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Oral Health & HIV

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1. Introduction

HIV and AIDS continues to ravage developing countries especially those in sub-Saharan Africa three decades into the pandemic. It has eroded social, political and economic health gains and dealing with its consequences is one of the greatest challenges of the new the millennium. There has been a decline in life expectancy in adults, an increase in child mortality rates that has reversed the years of hard won gains achieved in child survival, deepening poverty and exacerbation of food shortages. Susceptibility to HIV AIDS is sustained by the lack of economic, social and political influence, including the capability to effectively inform decision makers and policymakers. The majority of people affected by HIV/AIDS are vulnerable populations who live under conditions of gender-based violence, poor access to health care and inadequate living conditions. There is an urgent need for strategies to remove the barriers that prevent them from voluntary testing and counselling and accessing oral health promotion, prevention and treatment services.

2. Importance & relevance of oral lesions in HIV/AIDS

Oral lesions are a common occurrence in individuals with HIV infection. Oral manifestations include fungal, viral and bacterial infections. Neoplasms, periodontal disease, salivary gland disease and lesions of uncertain origin are also seen. Several studies have emphasised the prognostic significance of oral candidiasis and oral hairy leukoplakia as predictors of immuno suppression and AIDS-defining conditions. Lesions such as oral candidiasis, herpetic ulcers and Kaposi's sarcoma are among the first symptoms of HIV-infection. These conditions, although not life-threatening, impact on the quality of life (Yengopal & Naidoo, 2008) and are often associated with significant pain, discomfort, eating restrictions and consequent diminished nutritional intake. The oral cavity may also be a primary source of infection in any individual, which may spread via the mucosal associated lymphoid tissue or stimulate systemic inflammatory immune responses (Chapple & Hamburger, 2000).

The treatment and management of oral HIV lesions can considerably improve well being. Antiretroviral treatment and improved management of HIV, has been shown to reduce the prevalence of oral manifestations, but there may be an increase in oral warts and salivary gland disease (Greenspan et al., 2001). Oral examination is quick and inexpensive and may have a place, especially in primary health care settings and for screening populations at greater risk of HIV. Early detection of HIV-related oral lesions can be used to diagnose HIV infection, elucidate progression of the disease, indicate a response to antiretroviral therapy

and by predicting immune status can result in timely therapeutic intervention. For HIV infection in women it may be useful in ante-natal screening so that appropriate drug management can be instituted to reduce vertical transmission.

3. Oral lesions: Types and management

3.1 Oral candidiasis

Oral candidiasis is the most common oral lesion with a variable clinical presentation. *Candida albicans* is the most common cause. It is manifested as creamy white pseudomembranous plaques, erythematous patches, non-scrapable hyperplastic plaques or as angular chelitis. Once colonisation and super-infection by *Candida* spp. are established, deeper penetration into submucosal tissue may be facilitated by concomitant mucosal infections caused by bacteria and herpes simplex virus (HSV). In children, lesions are often characteristically of the pseudomembranous and erythematous types. Early presentations are often asymptomatic. Candidiasis may be accompanied by pain and an altered taste sensation, both of which interfere with nutrition and hydration and may be exacerbated by decreased salivary flow.

Pseudomembranous candidiasis is the most common variant and presents as creamy white or yellow, loosely adherent plaques anywhere in the mouth and it can be wiped off to reveal an erythematous surface with or without bleeding (Figures 1 and 2). Erythematous candidiasis presents as multiple, flat, diffuse or discrete red non-removable plaques. It is usually found on the palate, tongue and occasionally on the buccal or labial mucosa (Figure 3). A variant of erythematous candidiasis is median rhomboid glossitis – presenting as a smooth, red, depapilated area in the middle of the tongue (Figure 4).

