Review Article

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Oral Lesions in Borderline Tuberculoid Hansen's Disease: A Rarity

V Sairam[1] Turri Sabir Ahmed[2*] Praveen Kumar[3] G Naresh[4] G Vikas Reddy[5]

[1]Professor and Head, Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.
[2]PG Student, Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.
[3]Reader, Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.
[4]Reader, Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.
[5]Senior Lcturer, Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.

ABSTRACT

Background: Leprosy is a chronic disease affecting primarily the peripheral nerves and skin. It is contagious in nature and the causative organism is Mycobacterium lepra. Involvement of internal organs and mucosa has also been reported1. Leprosy presents as a spectrum of clinical manifestations that have bacteriologic pathologic and immunologic counterparts. The spectrum from polar tuberculoid (TT) to borderline tuberculoid (BT) to mid-borderline (BB, which is rarely encountered) to borderline lepromatous (BL) to polar lepromatous (LL) disease is associated with an evolution from asymmetric localized macules and plaques to nodular and indurated symmetric generalized skin manifestation an increasing bacterial load and loss of M. leprae-specific cellular immunity.2 The occurrence of oral lesions is a rarity occurring primarily in lepromatous form of leprosy.3 In this paper, we are reporting a case of oral lesions occurring in tuberculoid borderline leprosy in a 22 yr old female patient involving the maxillary and mandibular gingiva on the buccal and palatal as well as labial and lingual regions. The patient was put on MULTIDRUG THERAPY [MDT] and is showing signs of improvement with a regular follow up.

Keywords: Granulomatous infections, Leprosy, tuberculoid Hansen`s disease.

INTRODUCTION

Leprosy is a chronic infectious contagious disease affecting the skin and peripheral nerves. Involvement of internal organs and mucosa has been also reported. Ridley and Jopling (1962)[4] classified leprosy in several clinical forms based on clinical, bacteriological, histopathological and immunological criteria. Indeterminate leprosy is the initial form that either resolves spontaneously or evolves into the other forms according to the degree of cell-mediated immunity to the infectious agent.

The tuberculoid form represents the strongest response, whereas a relatively anergic state is reflected by the lepromatous form. The borderline group represents an intermediary position between these 2 forms and is divided into 3 subgroups: (1) close to the tuberculoid pole (borderline-tuberculoid); (2) in between the tuberculoid and the lepromatous poles (borderline-borderline); or (3) close to the lepromatous pole (borderline-lepromatous).

In 1982 WHO divided leprosy into paucibacillary and multibacillary types basing on the number of organisms for therapeutic purposes. Patients with a bacterial index less than 2+ were termed paucibacillary and those with a bacterial index greater than or equal to 2+ were termed multibacillary. Tuberculoid, borderline-tuberculoid, and indeterminate types are grouped into

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***Correspondence Dr. Turri Sabir Ahmed.**
Department of Oral Medicine and Radiology, G Pulla Reddy Dental College & Hospital, Kurnool, India.
Email: drsabirahmed@gmail.com

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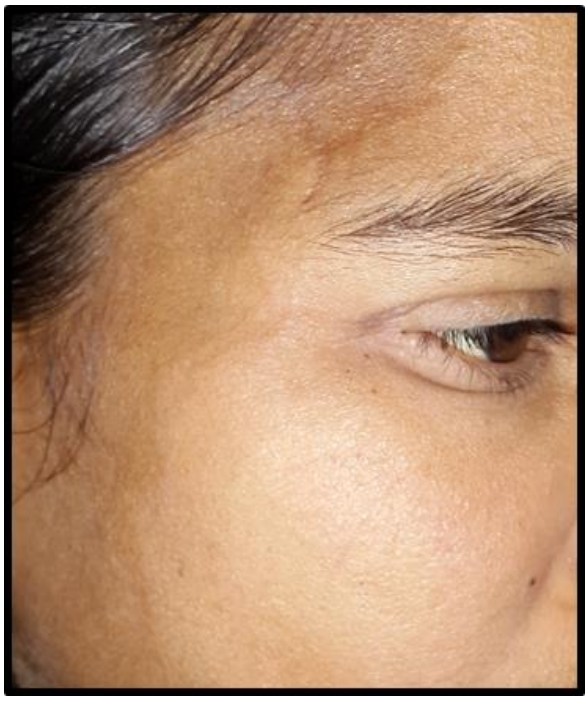

Fig 1: Extra oral picture.

