SCIENTIFIC ARCHIVES OF DENTAL SCIENCES (ISSN: 2642-1623)

Volume 2 Issue 9 September 2019

Review Article

Oral Health and AIDS - A Brief Update

Sarvesh Vijay[1]* and Shalu Srivastava[2]

[1]Co-Investigator, Oral Research Wing, M.S Clinical Research (P) Ltd, Halsuru, Bengaluru, Karnataka, India

[2]Resident Dental Surgeon, Just Dental- A Corporate Dental Chain, Basaveshwara Nagar, Bengaluru, Karnataka, India

***Corresponding Author:** Sarvesh Vijay, Co-Investigator, Oral Research Wing, M.S Clinical Research (P) Ltd, Halsuru, Bengaluru, Karnataka, India.

Received: August 08, 2019; **Published:** August 22, 2019

Abstract

Oral health is an important component of overall health status in AIDS. Even common dental diseases such as caries and periodontal disease have greater impact on patients with AIDS. Painful oral complications such as Necrotizing Ulcerative Periodontitis (NUP), Necrotizing Ulcerative Gingivitis (NUG), Aphthous ulceration, Herpes Simplex, Non-Hodgkin's Lymphoma (NHL), Oral Candidiasis, etc. are clinically significant and prevalent component of AIDS complex. A thorough examination of the oral cavity can easily detect most of the common lesions and good understanding and knowledge of these conditions by an experienced dental professional can herald the treatment in such patients if undiagnosed or may require change in treatment (be more aggressive) of a known patient living with AIDS. An understanding of the recognition, significance, and treatment of these oral complications is essential for long term evaluation and well-being of people living with AIDS.

Keywords: AIDS; Epidemic; Diagnosis; Human Immunodeficiency Virus; Oral Health; Oral Candidiasis

Introduction

Acquired Immune Deficiency Syndrome (AIDS) has emerged as a global crisis since its discovery in the summer of 1981 in the United States [1]. Since the beginning of the epidemic more than 20 years ago, over 60 million individuals have been infected by HIV worldwide and more than 20 million of them have died [2,3]. AIDS is the abbreviation for Acquired Immune Deficiency Syndrome. The disease is caused by virus known as human immunodeficiency virus [4,5]. The virus can remain in a person's body for many years without causing serious health problems [6]. Oral health is important to the overall health of all individuals, young and old, well and ill [7]. Oral manifestations are the earliest and most important indicators of AIDS infection. Oral lesions can not only indicate infection with HIV, they are also among the early clinical features of the infection and can predict progression of HIV disease to AIDS [3]. The main signs and symptoms observed in AIDS patients include diarrhoea, generalised multiple lymphadenopathy, weight loss, and other symptoms related to secondary or opportunistic infections, such as fever, cough, and nodular swelling of the skin [8]. Dental expertise is necessary for proper management of oral manifestations in HIV infection. Dental professionals should be able to recognize HIV associated oral disease and to provide appropriate care and referral [9]. Acquired Immune Deficiency Syndrome was first defined by Centre for Disease Control (CDC) Atlanta in 1982 as "A disease at least moderately predictive of a defect in cell mediated immunity, occurring in a person with no known cause, for diminished resistance to that disease".

In August 1987, the Centre for Disease Control (CDC) published a major revision of the adult case definition for surveillance, broadening the definition in three ways [10]:

1. Inclusion of HIV encephalopathy and HIV wasting syndrome.
2. Inclusion of diagnoses made presumptively in cases with laboratory evidence for HIV infection.

3. Elimination of exclusions due to other causes of immunodeficiency in cases with laboratory evidence for HIV infection.

The HIV/AIDS pandemic has become a human and social disaster, particularly in resource limited settings. HIV related oral abnormalities occur in 30 to 80 percent of the affected patient population [7]. Oral health services and professionals can contribute effectively to the control of HIV/AIDS through health education, patient care, infection control and surveillance [11]. Hence this paper is aimed at a broad review, detailed and comprehensive study of the oral manifestations of AIDS to understand their nature, behaviour, clinical, features, which would be of diagnostic significance, in accordance with the important classifications.

