Atrophic Rhinitis: A Review of 242 Cases

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ABSTRACT

Atrophic rhinitis is a debilitating nasal mucosal disease of unknown etiology. It is characterized by progressive nasal mucosal atrophy, nasal crusting, fetor, and enlargement of the nasal space with paradoxical nasal congestion. Primary atrophic rhinitis has decreased markedly in incidence in the last century. This probably relates to the increased use of antibiotics for chronic nasal infection. Secondary atrophic rhinitis resulting from trauma, surgery, granulomatous diseases, infection, and radiation exposure accounts for the majority of cases encountered by the rhinologist today. Excessive turbinate surgery has been both acquitted and accused in the literature as an etiology for secondary atrophic rhinitis. We saw 242 patients with the diagnosis of atrophic rhinitis between 1982 and 1999. The diagnosis was confirmed by physical examination, biopsy, and imaging studies. Patients were diagnosed with primary atrophic rhinitis if their condition developed in a previously healthy nose and secondary atrophic rhinitis if their condition developed after sinonasal surgery, trauma, or chronic granulomatous disease. Prevention and treatment of the disease is discussed. (American Journal of Rhinology 15, 355–361, 2001)

Atrophic rhinitis is a chronic debilitating disease of the nasal passages that is characterized by progressive nasal mucosal atrophy, nasal crusting, fetor, and enlargement of the nasal space with paradoxical nasal congestion. Patients with atrophic rhinitis also may complain of a disordered sense of smell, although they do not usually suffer complete anosmia until late in the disease process. Reports of the disease date back to ancient times, and historical remedies included intranasal installation of date wine, mother's milk, and passage of a red hot iron into the nose.[1] Much confusion has existed in the literature regarding the diagnosis, etiologic factors, and treatment of atrophic rhinitis. Terminologies of atrophic rhinitis, rhinitis sicca, and ozena have been used interchangeably in the literature. This confusion has made investigation of the causes and treatment of the condition difficult. Rhinitis sicca or "dry rhinitis" is a distinct disorder of uncertain etiology characterized by drying of the nose and hypertrophy, rather than atrophy, of the nasal mucosa.[2] Crusting may result, but overwhelming infection is rare in this condition.

Ozena, a Greek term denoting stench, often is used interchangeably in the literature with "atrophic rhinitis" to describe chronic nasal disease. More appropriately, ozena is a chronic nasal condition characterized by progressive atrophy of the nasal mucosa and underlying bone of the conchae, as opposed to atrophic rhinitis, which rarely involves the submucosal tissues until late in the disease process.[2,3] Dr. Francke Bosworth, in his 1881 text *A Manual of Diseases of the Nose and Throat*, stated that "the breath is often so penetrating as to render the near presence of the sufferer not only unpleasant but almost unendurable."[1] Interestingly, patients with ozena typically suffer anosmia secondary to the disease process affecting their olfactory nerve endings, so they usually do not detect the offensive odor that is so obvious to others around them.[4] The etiology of ozena is unknown, but various authors have proposed bacterial infection with *Klebsiella ozaenae* and *Bacillus foetidus*, chronic sinusitis, endocrine factors, inherited disorders, and nutritional deficiencies as possible factors.[2,5] The incidence of ozena has decreased markedly in the Western world with the increased use of antibiotics, but reports of the disease occasionally surface from China, Egypt,[6] India,[7] and even rarely the United States.[5]

Since the middle of this century, various authors have divided atrophic rhinitis into two separate entities: primary

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atrophic rhinitis of spontaneous onset, slow progression, and unspecified etiology and secondary atrophic rhinitis developing after chronic rhinosinusitis, chronic granulomatous disease, reductive nasal surgery, trauma, or irradiation.[8,9] Primary atrophic rhinitis actually may represent early ozena before the submucosal destructive processes brought on by inheritable or infectious causes have progressed to their end state.[8] Secondary atrophic rhinitis is much more commonly encountered, although it is no more completely understood. Characteristic findings in both forms include nasal crusting, enlarged nasal cavities, resorption of the turbinates, mucosal atrophy, and paradoxical nasal congestion.[3,4] The distinction in the two diseases is the etiology. Frequent attention has been given to secondary atrophic rhinitis in this country because of the numerous debates that have addressed the association between atrophic rhinitis and modified or total reductive turbinate surgery.

