A CASE OF SECONDARY ATROPHIC RHINITIS WITH HANSEN’S DISEASE
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PRESENTATION OF THE CASE
Atrophic rhinitis is an uncommon disorder in modern societies and its incidence varies from 0.3-7.8% of the population.1 It can be classified as primary atrophic rhinitis which arises de novo or secondary atrophic rhinitis which may occur as a sequela of granulomatous diseases such as leprosy, syphilis, etc., comprises only 1% of the cases.2 Thus such cases are of clinical importance and rarity. Atrophic rhinitis (AR) is a debilitating chronic nasal mucosal disease of unknown aetiology. The condition is characterized by progressive nasal mucosal atrophy, progressive atrophy of the underlying bone of the turbinates, abnormal widening (roomy) / patency of the nasal cavities (with paradoxical congestion) and viscid secretions and dried crusts leading to a characteristic fetor (ozaena).3,4,5 On the basis of causes AR can be classified as; Primary Atrophic rhinitis which has decreased markedly in incidence (0.3-1%) and the aetiology is unknown2,6 and secondary atrophic rhinitis is mostly common in developed countries. The most common cause is sinus surgery, it alone comprises of 90% of secondary atrophic rhinitis, common procedures include partial and total turbinectomy (80%), without turbinectomy (10%) and partial maxillectomy (10%), followed by radiation (2.5%), trauma (1%) and granulomatous or infectious diseases (1%).2,7 The malady of “empty nose syndrome is associated with extensive turbinectomy surgeries causing secondary Atrophic Rhinitis”.8 Little is known about this secondary form and it remains incompletely characterized. It has occurred in association with chronic granulomatous diseases of the nose including leprosy, sarcoidosis, rhinoscleroma, Wegener’s granulomatosis and infectious diseases like TB and syphilis. The onset after treatment with antiangiogenic drug underlines the role of the microvasculature in the pathogenesis.2,9 While these causes were once common, now only comprise of 1-2% cases, thus rare.2 Secondary atrophic rhinitis is typically seen in older population.

Hansen’s disease (Leprosy) is a chronic granulomatous disease caused by Mycobacterium leprae. Leprosy has been officially eliminated in India since 31st December 2005, yet significant number of cases are diagnosed every year, so it is important to stay well aware about the disease.10,11 This clinical report of a case of secondary atrophic rhinitis with Hansen’s disease is aimed at showing the nasal finding in leprosy along with its treatment and reconstruction of the nose.

In our experience, a 34-year-old male patient came to ENT outpatient department of SIMS presented with atrophic rhinitis, a small septal perforation and saddle nose deformity due to Hansen’s disease (secondary atrophic rhinitis), which was diagnosed and treated, followed by nasal reconstruction via open septrhinoplasty approach using an autologous conchal cartilage graft with columellar reconstruction, yielding a successful postoperative result.

DIFFERENTIAL DIAGNOSIS
Sarcoidosis, Rhinoscleroma, Wegener’s granulomatosis, Lupus erythematosus, Long-standing purulent sinusitis, Radiotherapy of the nose, Empty nose syndrome and Infectious diseases like TB and syphilis.

CLINICAL DIAGNOSIS
In this case, a 34-year-old male patient, generator mechanic by occupation with chief complaints of excessive unilateral left sided nasal crusting since 5 years, altered smell sensation since 4 years, excessive bilateral crusting and depression over the dorsum of the nose since 1 year, tingling sensation over the toes started 4 months back and thickening and tingling sensation over bilateral pinna was observed one month back. Bilateral crusting started 1 year back which required cleaning morning and evening, it was accompanied by nasal obstruction which was relieved on clearing of crust, there is no history of bleeding. No past history of similar complaints. No history of Tuberculosis, diabetes mellitus, Jaundice, Asthma or Allergy. Patient is using Betrovate N cream at night in both ears.

