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Oro-Facial Manifestation In Advanced Leprosy-A Case Report

[1*]Dr. B.N Padmavathi, [2]Dr. Pooja Dhakad, [3]Dr. Nishita Gautam, [4]Dr. Saba Khan
[1*]MDS, Professor & HOD, [2,3]PG Student, [4]MDS, Professor, PhD Scholar,
Department of Oral Medicine and Radiology,
Darshan Dental College and Hospital, Loyara, Udaipur-313011, Rajasthan, India

***Corresponding Author:**
Dr. B. N Padmavathi
MDS Oral Medicine and Radiology, Professor and HOD, Dept of Oral Medicine and Radiology
Darshan Dental College and Hospital, Loyara, Udaipur-31301,1Rajasthan, India.

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Abstract

Leprosy is a chronic multisystem granulomatous illness caused by Mycobacterium leprae. Primarily it affects skin and peripheral nervous system. Oral manifestations appear in late stages of lepromatous leprosy and occur in 20-60% of cases. The case report discusses oro-facial features seen in a leprosy patient with advanced disease, imaging features and treatment options

Keywords: NIL

Introduction

Leprosy is a chronic infectious disease with interpersonal transmission caused by Mycobacterium leprae.[1] It is an intracellular parasitic gram positive organism. It is 0.3−0.4 × 4.0−7.0 μm in size and multiplies very slowly, with a generation time of 12–14 days. Optimal growth occurs at about 30 °C; hence, M. leprae prefers the cooler areas of the human body.[2] Primarily it affects the skin and peripheral nervous system but it may also involve another system of the body like gonads, joints, lymph nodes, liver, kidney, eyes and oral mucosa.[1,3]

M. leprae invades and lives within macrophages, dendritic cells and Schwann cells. Bacteria multiply within the Schwann cells. The incubation period between initial infection with the organism and the development of clinical features is usually between 2 and 7 years.[2,4] Spread of leprosy occurs due to close and prolonged contact between a susceptible individual and a bacillus-infected patient through inhalation of the bacilli contained in nasal secretion or Flügge droplets. The main route of transmission is the nasal mucosa, less common route of transmission is skin eruptions. Other transmission routes are blood, vertical transmission, breast milk, and insect bites.[5]

It is commonly present in less developed countries and most often affects the low socioeconomic status countries. Currently India has 0.54 cases per 10,000.[6] However, due to lack of awareness there are advanced leprosy cases

Case Report

A 53 year old male patient complained of pain in upper right back teeth from 10-15 days. Pain aggravated from dull-aching to sharp-shooting continuous pain in the last 3-4 days. It radiated to right forehead region. Swelling was present on right side of the face from 3-4 days.

He was diagnosed with leprosy 25 years ago. He was on MDT including Rifampicin and Dapsone. He was also on Ferrous fumarate and folic acid tablets for anemia and Tab Nortryptiline 25 mg for neuropathic pain. He failed to take the drugs regularly. He consulted a dermatologist from 2002 only for symptomatic treatment.

He was asthenic, malnourished and walked with assistance. The vital signs were within normal limits. Skin was dry and scaly with an active skin eruption on right leg Hoarseness of voice and pallor was noted. Bilateral submandibular lymph nodes were palpable measuring approx 0.5 cm in size, mobile, non-tender. There was partial loss of digits (subphalanges) in middle and little fingers of both the hands and partial loss of first and second toe (distal phalanges) of left foot.

Diffuse swelling with obliteration of nasolabial fold was seen on right side of the face. It was soft, tender and fluctuant on palpation. Frontal bossing, coarse hair with scanty eyebrows and eyelashes bilaterally, deformed nose, saddle nose appearance was noted. Palpation revealed hyperesthesia in relation to bilateral cheek region (Fig A).

Sloughing was present on the upper labial mucosa. Multiple painless lepromatous lobulated nodular-ulcerative lesions were present in the gingiva and hard palate. Deep fissuring of hard palate, loss of uvula, bluish red gingiva with multiple soft swellings, bleeding and ulceration were noted (Fig B).

Extensive bone loss with loss of floor and medial wall of bilateral maxillary sinus, loss of floor of right orbit, floating tooth appearance in the maxillary arch, displaced 23 were noted on panoramic radiograph and CBCT images. (Fig C&D).

The canine space abscess was treated with tab Ofloaxcin 400 mg, tab Ibuprofen with Paracetamol for 7 days. Recall visit showed improvement in pain but no improvement in the swelling. He was referred to Leprosy centre for MDT therapy.

Discussion

Mycobacterium leprae enters the body through the nose and then invades skin and nerves via circulation which produces a physiological response mounted by the host that dictates the clinical phenotype to develop.[3,8] People with leprosy show a spectrum of clinical types. Tuberculoid disease is the outcome of high cell-mediated immunity with a largely Th1 type immune response where as lepromatous leprosy is characterized by low cell-mediated immunity and a humoral Th2 response.[5,9]

Lepromatous disease may be undetected for many years before diagnosis. The most common organs involved are the skin, monocyte–macrophage system and peripheral nervous system.[1] The early skin changes are widely and symmetrically distributed macules, poorly defined with mild hypopigmentation and erythema. Flesh coloured or erythematous papules and nodules may be present. Peripheral oedema of the legs and ankles leads to increased stasis. If skin is left untreated it leads to thickening which causes dermal infiltration and gives rise to the 'leonine facies'. Hair is lost from affected skin areas mainly from eyelashes and eyebrows.[3,7.]