We diagnosed and treated 242 patients with atrophic rhinitis between 1982 and 1999. Patients were diagnosed by physical examination with nasal endoscopy, and some diagnoses were corroborated further with biopsy, culture, and imaging studies. The patients were divided into primary atrophic rhinitis and secondary atrophic rhinitis. Symptomatology, etiologic factors, and treatment algorithms for the groups are discussed.

MATERIALS AND METHODS

The charts of all patients who presented to us between 1982 and 1999 with sinonasal complaints attributed to atrophic rhinitis were reviewed retrospectively. Two hundred forty-eight patients were identified. Six patients were not completely examined and diagnosed by an otorhinolaryngologist, so they were excluded from the study. The diagnosis of atrophic rhinitis was made by complete history (including past medical/surgical history, social history, and medications and allergies) and complete head and neck examination with nasal endoscopy. The presence of nasal congestion, nasal crusting, fetor, apparent enlargement of the nasal space, and turbinate resorption were noted. Nasal biopsies with histological examination or culture were used to confirm the diagnosis in 214 patients, and the diagnosis was made by history and physical exam alone in the remaining 28 patients. Computerized tomographic (CT) scanning of the nose and paranasal sinuses was completed in 194 patients, and rhinomanometry was performed in 135 patients. Then, the 242 patients were divided into primary and secondary atrophic rhinitis groups. To be diagnosed with primary atrophic rhinitis, the patient's symptoms must have started without any antecedent nasal trauma, sinonasal surgery, chronic granulomatous disease, or radiation therapy to the sinonasal area. The patients were evaluated for the symptoms of nasal crusting, fetor, epistaxis, facial pain, nasal congestion, anosmia, headache, and depression.

RESULTS

We authors diagnosed 242 patients with atrophic rhinitis between 1984 and 1999. The group consisted of 138 women and 104 men. The average age of the patients was 54 years, with a range of 12–89 years (Table I). All patients complained of bilateral nasal congestion, and all patients complained of daily nasal crusting and dryness. Facial pain and pressure was a complaint of 115 patients (48%), and 80 (33%) of the patients complained of intermittent epistaxis. Anosmia was present in 35 patients (16%), and 125 (52%) of the patients were diagnosed with depression by the Minnesota Multiphasic Personality Inventory and/or consultation with a psychiatrist (Table II).

On physical examination, every patient was found to have abnormal anatomy of the nasal sidewall. Inferior turbinate tissue was partially absent in 152 (62%) of the patients and totally absent in 90 (37%) of the patients. Absence of the middle turbinate was found in 137 (57%) of the patients. Seventy-eight (32%) of the patients were found to have either no recognizable turbinate tissue or only very small remnants of turbinate tissue remaining. Nasal septal perforation was found in 24 (10%) of the patients. Yellow, brown, or green crusts were bound covering the sidewalls and floor of the nose in all of the patients, and 125 (52%) patients had mucopurulent drainage from the maxillary and ethmoid sinuses.

Nasal biopsy was used to confirm the diagnosis in 194 (80%) patients. Findings consisted of squamous metaplasia with loss of the normal pseudostratified columnar epithelium, serous and mucous glandular atrophy, and diffuse endarteritis obliterans. One hundred seven (44%) patients had positive nasal cultures with the presence of pathogenic organisms. *K. ozaenae* was isolated in 48 cultures. Other common pathogens included *Staphylococcus aureus*, *Proteus mirabilis*, and *Escherichia coli.* Computerized tomographic studies of the nose and paranasal sinuses were performed on 158 (65%) patients. The predominant findings on tomographic scan were mucosal thickening of the paranasal sinus lining and enlargement of the intranasal space with bowing of the nasal walls. Resorption or destruction of the ethmoid sinus cavities was seen on all scans and hypoplasia of the maxillary sinuses was seen on 126 scans. Bony resorption or destruction of the inferior and middle turbinates with atrophic thinning of the mucosa over the turbinate remnants was seen on all scans. Rhinomanometry was performed on 135 patients, and the results were con-

TABLE I

Study Group Atrophic Rhinitis Patients

	Number	Men	Women	Age (Range)
Total	242	104	138	54 years (12–89 years)
Primary	45	20	25	52 years (19–89 years)
Secondary	197	84	113	56 years (12–66 years)

