nature than samples of tissue collected for histopathological evaluation. In a previous study on feline chronic idiopathic rhinosinusitis, suppurative inflammation was the invariable cytologic finding (Cape 1992). As a consequence, cytology does not appear to be a reliable means of detecting chronic changes. Previous studies have compared cytology with histopathology as a diagnostic methodology in nasal diseases, principally in tumours (Clercx et al 1996, Caniatti et al 1998). In one study in cats, six of 36 brush samples classified by cytology as inflammatory were subsequently diagnosed as neoplastic by histological examination (Caniatti et al 1998). In the same study, one of 17 brush samples classified by cytology as neoplastic was subsequently diagnosed as inflammatory by histological examination (Caniatti et al 1998). In a study in 54 dogs with nasal tumours diagnosed on histopathology, neoplasia was diagnosed more frequently by imprint cytology (81% of the cases) than by brush cytology (56% of the cases) (Clercx et al 1996). A suggestion to improve the agreement between cytology and histopathology in cats with non-specific chronic nasal disease would be to use imprint cytology rather than brush cytology. However, volume and number of biopsies taken do not always allow the preparation of impression smears of good quality. It should also be emphasised that such superficial specimens probably affect the results of bacterial culture and sensitivity. Therefore, mucosal biopsies rather than superficial specimens should ideally be obtained for this purpose, whenever possible.

The demonstration of an acute inflammatory process, either by cytology or histopathology could incite the clinician to use an antibacterial drug in the immediate treatment regimen administered to the patient. By contrast, the diagnosis of chronic lymphoplasmacytic infiltration on histopathology could explain a failure of single antibiotic therapy, and could be a factor justifying the use of glucocorticoids.

Consequently, there is a need for further prospective studies with predefined treatment regimens. In such studies, a complete examination including cytological and histopathological assessment will be needed and should be regularly repeated to assess any potential benefit of specific intranasal or aerosol-delivered therapies.

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