Important historic milestones

- The history of AIDS epidemic in the United States is very old. On June 5 1981, the first description of AIDS appeared in Centre for Disease Control (CDC) Morbidity and Mortality Weekly Report [7].
- Between June 1981 and May 1982 similar cases were reported to Centre for Disease Control (CDC) in the increasing numbers. At first only homosexual and bisexual men were thought to be affected and some aspect of the gay lifestyle was hypothesized to be the probable cause of the immunodeficiency, and hence the disease was termed as Gay-Related Immunodeficiency [9].
- In September 1982 the Centre for Disease Control (CDC) designated the disease as AIDS [10].
- In early 1983, researchers stated that a human T-cell leukemia/lymphotropic virus might be the causative agent of AIDS [11].
- In May 1984, Robert Gallo and other investigators at the National Institute of Health reported the isolation of a group of cytopathic retroviruses and antibodies against viruses in persons with AIDS. They termed it as human T-lymph tropic virus type III [11].
- In the summer of 1986 the international society on the taxonomy of viruses arbitrated changing the name to human immunodeficiency virus type-1. Montagnier and Gallo have been recognized as co- discoverers of the virus [11].

Classifications

Classifying oral manifestations of AIDS has always been a challenge. As more than 40 different oral lesions may be associated with the HIV infection. There is a need for classifying these lesions as a tool to be used in treating cases. Until recently most classifications was based on a mixture of clinical and pathological features, and there was little agreement on clinical staging classification. Some of the most accepted classifications are [12]:

Classification by Robert Gallo and Luc Montagnier (1983) [13,14]

- **HTLV-III:** Similar in shape to human T-lymphotropic viruses.
- **LAV:** Lymphadenopathy associated virus.
- Person presenting with swelling of the lymph node of the neck and physical weakness.
- The core protein of this virus were immunologically different from those of HTLV-III.

Classification by Pindborg (1989) [15]

The first classification of the oral manifestations associated with HIV infection was given by Pindborg. It was based on etiological aspects and the lesions can be classified into those with:

- Fungal.
- Bacterial.
- Viral origin.
- Other Subgroups:
- Neoplasms.
- Neurologic disturbances.
- Lesions of unknown cause.

The proposal was neither final nor exhaustive and is forwarded as a basis for epidemiologic surveys.

Classification by EC-Clearing House (1993) [16]:

I. Group 1- Strongly Associated to HIV/AIDS Infection

- Oral Candidiasis
- Leukoplakia
- Kaposi's Sarcoma

- Lymphoma
- Lineal Erythema

II. Group 2 - Less Frequently Associated to HIV/AIDS Infection

1. Papillota
2. Multifocal Epithelial Hyperplasia
3. Melanic Hyperpigmentation.
4. Labial Herpes.

III. Group 3- HIV/AIDS Infection

5. Histoplasmosis.
6. Recurrent Aphthous Stomatitis.
7. Molluscum Contagiosum

Classification by Greenspan (1993) [17]:

1. Group 1:

- Oral candidiasis
- Hairy Leukoplakia
- Kaposi Sarcoma
- Linear Gingival Erythema
- Necrotizing Ulcerative Periodontitis
- Necrotizing Ulcerative Periodontitis
- Non-Hodgkin Lymphoma

2. Group II:

- Atypical Ulcers
- Salivary Gland Diseases
- Viral Infections-CMV, HSV, HPV, HZV.

3. Group III-

- Diffuse Osteomyelitis
- Squamous Cell Carcinoma
- Other Rare Lesions

Classification by WHO - Clinical Staging of HIV/AIDS and Case Definition (2007) [18-20]:

1. Primary HIV Infection-

- Asymptomatic
- Acute retroviral syndrome

2. Clinical Stage 1

- Asymptomatic
- Persistent generalized lymphadenopathy

3. Clinical Stage 2

- Moderate unexplained weight loss (<10% of presumed or measured body weight)
- Recurrent respiratory infections (Sinusitis, Tonsillitis, Otitis media, and Pharyngitis)
- Herpes zoster
- Angular cheilitis
- Recurrent oral ulceration
- Papular pruritic eruptions
- Seborrheic dermatitis
- Fungal nail infections

4. Clinical Stage 3

- Unexplained severe weight loss (>10% of presumed or measured body weight)
- Unexplained chronic diarrhoea for >1 month
- Unexplained persistent fever for >1 month (>37.6°C, intermittent or constant)
- Persistent oral candidiasis (thrush)
- Oral hairy leukoplakia
- Lymphoma (cerebral or B-cell non-Hodgkin)
- Invasive cervical carcinoma
- Atypical disseminated leishmaniasis
- Symptomatic HIV-associated nephropathy
- Symptomatic HIV-associated cardiomyopathy