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TABLE II

Symptoms of Atrophic Rhinitis Patients

Symptom	Number of Patients
Congestion	242 (100%)
Crusting	242 (100%)
Facial pain	115 (48%)
Epistaxis	80 (33%)
Depression	125 (52%)
Anosmia	37 (15%)
Sinusitis	125 (52%)

sistent with the paradox of subjective nasal obstruction in these patients with widely patent nasal cavities. The mean total nasal resistance in these patients was 0.12 Pa/cm^3 per second (SD = ± 0.03 Pa/cm^3 per second). Normal total nasal resistance in healthy subjects is on the order of 0.15–0.30 Pa/cm^3 per second.[10]

The patients were subdivided into two groups: those who had a history of prior nasal destructive processes (secondary atrophic rhinitis) and those with no prior history of sinonasal trauma, surgery, radiation, or chronic granulomatous disease (primary atrophic rhinitis). The secondary atrophic rhinitis group consisted of 197 (81%) patients, 113 women and 84 men. One hundred seventy-six of the 197 patients in the secondary atrophic rhinitis group had undergone prior nasal surgery. The mean number of surgical procedures that the patients had undergone was 2.3, and 157 of the patients had a history of turbinectomy as part of their nasal surgery. One hundred ten patients had undergone partial inferior and/or middle turbinectomy, and 47 patients had total removal of the middle and inferior turbinates performed through one or more procedures. All of these patients reported that they had undergone turbinectomy in an effort to alleviate symptoms of refractory nasal congestion. Other nasal destructive processes noted in the history of the secondary atrophic rhinitis patients included endonasal sinus surgery without turbinectomy (19 patients), partial maxillectomy for neoplasm (12 patients), sinonasal irradiation (5 patients), significant nasal trauma with reconstruction (2 patients), and chronic granulomatous disease (2 patients; Table III).

The primary atrophic rhinitis group consisted of 45 (19%) patients, and it was comprised of 25 women and 20 men. The average age of these patients was 52 years, with an age range of 19–89 years. By definition, none of these patients had any history of trauma, surgery, chronic debilitating medical illnesses, or radiation before the development of their symptoms. Cultures were positive for *K. ozaenae* in all 45 patients. Five of the patients had a positive family history (affected sibling) of atrophic rhinitis. Radiographic and rhinomanometric findings in the primary atrophic rhinitis group were similar to those of patients in the secondary atrophic rhinitis group.

Treatment approach was similar in both groups of patients. After diagnosis, medical therapy was instituted with topical gentamicin solution (80 mg gentamicin sulfate dissolved in 1 L of normal saline solution) nasal irrigations of varying frequency tailored to the individual patient's clinical needs and tolerance. Appropriate systemic antibiotic treatment was added for persistent clinical infection or sporadic exacerbation of sinusitis symptoms. Generally, this regimen was well tolerated, and no instances of aminoglycoside toxicity from systemic absorption were noted. Patients also were instructed to follow the antibiotic irrigation and nasal debridement with glycerine solution applied topically to the atrophic mucosa to improve nasal humidification. Some patients also chose to apply mineral oil drops scented with rose geranium oil to the nose to relieve fetor. After achieving improvement in crusting and fetor, patients were converted to daily isotonic saline nasal irrigations or varying frequency with a bulb syringe followed by use of a glycerine solution or mineral oil drops.

Discussion with all patients stressed the chronic, unrelenting nature of the disease. Patients were instructed to continue their irrigation and humidification therapy daily without cessation. Frequent follow-up was arranged for those patients in the local area, and follow-up by correspondence with the patient or care provider was arranged for the rest of the patients. Two hundred twelve (88%) of the patients noted "significant improvement" in symptoms of nasal crusting, dryness, and fetor with this therapy. Physical examination in these patients supported their claims of decreased nasal crusts and purulent secretions. Twenty-five (10%) patients noted "some improvement" with medical therapy, and 5 (2%) patients noted "no improvement." No patients were able to cease irrigations and humidification without return of symptoms. Follow-up for these patients ranged from 18 years to 1 year, with the average follow-up being 8.2 years.