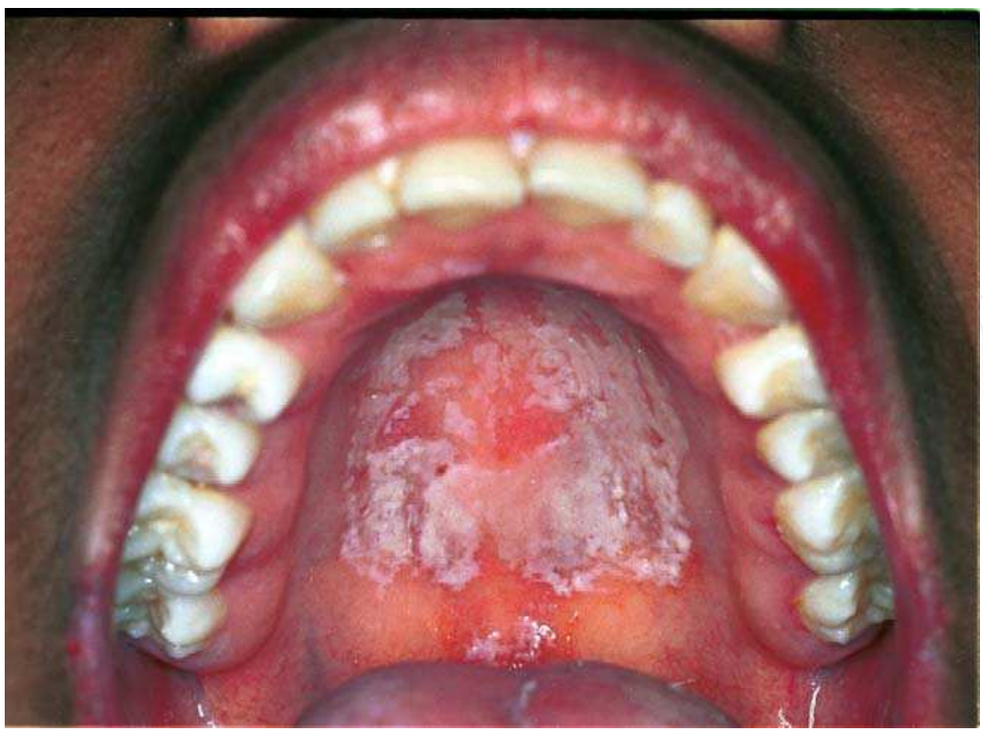

Fig. 1. Pseudomembranous Candidiasis Palate

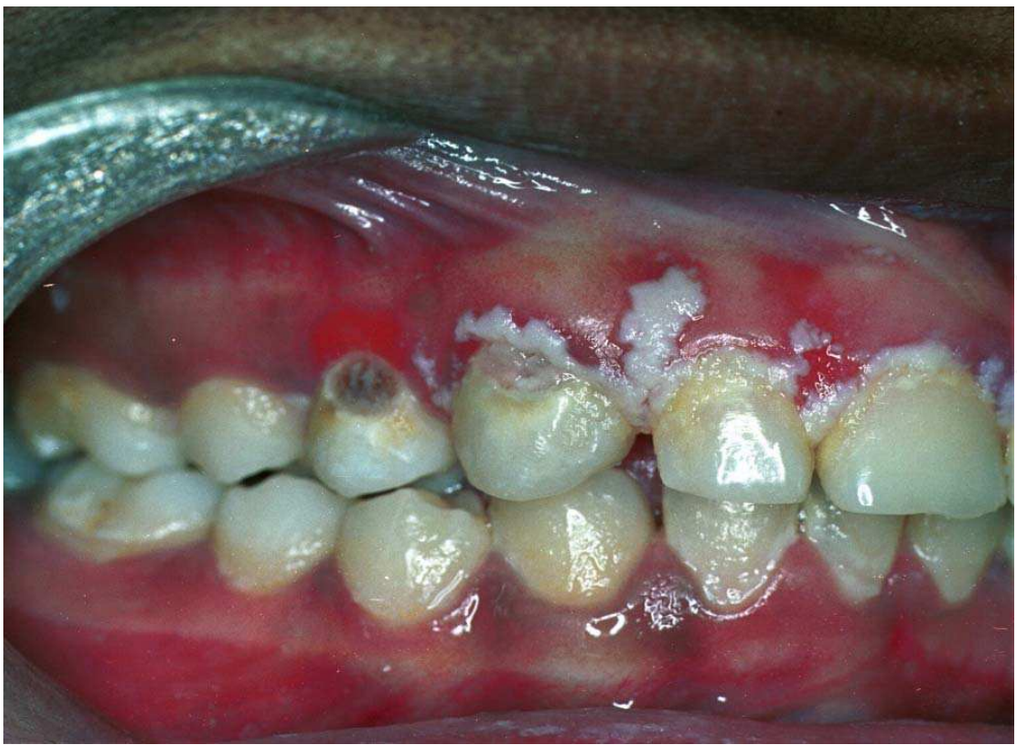

Fig. 2. Pseudomembranous Candidiasis Gingiva (with caries)

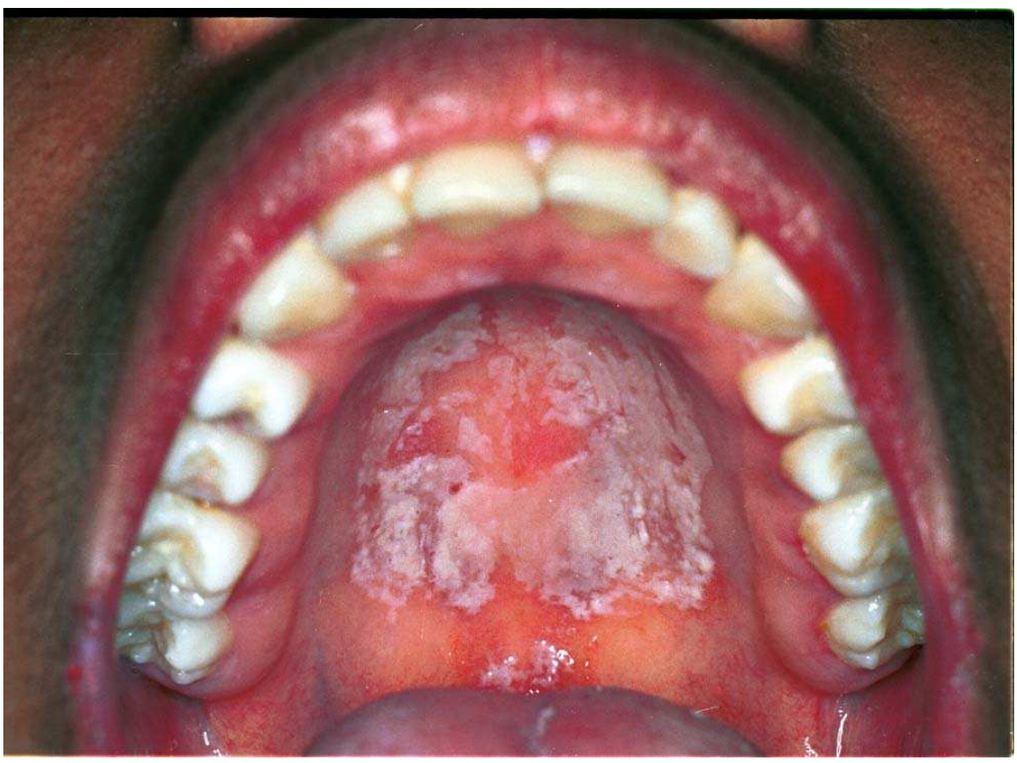

Fig. 3. Erythematous Candidiasis Palate

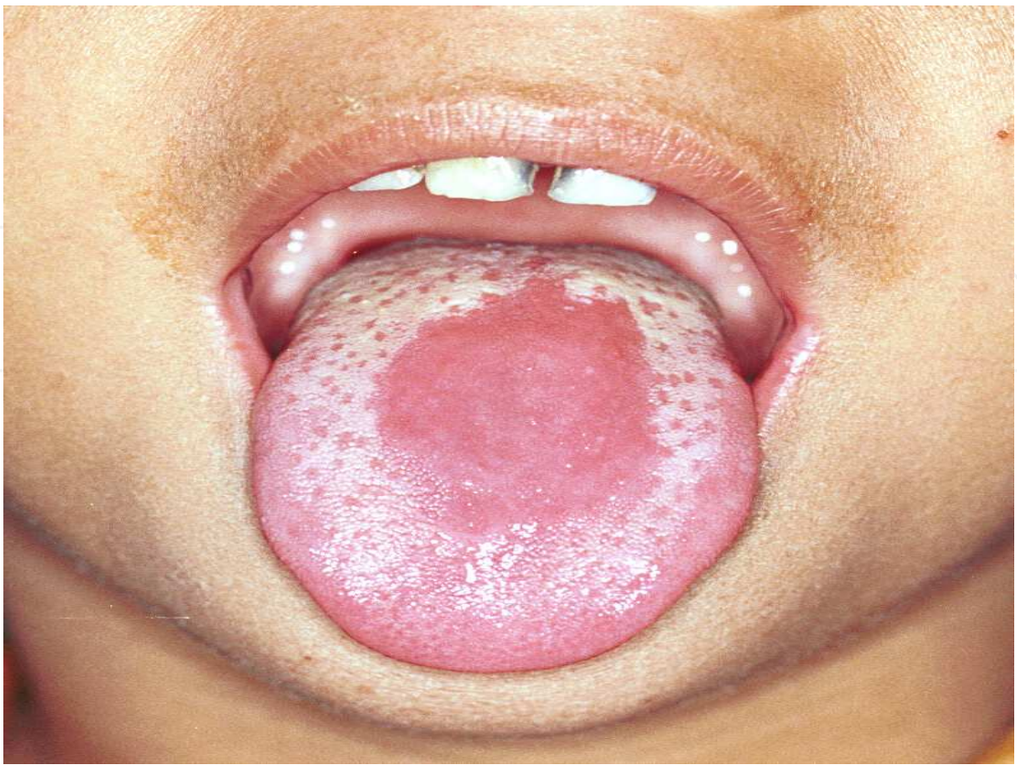

Fig. 4. Median Rhomboid Glossitis Tongue

Hyperplastic candidiasis is usually seen on the buccal mucosa as diffuse, white, adherent lesions (Figure 5). It needs to be distinguished from oral hairy leukoplakia.

Angular cheilitis presents as fissures or linear ulcers at the corners of the mouth, with varying degrees of inflammatory erythema. Hyperkeratosis may be present peripheral to the fissure. Concurrent with angular cheilitis, intraoral candidal involvement is a common clinical finding. The lesions are usually painful and slow to heal because of the repeated opening of the mouth (Figure 6).