Fig 2: Gingival lesion.

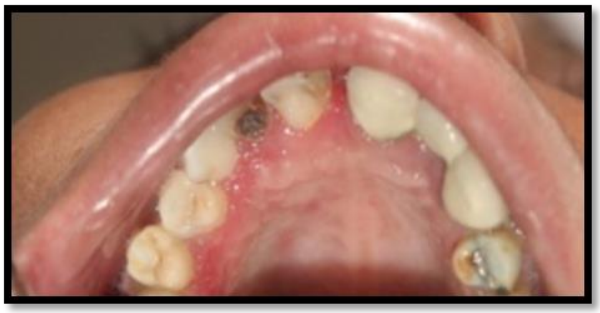

Fig 3: Palatal lesion.

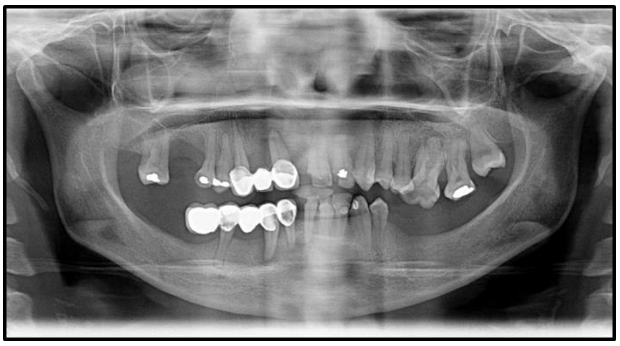

Fig 4: Orthopantomograph.

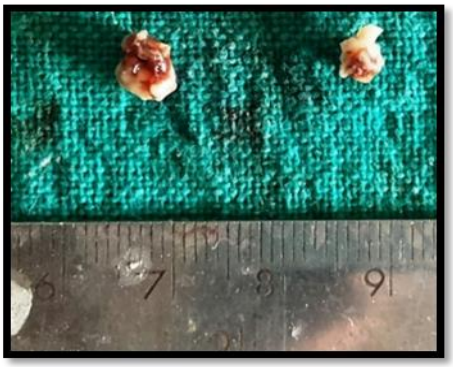

Fig 5: Punch biopsy.

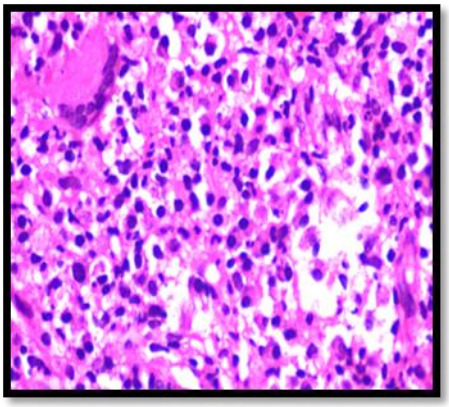

Fig 6: Pathological picture.

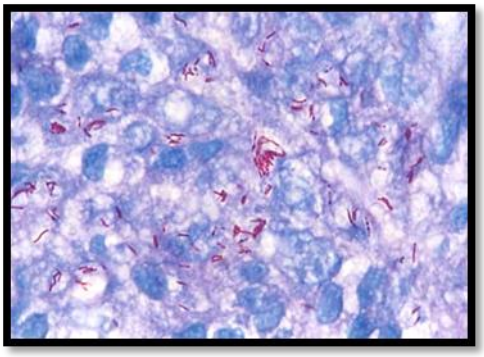

Fig 7: Acid fast staining.

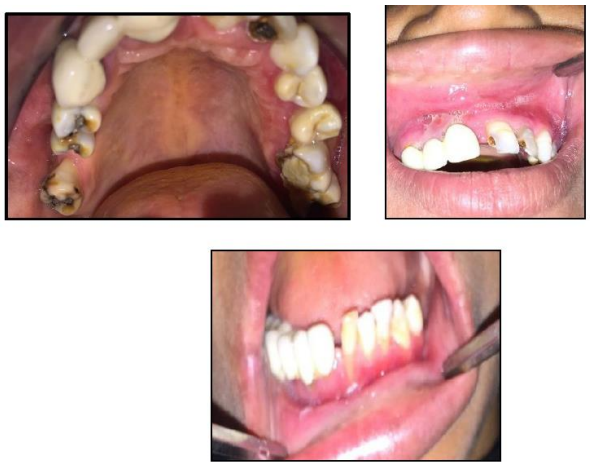

Fig 8: Three month post treatment.

