HIV/HCV Co-Infection in a Teaching Hospital in Nigeria: A Short Review

Co-infection and opportunistic infections are a major source of concern in HIV positive patients on Highly Active Antiretroviral Therapy (HAART) [1]. The hepatitis viruses are of particular importance and also the re-emergence of Tuberculosis in parallel with incidence of HIV [2]. Hepatitis C Virus Co-infection (HCV) with HIV though less common than hepatitis B virus co-infection with HIV is considered a major source of public health concern [3,4]. This is due to the effect of HIV on the life cycle of HCV and subsequently on the hepatic system.

The mechanisms underlying accelerated liver disease in hepatotropic viruses/HIV co-infected individuals are poorly understood but may include the following: direct viral effects on hepatocytes and hepatic stellate cells, and immunologic alterations such as immune activation, apoptosis and diminished HCV specific T-cell responses. Also to be considered is the liver toxicity of antiretroviral drugs and the burden of metabolic disease contributing to a faster progression of liver fibrosis in HIV/HCV co-infection. HIV immune activation induces cytokine changes (e.g., IL -4, IL -5, and IL -13, TGF -β) that increase liver inflammation and fibrosis [5-7].

Co-infection also increases apoptosis of hepatocytes through a Fas/Fas L pathway that could account for accelerated liver disease. Accumulation of cytotoxic CD8 T cells in the liver that increases inflammatory mediators in co-infected compared to HCV monoinfected patients may also lead to increased tissue damage in co-infected patients. Recent evidence shows HIV – specific CD8 T-cells accumulate in the liver in co-infection and produce TNF -α, which is associated with liver fibrosis [8,9].

The reported prevalence of HIV/HCV co-infection notoriously varies significantly among studies even within the same geographical location, suggesting that an environmental factor probably hygiene is operating but yet to be identified. HIV and HCV are both transmitted through parental, sexual and vertical exposure but differ in the transmission efficiencies of these routes. The parenteral routes being more efficient for infection of HCV hence nosocomial infections and intravenous drug users being more susceptible to HCV infection than HIV.

However it appears though that the effects of co-infection with these viruses on the liver are somewhat limited to developed countries or the western world. This could be considered to be a wide assumption however numerous reports of severe hepatic damage have been cited in Europe and America but very little from Africa [10-12]. The majority of studies carried out in Africa are mainly descriptive and do not reveal actual incidence rates or occurrence of liver disease as opposed to HIV prevalence in these patients some studies even report hepatotoxicity as being uncommon [13-15]. In a study carried out in southwest Nigeria in 2013, the prevalence rate of HCV among HIV patients in our facility was found to be 23.3% [16]. It is generally accepted that one third of HIV patients are co-infected with HCV. Three years later of all the co-infected patients screened at the time none have developed any significant clinically overt liver damage. The main findings among them is a persistently low CD4 count despite being on highly active antiretroviral therapy (HAART including Zidovudine, Lamivudine and Nevirapine) which is not unexpected and mildly raised liver enzymes (serum aspartate transaminase and serum alanine transaminase). None were on treatment for a Fas/Fas L pathway that could account for accelerated liver disease. Accumulation of cytotoxic CD8 T cells in the liver that increases inflammatory mediators in co-infected compared to HCV monoinfected patients may also lead to increased tissue damage in co-infected patients. Recent evidence shows HIV – specific CD8 T-cells accumulate in the liver in co-infection and produce TNF -α, which is associated with liver fibrosis [8,9].

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References