Classification of Oral Lesions Associated with HIV Infection Based on Intensity and Features (2005) [21]:

1. GROUP I- Seven Cardinal Lesions that are Strongly Associated with HIV Infection

- Oral candidosis
- Hairy leukoplakia

- Kaposi sarcoma
- Linear gingival erythema,
- Necrotizing ulcerative gingivitis
- Necrotizing ulcerative periodontitis
- Non-Hodgkin lymphoma

2. **GROUP II**
   - Atypical Ulcers
   - Salivary glands diseases
   - Viral infection such as Cytomegalovírus (CMV), herpes simplex virus (HSV), papillomavirus (HPV), and herpes zoster virus (HZV).
3. **GROUP III Lesion Rarer than those on Groups 1 and 2**
   - Diffuse osteomyelitis
   - Squamous cell carcinoma

Oral Lesions in Infection with AIDS

Oral candidiasis

Oral candidiasis is one of the most common, treatable oral mucosal infections seen in persons with Human Immunodeficiency Virus (HIV) infection or Acquired Immune Deficiency Syndrome (AIDS) [22]. Oral candidiasis can be a frequent and significant source of oral discomfort, pain, loss of taste, and aversion to food. *Candida albicans* carriage and a history of oral candidiasis are other significant risk factors for oral candidiasis [23]. The infection is caused by *Candida albicans*, a dimorphic fungal organism that typically is present in the oral cavity in a non-pathogenic state in about one-half of healthy individuals. Normally present as a yeast, the organism under favourable conditions, has the ability to transform into a pathogenic (disease causing) hyphae form. Conditions that favour this transformation include broad-spectrum antibiotic therapy, xerostomia, immune dysfunction (secondary to systemic diseases such as diabetes or the use of immune suppressant medications), or the presence of removable prostheses. Unless the patient is severely immunocompromised, the infection is generally limited to the superficial mucosa and skin [24]. Reports describe oral candidiasis during the acute stage of HIV infection, but it occurs most commonly with falling CD4+T-cell count in middle and late stages of HIV disease. Persons with HIV infection carry a single strain of candida during clinically apparent candidiasis and when candidiasis is quiescent. Advanced clinical stages of HIV infection also have been associated with more frequent oral candidiasis than the early stages. In the early stages, it affects mainly the oral mucosa, involving the esophageal mucosa in advanced stages. The development of oral candidiasis-particularly in young adults without a local predisposing cause such as xerostomia or treatment with antibiotics, corticosteroids or other immunosuppressive agents is highly suggestive of HIV infection [25]. The presence of oral candidiasis in subject with HIV infection has been associated with the occurrence of esophageal candidiasis and more frequent progression to AIDS. Oral candidiasis is an early oral sign of immunodeficiency and it has been used as a marker of severity in HIV infection [26].

Kaposi's sarcoma

Kaposi's sarcoma (KS) is an angioproliferative disorder characterized by proliferation of spindle-shaped cells (SC), neo-angiogenesis, inflammation and edema, categorized as an intermediate neoplasm due to the absence of conventional features of malignancy [27]. Kaposi's sarcoma (KS) is a malignant, multifocal systemic disease that originates from the vascular endothelium and has a variable clinical course. Kaposi Sarcoma is caused by human herpes virus 8 (HHV-8), which is transmitted sexually or via blood or saliva [21]. A sudden increase in the number of Kaposi sarcoma cases along with pneumonia is caused by pneumocystis carinii (PCP) and some other opportunistic infections, was observed among the victims of the epidemic of acquired immune deficiency syndrome (AIDS) [28]. Kaposi's sarcoma is the most common intraoral malignancy associated with HIV infection and it accounts for more than 90% of all cancers in HIV positive patients. Recognition of the lesion is essential, since Oral Kaposi Sarcoma is often the first manifestation of the disease and is a diagnostic criterion for AIDS [17]. Kaposi sarcoma was named by Dr. Moritz Kaposi, a prominent Hungarian dermatologist, who first described the rare classical form of Kaposi Sarcoma as ''idiopathic multiple pigmented sarcoma of the skin" in 1872 [29]. The discovery of the causative agent of Kaposi Sarcoma, however, was not intensively pursued until the early 1980s, when the incidence of Kaposi Sarcoma dramatically increased in homosexual and bisexual HIV-positive individuals during the AIDS epidemic [21-25]. The sudden surge of Kaposi Sarcoma incidence among HIV-infected individuals strongly suggested an infectious agent was involved in the development of Kaposi Sarcoma [30].

