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IP Journal of Otorhinolaryngology and Allied Science

Journal homepage: https://www.joas.co.in/

Case Report

Isolated nasal cavity *Hansen's disease*: A case report in a tertiary care hospital of tribal area of Odisha

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ARTICLE INFO

Article history:
Received 18-07-2023
Accepted 05-10-2023
Available online 27-10-2023

Keywords:
Nasal
Hansen's disease
Rhinosinusitis

ABSTRACT

Isolated nasal cavity *Hansen's disease* is very rare without other manifestations like skin and neural lesions, in a country like India where the disease prevalence is the highest among all countries of the world. Here we present a case of nasal cavity leprosy who presented clinically features of chronic rhinosinusitis with atrophic changes because of this unusual presentation.

1. Introduction

Leprosy is a chronic granulomatous infectious disease which is not so common now a days, that has a chronic evolution caused by the *Mycobacterium leprae* or Hansen's bacillus, an alcohol and acid fast bacillus. It may affect the nasal cavities mucosa independent of its clinical form, even before skin lesions or lesions to other parts of the body.[1] Though because of advanced health care management by different countries around the world the incidences and prevalences of the disease loads in the society have been reduced to many extent, but this stigmatic disease is still seen in many parts of this planet. According to Lombardi et al. (1990),[2] the transmission of the disease requires different social environments and can be correlated between poverty and low-income populations, which may be related with nutritional status, over crowding or presence of other concomitant diseases in poverty *Mycobacterium leprae* affects different parts of the body surface, like skin of the extremities, earlobes, helix, antihelix, tragus, alar cartilage, septum and testicles because of comaprative lower temperature in these area in relation to the body, that favors the mycobacterial growths.[3] The nasal mucosa is considered as entry and exit doors for *Mycobacterium leprae.* as per WHO fact sheets published on 27 January 2023. The disease is transmitted through droplets from the nose and mouth. Prolonged, close contact over months with someone with untreated leprosy is needed to catch the disease. As noted by Centers for Disease Control and Prevention, 1600 Clifton Road NE Atlanta, GA 30329,the distribution of new leprosy cases by country among 136 countries that reported to WHO in 2015. India reported 127,326 new cases, accounting for 60% of the global new leprosy cases and Brazil, reported 26,395 new cases, (13%) of the global new cases; Indonesia reported 17,202 new cases (8%) of the global case load. Universally nasal involvement occurs in all lepromatous leprosy and also occurs early in the course of the disease with nasal symptoms of leprosy like nasal obstruction, crusting, bleeding, and hyposmia.[1,4] The nose becomes infected very early in lepromatous form of the disease process in which nasal discharge shows heavily bacillated, that is the most potent source of spread of *Mycobacterium leprae.*[5] Isolated

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https://doi.org/10.18231/j.ijoas.2023.019

nasal cavity leprotic lesions which present with clinical features of rhinosinositis and atrophic rhinitis without other forms of manifestations of leprosy, like skin and neural lesions is very rare in a country like India where prevalance rate of this disease is the highest in the world. Here we present a case of nasal leprosy with such presentation, because of its rarity and simularity with rhinoscleroma.

2. Case Report

A 26 years male patient, literate, from low socio-economic and rural back ground from tribal community came to the S L N Medical College, Koraput, Odisha with chief complains of scanty mucopurulent of nasal discharge since one year, occaisional headache without fever for one year, crust expulsion from both the nasal cavities for more than six months.There was history of mild recurrent sore throat during these periods. He observed nasal blockage of the nasal passage since six months. There was history of occasional bleeding from the nose for last six months. He had no history of Diabetes Mellitus, hypertensions and any other sysemic disorders in past and also now. There was no ontact history of tuberculosis, syphilis, or leprosy. The patient takes bath in ponds and rivers.

On examination, he was of average body built, well oriented,conscious and cooperatve.Vitals were within normal limits with no pallor, cyanosis, icterus, pedal oedema and lymphadenopathy.

