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THE ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

HIV: There exist two recognized types of HIV: HIV-1 and HIV-2. Both have the same modes of transmission, and both may cause immunosuppression and AIDs.

The prime modes of transmission for HIV are :

-unprotected penetrative sex between men

-unprotected hetero- sexual intercourse،

-injection drug use ،

-unsanitary injections and blood transfusions

-Mother to child spread during pregnancy, delivery, or breast-feeding.

*In 5-15% of cases, HIV infection shows no clinical progress for periods up to 10 years, possibly because of variations in pathogenicity of HIV strains, or stronger host immune responses.

Diagnosis

-The diagnosis of HIV infection is obtained by appropriate laboratory testing. The standard HIV-antibody test (by taking plasma or serum which give positive test), however, cases of recent HIV infection may be missed as it takes several weeks (even months) for a measurable antibody response to develop.

-Detection of viral antigens, such as p24 protein, in the blood provides a more direct and reliable indicator of infection.

Pathogenesis:

For untreated HIV infection, a common pattern of disease progression has been established consisting of three phases: (1) primary infection, (2) prolonged (median=10 years) period of clinical latency, and (3) the appearance of clinically apparent disease.

HIV is a spherically shaped retrovirus whose outer coat, or envelope, consists of two layers of fatty molecules called lipids; these lipids are actually taken from the human cell membrane when newly formed virus particles bud from the cell. Within the envelope is the bullet-shaped core or capsid, which consists of about 2,000 copies of the HIV protein p24. The capsid encircles two single strands of HIV ribonucleic acid (RNA) and the enzymes (reverse transcriptase, integrase, and protease). The human immunodeficiency virus directly infects lymphocytes and other cells, such as some macrophages, which carry the CD4 marker and causes lymphopenia so the virus kills T helper (CD4) cells and reverses the ratio of helper to suppressor lymphocytes.

The predominant portal of entry for HIV is through blood and/or mucosal exposure and dendritic cells at or near the mucosal surface of the involved sites play an important role in the initiation of HIV infection. Infection is enhanced where the mucosal tissues are ulcerated or inflamed, upon initial infection, a rapid sequence of virologic, immunologic, and clinical events occurs within the first 4 to 8 weeks of infection (i.e. primary infection). There is a rapid rise in plasma viremia and rapid dissemination of virus to lymphoid organs, particularly the gut-associated lymphoid tissue, are major factors in the establishment of the chronic and persistent infection that is a hallmark of HIV disease.

The clinical symptoms are characterized by varying degrees of fever, fatigue, maculopapular rash and headache, lymphadenopathy (generalised lymphadenopathy syndrome) with wide- spread persistent enlargement of lymph nodes which is a typical early sign, pharyngitis, myalgia, arthralgia, gastrointestinal distress, night sweats, and oral or genital ulcers.

The key feature of AIDS :

•Caused by a retrovirus — usually HIV-1

•Transmitted sexually or by intravenous drug abuse, and by blood or blood products

•Progressive deterioration mainly of cell-mediated immunity and deaths mainly due to opportunistic infections

•Immunodeficiency leads to opportunistic infections such as Pneumocystis carinii pneumonia

•Common oral lesions include candidiasis and hairy leukoplakia

•Greatly increased frequency of Kaposi's sarcoma and lymphomas, often in oral regions

•Neurological and psychological disorders may be associated.

ORAL LESIONS IN HIV DISEASE

Clinical indicators of a poor prognosis are oral thrush, herpes zoster or other persistent or recurrent infections, hairy leukoplakia, unexplained constitutional symptoms and lymphadenopathy. More than 90% of acquired immune deficiency syndrome (AIDS) patients have oral candidiasis and the infection is considered a portent of AIDS development.

Oral candidiasis:

The most common types of oral candidiasis in conjunction with HIV are pseudomembranous candidiasis, erythematous candidiasis, angular cheilitis, and

chronic hyperplastic candidiasis. The main effect of the immunodeficiency and chief cause of death is infection by a great variety of microbes.

Viral mucosal infections

Herpetic stomatitis is less common than might be expected but can cause atypical or chronic ulceration. Severe orofacial zoster may indicate a poor prognosis. Cytomegalovirus can be found in some oral ulcers and the Epstein-Barr virus is the cause of hairy leukoplakia. Papillomaviruses have been isolated from proliferative lesions, such as verruca vulgaris, condyloma acuminatum and focal epithelial hyperplasia.

Bacterial infections

Infections by bacteria which rarely involve the oral tissues, such as Klebsiella pneumoniae, Enterobacter cloacae, and Escherichia coli, can develop.