Non-Hodgkin's lymphoma

Non-Hodgkin's Lymphoma (NHL) is the second most common HIV-associated tumour. As with Kaposi Sarcoma, the frequency of this tumour has fallen with the introduction of ART; however, it is still a very common tumour of HIV-infected individuals in the developing world [17]. A variety of Non-Hodgkin's Lymphoma (NHLs) can arise in the mouth in HIV disease; in fact, a rare type called Plasmablastic lymphoma seems to nearly always arise exclusively in the mouth has a reported prevalence of about 5% in developing countries [21]. Non- Hodgkin's Lymphoma, in contrast to Kaposi's sarcoma, occurs most commonly in intravenous drug abusers with AIDS. The lesions of Non-Hodgkin's Lymphoma are red and exophytic, and commonly involve the alveolar ridge, the gingiva and the palate. The disease can also present as a rapidly enlarging neck mass. Most lesions are of large B-cell origin and originate extranodally [31]. High grade B cell non-Hodgkin's lymphoma (NHL) has been classified as an AIDS defining illness. In the era that preceded the introduction of highly active antiretroviral therapy (HAART) in 1996, Non-Hodgkin's lymphoma (NHL) was diagnosed approximately 60 times more often in the HIV positive population than in the general population [15]. High-grade B-cell Non-Hodgkin's Lymphoma (NHL) is, together with Kaposi's sarcoma (KS) an AIDS-defining disease [22]. It occurs late in the natural history of HIV infection and is generally associated with severe lymphocytopenia. Recent therapeutic developments have led to the introduction of new antiretroviral therapies which resulted in a significant decline in the incidence of various opportunistic infections and Kaposi Sarcoma but not in the incidence of systemic AIDS-related lymphoma ; a decrease in the occurrence of primary AIDS-related primary central nervous system (CNS) Non- Hodgkin's Lymphoma (NHL) is still controversial [32]. Non-Hodgkin's lymphomas (NHLs) encompass several unique malignant lymphoid disease entities that vary in clinical behaviour, morphologic appearance, immunologic, and molecular phenotype. The various types represent neoplastic lymphoid cells arrested at different stages of normal differentiation [33].

Gingival and periodontal disease

Gingival and periodontal disease is common in HIV infection, particularly in individuals residing in or who have migrated from the developing world. Chronic periodontal disease has been described to be more common and more aggressive in HIV-infected patients [8,9]. The possible occurrence of HIV-specific periodontal disease has been observed in some but not all groups of HIV-infected patients, suggesting that HIV infection alone does not predispose patients to pocketing, attachment loss, or bleeding on probing [21]. The reported prevalence of HIV-related gingival and periodontal disease (excluding opportunistic infections and malignancy) varies from 0 to 47% in adults and from 0 to 20% in children; Necrotising Ulcerative Gingivitis (NUG) and Necrotising Ulcerative Periodontitis (NUP) are less prevalent, varying from 2.2 to 5% [34]. Although aspects of HIV-induced immunosuppression have been proposed as the likely cause of HIV-related gingival and periodontal disease, HIV-infected patients often have other relevant risk factors, such as tobacco smoking and poor oral hygiene, and these factors alone can explain the increased prevalence of the disease [21]. Since the first descriptions of the HIV in 1981, a considerable number of researches have focused on the periodontal changes specifically associated with HIV infection. Earlier reports included unusual and severe forms of periodontal disease in HIV-infected individuals, particularly among homosexual males [35].

Mycobacterium tuberculosis

Tuberculosis (TB) is a communicable granulomatous disease caused by *Mycobacterium tuberculosis*. It usually affects the lungs but may affect any organ or tissues in the body. Typically, the centres of the tubercular granuloma undergo caseous necrosis [36]. Rarely, tuberculosis is caused by Mycobacterium bovis, although it is now recognized that a variety of other species of mycobacterium are also potentially pathogenic for humans [37]. Tuberculosis is seen as HIV co-infection. Amongst the large number of reports available pulmonary tuberculosis is being reported more than any other clinical forms. All forms of tuberculosis, in HIV-positive patient, except when cavitation occurs, are paucibacillary in nature [38]. The incidence of tuberculosis has been rising all over the world due to development of Mycobacterium tuberculosis forms resistant to the usual therapy and an increasing number of HIV infected patients It is estimated that oral lesion of tuberculosis are very rare and occur only about 0.05% to 5% [39]. As immunosuppression of HIV infected patient progresses, extra-pulmonary tuberculosis (TB) becomes more common and affects many internal body sights. Occasionally, mycobacteria caused lesions might occur in mouth [40]. In primary disease mouth may be the primary site and more likely occur in younger rather than adults while secondary orofacial tuberculosis (TB) may reflect oral inoculation with sputum or hematogenous spread of mycobacteria [35-38].