The sensory, motor, and autonomic functions of perip heral nerves are all affected by nerve involvement in l eprosy. Sensory loss is the earliest and most frequently affected modality but a predominantly motor loss can also occur which leads to disability and deformity.[3,10] Bacteria multiplies within the Schwann cells, which leads to foamy degeneration of the cells and loss of ability to regenerate which leads to hyperesthesia. The persistence of the bacteria and the subsequent destruction of nerve fibres are the cause of anesthesia in hands and feet.[11]

The nasal mucous membrane is frequently involved leading to lepromatous rhinitis, chronic nasal discharge, atrophy and ulceration, followed by involvement and necrosis of the alar and septal cartilages. It leads to development of a saddle nose. Other signs seen are hoarseness, stridor, asphyxia (laryngeal infiltration).[3-12]

Oral manifestations usually appear in late stages of lepromatous leprosy and occur in 20-60% of cases. The lesions are located on hard and soft palate, uvula, on the underside of the tongue, lips and gums. These ulcers lead to perforation of palate and loss of periodontal support for the upper incisors, which exfoliate eventually. In very advanced cases this process may spread and involve canines.[1,3-15]

Usually 95% of bone invasion occur indirectly via lesions in the surrounding soft tissues or blood and secondary to peripheral neuropathy. The skin areas anaesthetized by the nerve infection are more prone to tissue infection. This causes non-leprous arthritis and septic osteomyelitis, as well as bone degeneration and limited movement due to muscular paralysis. The phalanx and metacarpal or metatarsal bones are often affected, with marked shortening that may leave only a stump (leprous osteitis).[3,16]

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Many additional organs and organ systems, including the liver, kidneys, eyes, lymph nodes, bones and joints, and gonads, show a slight to moderate degree of infiltration.[1,3]

MDT therapy which contains Rifampicin 600 mg, Dapsone 300 mg and Ofloaxcin 100 mg is given for 12 months. One especially important measure is the provision of appropriate footwear for patients with anesthetic feet liable to plantar ulceration (simple sandal, fitted wih a microcellular insole).[17,18] For claw hands, a home self care exercise program, and intermittent use of dynamic orthoses, elective nerve surgery are recommended.[19]

Before elective dental reconstruction, a full sensory, motor assessment, occupational and functional appraisal is required. MDT therapy must be completed and negative skin smear by slit-scrap technique must be obtained.[19]

Soft tissue flaps like buccal fat pad, free mucosal graft, temporalis muscle graft or tongue graft can be used for reconstruction. Hard and soft palate obturator can be given. Education of the diseased individual and their family plays a crucial role in the management of leprosy. General community-based projects involving family and the wider community have been shown to help best with rehabilitation.[3,19-20.]

Conclusion

It needs to be highlighted that while examining a leprosy patient, examination of the oral mucosa should be an integral part. The role of dental specialist is of great importance in early diagnosis of oral lesions. It is believed that oral lesions along with nasal lesions form an important source of bacillary transmission in the community. Early detection of disease and effectiveness of MDT therapy lead to the reduced number or absence of patients exhibiting oral manifestations of leprosy.