Selected patients have been identified as having paradoxical subjective nasal obstruction, which is relieved in the

TABLE III

Nasal Destructive Processes in Secondary Atrophic Rhinitis Group

Process	Total Turbinectomy	Partial Turbinectomy	Sinus Surgery	Partial Maxillectomy	Sinonasal Irradiation	Other
Number	47 (24%)	110 (56%)	19 (10%)	12 (6%)	5 (3%)	4 (2%)

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office by augmenting their nasal resistance with rolled cotton placed in the valve region. In 7 of these patients, we performed endonasal microplasty with subcutaneous placement of autogenous cartilage and acellular dermis (Alloderm; Lifecell, Branchburg, NJ) or irradiated rib. All 7 patients have been seen in follow-up (6 months to 4 years; average, 10 months) with subjective relief of nasal crusting and nasal obstruction. No extrusion of the implants has occurred, but the follow-up is insufficient to assess nasal mucosal regeneration or resorption of the implants.

DISCUSSION

Atrophic rhinitis is a clinical diagnosis that should be entertained during the workup of patients with chronic rhinitis and crusting, particularly if they have any history of extensive nasal trauma or insult. The constellation of symptoms of thick adherent crust formation, foul odor, and nasal obstruction are essential to the diagnosis. Although not essential to the diagnostic process, further confirmation can be found in histopathological study of a nasal biopsy specimen. The normal nasal lining is a ciliated pseudostratified columnar epithelium (Fig. 1). In the patient with atrophic rhinitis the epithelium undergoes metaplasia to islands of squamous epithelium (Fig. 2). Universal histological findings of atrophy of serous and mucinous glands, loss of cilia, loss of goblet cells, and inflammatory cell infiltrate are seen in atrophic rhinitis.[11] Characteristic inflammatory vascular changes also are prominent in the submucosal layer. Endarteritis obliterans with thickening of the media and dilatation of the subepithelial capillaries leads to impaired mucosal regeneration and fragility of the epithelium.[5] This constellation of histological changes results in defective or absent mucociliary clearance. Moisture is absent in the nose from the glandular atrophy, causing disappearance of the sol and gel layers of the normal mucociliary blanket. Loss of cilia results in stasis of any secretions that are produced by the remaining glandular cells. The result is exposed and damaged epithelium covered by chronically superinfected crusts. Overall, the nose has impaired defense mechanisms that contribute to continual rhinitis and sinusitis.[12] Cultures of the nasal mucosa in patients with atrophic rhinitis frequently will show colonization or infection with pathological organisms.

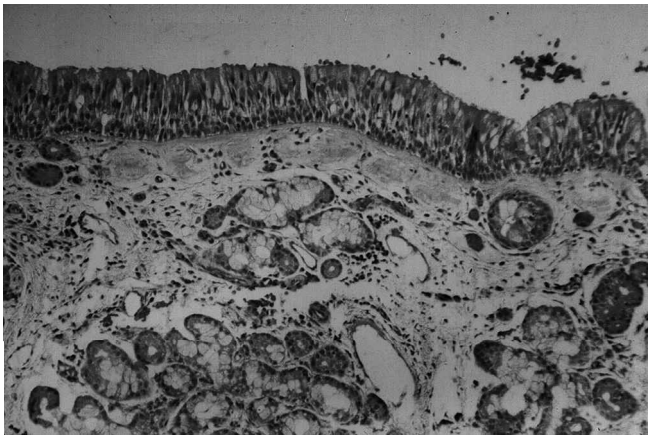

Figure 1. *Photomicrograph of the nasal lining of a normal inferior turbinate showing ciliated pseudostratified columnar epithelium with abundant seromucous glands.*