HIV-associated salivary gland disease

HIV-associated salivary gland disease (HIV-SGD) is disfiguring and causes significant morbidity in the HIV population. Evidence detailing the epidemiology of HIV-associated salivary gland disease (HIV-SGD) suggests the involvement of a viral opportunist in its pathogenesis, yet the specific etiology of HIV-associated salivary gland disease (HIV-SGD) remains unclear [41]. Salivary gland disease is well established as an important Human Immunodeficiency Virus (HIV) associated oral lesion and salivary gland enlargement may be its initial clinical manifestation. The term HIV-associated salivary gland disease was first described by Schiodt., *et al.* as salivary gland swelling involving one or both parotid glands with or without xerostomia [42]. These lesions may lead to considerable morbidity due to facial disfigurement, pain and discomfort. In the context of HIV, the swelling may be due to a wide spectrum of pathological conditions that include reactive or inflammatory disorders, acute and chronic infections, and neoplasms [39]. Parotid gland enlargement is typically an early manifestation in the HIV-positive patient and should alert healthcare professionals to the likelihood of HIV infection. FNAC of the parotid gland is required to confirm the diagnosis and instituting HAART forms an important part of the management [43].

Herpes simplex virus (HSV) infection

Herpes simplex virus (HSV) infection is a common childhood infection that is not specifically related to HIV status [7,8]. The primary disease, referred to as primary gingivo stomatitis, and the secondary disease, referred to as recurrent HSV (Herpes simplex virus) infection, may develop in children with HIV infection [17]. Most studies do not distinguish between the two forms of the disease. The reported prevalence for Herpes simplex virus (HSV) infections in HIV-infected children ranges from 1.7% to 24% [21]. In immunocompetent individual it affects only the gingiva and hard palate. Usually a prolonged or severe infections and herpetic ulceration persisting for more than a month is an AIDS defining illness [35]. Herpes Labialis represents a recurrent infection which presents with multiple grouped, fragile vesicles or ulcers on the vermillion border of the lip or adjacent skin [36,37]. The vesicles may coalesce to form larger vesicles which heal slowly. In HIV/AIDS lesion may occur in any oral sites and are more severe and prolonged [31]. Ulcers might persist for more than 1 month and their presence is indicative of immunosuppression. Persisting painful lesions can result in reduced intake of food and weight loss, which worsen the morbid condition [40].

Cytomegalo virus

Oral Cytomegalovirus is a member of the human herpes viruses that have an extremely high seroprevalence in all populations studied. These viruses are increasingly important in the modern era of immunosuppression, whether due to AIDS or in the transplant or cancer chemotherapy population, and their reactivation gives rise to a wide spectrum of neurological diseases. The pathogenesis of these infections is not completely understood, but certainly multifaceted. In cytomegalovirus (CMV) lytic infection damages systemic tissues directly [44]. Infection with cytomegalovirus (CMV) is common and occurs throughout life. Between 50 and 100 per cent of the adult population may be seropositive for CMV. When clinical illness develops it is usually an infectious mononucleosis-like syndrome [35-37]. After primary infection, CMV remains latent within the host and the virus is shed from multiple sites, for example, saliva, tears, urine, semen, cervical secretions, and breast milk. This shedding may continue for several years. In patients with immunosuppression, latent virus may reactivate producing a variety of diseases such as pneumonia, colitis, encephalitis, etc [21]. Reactivation of CMV is a common finding in HIV-seropositive patients and studies indicate that up to 90 per cent of patients with AIDS develop active CMV infection during the course of the illness. CMV-related oral ulcerations have been reported. These ulcers are non-specific in their clinical appearance, are generally painful and may be confused with recurrent aphthous stomatitis [45].