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Figures Legends

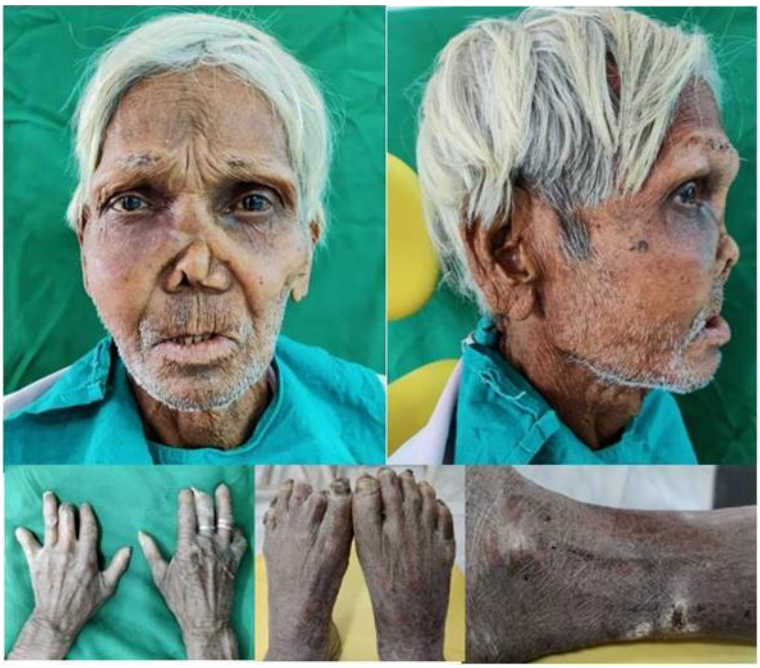

Fig A- 1. Facial swelling on right side of the face, 2. Frontal bossing

3.4. Partial loss of subphalanges in both the hands and left leg. 5. Active skin eruption noted on right leg

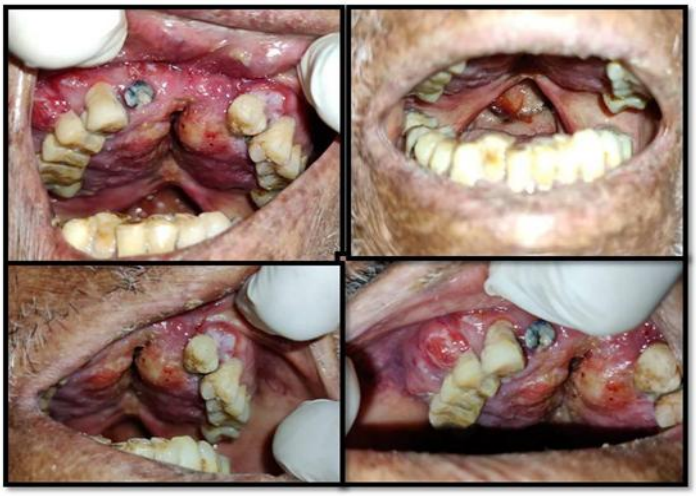

Fig B- 1.Deep fissuring with multiple lepromatous nodules seen in hard palate,2.Loss of uvula, bleeding, 3,4. Multiple swellings with ulceration noted in gingiva of maxillary arch

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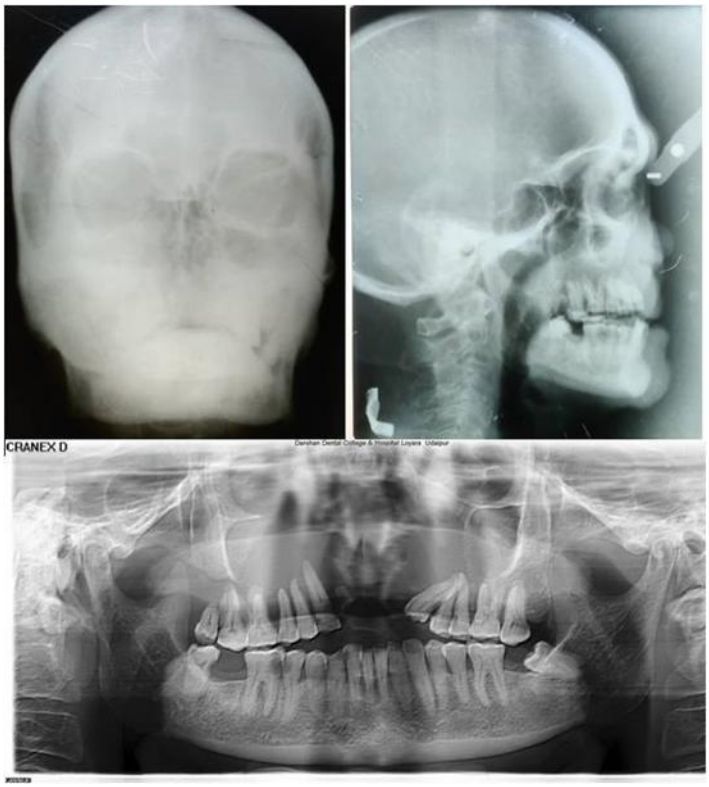

Fig C- 1. Complete opacification of right maxillary sinus and partial opacification of left maxillary sinus noted. 2. Frontal bossing noted. 3. Extensive bone loss noted in maxillary arch with loss of floor and medial wall of maxillary sinus

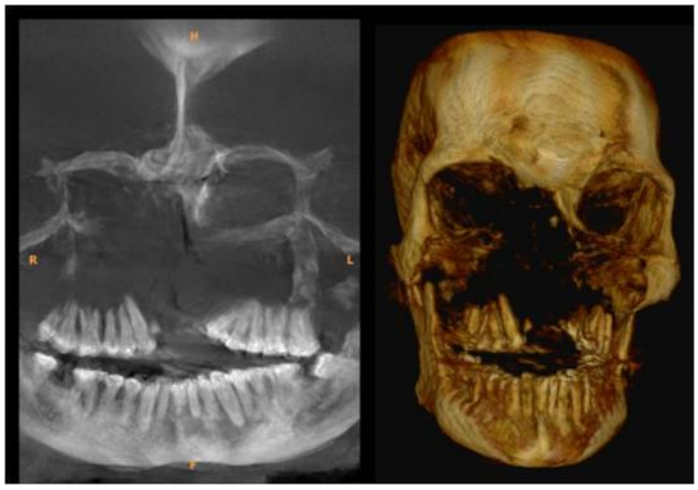

Fig D- CBCT reveals extensive bone loss in maxillary arch, loss of medial and floor of maxillary sinus bilaterally with loss of floor of right orbit

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