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Oral reflections of hansen's disease: A brief review

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Abstract
Hansen's disease is a non- fatal contagious disease caused by Mycobacterium Leprae. This disease can manifest widely in the oral cavity. Hence regular and proper evaluation of the oral manifestations of patients with Hansens disease can further avoid wide complications as this disease can cause cutaneous lesions, lesions of peripheral nerve, and several orofacial manifestations which we will be discussing in this brief review.

Keywords: Hansen's, widely peripheral, Leprae

Introduction

Leprosy is a chronic contagious disease caused by a gram-negative, acid fast bacillus Mycobacterium Leprae [1-5]. Hansen's disease, also called as leprosy is a public health problem globally [7]. Leprosy can be divided into two types depending on the bacillary load as: paucibacillary and multibacillary [1]. The paucibacillary type is mainly due to low bacillary load and is characterised by small well-defined borders of the anaesthetic lesions along with hypopigmentation [1]. this type affects the peripheral nervous system in its early stages and is mediated by T-Helper 1 immune response [1]. The other type namely multibacillary is due to high bacillary loads, and infiltrative skin lesions which may affect the peripheral nerves and may involve the internal organs [1]. This type is mediated by Th2 immune response [8].

Lesions of oral cavity are common in the multibacillary type [9]. In advanced stages of the disease, there is involvement of the hard plate. The involvement of oral mucosa is of great epidemiological significance in leprosy patients, as the association of oral mucosa along with nasal mucosa forms an important pathway and source for the transmission of this gram negative bacilli [6, 10, 11].

The clinicopathologic picture of Hansen's disease is determined by the association between the invasion of the organism and the patient's immunity in responding to it [6]. Mainly the immune system, the skin and the peripheral nervous system are affected by this disease [6]. Other organs like the liver, eyes, kidneys, oral mucosa, bones, joints, lymph nodes may also be affected slightly to a lesser degree due to mild infiltration of the disease [6].

Epidemiology and Pathogenesis

The prevalence of involvement of oral cavity in patients with Hansen's disease is about 19-60% in the world. The oral lesions are usually associated with multibacillary type as compared to the paucibacillary type of leprosy [6, 1, 12, 13]. Martins *et al* recently studies that lesions associated with Hansen's disease are not seen in patients undergoing treatment due to a fair response to the multidrug therap [6, 14]. Mycobacterium leprae usually sustains better in cold regions of the body [6]. This throws light on the preferential sites that are affected in leprosy, mainly the nasal mucosa and peripheral nerves [1]. The inclination of gender predilection in case of Hansen's disease tends to bend towards males as women may consult a doctor earlier due to esthetical issues [22, 26].

The skin and the nasal mucosa are the two main exit routes of M. laprae in human body [40]. Literatures quote that M. Laprae may be found in the superficial keratin layer of the skin of patients with Hansen's disease with a lepromatous type of leprosy implying that M. Laprae could exit via sebaceous secretions [41, 42]. Patients with lepromatous type of leprosy may contain

10,000-10,000,000 of bacilli in their nasal mucosa and in their nasal secretions. These bacilli can be collected by asking the patient to blow his nose [43, 44].

The minimum incubation period of Hansen's disease as reported in a few case studies is as short as of a few weeks and is based on rare occurrence of this disease in pediatric patients, however measuring this period may be difficult due to lack of availability of immunological tools and a slow onset of this disease [45].

A maximum incubation period of approximately 30 years or over is reported among war veterans otherwise residing in non-endemic areas but exposed in endemic areas for short durations. It is discussed though that the average incubation period of Hansen's disease may be between three and ten years [46].

An observation proved that skin was associated with a high bacterial index along with a low mean skin surface temperature which was found to be 32.5°C when compared to the high mean temperature of skin which is 33.46°C. The occurrence of oral lesions is directly proportional to the direction of disease, which implies it as a late manifestation [16].

Scheepers *et al* [6]. found that the most affected site of oral involvement in patients with Hansen's disease is the hard palate, wherein tongue, lips, soft palate, buccal mucosa, labial maxillary gingiva, labial mandibular gingiva being the other less common sites of involvement [6]. These sites were found to be associated with their mean surface temperatures [6, 17]. This implies that lower the mean skin surface temperature, higher will be the frequency of involvement [6]. The anterior palate possesses a mean surface temperature of 27.4°C in about 75% of cases with oral lesions [6]. the patients with lepromatous leprosy usually suffer from mouth breathing due to nasal blockage and obstruction leading to stuffiness [6]. This leads to low mean surface temperatures, especially involving the tongue and the hard and soft palate [6, 18, 19].