Management: Candidiasis should be treated promptly and vigorously and early treatment is warranted, not only because of discomfort, but also because infection may spread to the pharynx and oesophagus. Either topical or systemic antifungal agents may be recommended. Topical treatments include 0.5% gentian violet aqueous solution painted in the mouth three times daily and nystatin suspension 100 000IU/Ml rinse. In severe cases apply 2% micoazole oral gel two or three times daily for ten days and suck amphotericin B lozenges 10mg six hourly for ten days.

Recurrences are common and if there is no response in one or two weeks, systemic agents may be required: Fluconazole 50-100mg daily for seven days, Ketoconazole 200-400mg daily for seven days. Itraconazole 200mg daily for seven days may be useful for azole-resistant *C.albicans* infection. Angular cheilitis is best managed with topical nystatin or for a mixed flora infection miconozole gel.

Due to the high sugar content of some formulations, topical fluoride should be used daily, if frequently prescribed. Dentures should be removed when using medication and local contributory risk factors like continuous denture wear, poor denture hygiene and xerostomia should be eliminated.

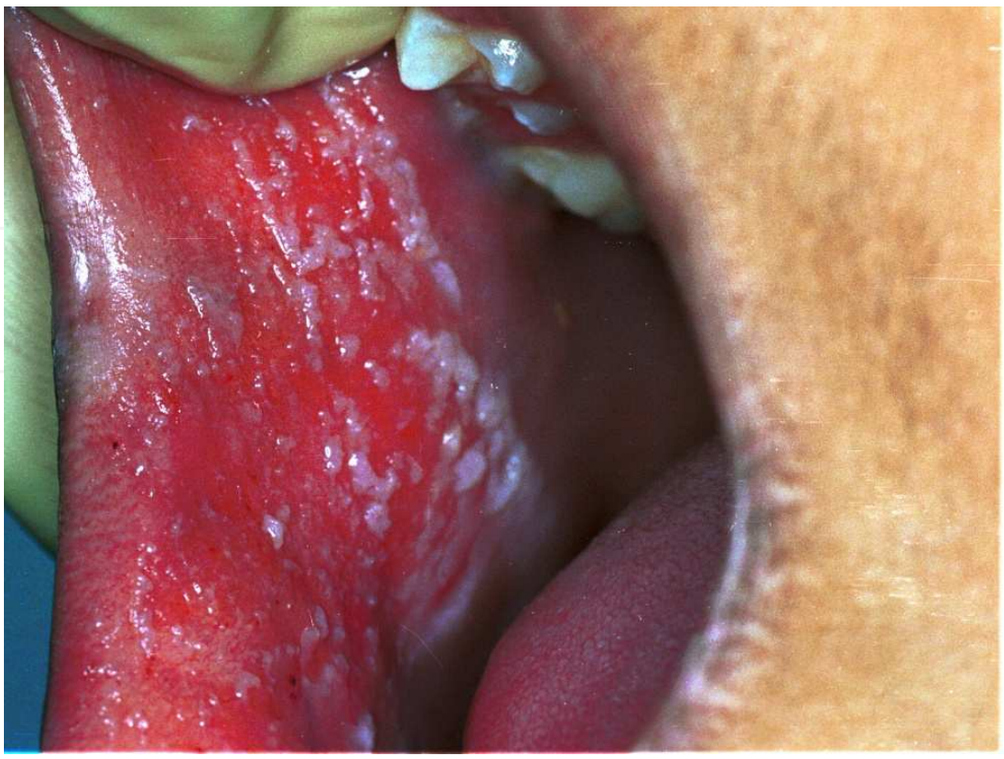

Fig. 5. Hyperplastic Candidiasis Buccal mucosa

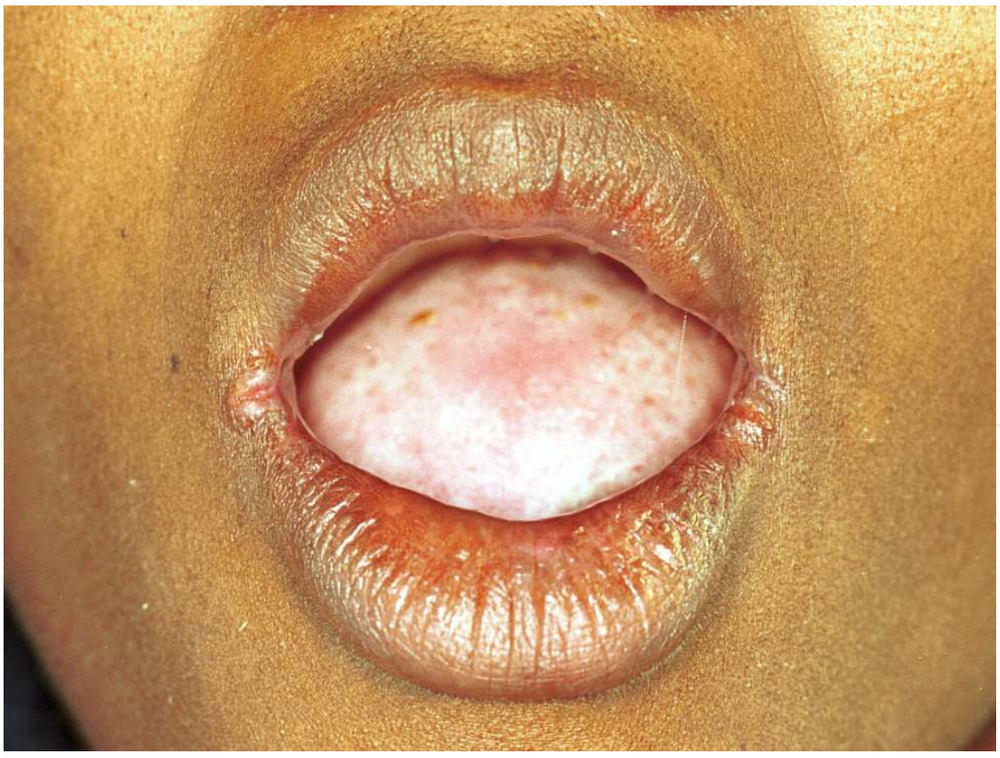

Fig. 6. Angular cheilitis

3.2 Oral hairy leukoplakia

Oral Hairy leukoplakia (OHL) usually presents as white, vertically corrugated projections on the lateral borders of the tongue, unilateral or bilateral and cannot be rubbed off (Figure 7). It is a benign, usually asymptomatic and more common in men. Studies have shown that it is associated with intraepithelial proliferation of the human herpesvirus, the Epstein-Barr virus (EBV) and that multiple strains for the virus are often present in OHL tissues (Ammatuna, 2001). OHL is the only lesions caused by EBV in which the virus replicates and is commnly shed in saliva (Sitki-Green et al., 2002).