His Cardiovascular, central nervous system, peripheral nervous system and skin examination revealed no abnormalities. On otorhinolaryngeal examination, there was neither external nasal deformity nor any other abnormal skin canges or swelling over facial regions. There was mild maxillary sinus tenderness. Anterior rhinoscopy revealed formations of crusts in either sides of the nasal cavities with dry and pale nasal mucosa. The septum was bilaterally thickened and ulcerated with few granulomatous changes towards lower half, anteriorly and in midportions .Granulomatous masses were found in right nasal cavitie. Examination of oral cavities oropharynx and nasopharynx on posterior rhinoscopy showed no significant changes. Examinations of hypopharynx and larynx and ears also revealed no abnormalities.

The patient was diagnosed chronic rhinosinusitis with atrophic changes and nasal masses in both sides and was admitted for endoscopic biopsy and general examinations.

Routine Blood examinations were within normal limits. The serological screening tests for HIV, hepatitis and syphillis were non specific. NCCT scanning of the nose and paranasal sinuses showed mild septal deviation to the left, mild mucosal thickening of bilateral maxillary and ethmoid sinuses with bilateral concha bullosa. Billateral nasal masses with obstruction of bilateral osteomeatal complexes (Figure 1 A and B)

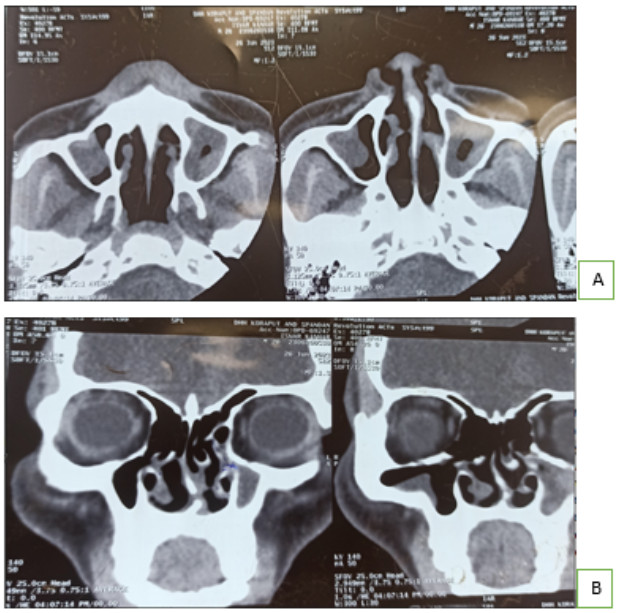

Fig. 1: A: Mucosal thickening of bilateral maxillary sinuses in axial NCCT of Paranasal sinuses, **B:** Mucosal thickening of bilateral maxillary sinuses and soft tissue type swelling of right inferior turbinat in coronal plane of NCCTPNS.

The tissues were taken endoscopically for histoathological studies under local anaesthesia [2% lignocaine + Adrenaline 1:1000 injection,topical 4% lignocaine] and with premedications of sedatives [tramadol inj.im] and diclofenac injection intragluteally. Endoscopic observations showed crusts formations in left nasal cavity and uclerations over septum. Right nasal examination revealed granuloma formations over inferior and middle turbinares and ulceration and thickening of the septum(Figure 2 and 3). Nasal packs were given for 24 hours and injectable antibiotics[ceftriaxone+sulbactam 11 gm IV] was administerred. The patient was discharged next day with advice of doxycycline capsules[100 mg daily orally for 7 days and ciprofloxacin tablets 500 mg twicedaily for 7 days] and daily regular alkaline nasal douching.

