Bilateral Parotitis, Cervical Lymphadenopathy and Oral Hairy Leukoplakia Presenting as the Initial Clinical Manifestation Of HIV Infection

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INTRODUCTION

The clinical signs and symptoms of the oral and perioral regions play a very important role in the early diagnosis of the Human immunodeficiency virus (HIV) infection. The altered immunity in HIV infection gives rise to a variety of changes in the head and neck regions. These include generalised lymphadenopathy, bacterial and fungal infections of the oral mucosa, salivary gland enlargement and xerostomia. Recognition of these changes by the clinician is paramount to the long term symptomatic treatment of HIV patients. We report a case of HIV infection in a young healthy woman who presented with bilateral parotitis, cervical lymphadenopathy, oral hairy leukoplakia and xerostomia.

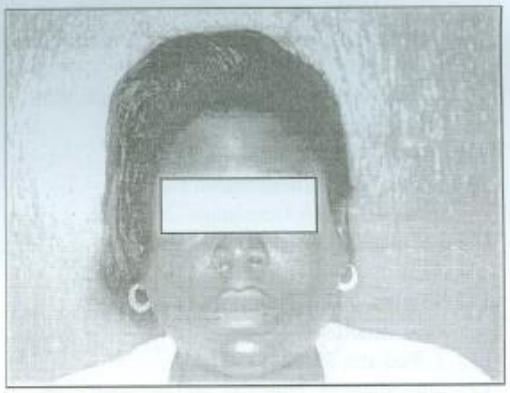

FIGURE 1: Patient showing bilateral parotid enlargement and elevated lower lobes of the ears

CASE REPORT

A 20 year-old female was referred to the Oral Diagnosis Clinic at the University of Ghana Dental School, with a four- month history of painful bilateral parotid gland enlargement. The salivary gland enlargement started as a small non-tender swelling which gradually increased in size. She had dry mouth, which was particularly severe at night. Her other complaints were malaise, furunculosis, sore throat and difficulty in swallowing.

The extra-oral examination revealed bilateral, very tender and firm parotid gland swelling (Fig 1). There was lymphadenopathy of the submental, submandibular and the superficial cervical lymph nodes. Intra-orally, there was excessive keratinization of the filiform papillae of the dorsum of the tongue. The oral mucosa appeared dry, although the orifices of the major salivary glands were patent. There was hairy leukoplakia which extended from the left retro molar tissues to the buccal mucosa adjacent to the first molar tooth(Fig 2).She had bilateral pericoronitis and enlarged tonsils (Fig 3). The systemic examination was unremarkable. The initial laboratory results were as follows: haemoglobin, 9.4gm/dl; erythrocyte sedimentation rate (ESR), 80mm fall/hr (WEST), leucocyte count, 7.6x10 ^{9}/L; neutrophils, 40%, lymphocytes, 58%; eosinophils, 1%; and basophils, 1%; Two months after the initial referral, the laboratory studies revealed the following changes: hemoglobin, 8.2 gm/dl; ESR, 145mm fall/hr; leucocyte count, 5.8 x 10 ^{9} /L; neutrophils, 40%; lymphocytes, 53% and eosinophils, 7%

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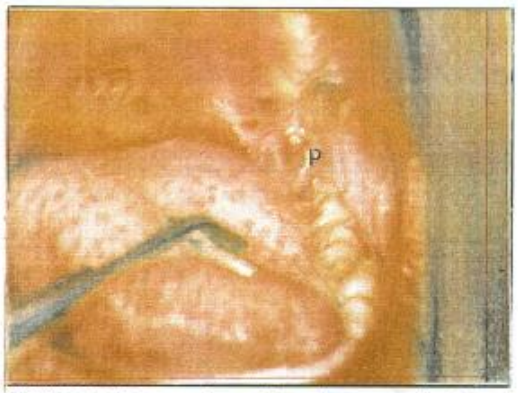

FIGURE 2: Retro molar and buccal mucosa showing leukoplakia and pericoronitis (P) of the third mandibular molar tooth.

Histopathological examination of the labial salivary gland biopsy showed a diffuse chronic inflammatory cell infiltrate. There was destruction of the salivary acini by the inflammatory infiltrate and the intercalated ducts were dilated (Fig 4). The clinical presentation of oral hairy leukoplakia, bilateral parotitis and cervical lymphadenopathy necessitated serological testing for HIV, which was reactive and positive

