International Journal of Applied Research 2017; 3(4): 801-803

ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2017; 3(4): 801-803
www.allresearchjournal.com
Received: 12-02-2017
Accepted: 13-03-2017

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AIDS related complex- A case report

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Abstract
AIDS is defined as reliably diagnosed opportunistic diseases in an adolescent or an adult, at least moderately indicative of underlying cellular immunodeficiency and with no other known cause of underlying cellular immunodeficiency or any other reduced resistance reported to be associated secondarily immune-deficiencies associated with immunosuppressive therapy, lympho-reticular malignancies or starvation. The present case report discusses the diagnosis and management of AIDS related complex.

Keywords: AIDS, complex, hairy leukoplakia, western blot

Introduction
HIV belongs to the sub group of family retrovirus. It is spherical enveloped virus about 90-120nm in size. The most outer part of virus is consists of projecting knob-like structure made up of glycoproteins [1, 2]. This constitutes the major surface component of the virus which binds to the CD4 receptors on susceptible host cells. The next layer is lipi d by layer which present next to the outer glyco protein layer. The nucleo-cuspid has an outer ico-sahedral shell and an inner cone shaped core enclosing the ribonucleo proteins. The genume is diploid composed of two identical single stranded RNA. Along with this RNA the enzyme reverse transcriptase which is characteristic features of retro-virus. It mainly helps viral multiplication.

Literature Review: Once the virus enters the blood or tissues it binds the cell which contain CD4 antigen. Because the receptor for the virus is CD4 antigen [3] This is primarily helper or inducer T-lymphocytes. This virus also affects macrophages, monocytes and glial cells of C.N.S. after binding of virus to CD4 receptor fusion of virus and host cell takes place. This is brought about by transmembrane glycoprotein 41. After fusion of virus with host cell membrane HIV genome is uncoated and enters into the host cell. Now RT transcribes viral RNA into double stranded DNA which is integrated into chromosome of infected host cell. Due to this whole function of host cell is suppressed without structural damage. The infected T4 cell does not appear to be produce normal amount of interferon, interleukin and lymphokinins. It severely affects cell-mediated immunity. The helper T-cell activity is essential for optimal B-cell function [4, 5]

Clinical features: According to W.H.O if the individual said to HIV +ve he should have atleast 2 major signs associated with one minor sign in the absence of known secondary causes of immuno suppression.

Major signs: Weight loss of >10% of body weight, Chronic diarrhoea of > 1 months duration, Prolonged fever. Minor signs: Recurrent oro-pharyngeal candidiasis, Persistent generalized lymphadenopathy, Persisted cough for > one month, generalized pruritic dermaititis, recurrent herpes zoster and progressive disseminated herpes simplex infection.

Oral-manifestation of HIV infection [6]
Oral hairy leuko-plakia: It is a keratotic lesion mainly found in lateral borders of tongue with bilateral distribution, sometime it may extend into ventrum of the tongue, soft palate, buccal mucosal, also it is mainly caused by Epstein bar virus.

Microscopically, the lesion shows a hyperparakarotic surface with projections which resemble hairs.

Oral candiasis: It is most common oral lesion in HIV diseases and has been found in approximately 90 of the AIDS patients, caused by candida albicans. It usually has one of four clinical presentations.

Erythematous candidiasis: It appears as rod patch on buccal mucosal usually associated depapillation of tongue.

Pseudomembraneous candidiasis: Painless white patches on buccal mucosal which can be easily scraped or separated from oral mucosa.

Hyper plastic candidiasis: It is least common and usually located in buccal mucosa, and tongue.

Kaposis sarcoma: It is an rare multifocal vascular neoplasm found in skin of buccal, mucosa, gingiva. In oral cavity it manifest as painless, redish purplemacules. As the lesion progress they frequently become nodular and easily confused with oral vascular entities, such as hemangioma, hematoma. It is viral origin caused by human herpes virus-8 (HHV-8). Microscopically the kaposis sarcoma consists of 4 components. Endothelial cell proliferation with formation of atypical vessels, extra vascular hemorrhage with hemosiderin deposition. Spindle cell proliferation & mono-nuclear inflammatory cell infiltration.

Oral hyper pigementation: An increased incidence of oral hyperpigmentation has been described in HIV infected individuals. It often appear as spots or striations on buccal mucosa palate, gingiva and tongue. In some times these pigmentation also due to drugs like zidovudine, keta-conazole etc. zidovudine is also causes excessive pigmentation of nails and skin.

Linear gingival erythema: A persistent linear easily bledding erythematous gingivitis has been described in some HIV positive patients. It may be localized or generalized in nature. The erythematous gingivitis may be limited to marginal tissue or extend into attach gingiva or alveolar mucosa. The exact etiology is unknown. But it is thought that it may be caused by candidal infections.

Necrotizing ulcerative stomatitis (NUS): The acute painful necrotizing ulcerative stomatitis has been occassionally reported in HIV positive patients. It is characterized by necrosis of significant areas of oral tissues and underlying bone. It is commonly associated with severe depression of CD4 immune cells.

Necrotizing ulcerative periodontitis (NUP): It is an rapidly progressive form of periodontitis occur more frequently in HIV positive patients. It is characterized by soft tissue necrosis, rapid periodontal destruction with interproximal bone loss. Lesion may localized or generalized. Here bone is often exposed resulting in necrosis and sequestration.

Management: Anti-retrovial therapy: *Conservative approach is:* Zidovudine + lamivudine; Zidovudine + didanosine or Zidovudine + Zalctabine while.

