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SHORT REPORT

Atrophic Rhinitis: A Case Report*

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Primary atrophic rhinitis, or ozena, is a progressive chronic nasal disease and a form of chronic rhinitis characterized by mucosal atrophy with resorption of underlying bone, formation of thick crusts, nasal obstruction, anosmia and a distinct fetid odor (1). The etiology is still unknown, but some bacteria such as *Klebsiella ozaenae*, *Proteus*, *Escherichia coli* and *Bacillus pertusis* have been isolated from the nasal secretions of patients as causative organisms (2,3). It is unknown whether primary atrophic rhinitis is purely an infectious entity or a combination of infectious, hereditary, nutritional deficiencies, estrogen deficiency and vascular disorders of the paranasal sinuses (1,4). Primary atrophic rhinitis occurs in a previously healthy nose. Secondary atrophic rhinitis occurs most commonly as a late postoperative complication following excessive surgical destruction of the nasal mucous membrane (1,4). The diagnosis is made clinically by the presence of characteristic changes inside the nose such as enlargement of the nasal cavities, atrophy of the mucosa and the presence of adherent, thick and green-yellow crusts, or microbiologically by isolation of putative bacteria such as *K. ozaenae* from nasal cultures (2,5). Although there is no cure for atrophic rhinitis, some surgical techniques like cavity-narrowing or denervation have been suggested (6).

Case Report

A woman aged 47 years with a 3-year history of nasal obstruction, fetid odor and anosmia was admitted. The fetid odor was detectable as soon as she entered the room. Her history revealed that her family regularly consumed home-made bread baked on a brazier by the patient. Diagnostic nasal endoscopy revealed green-yellow crusts in the nasal cavity and atrophy of the turbinates. A swab from the nasal secretions was cultured and this resulted in the isolation of *K. ozaenae*, which showed susceptibility to ciprofloxacin. Her blood iron level was as low as 44 µg/dl (59-158 µg/dl normal range), while folate and vitamin B12 blood levels were within the normal range. Seroimmunologic tests showed positive total ANA, anti-DNA and RNP and an increase in C3 levels, whereas IgA and IgM levels were within the normal range. Her VDRL was negative and her blood profile showed no abnormality. Coronal CT scans of the paranasal sinuses revealed the bilateral absence of the superior and middle turbinates and extreme atrophy of both inferior turbinates. Significant enlargement of the nasal cavities with hypoplasia of the maxillary sinuses, resorption of the ethmoid sinuses and the lateral nasal wall leading to erosion of the uncinat process, diffuse mucosal thickening of the nasal cavity and bilateral

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maxillary sinuses were also found on CT scans (Figure 1). The fetid odor disappeared after the first week of ciprofloxacin therapy and, at that time, the patient underwent endoscopic nasal surgery. All the crusts were cleaned up and biopsies were taken from the turbinates. Histopathologic examination of the specimen revealed an infiltration of the mucosa consisting of metaplastic squamous epithelium with lymphocytes and plasma cells. In some areas, seromucous glands and vascular structures were atrophied because of lymphoid infiltration. After surgery the nasal obstruction and fetid odor improved and crusting diminished.

Atrophic rhinitis is still a common disease in developing countries, whereas it is unusual in US and other developed countries (7). The disease appears to be endemic in subtropical and temperate regions like South Asia, Africa, Eastern Europe and the Mediterranean, and the patients are usually poor and live in unhygienic conditions (6). There is a slight female predominance (1.4 to 1) (1). The patient in the present study was a woman living in poor conditions.

Mickiewicz et al. found atrophic rhinitis in workers exposed to phosphorite and apatite dusts (8). Our patient was exposed to flour dust as well as sulfur oxide and other aerial pollutants in charcoal smoke from a brazier whilst baking bread over several years.

K. ozaenae is the most commonly found pathogen in cultures as a causative organism (5). This organism was isolated from the nasal secretions of our patient, who was given ciprofloxacin. The fetid odor disappeared and crusting diminished after antibiotherapy, indicating that *K. ozaenae* was responsible for the infection and odor.

Another important factor suggested as a cause of atrophic rhinitis is iron and vitamin A deficiency (6). In our case, iron deficiency was found, whereas B12 and folate levels were normal. On the other hand, iron deficiency has not been found in all cases of atrophic rhinitis (9).

Atrophic rhinitis has been described as an autoimmune disease (10). Total ANA, anti-DNA and RNP were positive, indicating the presence of an autoimmune process.



Figure 1. Coronal CT scan shows bilateral absence of the superior and middle turbinates and extreme atrophy of both inferior turbinates; significant enlargement of the nasal cavities with hypoplasia of the maxillary sinuses, resorption of the ethmoid sinuses and the lateral nasal wall leading to erosion of the uncinat process, and diffuse mucosal thickening of the nasal cavity and bilateral maxillary sinuses.

Because of the high incidence of concurrent sinusitis, CT is frequently included in the diagnostic evaluation of atrophic rhinitis. Pace-Balzan et al. list characteristic changes identified by CT as the following:

- 1) Mucosal thickening of the paranasal sinuses,
- 2) Loss of definition of the ostiomeatal complex secondary to resorption of the ethmoid bulla and uncinete process,
- 3) Hypoplasia of the maxillary sinuses,
- 4) Enlargement of the nasal cavities with erosion and bowing of the lateral nasal wall,
- 5) Bony resorption and mucosal atrophy of the inferior and middle turbinates (5).

In our case, the most prominent finding was expansion of the nasal cavities at the expense of the maxillary sinuses, which appeared hypoplastic (Figure 1). It was reported that these findings occurred late in the disease as a result of the changes in the nose (5). In our case, the absence of the superior turbinates was detected in addition to the findings mentioned above.

Histopathologic examinations in atrophic rhinitis showed chronic nonspecific inflammation with lymphocytes and plasma cells, absence of columnar and goblet cells, and metaplasia from columnar ciliated to squamous epithelium (6). Hagrass et al. noted round cell infiltration of the tunica propria, formed mainly of

lymphocytes and plasma cells together with fibrosis (11). We saw similar histopathologic features in our patient and there were periarteritis and endarteritis of the terminal arterioles.

There is no satisfactory treatment for atrophic rhinitis. The mainstays of conservative treatment are the removal of crusts from the nose and the use of antibiotics (5).

In conclusion, the exact pathophysiologic mechanism is still unknown in atrophic rhinitis. However, as supported by the literature, exposure to flour dust and smoke may have facilitated colonization of the nasal mucosa by *K. ozaenae* in our patient and an autoimmune process leading to atrophic rhinitis with iron deficiency was initiated. CT is very useful for the diagnostic evaluation of atrophic rhinitis. In our opinion, ciprofloxacin therapy, endoscopic removal of the crusts and long-term nasal irrigation were successful in the management of this patient.

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