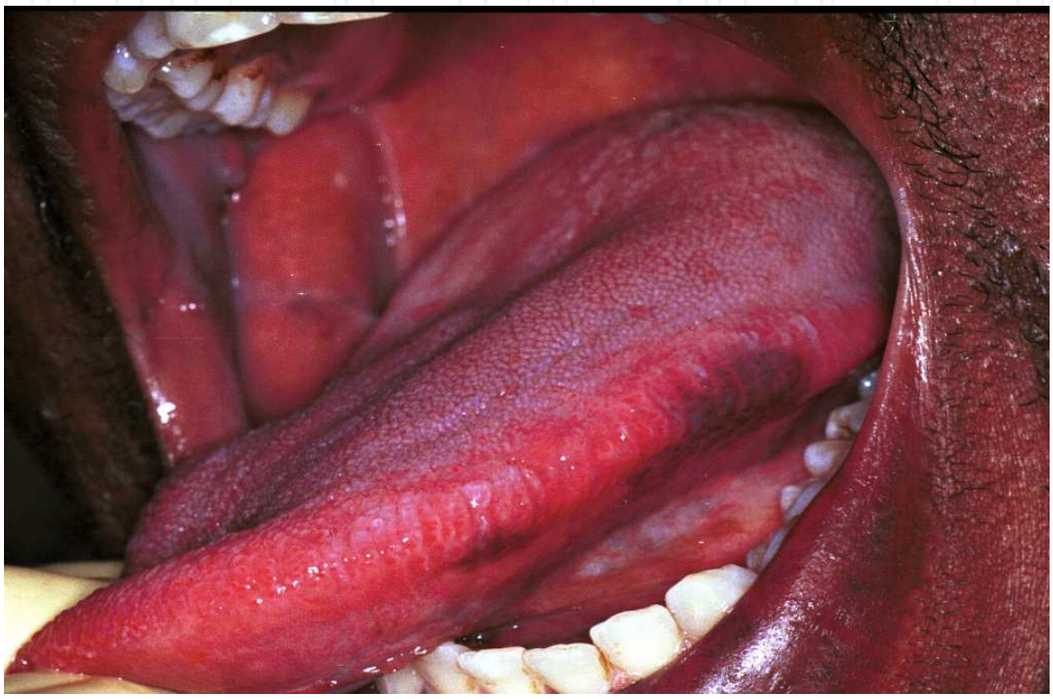

Fig. 7. Oral Hairy Leukoplakia Tongue

Although it may be a source of concern and discomfort to the patient, specific treatment is rarely indicated. Antiretroviral therapy may clear the lesions but the underlying EBV infection is rarely eliminated. Aciclovar 800mg three to five times daily for ten days may be recommended for patients complaining of discomfort. The presence of oral candidiasis and oral hairy leukoplakia in patients receiving antiretroviral medication is indicative of the failure of therapy.

3.3 Oral ulceration

People with HIV present with oral ulcerations of diverse aetiologies. The ulcers may be infectious (viral, bacterial or fungal) or atypical (aphthous or drug-induced) in nature. Herpetic stomatitis caused by the herpes simplex virus 1 (HSV) is commonly seen in HIV-infected patients and has a tendency to recur. Lesions may be seen on the gums, hard palate, vermillion border of the lips and adjacent facial skin (Figure 8). Early vesicles soon rupture to become painful irregular ulcers. In HIV-infected people, the lesions are chronic, recurrent and may progress rapidly to cause extensive mucocutaneous involvement that may persist for several weeks and extend into the oesophagus.

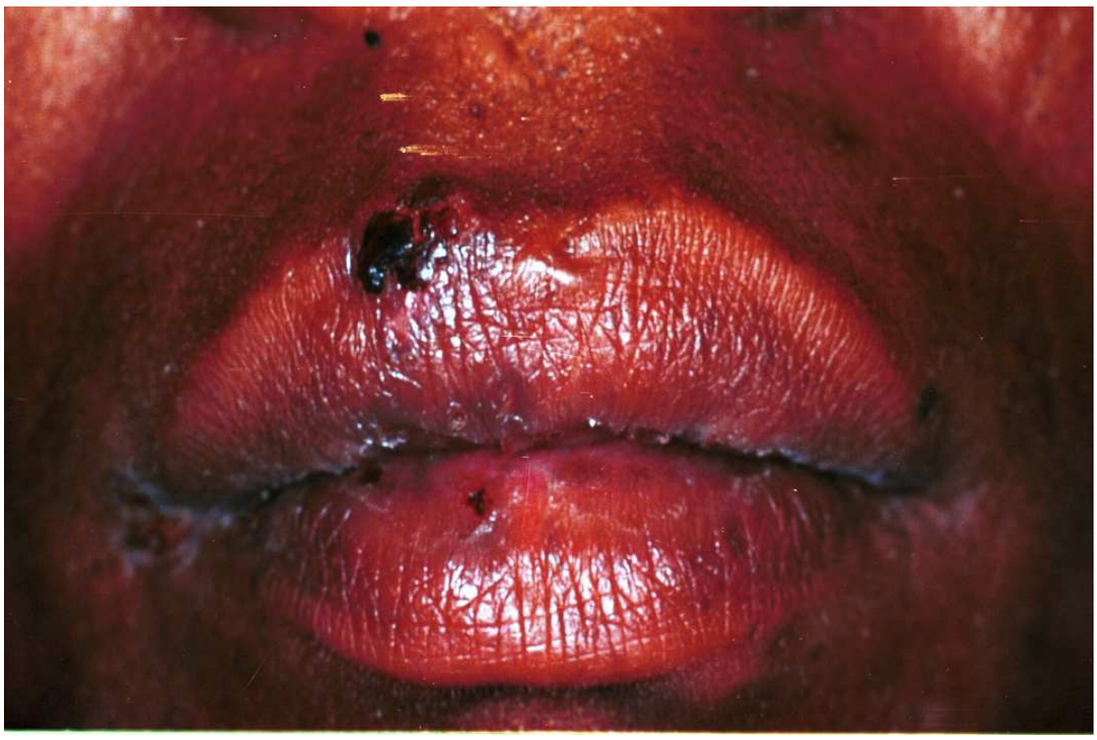

Fig. 8. Herpes Zoster (healing)

It is widely believed that herpes zoster infection (HZI) is a reactivation of latent varicella zoster virus in the dorsal root ganglia. The occurrence of the secondary form of HZI in HIV positive patients, manifests on the skin as localised, disseminated or typical generalised zoster and may herald a poor prognosis.

Aphthous ulcerations can be small or large, single or multiple (minor, major or herpetiform types). Minor aphthous ulcers are painful, round, well-circumscribed with a whitish covering surrounded by an erythematous halo, usually limited to the non-keratinised mucosa of the soft palate, buccal mucosa, tongue or tonsillar area but can occur anywhere in the mouth. Herpetiform ulcers are the least common type and are pinpoint, round and with a perilesional erythema. They are usually found in batches up to a hundred, appearing on nokeratinised mucosa such as the ventral surface of the tongue and soft palate.

Severe recurrent aphthous ulcers (RAU) may occur in the mouth, oropharynx, and oesophagus of untreated HIV/AIDS patients. Aphthous ulcers are deeper than herpetic ulcers and have a well-defined edge, unlike herpetic ulcers, which are shallower with an irregular border. Preceding vesiculation is characteristic of herpetic lesions. Large lesions are progressive, chronic and heal slowly. They often interfere with speech and swallowing and may contribute to inadequate oral intake and rapid weight loss.

Management of oral ulcerations: Early diagnosis and treatment of lesions is important as severe, long-standing, painful lesions may interfere with nutrition and hydration. Treatment is focused on providing symptomatic relief and adequate pain control is essential - paracetamol or paracetamol-codeine with topical 2% viscous lidocaine gel and analgesic mouth rinses are helpful. Herpes simplex lesions often heal spontaneously. A 0.5% gentian violet aqueous solution painted in the mouth three times daily or 1% topical povidone-iodine may be useful for small ulcers. Aciclovir is very useful if administered early – 400mg

eight hourly for five days will effectively treat large ulcers. Intravenous foscarnet has been used to treat acyclovir-resistant HSV infection. Patients sometimes have prolonged bouts and frequent recurrences that are accompanied by severe pain and local tissue destruction. Short courses of topical corticosteroids should be tried, but these ulcers may be resistant to conventional treatment. Thalidomide has been successful in some cases. Viruses may occasionally become resistant and antibiotics may be necessary to limit super-infection.