Recurrent aphthous stomatitis

Recurrent Aphthous Stomatitis (RAS) occur in approximately 2% to 6% of the adult HIV infected population and are more common among HIV infected children, especially due to drugs such as Didanosine (DDI) that may induce lesions [21]. Recurrent Aphthous Stomatitis (RAS) is a disorder characterized by painful ulcers with variable size and duration typically found in non-keratinized sites of the oral mucosa. The three major types of aphtous stomatitis are minor, major and herpetiform [27,28]. They are differentiated by number, size and duration, however such differentiation is not always clear. The immunodeficiency state in HIV infected patients has been the cause of severe episodes of Recurrent Aphthous Stomatitis (RAS) [41]. Studies show that recurrent aphthous stomatitis has a higher prevalence in young adults, with decreased incidence and severity with increasing age. The etiology remains

unknown although there is evidence for immunogenic factors. Other possible causes are trauma, drug use, lack of B12 vitamin, folic acid, iron and other dietary factors, stress, hormonal changes, metabolic diseases and infection by microorganisms [46].

Cat-scratch disease

Cat-scratch disease (CSD) in an immunocompetent hosts (AIDS) is usually a self-limited illness with characteristic lymph node pathologic findings that have recently been shown to be bacterial in origin [47]. It is the most common human infection caused by Bartonella species. Cat-scratch Disease (CSD) has worldwide distribution. Patients with acquired immune deficiency syndrome (AIDS) can have a variety of histopathologic lymph node changes and are subject to a wide variety of opportunistic infections that rarely cause severe disease in immunocompetent hosts [48]. In immunocompetent humans, *B. Henselae* causes cat scratch disease, which is most often a relatively benign and self-limiting illness. In contrast, *B. henselae* infections are often severe in immunocompromised individuals, and can be fatal without antibiotic treatment [49]. Other species of *Bartonella* have also been linked occasionally to human illnesses, with varying levels of evidence for a causative role [50].

Conclusion

Evaluation of oral health status is important at every stage in the management of HIV disease. The necessity to identify HIV-related oral lesions involves all health care professionals- primary health care workers, infectious diseases specialists, oral health and public health professionals to closely collaborate to provide the best care, health promotion and prevention possibilities for patients infected with AIDS. A major challenge lies in the part of HIV infected persons unaware of their HIV status. Oral health practitioners must take their role in recognizing the potential significance of the oral manifestation of HIV. Virtually everyone infected with human immunodeficiency virus (HIV) will have oral disease during their illness. Conditions such as Oropharyngeal Candidiasis, Hairy Leukoplakia, and Oral Kaposi's Sarcoma frequently constitute the sentinel event leading to HIV diagnosis. As some oral lesions are independent markers for deteriorating immune function, their prompt identification has prognostic and therapeutic implications. At least 90% of HIV-infected patients will have at least one oral manifestation at some time during the course of their disease. Oral lesions might herald underlying immunodeficiency. To conclude we can say that much has been learned about AIDS in past few years. However, no cure is available till now. It is estimated that most if, not all of the currently infected will progress to overt AIDS disease, which will continue to place an incredible burden on the health and immunity service system.

Bibliography

1. Samarannayake LP, Holmstrup P. Oral Candidiasis And Human Immunodeficiency Virus Infection. J Oral Pathol Med. 1989;18(10):554-564.

2. J Campo, Perea MA, del Romero J, Cano J, Hernando V, Bascones A. Oral Transmission Of HIV, Reality Or Fiction? An update. Oral Dis. 2006;12(3):219-228.

3. LL Patton, Phelan JA, Ramos-Gomez FJ, Nittayananta W, Shiboski CH, Mbuguye TL. Prevalence And Classification Of HIV Associated Oral Lesions. Oral Dis. 2002;8(2):98-109.

4. Burket's Oral Medicine, Diagnosis and Treatment. 10th edition 2003:538-556.

5. Moniaci D, Cavallari M, Greco D, Bruatto M, Raiteri R, Palomba E, Tovo PA, Sinicco A. Oral Lesions In Children Born To HIV-1 Positive Women. Journal of Oral Pathol Med. 1993;22(3):8-11.

6. PedersonC, J Gerstoft, B Lindhardt and J Sindrup. Candida Esophagitis Associated With Acute Human Immunodeficiency Virus Infection. J Infect Dis. 1987;156(3):529-530.

7. Garcia-Cuesta C, Sarrion-Pérez MG, Bagán JV. Current Treatment Of Oral Candidiasis: A Literature Review. J Clin Exp Dent. 2014;6(5):576-582.