Hypopigmented patch of size 5x3 cms over the right thigh. Ulnar nerve thickening felt over the left elbow. On Local examination of nose, External nose skin appears normal, depression over the dorsum of the nose is seen (Figure 1). Anterior rhinoscopy shows nasal cavity is covered with greenish yellow crusts, both nasal cavities appear roomy and were foul smelling, atrophy of inferior turbinate seen, small septal perforation was seen (Figure 2A). Posterior rhinoscopy shows crusts in nasopharynx. On examining oral cavity and oropharynx, teeth are tobacco stained, oral cavity, tongue, floor of the mouth, hard palate and soft palate...
appears normal. Post nasal drip with crusting is seen in the posterior nasal wall (Figure 2b). Ear examination, inspection shows bilateral pinna thickened and shiny in appearance, post and pre-auricular areas are normal.

On palpation, bilateral pinna thickened and rubbery in consistency. 1 nodule of size 0.5x1 cm felt over the helix of left pinna, it is mobile adherent to the skin but not to the underlying cartilage. Sensation over the pinna is reduced. DNE shows bilateral crusting, small septal perforation and atrophied turbinates (Figure 2c). CT Scan shows atrophy of inferior and middle turbinates, mucosal thickening of maxillary and ethmoid sinuses (Figure 2d). In view of leonine facies and nodules the patient was advised Silt Skin Smear both ear lobules. Z N stain shows stout bacilli singly arranged intracellularly in cigar bundle appearance (globi), with bacteriological index 4+ (Ridley Jopling) suggestive of lepromatous leprosy (Figure 2e).

Patient was treated with Rifampicin 600mg once a month, Dapsone 100mg OD, Clofazimine 300mg once a month 50mg OD for 18 months and then remained quiescent for 1 year after which nasal reconstruction surgery for the saddle nose deformity by augmentation rhinoplasty (open) using autologous auricular cartilaginous graft and along with columellar reconstruction was done (Figure 3). Post-operative recovery was satisfactory and good cosmetic result and functional improvement was achieved (Figure 3).

**DISCUSSION OF MANAGEMENT**

In leprosy (Hansen’s disease), the nose is an important portal of entry for this bacterium and is therefore frequently affected. Depending upon the host immune response, nasal symptoms of leprosy may include epistaxis, gross nasal deformity and destruction. Patient may complain of anosmia and nasal obstruction. On nasal endoscopy friable granulomatous intranasal lesion involving the septum with associated crusting. In the early stage, yellowish discolouration of the mucosa, nodules or pale plaques may be visualized involving the septum and inferior turbinates. As the disease progresses the mucosa becomes thickened resulting in purulent nasal discharge that contains a high concentration of the organisms. This disease has a predilection for nasal invasion resulting in decreased nasal sensation. Late stage leprosy is characterized by dryness, crusting and septal cartilage destruction. This disease is very slowly progressive but once the diagnosis is confirmed, broad spectrum antibiotic treatment should be initiated to prevent disfigurement. Sinonasal symptoms can occur and can be refractory to systemic treatment. The clinical spectrum ranges from localized involvement of the external nose and extensive destruction of the septal cartilage and bone resulting in nasal septal perforation and saddle nose deformity. Principally, systemic treatment should precede any local surgical procedure. Menger and coworkers reconstructed 24 saddle nose deformities of different severity caused by leprosy. In all cases external approach was used. A inverted V procedure was used in 4 patients in order to lengthen a retracted columella. The nasal septum and upper lateral cartilages were reconstructed by implantation of a dorsal onlay graft. An absent anterior nasal spine was reconstructed by implantation of a caudally extended columnellar structure. An autologous costal and auricular cartilage was exclusively used. Wound infection, extrusion or warping of implants was not observed in any patient. Functional and aesthetic improvement was observed (15/17) patients. The rate of implant resorption was dependant on the implant site. Least resorption was observed for dorsal onlay grafts (4/17). Moderate resorption was observed for columnellar structure and shield grafts (7/17). In general, conchal cartilage grafts were associated with less resorption than costal cartilage grafts overall, advantages are dominating so that authors advocate reconstruction of saddle nose deformity in leprosy using autologous cartilage grafts. Septal or Conchal cartilage is easily available, causes minor donor site morbidity, suffices to compensate most of simple and complex saddle nose deformities. Some patients may require more graft material like costal cartilage (5%) to substitute for the major structural losses. Also bone grafts like autologous iliac crest, calvarian bone, have been used but the rate of complications such as wound infection and graft resorption was 50%. This case report reinforces the importance of an otolaryngologist for early diagnosis and multi-disciplinary evaluation of patients with leprosy.
REFERENCES