Treatment for aphthous ulcerations depends on severity of ulcerations. The ulcers respond to topical steroids like triamicinolone acetonide 0.1% in a sodium carboxymethylcellulose base given eight hourly. A 0.2% chlorhexidine digluconate mouth rinse two to four times daily or 1% topical povidone-iodine may also be useful. For large persistent ulcers beclomethasone spray (one or two puffs twice daily on the ulcer), benzydamine hydrochloride mouthwash or betamethasone 0.5mg tablets (dissolved in 15mL water) may be used as a mouthwash for three minutes daily. For longstanding, intractable ulcers, systemic steroids like prednisolone may be used, but it is important to exclude cytomegalovirus (CMV) and herpes, as steroids may exacerbate these ulcers. Recurrences must be treated aggressively. If ulcers persist despite treatment patients should be referred for a biopsy to exclude malignancy or CMV infection.

3.4 Kaposi's sarcoma

Kaposi's sarcoma (KS) is a neoplasm of putative vascular origin. It is a multifocal neoplastic proliferation of endothelail cells. Oral lesions of KS lesions are red, blue and purple and occur commonly on the posterior hard palate and the facial gingiva (Figure 9), with or without ulcerations. Lesions on the palate are associated with pulmonary KS (Pozniak et al., 1992). The African form is characterised by lymph node enlargement. If untreated, they may spread and ulcerate. The human herpes virus 8 (HHV-8) has been identified in all forms of KS and its replication in the oral cavity and viral shedding in saliva are important factors for HHV-8 transmission (Pauk et al., 2000). Recent research has suggested that in certain *in vivo* conditions, oral micoorganisms like *Porphyromonas gingivalis, Fusobacterium nucleatum* and *Prevotella intermedia* can potentially activate HHV-8 to cause disease (Morris et al., 2007).

Lesions begin as flat red macules of variable size and irregular configuration. Although they may appear as a focal lesion, typical oral KS lesions are multifocal, with numerous isolated and coalescing plaques. Eventually the lesions increase in size to become nodular growths and may involve the entire palate (Figure 10).

Nearly two-thirds of patients with oral KS have pain, discomfort or dysphagia, or complain of poor aesthetics and require treatment. In the early stages, the differential diagnosis includes pyogenic granuloma and giant cell granuloma. A biopsy is essential for a definitive diagnosis.

Management: The incidence of oral KS has decreased with the introduction of antiretroviral treatment, but remains the most common oral malignancy in people with HIV. Treatment decisions are made on the basis of the extent of the disease. The early plaque and macular lesions are painless and do not require treatment. Nodular lesions may become unsightly and interfere with mastication. Isolated oral lesions can be treated by laser or surgical excision. Intralesional injections of 1% vinblastine sulphate can cause lesions to regress, however, in some patients it produces pain and may require repeated visits before a response is achieved. Systemic chemotherapy is indicated for patients with widespread progressive disease. Radiation causes mucositis. Benzydamine hydrochloride may cause some relief and is recommended mainly when there are obstructive symptoms. Good oral hygiene and plaque control is essential to prevent secondary infection.