Histopathological report revealed sheets of histiocytic cells some with foamy and vacualated cytoplasm admixed with cells of minimal amount of other inflammatory cells. Overlying squamous epithelium found focally. Isolated multiple clumps of acute inflammatory exudates and microbial agents were present. No fungal elements and malignant cells were found. Giemsa stains and GMS stains were negative for Acid Fast Bacilli. Fite staining showed presence of numerous acid fast bacilli [AFB] including Globi suggestive of *Hansen's disease* [*Leprosy*].(Figure 3 A, B, C and D)

The patient was sent for dermatological consultations. He was now under treatment of MDT [multi drug

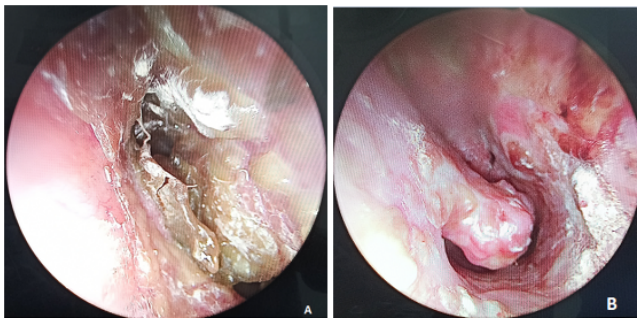

Fig. 2: A, Crusts formations in left nasal cavity and uclerations over septum, **B .** Granuloma formations over inferior and middle turbinares and ulceration and thickening of the septum

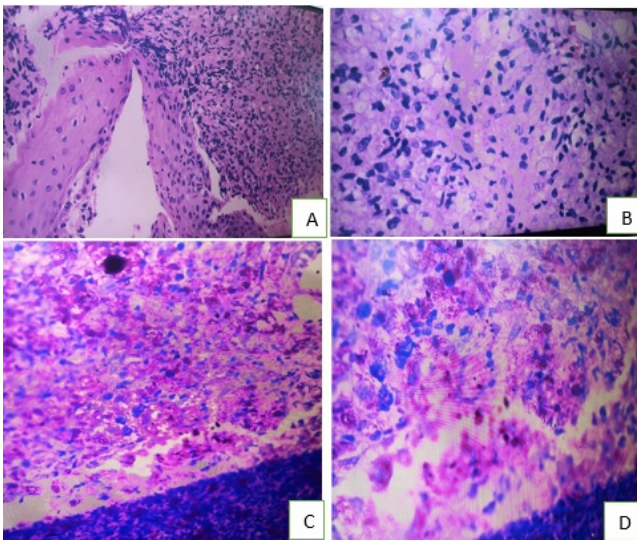

Fig. 3: A: Showing squamous epithelium and hitiocytes and chronic inflammatory cells .(on H& E stain), **B:** Showing sheets of histiocytic cells some with foamy and vacualated cytoplasm admixed with cells of other inflammatory cells.(on H & E stain in high power), **C:** Showing multiple clumps of acute inflammatory exudates and microbial agents with presence of numerous acid fast bacilli [AFB] including Globi suggestive of *Hansen's disease* (on Fite staining) **D :** Showing multiple clumps of acute inflammatory exudates and microbial agents were present with presence of numerous acid fast bacilli [AFB] including Globi suggestive of *Hansen's disease* (on Fite staining)

treatment regimen-Dapsone + Rifampicins+ Clofazimine] plus regular nasal alkalinne douching. He was also advised multivitamins.