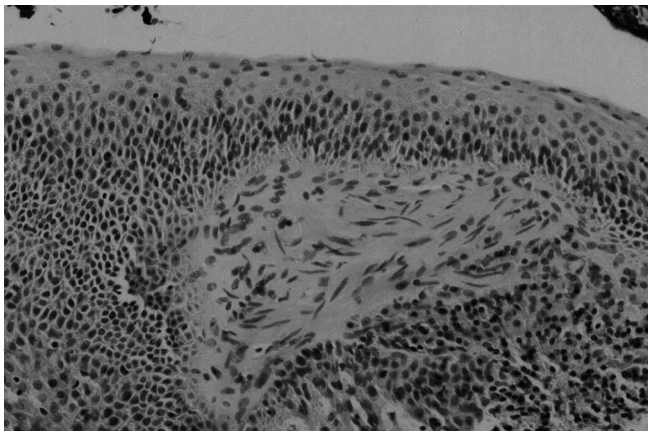

Figure 2. *Photomicrograph of the nasal lining of an inferior turbinate remnant from a patient with atrophic rhinitis. Squamous metaplasia occurs with loss of ciliated cells. The subepithelial layer shows abundant infiltration of inflammatory cells.*

CT of the nose and paranasal sinuses also will show characteristic findings in atrophic rhinitis. Our study showed some of the common findings of atrophic rhinitis as delineated by Pace-Balzan *et al.*: (1) mucosal thickening of the paranasal sinuses, (2) loss of definition of the ostiomeatal complex secondary to destruction of the ethmoid bulla and uncinate process, (3) hypoplasia of the maxillary sinus, (4) enlargement of the nasal cavities with destruction of the lateral nasal wall, and (5) bony destruction of the inferior and middle turbinates. The characteristic scan of an atrophic rhinitis patient shows the cavernous nasal airway, which is prominent in these patients. The absence of normal nasal structures is universal in these patients, and the symptoms of atrophic rhinitis coupled with a cavernous nasal airway lacking identifiable turbinate tissue has been termed "the empty nose syndrome" (Fig. 3).

The absence of any obvious impediment to airflow on CT and exam makes the universal symptom of subjective nasal congestion in these patients seems paradoxical. Some authors have recognized a similar nasal congestion in the absence of anatomic obstruction or nasal airflow resistance in their patients after turbinectomy. They have attributed this finding to the patient's inability to recognize normal baseline nasal sensation of breathing or to malingering.[13] The true cause of this paradoxical nasal congestion in the empty nose of atrophic rhinitis remains unknown, but several factors may contribute. Eccles and others have shown that stimulation of the trigeminal temperature receptors in the nasal valve region may be more significantly coupled to the sensation of nasal breathing than to actual airflow.[14] It is

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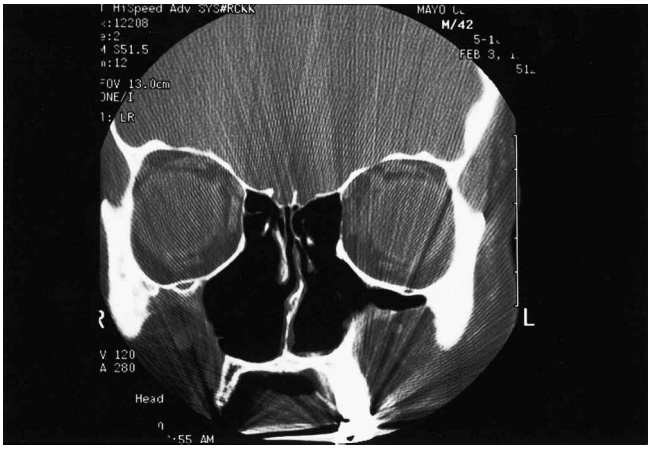

Figure 3. *Coronal CT scan of a patient with atrophic rhinitis showing common findings of the "empty nose syndrome." There is cavernous expansion of the intranasal airway with absence of the lateral nasal walls and inferior turbinates. The mucosa covering the middle turbinate remnants and paranasal sinuses is atrophied in this patient who had undergone four previous sinonasal surgeries for chronic sinusitis.*

already known from electron microscopic studies that patients with atrophic rhinitis have atrophy of the olfactory epithelial receptors leading to anosmia, and they also may develop atrophy or disorder of the pain and temperature receptors in the nasal lining.[4,15] This may lead to these patients' inability to sense air flowing to the nose and a resultant feeling of nasal congestion. Furthermore, the obliteration of all nasal resistance may not result in attainment of satisfying nasal respiration. Most people would agree that nasal breathing is far more satisfying than mouth breathing, but the nose provides one-half the resistance of the entire respiratory tract and 50% more effort is required for nasal breathing compared with mouth breathing.[16] Some nasal resistance provided by the intranasal structures may be necessary to balance the pulmonary resistance during inspiration.[17] This balance may be perceived as satisfying nasal airflow, and extreme lack of nasal resistance then actually may be perceived as paradoxical nasal obstruction.