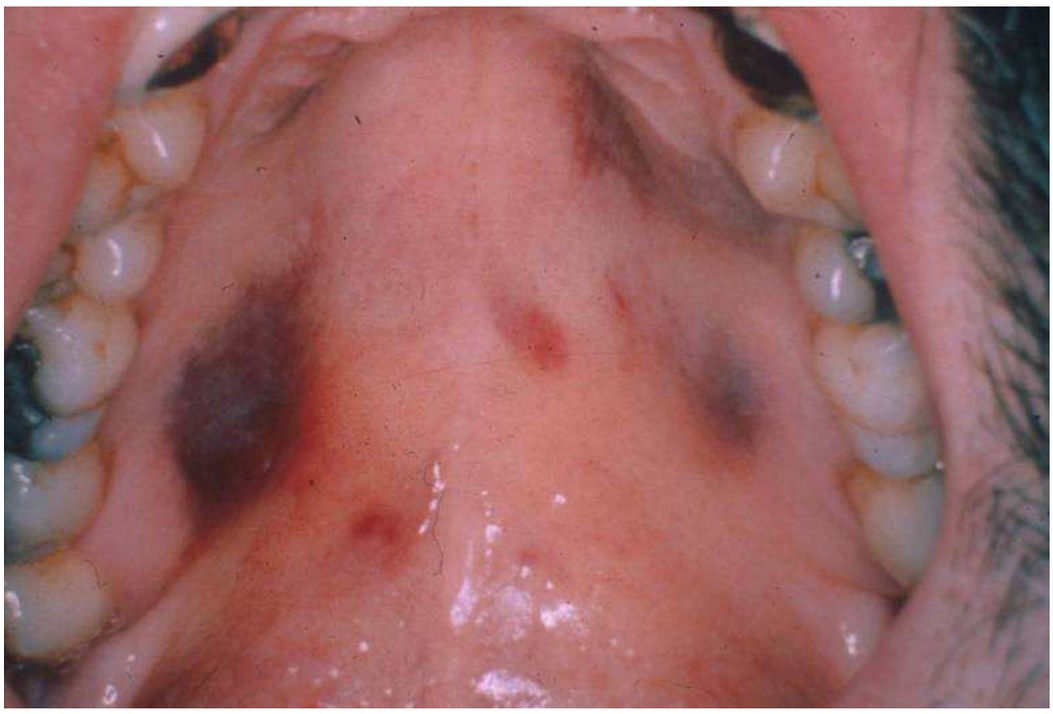

Fig. 9. Kaposi's sarcoma palate

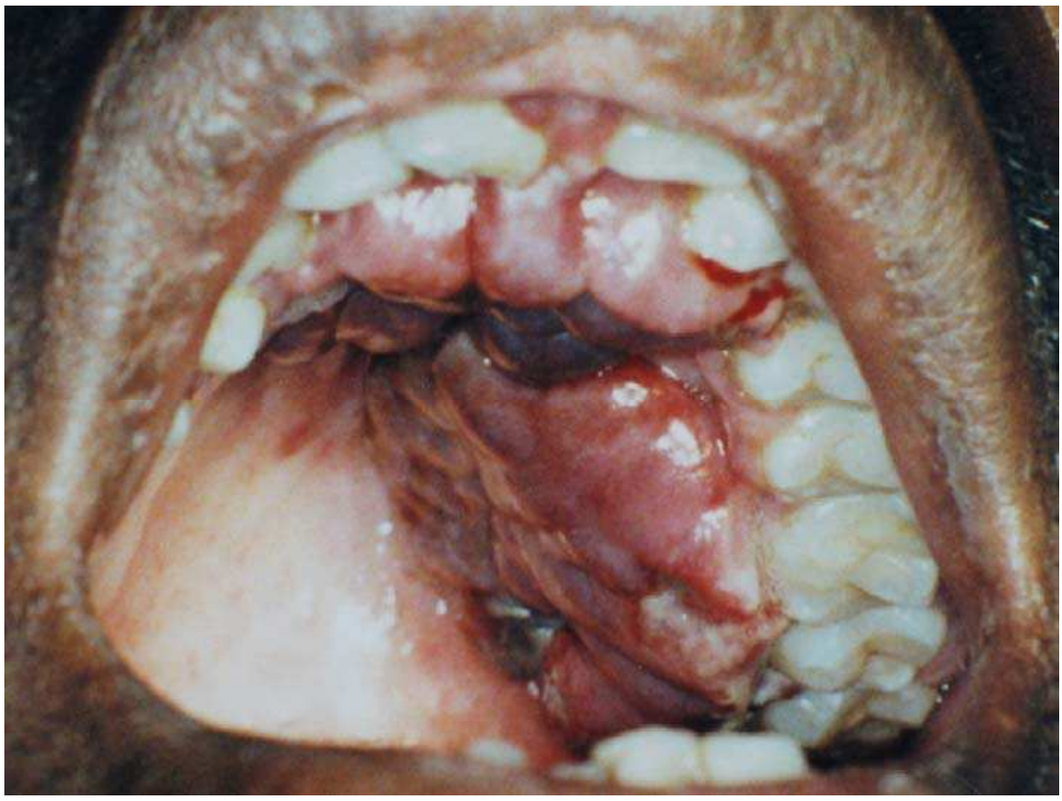

Fig. 10. Kaposi's sarcoma ulcerated

3.5 Non-Hodgkins lymphoma

Non-Hodgkins lymphoma (NHL) is the second most common malignant condition associated with HIV infection. It comprises a group of malignant lymphoproliferative diseases and is an AIDS-defining condition. Lymphomas present as focal soft oral swellings with or without ulceration. They may be red and inflamed and are commonly found on the gingival, palatal or alveolar mucosa (Cattaneo et al., 2005, Iamaroon et al., 2003). The lesions are painful and may progress rapidly. Lesions must be biopsied for a definitive histological diagnosis. Treatment requires systemic combination chemotherapy and occasionally radiotherapy. To reduce pain and interference with chewing and speaking, large exophytic or pedunculated lesions can be surgically removed. Treatment is almost always palliative. For patients with KS and lymphomas, good oral hygiene, plaque control and frequent professional cleaning needs to be encouraged to prevent the lesions becoming infected in the advanced stages. Patients on chemo- and radiotherapy require regular care and maintenance to manage specific oral complications of mucositis and xerostomia and prevent the associated increased risks of bacterial and fungal super-infections. Benzydamine hydrochloride mouth rinse may provide relief and is recommended when there are obstructive symptoms. The immune reconstitution associated with antiretroviral therapy may cause lesions to regress.

4. Gingival and periodontal lesions

The gingival and periodontal lesions associated with HIV infection include linear gingival erythema (LGE), necrotising ulcerative gingivitis (NUG), necrotising ulcerative periodontitis (NUP) and nectrotising stomatitis. Linear gingival erythema is characterized by a profound erythema of the gingival margin especially of the front teeth (Figure 11). In necrotizing ulcerative gingivitis there is destruction of one or more interdental papillae with bleeding, ulceration, necrosis and sloughing. Tissue destruction is limited to the gingival tissues and does not involve the alveolar bone. Necrotising ulcerative periodontitis is characterized by advanced necrotic destruction of the periodontium. There is rapid loss of the periodontal attachment, destruction and sequestration of bone and teeth may become mobile. It is often accompanied by severe pain and halitosis.

Several herpesvirus have been shown to be involved in periodontal disease, particularly human cytomegalovirus and EBV (Slots, 2000) - the latter being associated with destructive periodontal disease. Herpesviruses were shown to be positively associated with elevated levels of periodontopathic bacteria (Slots, 2007).

Management is based on plaque reduction and includes thorough curettage and debridement. Strict oral hygiene measures to be prescribed including meticulous brushing and flossing. A dental referral for professional scaling, local debridement, followed by sub-gingival irrigation and regular long-term maintenance is indicated. Mouth rinses and antibiotic therapy may also be indicated. A regimen of topical antiseptic agents such as 1% povidone-iodine solution and 0.2% chlorhexidine gluconate mouth rinse two or four times daily is often initiated as an adjunct therapy. This regimen should be continued until all the diseased hard and soft tissue has been removed and the patient is no longer symptomatic.

In severe cases, topical antimicrobial treatment should be supplemented by a short course of systemic antimicrobial therapy. Metronidazole 400mg 8 hourly for 5 days may be prescribed

for necrotising lesions. Clindamycin 300mg eight hourly for 7-14 days or co-amoxiclav 375mg eight hourly for five days may be used as alternatives. Mobile teeth may need to be splinted or extracted. Bony sequestrations should be removed under antibiotic cover.

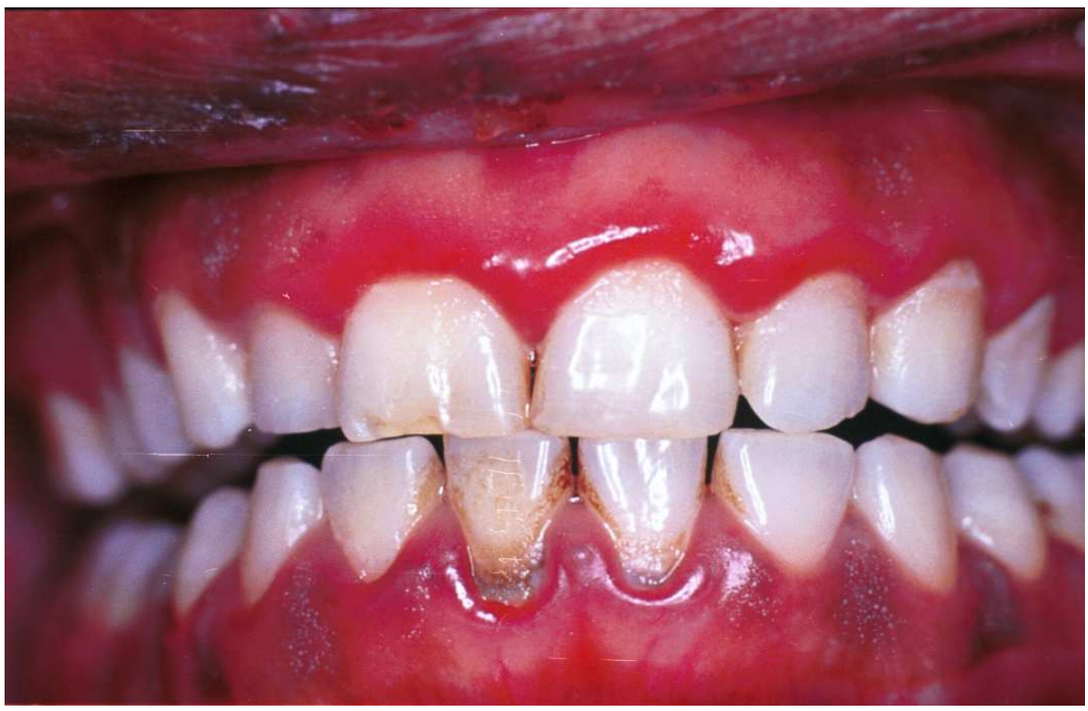

Fig. 11. Linear gingival erythema

5. Salivary Gland Disease (SGD)

Several salivary gland disorders are found in patients with HIV. Parotid gland enlargement often accompanies a syndrome of persistent generalized lymphadenopathy caused by lymphoid proliferation in response to HIV - a condition known as diffuse infiltrative lymphocytosis syndrome (DILS). Patients will DILS are at high-risk of developing lymphoma (Harris, 1999). It is associated with slow progression of the disease. Antiretroviral medication has decreased the prevalence of SGD in adults, but not in children. Parotid gland enlargement may manifest as a unilateral (Figure 12) or bilateral swelling (Figure 13) that may fluctuate in size but commonly persists. Recurrent bacterial parotitis may occur. Salivary gland disease is usually associated with reduced salivary flow and persistent dry mouth and may predispose to the development of dental caries.