Personal protective measures [7, 8] Wash thoroughly after each patient. Use surgical soap that contain lanolin which prevent drying and cracking of skin which could provide a portal of entry for viruses into blood stream. The anti-septics like 3% PCMXC P. chloro meta-xylenole) or 4% chlorhexidine can be used.

Protective eye-glasses or large plastic face shield should be worn.

A face mask should be worn in additional to eye-glasses. It should be changed after every patient to reduce the risk of the mast itself becoming a nidus of infection. Dome shaped mask are adequate barriers against HIV virus.

Disposable gloves should mandatory double gloving must be considered when the patient is known or suspected to harbor an infective organism.

Dentists or auxiliary person with exudative lesion should not perform or assist any procedures in patients suffering from HIV.

Operating gowns and hair covers should be worn when patient is known or suspected to be infectious.

To minimize self-injury extreme caution must be used in handling sharp instruments, needles, scalpels and blades. The holder should used to insert and remove sharp instruments.

All handles and switches should be covered by plastic bags or aluminium foil and not touched by contaminated persons.

At the end of each procedure, clean the entire unit with freshly prepared 0.5% sodium hypochlorite or 2% glutaraldehyde.

Case Report: A male patient reported to the Dept. of Oral medicine and Radiology, in a private dental college, Lucknow with chief complaint of burning sensation in his mouth since one and half months.

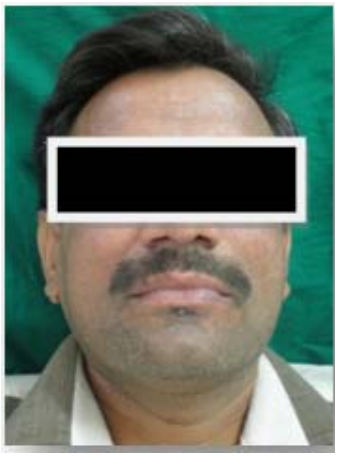

Fig 1: *Extraoral Photograph*

History of present illness: Patient was asymptomatic one and half months back when he noticed soreness in the mouth. Patient went to a local physician for treatment but was not completely relieved of symptoms. Patient had burning sensation on consuming spicy food. Then he visited present private dental college for treatment for the same.

Personal history: Patient used to chew gutka for 10-12 years and had left habit 02 months back. He used to chew 02 packets of tobacco each day and kept it in lower vestibule for 15 minutes and then spit it out. Patient brushed his teeth

once a day with toothbrush and toothpaste in the horizontal manner.

Area of chief complaint

Inspection - small multiple pin point ulcers are present on hard palate region. Site is junction of hard and soft palate. Size is around 0.2 mm. shape is oval. Base is erythematous.

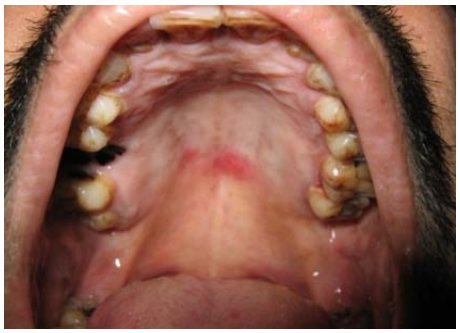

Fig 2: *Erythematous Candidiasis*

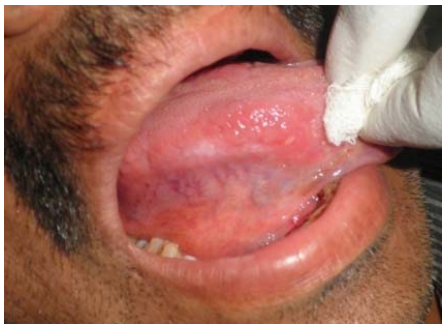

Fig 3: *Aphtuous Ulcers*

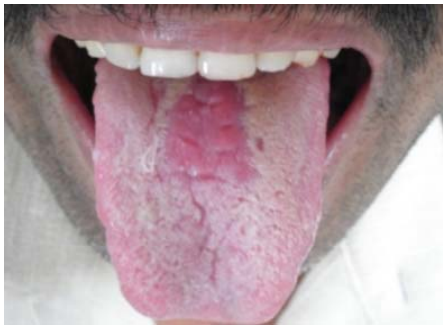

Fig 4: *Median Rhomboid Glossitis*

Median rhomboid glossitis present.
Aphthous ulcers were also seen.

Palpation - Inspectory findings were confirmed, ulcers were non tender. Edges were sloping & No induration was present.

Provisional diagnosis: Minor recurrent apthous ulcers on rt. lateral border of tongue, erythematous candidiasis on palate & dorsum of tongue.

Differential diagnosis: Erythematous candidiasis, AIDS related complex & herpetiform ulcers on hard palate.

Investigations: TRI-DOT test was done which was positive, HIV Triline also positive, HBs Ag ELISA-non-reactive & VDRL TEST-non reactive.

Fig 4: *Tri Dot Test*

Final diagnosis: AIDS related complex
Treatment plan: Emergency treatment- Candid mouth paint 3 times a day for two weeks, Cap B complex 2 times a day for two weeks, Follow up after 2 weeks & planned treatment.

Aggressive definitive approach: [9] Zidovudine+ Lamivudine + Indinavir

Recall visit: Patient was recalled after 1 month and healing of ulcers was normal.

Conflict of interest: Nil
Souirce of support: None

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