



Children with HIV Receiving Oral Health Care

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Description

The existence of one or more oral lesions is among the first manifestations in children with Acquired Immune Deficiency Syndrome (AIDS). Moreover, oral candidiasis and oral hairy leukoplakia are predictors of AIDS evolution and are related with CD4+T-lymphocyte cell count 200 cells. The prevalence of oral lesions in Human Immunodeficiency Virus (HIV)-infected children in developed countries is equivalent to 72%, meanwhile in developing countries it equates to 60%. For this reason health professionals should identify and treat the numerous oral manifestations in HIV-infected children. There are several oral lesions that could be present in HIV-infected children. However, the prevalence of oral lesions is considerably lower in children on Highly Active Anti-Retroviral Treatment (HAART) as compared to their equivalents not on HAART.

In regards to caries problems in children with HIV, the problem increases as the CD4 counts decreases however, in HIV-infected children taking HAART, the rate of decay is less associated with patients not receiving HAART. Furthermore, clinicians must know the side effects on their oral health of drug taken; the CD3+, CD4+, T-lymphocyte amount and proportion; and to solicit supplementary laboratory examinations including hepatitis, herpes, varicella zoster and papillomavirus with the purpose to offer secure management for HIV-infected children.

In general dental practice, children with AIDS disease in stages according to the American academy of pediatrics dentistry and World Health Organization classification (CD4 amounts), patients with absolute neutrophil count below 1,500 mm and with deranged liver functions tests will need antibiotic prophylaxis. Another important aspect is that patients with low platelet quantities (10,000 μ L-30,000 μ L) require a platelet transfusion prior to surgical procedures. Oral problems have an undesirable influence on the nutritional health status of HIV-infected children by decreasing food intake as a consequence of pain during ingestion as these patients have one or more oral manifestations. Malnutrition predisposes to periodontal disease, candidiasis and xerostomia.

To provide safe care for HIV-infected children, clinicians must know essential recommendations for treatment planning and prevention. Managing for these HIV-infected children requires close synchronization between the dentist, the paediatrician, the nutritionist and the child's parents or tutors. Preserving satisfactory oral health

through prevention associated with suitable treatment makes it feasible to maintain general health in these children.

Hepatocellular Carcinoma of Liver Fibrosis

Worldwide, approximately 10%-25% of Human Immunodeficiency Virus (HIV) infected people are also chronically infected with the Hepatitis C Virus (HCV). Since the introduction of antiretroviral therapy, life expectancy of HIV-infected patients has dramatically improved. As a consequence, HCV-related end-stage liver disease is presently an important cause of morbidity and the second-most important cause of death after HIV-related mortality in co-infected patients. Patients co-infected with HCV and HIV has increased mortality rates compared to those with HCV or HIV infection alone. Interestingly, co-infected patients develop hepatocellular carcinoma at a younger age than HCV-mono infected patients, and suffer an accelerated rate of progression to fibrosis. It is widely understood that HIV mono infection can contribute to liver fibrosis by multiple general mechanisms such as increase in bacterial translocation from the gut, increased mitochondrial toxicity and direct cytopathic effect, among others. However, it is unclear whether both viruses increase fibrosis progression *via* independent mechanisms, or act in conjunction, potentiating each other through synergistic pathways.

Hepatic fibrosis is a dynamic process initiated by liver injury that results in increased deposition of extra cellular matrix in the space of doses, the area in between the hepatocytes and the liver sinusoids, mainly inhabited by Hepatic Stellate Cells (HSC). Eventually, accumulation of extracellular matrix proteins and their decreased removal by matrix metallo proteinases results in a progressive replacement of the liver parenchyma by scar tissue, leading to liver cirrhosis and its complications. The production of Reactive Oxygen Species (ROS) is one of the main triggers for liver injury that results in the cascade that leads to fibrosis. ROS are oxygen-containing free radicals that cause injury through oxidation of lipids and proteins. Deregulation of mitochondria in the liver is the most recognized source of ROS during hepatic injury. Other stimuli include lipid peroxides and inflammatory cytokines, all of which will eventually lead to activation of HSCs with subsequent extracellular matrix accumulation and fibrosis. In addition, recent studies suggest that autophagy, a metabolic process in which cells digest their own organelles, activate HSC, initiating and stimulating liver fibrosis.

Chronic Infection of Bacterial translocation

Chronic infection with HCV is characterized by progression to liver fibrosis, resulting in cirrhosis and/or hepatocellular carcinoma. When HCV infects hepatocytes its core protein can modulate the mitochondrial membrane to induce ROS. In cellular models, the HCV non-structural protein NS5A modulates calcium homeostasis altering the concentration of cytosolic calcium and inducing oxidative stress with production of ROS. This increase in ROS results in fatty deposition in the sinusoids and eventual fibrosis. HCV-infected hepatocytes induce immune activation in the liver, with production of TNF-alpha and IL-1, which leads to activation of HSCs and creates an imbalance between matrix metallo proteinases and tissue-inhibitor metallo proteinases. Moreover, once HCV infection is established, HCV can exert modulation of chemokines gradients in the liver, which affects chemotaxis of immune cells into the liver. Chemokines, such as CCR2 can promote recruitment of inflammatory macrophages

into the sinusoids, thus increasing progression to liver injury. Because of the role of chemokines in liver fibrosis, they have become attractive targets for treatment of liver fibrosis. For example, CCR5 targeting is currently being developed as anti-fibrotic therapy for HIV/HCV co-infected patients. Kupffer cells infected with HCV increase expression and release of Tumor Growth Factor beta (TGF-beta), one of the best-characterized fibro genic cytokines. TGF-beta stimulates collagen I and alpha-smooth muscle actin, both of which are important components of the extracellular matrix. HCV-Infected hepatocytes also undergo apoptosis. Growing evidence suggests that following intake of apoptotic bodies, Kupffer cells express TNF and the death receptor component both of which worsens liver injury.

Hepatocytes in Insulin resistance

Although HIV does not replicate in hepatocytes, the HIV co-receptors CXCR4 and CCR5 are expressed in the hepatocyte surface, and the HIV protein gp120 can induce cell signaling in the liver through these co-receptors. HIV does, however, infect HSC. In these cells, gp120 induces activation of tissue-inhibitor metallic proteinases and the monocyte chemo-attractant protein 1 with subsequent HSC

chemo taxis and accumulation, leading to liver inflammation and fibro genesis. Outside of the liver, HIV infection leads to a substantial decrease of CD4+T cells in the gut, causing an increased "microbial permeability" in the gastrointestinal mucosa, with bacterial translocation into the portal and systemic circulation. The increase in lipopolysaccharide secondary to bacterial translocation can stimulate toll-like receptor 4 in the liver, stimulating TGF-beta production and prompting activation of the fibro genic cascade. Independent from direct viral effects, antiretroviral therapy for HIV, particularly nucleoside reverse transcriptase inhibitors, can induce mitochondrial toxicity. The lack of normal mitochondrial function impairs beta-oxidation of fatty acids with accumulation of these acids in the cellular cytosol leading to liver fibrosis. In addition, HIV-infected patients on antiretroviral therapy, particularly those on protease inhibitors, frequently experience lipodystrophy (known as HIV-associated lip dystrophy syndrome). Protease inhibitors can decrease peripheral lipolysis through inhibition of GLUT-4 activity increasing adipocyte size. These hypertrophic adipocytes in peripheral tissue and abdomen lose functional activity and become resistant to insulin. Consequently, insulin-resistant adipocytes secrete less adiponectin, which in turn increases body fat worsening deposition of liver fat and fibrosis.