3. Discussion

Leprosy is a chronic granulomatous infectious condition, caused by the Mycobacterium leprae or Hansen's bacillus, an alcohol and acid fast bacillus. M. leprae was discovered by Norwegian physician, Gerhard Henrik Armauer Hansen in 1873. It may affect the nasal cavities mucosa independent of its clinical forms, even before skin lesions or lesions to other parts of the body.[1] It is a major social stigmatic and disabling health problem in many parts of the world, especially in India which has the highest number of active Leprosy patients in the world.[6] M.leprae spreads through nasal mucosa to others by droplet spread and from skin by contageous spread.[7] The leprosy is divided into five forms depending upon clinical, histopathological, and immunological criteria like (1) tuberculoid polar leprosy (TT), (2)borderline tuberculoid (BT), (3) midborderline (BB),(4)borderline lepromatous (BL), and (5) lepromatous polar leprosy (LL).[8] In respect of operational point of view as per WHOStudy Group on Chemotherapy of Leprosy for Control Programmes(1981) classified Leprosy into two groups, multibacillary(MB) with a bacteriological index of 2+ or more at any site in the initial skin smears and pauciballary (PB) depending on bacterial positivity count on skin smear.[6] In the Ridley–Jopling classification multibaccillary groups include polar lepromatous (LL), borderline lepromatous (BL), and mid-borderline (BB) and paucibacillary group includes indeterminate (I), polar tuberculoid (TT) and borderline tuberculoid (BT) cases, with a bacteriological index of <2 count per field.[6,8]

WHO recommended guidelines for clinical diagnosis of Leprosy as per any one of the these criteria manifested like :1. definite loss of sensation in a pale (hypopigmented) or reddish skin patch; 2.a thickened or enlarged peripheral nerve, with loss of sensation and/ or weakness of the muscles supplied by that nerve; 3. The presence of acid-fast bacilli in a slit-skin smear.[6]

Nasal complications and nasal symptoms like nasal stuffiness,musty odor, epistaxis, nasal bridge collapse are usually presented in multibaccillary leprosy.[9] Gerami H observed nasal obstruction, anosmia and rhinorea and intranasal mass in lateral wall, alongside the inferior conchea and septum.[10] Giselle Mateus da Silva et.al. in their study observed, nasal obstruction(36.25%),crusts (30%), epistaxis (15%). And anosmia(13.7%) as most frequent symptoms and hypertrophy of the conchas (36.25%) and atrophy of conchas (22.5%), hyperaemia(22.5%) as most frequent signs along with ulceration in nasal mucosa, septal perforation and saddle nose in their 80 observed cases.[11] Leprosy compromises the nasal mucosa producing leprous rhinitis, including infiltration, lepromas, perforation, ulcerations and crust formation but however, under endoscopic examinations noted that infiltration, lepromas and hematic crusts were mostly seen in multibacillary patients(75%).[12]

Supplimenting clinical diagnosis there are a number of serological methods like enzyme-linked immunosorbent assays (ELISA). Polymerase chain reaction (PCR)-based assays are associated with higher diagnostic accuracy. But the gold standard test for establishing the diagnosis is bacteriological study.[9]

WHO has recommended guidelines for treatment of leprosy as such: Multibacillary leprosy-(MDT) rifampicin: 600 mg once a month + clofazimine: 300 mg once a month, and 50 mg daily + dapsone: 100 mg daily for 12 months in adults. The standard adult treatment regimen for PB leprosy is: Rifampicin: 600 mg once a month+ dapsone: 100 mg daily fo 6 months.

4. Conclusion

Though the *Hansen's disease* has been under declining pace it is still persisting as a threat to public health problem in many states of India. Nasal cavity leprosy without neural and skin involvement is very rare and may present as chronic rhinosinusitis or a chronic granuloma. This may also be missed from diagnosis or misdiagnosed as rhinoscleroma or atrophic rhinitis in ENT clinical practice.Therefore, it is stressed that, it is advised to be vigillant and to have a wide differential diagnosis during clinical examinations, about the possibility of *Hansen's disease* for early management.

5. Disclaimer

1. Competing interest /interest of conflict-None
2. Sponsorship / funding – None
3. Written consent of the patient – Taken

6. Source of Funding

None.

7. Conflict of Interest

None.

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Cite this article: Kar R, Acharya A, Mallik KC, Dash P. Isolated nasal cavity *Hansen's disease*: A case report in a tertiary care hospital of tribal area of Odisha. *IP J Otorhinolaryngol Allied Sci* 2023;6(3):81-84.