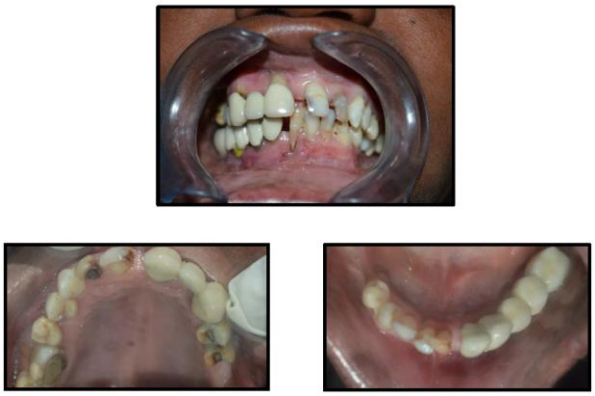

Fig 9: Six month post treatment.

paucibacillary and borderline-borderline, borderline-lepromatous, and lepromatous types are grouped into multibacillary types.[5]

The main clinical features of leprosy are erythematous or hypopigmented macules on the skin with reduced sensation, distinct sensory loss and peripheral nerve involvements, muscle weakness, nerve thickening, positive skin biopsy of acid – fast bacilli and positive skin smear. If a subject presents with one or two of these major findings should be considered as a leprosy case.[6] Oral manifestations are rare in leprosy if at all occurring in lepramotous form.[7]

Oral lesions are characteristic of advanced disease. They are rare, especially in the tuberculoid form; however, they may be recognized in about 20–60% of cases in the lepromatous type. [8,9]They are usually asymptomatic and may vary between yellowish spots or reddish papules or nodules that may develop sessile tissue necrosis and ulceration.[10] Hard and soft palate, uvula, tongue belly, lips, and gingiva are primary sites in addition to the destruction of the anterior maxilla and tooth loss.[11]It is important to note that clinical abnormalities of the oral cavity in patients with leprosy do not imply a specific lesion of the disease, and a histopathological confirmation is a must. Moreover, even in patients with normal clinical appearance, it is possible to see specific histopathological disease signs.[12, 8.] This paper presents a case of oral leprosy in a borderline tuberculoid patient, which is a rarity.

CASE REPORT

A 22 yr old female patient attended the department of oral medicine and radiology with a chief complaint of bleeding gums. Patient gives a history of pain, swelling and bleeding of gums since 3 years with intermittent relief. She was on different medications at different periods of time with gingival lesions healing intermittently.

On extra oral examination, swollen lips, hypopigmented area on the right side of the face, [fig1] rash at the corner of mouth was evident. On intra oral examination, inflamed, erythematous gingiva was seen in the palatal, buccal, labial and lingual regions involving the attached gingiva and extending up to the vestibular margin. [fig2, 3]. On hard tissue examination, generalized periodontal pockets with recession in relation to upper left first molar, lower left canine and premolar, lower right incisor and upper right premolar and second molar regions. Grade I mobility of upper left incisors and lower right central incisor was observed. A provisional diagnosis of chronic generalized periodontitis was given based on history and clinical examination. A differential diagnosis of inflammatory hyperplasia, plasma cell gingivitis, was made. Patient was subjected to radiographic [fig 4] and hematological examination which revealed generalized bone loss and raised ESR [60 mm/hr]. Patient underwent a punch biopsy in the upper right premolar region [fig 5]. On histopathological examination, inflamed and ulcerated squamous epithelium covered with necrotic inflammation and actinomycosis, submucosa showing dense acute and chronic inflammatory cells comprising of polymorphonuclear leucocytes, lymphohistocytes,

ill-defined granuloma with few multinucleated giant cells of Langhans type [fig 6] was revealed and a final diagnosis of granulomatous inflammation [leprosy/TB] was given. Acid fast bacilli staining of sputum samples was positive. [fig7]. Patient was refered to a dermatologist and the final diagnosis of Borderline Tuberculoid Hansen`s disease was confirmed, basing on hypopigmented area on the right side of face and impaired sensation in that region. Patient is on MULTIDRUG THERAPY since with relief in symptoms [fig 8, 9, 10, 11, 12, 13].