8. Maeve M Coogan, John Greenspan, Stephen J Challacombe. Oral Lesions In Infection With Human Immunodeficiency Virus. Bull World Health Organ. 2005;83(9):700-706.

9. Reznik A. Oral Manifestations Of HIV Disease. Top HIV Med. 2005-06;13(5):143-148.

10. PT Cohen, Merle Sande, Paul A Volberding. The AIDS Knowledge Base; A Textbook On HIV Disease From The University Of California, San Francisco And San Francisco General Hospital; 3rd Edition.

11. Peter J Ungvarski and Jacquelyn Haak Flaskerud. HIV/AIDS: A Guide To Primary Care Management. 4th Edition 1999.

12. Pindborg JJ. Oral Manifestations Of The HIV Infection: Classification Problems. J Dent Assoc S Afr. 1992;47(5):224-226.

13. RC Gallo, Sarin PS, Gelmann EP, Robert-Guroff M, Richardson E, Kalyanaraman VS, Mann D, Sidhu GD, Stahl RE, Zolla-Pazner S, Leibowitch J, Popovic M. Isolation Of Human T-Cell Leukemia Virus In Acquired Immune Deficiency Syndrome (AIDS). Science. 1983;220(4599):865-867.

14. Barre Sinoussi, Chermann JC, Rey F, Nugeyre MT, Chamaret S, Gruest J, Dauguet C, Axler-Blin C, Vézinet-Brun F, Rouzioux C, Rozenbaum W, Montagnier L. Isolation Of A T-Lymphotropic Retrovirus From A Patient At Risk For Acquired Immune Deficiency Syndrome (AIDS). Science. 1983;220(4599):868-871.

15. Pindborg JJ. Classification Of Oral Lesions Associated With HIV Infection. Oral Surg Oral Med Oral Pathol. 1989;67(3):292-295.

16. Bravo IM, Correnti M, Escalona L, Perrone M, Brito A, Tovar V, Rivera H. Prevalence Of Oral Lesions In HIV Patients Related To CD4 Cell Count And Viral Load In A Venezuelan Population. Med Oral Pathol Oral Cir Bucal. 2006;11(1):E33-E39.

17. Ponnam SR, Srivastava G, Theruru K. Oral Manifestations Of Human Immunodeficiency Virus In Children: An Institutional Study At Highly Active Antiretroviral Therapy Centre In India. J Oral Maxillofac Pathol. 2012;16(2):195-202.

18. Centers for Disease Control and Prevention. 1993 Revised Classification System For HIV Infection And Expanded Surveillance Case Definition For AIDS Among Adolescents And Adults. MMWR Recomm Rep. 1992;41(RR-17):1-19.

19. Centers for Disease Control and Prevention. Guidelines For National Human Immunodeficiency Virus Case Surveillance, Including Monitoring For Human Immunodeficiency Virus Infection And Acquired Immunodeficiency Syndrome. MMWR Recomm Rep. 1999;48(RR-13):1-27,29-31.

20. World Health Organization. WHO Case Definitions Of HIV For Surveillance And Revised Clinical Staging And Immunological Classification Of HIV-Related Disease In Adults And Children, 2007.

21. Prabhu, Vishnudas Prabhu, Laxmikanth Chatra, Prashant Shenai. Oral Manifestations Of HIV. J Trop Dis. 2013;1(3):111.

22. Greenspan D. Treatment Of Oral Candidiasis In HIV Infection. Oral Surg Oral Med Oral Pathol. 1994;78:211-215.

23. MacPhail LA, Hilton JF, Dodd CL, Greenspan D. Prophylaxis With Nystatin Pastilles For HIV-Associated Oral Candidiasis. J Acquir Immune Defic Syndr. 1996;12(5):470-476.

24. Pons V, Greenspan D, Lozada-Nur F, MacPhail L, Gallant JE, Tunkel A, Johnson CC, McCarty J, Panzer H, Levenstein M, Barranco A, Green S. Oropharyngeal Candidiasis In Patients With AIDS: Randomized Comparison Of Fluconazole Versus Nystatin Oral Suspensions. Clin Infect Dis. 1997;24(6):1204-1207.