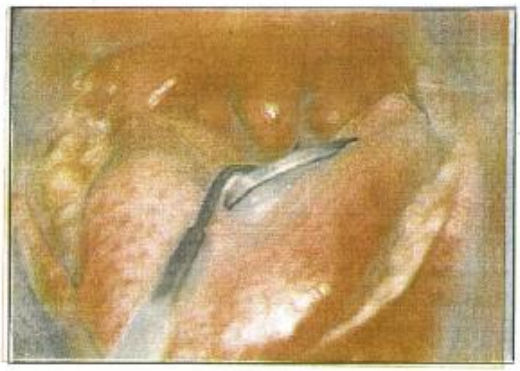

FIGURE 3: Oropharynx showing enlarged tonsils and uvula

DISCUSSION

The term "Human Immunodeficiency Virus – Associated Salivary Gland Disease" (HIV-SGD) is used to describe the presence of xerostomia and/or swelling of the major salivary glands in HIV/AIDS patients.[1]

A review of the literature shows that this condition is common among HIV infected children with vertical transmission of the disease but is uncommon in adults[2,3]. The salivary gland enlargement usually occurs early in the course of the disease and frequently affects the parotid glands. The submandibular and sublinqual glands are infrequently affected[1,2]. The enlargement of the salivary glands may be due to cystic degeneration of the ducts or as the result of the presence of large numbers of lymphocytes in the parenchyma of the salivary gland [2,3].

The origin of the lymphocytes in the parotid gland in HIV infection is not well understood. It has been suggested that the lymphocytes may arise primarily from proliferation within the intraparotid lymph nodes or as a result of massive lymphocytic infiltrate from extra glandular source [3,4]. In a review of 107 reported cases, Schiødt observed that the parotid glands were involved in 105 cases while the submandibular glands were involved in only two cases. The large numbers of lymphoid aggregates in the parotid tend to support the hypothesis that the lesion occurs primarily from proliferation of lymphocytes in intra-parotid lymph nodes. However the labial salivary gland which lacks lymphoid aggregates also exhibits extensive lymphocytic infiltrate as observed in this case report. It is possible that both primary proliferation of lymphocytes within intraparotid lymph nodes and lymphocytic infiltrate from extraglandular source may contribute to the diffuse lymphocytosis of the parotid glands.

Although the histopathological features of HIV-SGD and Sjögren syndrome are similar, the two conditions differ on immunohistopathological examination. The chronic inflammatory infiltrate in HIV-SGD is predominantly the CD8 lymphocyte while in Sjögren Syndrome it is mainly the CD4 type. Serological examinations also reveal elevated antinuclear antibodies and rheumatoid factors in Sjögren syndrome, but this is not a feature in HIV-SGD.[4]

In this case report, chronic cervical lymphadenopathy and associated enlarged tonsils were significant clinical findings. Lymphoid tissue enlargement in HIV/AIDS patient may be due to benign lymph node hyperplasia or to lymphocytosis due to malignant lymphoma [5,7]. A review of histopathological sections of cervical lymph nodes from HIV/AIDS patients showed that majority of the nodal enlargements were benign while a small percentage was malignant [8]. The lymphomas in these patients tend to be very aggressive, [6,7] therefore it is important to rule out malignant lymphoma in all HIV infections.

The possible association of oral hairy leukoplakia and HIV-infection was first reported by Greenspan et al[9]

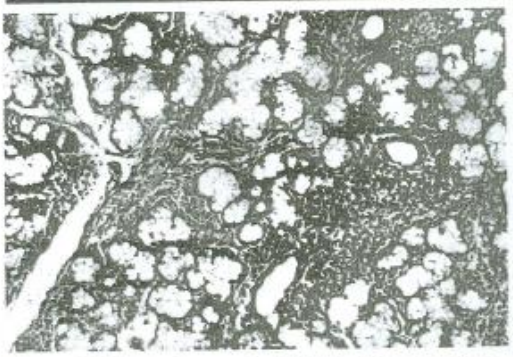

Figure 4: Photomicrograph of labial salivary gland biopsy showing chronic inflammatory cell infiltrate, destruction of the salivary acini and dilated ducts. (Haematoxylin and eosin stain. Original magnification x 100)

in 1984. Since this preliminary report, several studies[10,11] have shown that oral hairy leukoplakia occurs exclusively in immuno-compromised individuals, therefore the presence of oral hairy leukoplakia in the absence of obvious clinical immuno-suppression is predictive of HIV infection. The presence of oral hairy leukoplakia in this case report prompted the request for serological test for HIV.

The management of HIV-SGD patients should include a long-term oral and dental protocol to control the sequlae to xerostomia such as rampant caries, candida infections and mucosal ulcerations. The HIV-SGD patient should be introduced to saliva substitute or sugarless chewing gum to prevent drying of the oral mucosa. Application of topical fluoride gel to the teeth is recommended to prevent rampant caries and daily chlorhexidine mouth rinse should be prescribed to control candida infection.

CONCLUSION

The presence of parotid enlargement, cervical lymphadenopathy and oral hairy leukoplakia in a patient should warrant serological investigation for HIV infection.

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