DISCUSSION

In 1985 Wiesenfeld introduced the concept of orofacial granulomatosis to classify diseases causing granulomas in the orofacial region with the absence of any systemic manifestations. It includes a variety of clinical conditions such as tuberculosis, leprosy, sarcoidosis, Crohn's disease, and cheilitis granulomatosa and all are considered in the differential diagnosis as they show similar clinical presentation.[13]

Leprosy, a granulomatous disease is caused by mycobacterium leprae also called as Hansen's *bacillus* after the person who discovered the organism in 1874. Mycobacterium leprae is an acid fast *bacillus* similar to *Mycobacterium tuberculosis*. The organism affects almost any part of the body but has more predilection for skin and superficial nerves.[14]

CLASSIFICATION: [15]

D. S. Ridley and William Jopling [1962] proposed their classification for leprosy which includes:

- Tuberculoid (TT)
- Borderline tuberculoid
- Mid borderline
- BL
- Lepromatous (LL).

However in India, leprosy is classified and simplified into the following types:

- TT
- Borderline
- LL
- Intermediate
- Neuritic.

Chronic rhinitis leading to ulceration of the nasal mucosa is the first sign of lepromatous leprosy. Localised swelling, with deep furrows develop on the facial skin. Ulcerated nodules, swollen upper eyelids which create a sleepy appearance ensue later. Blindness subsequent to scleral and iris damage may happen. The nodular dermal manifestation often referred to as "leonine facies" is a facial deformity characteristic of leprosy. Multiple skin macules are present in various cooler regions of body appearing hypopigmented in dark skinned people and erythematous in those with light skin.[16] Oral lesions in leprosy are seen mostly in the lepromatous form.[17] These lesions are seen as enanthemas, ulcers, perforations and scars, papules, nodules (lepromas) and superficial erosions. They can involve the following areas:

1. Palate: Most commonly affected area.[18]The most varied types of lesions are observed: infiltration, ulceration, perforation, and reddish or yellow-reddish nodules, sessile or pedunculated, varying from 2 to 10 mm, some confluent, and prone to ulceration.

2. Tongue: The dorsal surface of anterior two-thirds of tongue is affected in 17% to 25% of the cases. Superficial erosions with loss of the papillae and longitudinal fissures have been described to nodular infiltration that could lead to a "paving stone appearance"[19] Scarring can also occur. The muscles of the tongue do not exhibit greater number of bacilli unlike other subcutaneous muscles.

3. Uvula: In extreme cases there is intense fibrosis with partial loss or even complete destruction of the uvula.[20]

4. Lips: Swelling of lips - macrocheilia (caused by infiltration) or microstomia (caused by ulceration and subsequent repair with fibrosis of perioral lip lepromas)[21]

5. Gums: They are usually affected in the area behind the upper central incisors, often by contiguity, of lesions of the hard palate. Chronic gingivitis, periodontitis and periodontoclasia may occur.[22]

Diagnosis of leprosy:

These three features are essential in diagnosis of leprosy

• Enlargement and/or tenderness of a peripheral nerve
• Loss of sensation in plaque type skin lesions
• Acid fast bacilli in smears.

Diagnosis of Tuberculoid Hansen`s disease: [23]

Clinical criteria:

1. Number of lesions 3 to 10
2. Presence of mother, daughter lesion.
3. Well defined borders of the lesion

MDT is the mainstay of leprosy treatment as it not only cures the patient, but also reduces the reservoir of infection thereby interrupting the spread of disease. Two types of drug regimens are generally used for treating leprosy patients and depending on the severity patients are divided as:

• Paucibacillary leprosy - 2 drug regimen for 6 months: Rifampicin (600 mg) monthly supervised and dapsone (100 mg) daily.

• MB leprosy - 3 drug regimen for 1 year: Rifampicin (600 mg) with clofazimine (300 mg) monthly supervised and dapsone (100 mg) with clofazimine (50 mg) daily. Symptomatic and supportive measures such as adequate oral hygiene maintenance.

CONCLUSION

Leprosy should always be considered in the differential diagnosis of chronic granulomatous lesions in an endemic country. The diagnosis depends upon the correlation of historical and clinical data with histopathological findings.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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