Management: No definite treatment is indicated for HIV-related salivary gland disease if there is no super-infection. Oral broad spectrum antibiotics should be used in the treatment of suspected bacterial salivary gland infection. Dry mouth should be treated with salivary substitutes containing methylcellulose. Glycerine may be useful. Sugarless chewing gum should be recommended to stimulate salivary flow. A dry mouth can predispose a patient to dental caries, therefore thorough oral hygiene and the daily use of topical fluoride rinses, varnishes or gels should be recommended to prevent caries. The intake of sugar and sugary foods should be limited.

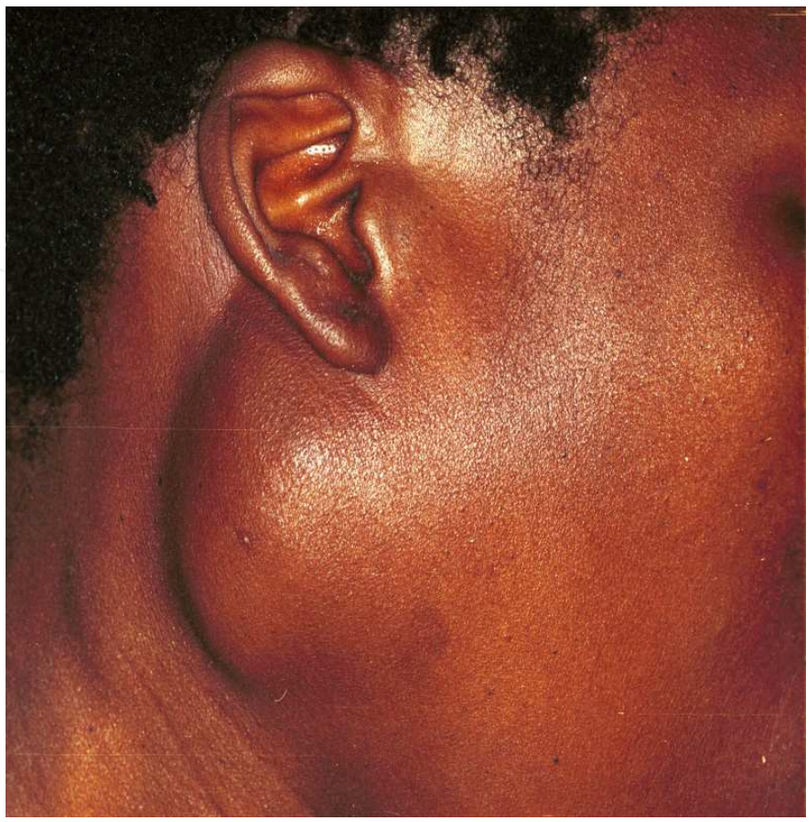

Fig. 12. Parotid gland swelling (adult)

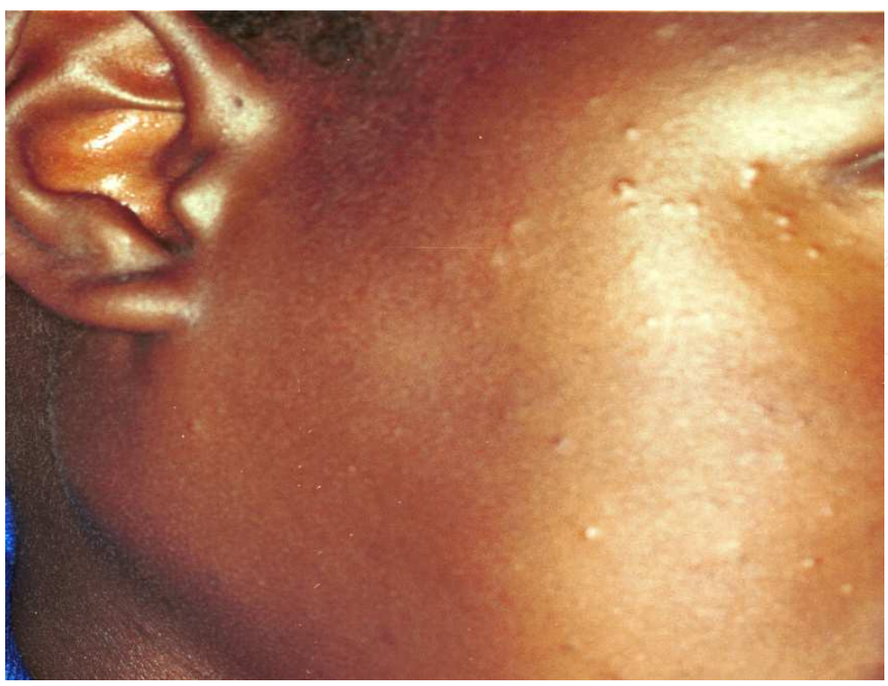

Fig. 13. Parotid gland swelling (child)

6. HPV infection and oral warts

Infections with human papilloma virus (HPV) have distinct appearances and a specific expression of an identified HPV genotype. A few HPV subtypes have been implicated with a subset of oral cancers due to their high-risk oncogenic potential (Nokta, 2008). Most cases of HPV-related oropharyngeal cancers (OPC) are caused by HPV-16 (D'Souza et al 2007) and occurs at higher rates in men who are HIV positive. HPV is particularly associated with head and neck squamous cell carcinomas of the lingual and palatine tonsils. Furthermore, the prevalence of HPV infection in the oral cavity of HIV-positive people without oral cancer was higher than those who were HIV-negative and was strongly associated with HIV infection, immuno-suppression and oro-genital contact (Kreimer et al., 2008).

The increasing prevalence of benign oral warts that has been found in patients on antiretroviral therapy has been associated with HPV-13 and HPV-32 (Greenspan et al., 2001).This was initially thought to be part of the immune reconstitution syndrome, but Lilly et al (2005) reported that the pathology of oral warts was independent of immune reconstitution. Warts are proliferative epithelial lesions and can be found anywhere in the mouth or lips and are cauliflower-like, spiked or raised with a flat surface. Most lesions are asymptomatic, but can interfere with mastication and raise cosmetic concerns. Treatment includes surgical excision, cryotherapy, laser ablation and topical application of keratinolytic agents, but they do tend to recur.

7. Oral health & HIV in children

The oral manifestations of AIDS in children are different from that of adults and can affect them more severely. Studies have emphasised the prognostic significance of oral hairy leukoplakia, Kaposi's sarcoma and Non-Hodgkin's lymphoma as AIDS-defining conditions in adults. Oral candidiasis, parotid gland enlargement, recurrent oral ulcerations and gingival and periodontal disease are the most prevalent oral manifestations commonly seen in children with AIDS.