The etiology of primary atrophic rhinitis remains incompletely understood, and a number of causes have been proposed. Possible factors include infectious, hormonal, vascular, hereditary, hygienic, autoimmune, and dietary.[5,18] Infection has been considered a leading factor, and this study corroborated others that have found a high correlation between positive cultures for *K. ozaenae* and the presence of atrophic rhinitis.[19] Unfortunately, many of these patients are diagnosed late in the disease process. It often is difficult to determine if the infecting organism caused the tissue destruction and ensuing symptoms or if the organisms represent contaminants or opportunistic invaders of an environment with previously damaged mucosa and deficient defense mechanisms.

The cause of secondary atrophic rhinitis also remains a challenging and controversial puzzle. Extensive removal of nasal tissue in the form of turbinectomy has been postulated as an etiology of secondary atrophic rhinitis, with some authors reporting 15–71% of their own patients experiencing postoperative atrophic rhinitis symptoms.[20–22] Other authors report that they have never encountered a case of atrophic rhinitis or empty nose syndrome after total removal of the inferior turbinates.[23,24] Even the function of the turbinates and the alteration of those functions when the turbinates are disturbed are incompletely understood. Some claim that it is the middle turbinate that is completely responsible for nasal humidification, while the inferior turbinate regulates nasal resistance and airflow, and they recommend resection of the inferior turbinate but complete preservation of the middle turbinate.[24] Other authors recommend routinely resecting the middle turbinate during sinus surgery, and they assert that because of its diminutive surface area and less prominent position in the airway, it is of less functional significance than the inferior turbinate.[25] Delayed effect of nasal mucosal resection may contribute to the controversy surrounding turbinectomy and its relationship to secondary atrophic rhinitis. Martinez found only 3 patients with excessive dryness and crusting in a 2-year follow-up of 29 patients out of a total of 40 patients who had undergone total inferior turbinectomy for nasal obstruction.[26] He concluded that the advantages of total turbinectomy greatly outweigh its reported disadvantages. In a follow-up study on 18 of the same 40 patients after 3–5 years had elapsed since their surgery, Moore found that 89% now had bilateral nasal crusting and 39% had thick malodorous secretions.[3] He concluded that total inferior turbinectomy should not be performed because of the resultant morbidity. Others have performed similar long-term review of total turbinectomy patients and found no evidence of empty nose syndrome or atrophic rhinitis, although ~35% of the patients in these studies was lost to follow-up.[27,28]

In our series the patients were diagnosed with a secondary process if they had undergone turbinectomy, nasal trauma with tissue loss, radiation, or another nasal destructive process before exhibiting the characteristic atrophic rhinitis symptoms. A cause-effect relationship between removal of nasal tissue and atrophic rhinitis is unproved from this data or any other published reports. To prove or disprove this relationship, a prospective randomized study of carefully measured nasal tissue removal would need to be coupled with long-term (5–10 years) follow-up. Because one of the hallmarks of atrophic rhinitis is irreversible loss of viable nasal mucosa, and because the disease is so difficult to treat fully, we have been unwilling to subject our patients with intractable rhinitis and nasal obstruction to this experiment.

Treatment of atrophic rhinitis remains less controversial but still varied because of the dissatisfying number of "cures." Continual nasal hygiene with vigorous and regular intranasal irrigation remains the standard of conservative therapy. Sodium bicarbonate solution, aminoglycoside top-

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ical therapy, normal saline solution, and plain water have all been proposed as irrigants.[5–9] The addition of topical antibiotic irrigation should be guided by the purulent appearance of the secretions. In addition, systemic antibiotic therapy usually is necessary periodically, and its use should be guided by appropriate intranasal sinus cultures and sensitivities obtained at the onset of symptoms of acute sinusitis. Tetracycline, aminoglycosides, and, more recently, ciprofloxacin (250–500 mg twice daily for 4 weeks) have been reported to be successful.[11,29] We avoid vasoconstrictors and topical steroids in the nose of someone who already suffers from a compromised vascular and immune system. The goal of therapy is to restore nasal hydration and minimize crusting and purulent debris (Fig. 4).