Pinkerton in 1932 elucidated the concatenation of the various pathologic changes that occur in the nasal and mucous membrane [1]. Due to a positive response to multidrug therapy, there are less leprosy related lesions as observed by Martins et.al recently in his studies [5]. Mycobacterium Laprae is usually present in nasal mucosa and peripheral nerves which are relatively cooler regions of our body [5]. Mucosal congestion is the first change which in turn is followed by nodule formation and infiltration resulting in severe fibrosis and ulcerative lesions and atrophy of the mucosa ultimately leading to facial mutilation and dentofacial deformities and several functional abnormalities [1].

To understand the morphology of oral lesions occurring in Hansen's disease can be of a bit challenge as the lesions are initially asymptomatic in nature and may grow or progress gradually as time passes by [1]. The occurrence of these oral lesions may also be secondary to nasal changes [22, 23, 24].

The oral lesions in Hansen's disease may vary morphologically from enanthemas to ulcerations/ulcers. They can also progress to scars or perforations, passing through papules or lepromas (nodules) and superficial erosions [22]. The oral lesions may involve palate, tongue, uvula, lips and gums. Lesions of the palate can be varied and can be observed as infiltrations, ulcerations or perforations which may further lead to oroantral fistula. Many authors have found different regions of palate to be the sites of oral lesions. Some have found severe lesions in the mid-anterior Region of palate while some found them occurring on the soft palate, the latter being a common one [22, 27, 28]. In later stages, patients may experience functional abnormalities while difficulty in swallowing, eating or drinking. Some studies have reported that there may be destruction of the hard and soft palate due to erythema nodosum leprosum, a rare cause. In cases of lesions of the soft palate, the mucosa over the soft palate,uvula and the faucial pillars of tonsils may get infiltrated and may give rise to a military popular or nodular appearance. These lesions further get converted into superficial ulcers during leprosy reactions.

The dorsum and anterior 2/3rd of tongue are also the most common sites of involvement in approximately 17% to 25% of the cases. Superficial leions can transform from having loss of papillae and longitudinal fissures to having nodular infiltration[22] which leads to a "paved stone appearance" In some cases scarring can also be seen. Severe lesions may occur on the base of the tongue secondary to highly infectious nasal secretions passing from the nasal cavity to the oral cavity and this was suggested by Mukherjee [30]. and Bucci *et al* [31]. The uvula mat also be affected and in severe cases there may be severe fibrosis along with complete or partial destruction of the uvula [22].

Due to infiltration there may be macrocheilia of the lips or microstomia due to ulcerations and repair of perioral or lip lepromas with fibrosis [22, 32, 33]. The gums are affected usually in the resgion behind the upper central incisors especially as a continuation of lesions of the hard palate [23]. The gums may become swollen and may bleed easily. There may be decreased sensitivity to pain and the mucosa may appear shiny [1]. The buccal muscosa may appear pale initially while in the later stages it may lead to development of swelling, occurrence of nodules or papules or diffuse infiltrations or even ulcerations [1].

The oral mucosa in advance stage of Hansen's disease may lose its shine and may have a matt like appearance [34]. Some literature quote the occurrence of verrucous carcinoma in patients with Hansen's disease [35]. But their association is still not established [1]. Some literatures have quoted that Hansens disease may cause infections like gingivitis, periodontitis and periodontoclasia [1].

Martin., *et al*. reported an association between poor oral health and Hansen's disease irrespective of the presence of any facial destruction or the type of leprosy [36]. There may be involvement of the cranial nerve in Hansen's disease, the facial nerve and the trigeminal nerve being the most commonly affected nerves [37, 38]. The trigeminal nerve may affect its maxillary and mandibular branches and may cause ipsilateral anaesthesia to the face and may cause loosening within teeth. The examination of these two cranial nerves is of utmost importance as they may manifest widely. There may be difficulty in speech and mastication if the buccal and the mandibular branches get involved [39].

A histopathological examination may reveal a major difference between the presence of an enanthem and a specific lesion. An enanthem may show non-specific infiltrates while presence of a bacilli may be indicative of a specific lesion [25]. The lesions occurring in a treated case of Hansen's disease may show a dry surface and may often show melanin in the basal layer without in the melanocytes at the dermoepidermal junction [47]. The histopathological section at first may be suggestive of a pigmented lesion, but when clinically biopsied the lesion may be hypopigmented [47].

Conclusion

Patients with Hansen's disease must be treated and examined with great care as there may be risk of infection to the treating oral health care practitioner who is treating a patient with Hansen's disease. A dentist must routinely examine a patient of Hansen's disease with proper follow up in order to avoid wide complications. He must be aware of the transmissible infections and hence must wear all the precautionary eqipments like gloves, mask or a scrub. Oronasal lesions and infections may cause dissemination of Hansens disease amongst the population. Hence its very important to deeply evaluate the oral reflections of Hansen's disease in order to keep a tract on the oral health of the patient with Hansen's disease and provide them a nurturing oral care.

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