Molluscum contagiosum is commonly seen on the face. It is caused by the poxvirus that infects the skin and is characterized by clusters of variable sized pearly-white smooth papules, showing characteristic central umbilication. It is usually self-limiting, but in severely immunosuppressed children may be extensive and disfiguring (Figure 14). Management is difficult but repeated topical applications of potassium hydroxide, silver nitrate, liquid nitrogen may be helpful. Dental manifestations include and increased prevalence in dental caries (Naidoo & Chikte, 2004), delayed eruption (Hauk et al., 2001) and over-retention of primary teeth (Flaitz et al., 2001). Children with decreased salivary flow and xerostomia should maintain thorough oral hygiene and use topical fluorides (rinses, gels and varnishes) to prevent caries. Dietary control is essential to limit the intake of sugar and sugary foods. Sugar-based medication should be avoided.

8. Oral lesions in resource poor settings

Many patients may present with any of the lesions described above that are suggestive, but not diagnostic of HIV infection. In resource-poor settings definitive diagnosis may be problematic due to lack of sophisticated diagnostic tests. While oral lesions, may serve as useful indicators not only for disease progression in untreated HIV-positive patients, or

herald failure of treatment in those receiving antiretroviral agents, it should not replace traditional tests for viral loads, CD4 counts and other pathological measurements for positive confirmation.

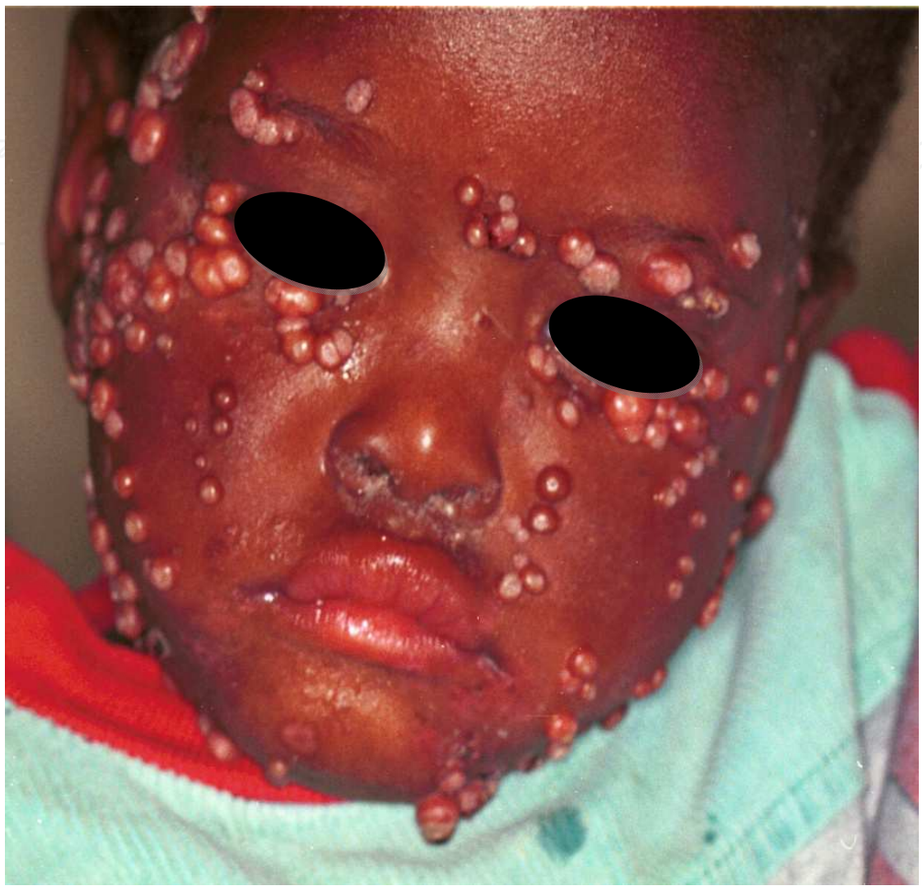

Fig. 14. Molluscum Contagiosum

Saliva has been shown to contain a variety of factors that protects against HIV infection including mucins, secretory leukocyte protease inhibitor, salivary agglutinin, defensins and secretory IgA. Mucosal IgA has been shown to exert antiviral activity by direct neutralization, blocking HIV attachment to epithelial or other target cell receptors and transcytosis of HIV across epithelial cells (Challacombe & Naglik, 2006; Kazmi et al., 2006; Weinberg et al., 2006). Collection of saliva is simple and non-invasive and in resource-poor settings or for large scale population screening, salivary tests may be used to determine HIV serostatus (Chen et al., 2007). More recently, the oral cavity has been viewed as a potentially useful site to initiate a vaccine induced oral mucosal immune response and it is thought that oral mucosal vaccines may induce cellular and humoral immune responses similar to systemic immunization (Stahl-Henning et al 2007).

9. Concluding remarks

Oral health care workers can contribute to the early diagnosis, prevention and treatment of HIV. The oral health management of HIV infected patients should focus on the provision of

dental care and treatment of the oral manifestations of the disease. Despite the fact that significant advances in the pathogenesis and management of the oral manifestations of HIV have been made, more research is required to elucidate the long-term effects of antiretroviral therapy, HPV involvement, the progression and significance of SGD and creative, innovative strategies to reduce the barriers of vulnerable populations seeking oral and general health care.

The health care worker would need to ascertain the patient's overall health status, immune status, prognosis, presence and history of opportunistic infections, risk for developing more serious opportunistic infections, current medications and their long-term survival. Usually no dental modifications are required for patients based on their HIV status. HIV-positive patients should always receive treatment which is standard in its substance (i.e. the same treatment which would be administered to HIV-negative patients) though the treatment may be non-standard in its manner (e.g. slower, more careful treatment, involving greater protective measures). Major concerns are impaired haemostatis, susceptibility to infections, drug interactions and the patient's ability to withstand the stress and trauma of the dental procedure. Treatment planning for HIV-positive patients needs to be carefully thought through and address numerous considerations.

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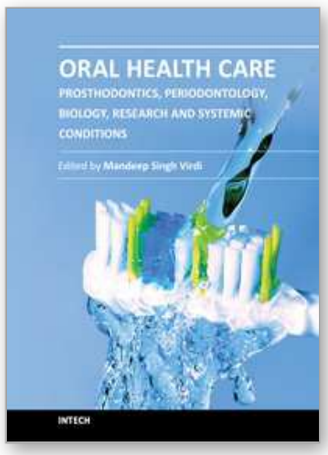

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Geriatric dentistry, or gerodontics, is the branch of dental care dealing with older adults involving the diagnosis, prevention, and treatment of problems associated with normal aging and age-related diseases as part of an interdisciplinary team with other healthcare professionals. Prosthodontics is the dental specialty pertaining to the diagnosis, treatment planning, rehabilitation, and maintenance of the oral function, comfort, appearance, and health of patients with clinical conditions associated with missing or deficient teeth and/or oral and maxillofacial tissues using biocompatible materials. Periodontology, or Periodontics, is the specialty of oral healthcare that concerns supporting structures of teeth, diseases, and conditions that affect them. The supporting tissues are known as the periodontium, which includes the gingiva (gums), alveolar bone, cementum, and the periodontal ligament. Oral biology deals with the microbiota and their interaction within the oral region. Research in oral health and systemic conditions concerns the effect of various systemic conditions on the oral cavity and conversely helps to diagnose various systemic conditions.

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