25. Rao PK. Oral Candidiasis - A Review. Scholarly Journal of Medicine. 2012;2(2):26-30.

26. Phyllis J Kanki. Human Immunodeficiency Virus Type 2 (HIV-2). AIDS. 1999;1:101-108.

27. Amador VR, Saavedra GA, Mata GM. Kaposi's Sarcoma Of The Head And Neck: A Review. Oral Oncology. 2010;46(3):135-145.

28. Raju SB, Rajappa S. Isolation And Identification Of Candida From The Oral Cavity. ISRN Dentistry. 2011:487921.

29. Olsen I. Oral Adhersion Of Yeast. Actaodontol Scandal. 1990;48(1):45-53.

30. Samaranayake LP, MC Farlane TW. The Effect Of Dietary Carbohydrates On The Invitro Adhesion Of Candida Albicans To Epithelial Cells. J Med Microbial. 1982;15(4):511-517.

31. M Ukpebor, OB Braimoh. HIV/AIDS; Oral Complications And Challenges, The Nigerian Experience. Benin Journal of Postgraduate Medicine. 2007;9(1).

32. Silvestris N, Crucitta E, Lorusso V, Gamucci T, Lena MD. AIDS-Related Non- Hodgkin's Lymphoma: Clinico-Pathological Characteristics And Therapeutic Strategies (Review). International Journal Of Oncology. 2002;20(3):611-615.

33. Rosenberg S, Berard C, Brown Jr. B, Burke J, Dorfman R, Glatstein E. National Cancer Institute Sponsored Study Of Classifications Of Non-Hodgkin's Lymphomas: Summary And De-

scription Of A Working Formulation For Clinical Usage. The Non-Hodgkin's Lymphoma Pathologic Classification Project. Cancer. 1982;49(10):2112-2135.

34. Scully C, Cawson RA. Medical Problems In Dentistry. 2nd Edition Bristol: Wright, 1987.

35. Agbelusi GA, Eweka OM, Ùmeizudike KA, Okoh M. Oral Manifestations Of HIV, 2012.

36. Aguiar MC, Arrais MJ, Mato MJ, de Araújo VC. Tuberculosis Of The Oral Cavity: A Case Report. Quintessence Int. 1997;28(11):745-747.

37. Chamie G, Luetkemeyer A, Charlebois D, Havlir DV. Tuberculosis As Part Of The Natural History Of HIV Infection In Developing Countries. Clinical Infectious Diseases. 2010;50(S3):S245-S254.

38. Banerjee U. Progress in diagnosis of opportunistic infections in HIV/AIDS. Indian J Med Res. 2005;121(4):395-406.

39. Miziara ID. Tuberculosis Affecting The Oral Cavity In Brazillian HIV Infected Patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005;100(2):179-182.

40. Askinyte D, Matulionyte R, Rimkevicius A. Oral Manifestations Of HIV Disease: A Review. Stomatologija. 2015;17(1):21-28.

41. Jeffers, Webster-Cyriaque. HIV-Associated Salivary Gland Disease: A Role For BK Birus. Infectious Agents and Cancer. 2010;5(1):A33.

42. Jeffers L, Cyriaque W. Viruses And Salivary Gland Disease (SGD): Lessons From HIV SGD. Adv Dent Res. 2011;23(1):79-83.

43. Ebrahim S, Singh B, Ramklass S. HIV-associated salivary gland enlargement: a clinical review. SADJ. 2014;69(9):400-403.

44. Tselis A. Epstein-Barr Virus And Cytomegalovirus Infections. Birkhauser Advances In Infectious Diseases. 2013:23-46.

45. Mccullough MJ, Firth NA, Reade PC. Human Immunodeficiency Viru Infection: A Review Of The Mode Of Infection, Pathogenesis, Disease Course, And The General And Clinical Manifestations. Australian Dental Journal. 1997;42:(1):30-37.

46. Gomes MAG, Zaroni FM, Martins MC, Lima AAS. Major Recurrent Aphthous Stomatitis In Mother And Son With HIV/AIDS Infection - Case Report. Pediatria Polska. 2015;90(3):256-259.

47. Pilon VA, Echols RM. Cat-Scratch Disease In A Patient With AIDS. AJCP. 1989;92(2):236-240.

48. Cat Scratch Disease And Other Zoonotic Bartonella Infections. CFSPH 2012:1-12.

49. Chomel BB. Cat-Scratch Disease. Rev Sci Tech Off Int Epiz 2000;19(1):136-150.

50. Klotz SA, Ianas V, Elliott SP. Cat-Scratch Disease. American Family Physician. 2011;83(2):152-155.

Volume 2 Issue 9 September 2019