Bacillary angiomatosis

Bacillary angiomatosis is a vascular proliferative disease caused by Bartonella henselae and should respond to antimicrobial therapy. However, it can mimic Kaposi's sarcoma clinically and, to some extent, histologically. It affects the skin more frequently than the oral cavity. Biopsy is essential to exclude Kaposi's sarcoma.

Systemic mycoses

Histoplasmosis or cryptococcosis can give rise to proliferative or ulcerative lesions.

Hairy leukoplakia

Hairy leukoplakia is highly characteristic of HIV infection.

Tumors

Nearly 50% of patients with AIDS have a malignant tumor at the time of presentation; the most frequent are Kaposi's sarcoma and non-Hodgkin lymphomas. Unlike non-AIDS patients, these tumors are particularly frequent in the head and neck region.

Kaposi's sarcoma in the mouth, particularly in a young male is pathognomonic of AIDS. It is usually associated with a CD4 lymphocyte count of less than 200/µL and frequently associated with other effects of HIV infection such as candidiasis, hairy leukoplakia or HIV-associated gingivitis. Though oral Kaposi's sarcoma may be the cause of early symptoms, the tumor is usually multifocal, with lesions affecting skin, lymph nodes and viscera.

KS is a multicentric neoplastic proliferation of endothelial cells and can involve any oral site, but most frequently involves the attached mucosa of the palate, gingiva, and dorsum of the tongue. The palate is the most frequent site and the lesions begin as blue purple or red purple flat discolorations that can progress to tissue masses that

may ulcerate. The lesions do not blanch with pressure. Initial lesions are asymptomatic but can cause discomfort and interfere with speech, denture use, and eating when lesions progress. The differential diagnosis includes ecchymosis, vascular lesions, salivary gland tumor, and metastatic disease. Definitive diagnosis requires biopsy.

Lymphomas

AIDS-related lymphomas can develop in intraoral sites or salivary glands far more frequently than in HIV-negative persons. Typical sites within the mouth are the palate or gingiva, where the tumors form soft painless swellings which ulcerate when traumatized ex. Burkitt's lymphoma.

Autoimmune disease

The most common autoimmune phenomenon in AIDS is thrombocytopenic purpura. This can give rise to oral purple patches which may be mistaken for Kaposi's sarcoma, petechiae or blood blisters. Other autoimmune diseases reported in AIDS are lupus erythematosus and a Sjogren-like salivary gland disease.

Gingivitis and periodontitis

HIV-related periodontal disease includes necrotising gingivitis and accelerated periodontitis.

Salivary gland disease

"HIV salivary gland disease" (HIV- SGD), it encompasses a range of conditions, including xerostomia and benign (unilateral or bilateral) salivary gland enlargement in HIV-positive patients. The etiology of HIV-SGD is poorly understood, but the reactivation of a latent virus has been hypothesized. HIV-SGD is associated with a CD8+ cell lymphocytosis of the salivary glands and with the diffuse infiltrative lymphocytosis syndrome, also the chronic parotitis, possibly due to Epstein-Barr virus or cytomegalovirus, appears to affect children with AIDS particularly .

Clinical Manifestations: the primary sign of HIV-SGD is salivary gland swelling, primarily in the parotid glands and frequently bilateral. Xerostomia is a common symptom and salivary flow rates may be decreased, which may occur relatively early in HIV infection.

Diagnosis:

-HIV-SGD frequently resembles Sjögren's syndrome but can be distinguished it by lacking the anti-SS-a and anti-SS-B autoantibodies in the HIV-SGD population.

-Using immunohistochemical stains to differentiate the infiltrating cells, there is a preponderance of CD8+ cells in HIV-SGD compared with the CD4+ infiltrates that predominate in Sjögren's syndrome.

-Biopsy: the histopathology of an HIV–involved major gland demonstrates hyperplastic lymph nodes, lymphocytic infiltrates, and cystic cavities, with persistent enlargement of a major gland, a biopsy of the affected tissue may be necessary to exclude neoplasia particularly concern are lymphoma and Kaposi's sarcoma, both of which have been reported in the salivary glands of HIV-infected individuals.

Miscellaneous oral lesions

Mucosal ulcers and Major aphthae. They become more frequent and severe with declining immune function.

Oral hyperpigmentation, pigmentation may be secondary to Addison's disease due to fungal destruction of the adrenals .

Neurological disease

Orofacial effects include facial palsy and trigeminal neuropathy.

Thank you