Various surgical procedures, ranging from conservative debridement and even further tissue removal to radical alteration of the nasal anatomy with closure of the nostrils have been reported.[30–32] Young described bilateral closure of the nostrils at staged 3-month intervals to prevent further degeneration of the nasal mucosa.[31] Modification of Young's procedure has been described as partial closure of the nostril to allow for serial endoscopy.[32] Reports of the operations have shown disappearance of the crusting at 6 months after operation and increase in length, but not in number, of the cilia.[32] Serial endoscopy shows some mucosal regeneration and an appropriate time to reopen the nostrils, usually 3–5 years after closure. Artificial implantation of various materials to restore intranasal volume also has been proposed by many authors. Short-term results have been encouraging, but long-term reports of extrusion of artificial material in up to 80% of cases and resorption of dermofat grafts and bone have tapered enthusiasm for the procedures.[33,34] We currently select surgical candidates from patients who show modest to unsatisfactory response to medical therapy but resolution of nasal congestion with temporary partial closure of the nasal vestibule with cotton. In these patients, we perform endonasal microplasty by creating a subepithelial pocket along the nasal floor and implanting autogenous cartilage and/or dermofat grafts in an attempt to decrease partially the patency of the nasal lumen. Initial resolution of nasal congestion and crusting has been encouraging, but long-term follow-up of the patients is necessary to evaluate resorption and permanence of improvement.

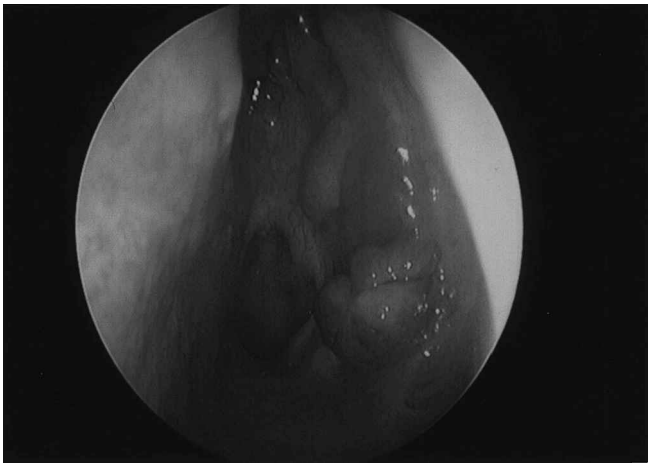

Figure 4. *Intranasal photograph of a patient with atrophic rhinitis that is well managed with nasal irrigations and application of humidifying spray. The goal of treatment is prevention of crust formation and drying of the atrophic nasal lining.*

CONCLUSION

Atrophic rhinitis is a chronic debilitating disorder diagnosed by a constellation of symptoms of nasal congestion, crusting, and fetor in a patient with an abnormally patent empty nose. Diagnosis made by symptomatology can be confirmed with imaging studies of the paranasal sinuses and intranasal biopsy with histological study. The disease can exist in a primary form of uncertain etiology, which usually is accompanied by chronic bacterial infection. It also can exist in a more commonly seen secondary form after nasal trauma, surgery, irradiation, or chronic granulomatous disease. The time course and cause–effect relationship of trauma to development of symptoms are incompletely understood. Until these processes are better understood, we advise against extensive removal of intranasal structures during nasal surgery.

Medical management remains the most effective therapy, and control, rather than cure, of symptoms is the usual outcome. Patients should be educated about the chronicity of the disease, and the importance of diligence in maintaining their nasal hygiene should be stressed. Daily irrigations with isotonic saline solutions are prescribed with varying frequency dependent on the degree of crusting and fetor developing per patient. Topical aminoglycoside irrigations and systemic antibiotics are prescribed based on symptoms, culture, and sensitivities performed on intranasal smears. Frequent follow-up and communication is encouraged to prevent deterioration in the nasal hygiene of the patients. Surgical therapy with endonasal microplasty is beneficial in selected patients, and continual modification and follow-up of this procedure may delineate the ideal implant material and location of placement. Prevention of the disease is a crucial part of the management, and further investigation of these patients may illuminate a critical amount of nasal mucosa that should be preserved during intranasal destructive procedures.

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