Case Report

Alcohol-Induced Hepatitis with HIV Infection: A Rare Case Report

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ABSTRACT

Human immunodeficiency virus (HIV) is a virus spread through certain body fluids that attacks the body's immune system, specifically the CD4 cells, often called T cells which result in damage to the immune system. Opportunistic infections or cancers take advantage of a very weak immune system and signal that the person has acquired immunodeficiency syndrome (AIDS). HIV-infected patients with controlled disease are prone to developing liver diseases from simple and common causes such as alcoholic and Non–Alcoholic fatty liver disease (NAFLD), viral hepatitis, and aging in addition to more HIV-specific processes such as Highly Active Antiretroviral Therapy (HAART)-related toxicity and direct injury to the liver by the HIV itself. Alcohol increases concentrations of Reactive oxygen species (ROS) throughout the body by inducing systemic hyperhomocysteinemia. Hepatitis viruses replicate primarily in the liver cells. Inflammation leads to the enlargement of the liver (hepatomegaly) in over 60% of the people infected with hepatitis. In this case report, we summarize regarding a patient who has been diagnosed with AIDS and alcohol-induced hepatitis which would further complicate the situation and the patient has been advised with ART therapy for a retroviral disease which is also a risk factor for inducing hepatitis.

Keywords: Non–Alcoholic fatty liver disease (NAFLD), Hyperhomocysteinemia, Hepatomegaly.

INTRODUCTION

As HIV-infected patients live longer, non-AIDS illnesses are becoming increasingly important sources of morbidity and mortality in the HIV-infected population. In particular, liver-related diseases are becoming increasingly prominent in HIV-infected patients. The liver-related disease has been estimated to account for 13-18%of all-causes of mortality in HIV-infected patients and is one of the leading causes of non-AIDS-related death. HIV-infected patients with controlled disease are prone to developing liver diseases from simple and common causes such as alcoholic and Non –Alcoholic fatty liver disease(NAFLD), viral hepatitis, and aging in addition to more HIV-specific processes such as Highly Active Antiretroviral Therapy (HAART)-related toxicity and direct injury to the liver by the HIV itself. Typical mechanisms of liver disease in HIV infected patients include oxidative stress, mitochondrial injury, lipotoxicity, immune-mediated injury, cytotoxicity, toxic metabolite accumulation, gut microbial translocation, systemic inflammation, senescence, and nodular regenerative hyperplasia. Alcohol increases concentrations of Reactive oxygen species (ROS) throughout the body by inducing systemic hyperhomocysteinemia[1,2]. Hepatitis viruses replicate primarily in the liver cells. Inflammation leads to the enlargement of the liver (hepatomegaly) in over 60% of the people infected with hepatitis and can cause the fibroelastic sheath (Glisson's capsule) surrounding the liver to stretch, which may be the cause of pain in the liver area. Alcohol liver disease is a frequent cause of enlarged liver symptoms. Microcytic hypochromic anemia is the type of anemia in which the circulating RBC's are smaller than the usual size of RBC's and have decreased red color. The most common cause of this type of anaemia is decreased iron reserves in the body which may be due to multiple reasons. Alchohol and iron may increase Oxidative stress and risk of alcohol-related liver diseases[3].

CASE REPORT

A 35-year-old man with greater than a 7-year history of alcohol misuse was presented to the hospital with a 10-day history of generalized weakness. Social history was remarkable for drinking whiskey and beer very often. Physical examination shows pale color of the skin especially on the face and palms which has occurred due to illness and anemia. The laboratory findings on admission are shown in the table-1 &2. The patient has Microcytic Hypochromic Anaemia with mild Leukocytopenia which indicates that the patient has iron deficiency Anemia. Liver function tests showed an increase in Bilirubin levels and liver transaminases. Abdominal sonography revealed an enlarged liver showing mild hepatomegaly with gallbladder wall edema and minimal ascites. A rapid HIV TRI-DOT test has been done to the patient which gave a positive result which means that the patient has been infected with the HIV virus. This indicates that the patient has a Retroviral disease. CD 4 cell count has done and the laboratory reports show a decline in CD 4 Immune cells. The patient was discharged after 5 days with the treatment provided for his symptoms and abnormal laboratory findings. A concerned doctor from the department of General Medicine has advised the patient to refer to ART therapy.

Table 1: Liver function test

Parameter	Observed value	Reference value
Total bilirubin	5.2mg/dl	0.3-1.0mg/dl
Direct bilirubin	1.3mg/dl	<0.3mg/dl
Indirect bilirubin	3.9mg/dl	0.2-0.7mg/dl
SGOT	200IU/L	5-40IU/L
SGPT	158IU/L	5-35IU/L
ALP	146IU/L	35-130IU/L

Table 2: Haematology report

Parameter	Observed value	Reference value
Hemoglobin	7.9gm/dl	13-18gm/dl
Total RBC	3.0 mill/cumm	4.0-6.5 mill/cumm
Total WBC	3,700 cells/cumm	4000-11000 cells/cumm
Neutrophils	59%	35-75%
Lymphocytes	32%	20-45%
Eosinophils	05%	01-08%
Monocytes	04%	01-06%
Basophils	00%	0-1%
Platelets	4.2 lakhs/cumm	1.5-4.5lakhs/cumm

DISCUSSION

Managing liver disease is an increasingly important component to the care of individuals infected with human immunodeficiency virus -1 (HIV-1). In some studies, nearly half of deaths among hospitalized HIV- infected patients in the ART- era have been attributed to liver disease. Although alcoholic liver disease is responsible for nearly half of all deaths due to chronic liver disease, the role of alcohol abuse on liver disease in HIV infected populations has not been well defined[4]. The role of exogenous agents, including hepatotoxic drugs and alcohol, is less well defined. In one study of 1358 HIV-infected individuals at an urban center, 10% reported hazardous drinking, which was independently associated with an elevated surrogate for hepatic fibrosis. These results suggest that alcohol abuse is prevalent among HIV-infected individuals and can independently contribute to liver disease progression. As a modifiable risk factor for liver disease, it is important that physicians provide counseling regarding alcohol consumption in this population[5]. The viral population undergoes remarkable changes from the time of initiation of infection to the time of over immunodeficiency. The large viral population in an infected person is usually founded by a single infected $CD4^+$ Tcell in the mucosal tissue proximal to the site of exposure. For much of the time course of the infection, viral evolution is apparent, a result of evading the humoral and cell-mediated immune responses, while the virus continues to replicate in $CD4^+$ T cells using CCR5 as the coreceptor. Initially, T cells in the gut-associated lymphoid tissue (GALT) are massively depleted even though a majority of these cells are not in the activated state, which is preferred for HIV-1infection in cell culture. The massive loss of GALT $CD4^+$ T cells happens early and therefore cannot be the direct cause of immunodeficiency, which occurs late. However, the GALT is likely the source for a significant fraction of the virus in the blood, although the relationship between the production of virus in lymphoid tissue and its transfer to the blood is unknown[6]. The indication of acquired immunodeficiency syndrome (AIDS) pathogenesis is progressive depletion of $CD4^+$ T-cell populations in close association with progressive impairment of cellular immunity and also increase susceptibility to opportunistic infections (OI). Disease progression in untreated human immunodeficiency virus (HIV) infection can take many years, and it was originally hypothesized to be a repercussion of slow, viral-mediated $CD4^+$ T-cell destruction. But, massive $CD4^+$ memory T-cell destruction is known to occur quite early in infection, almost always without overt immunodeficiency[7]. In patients infected with human immunodeficiency virus type-1 (HIV-1) Liver toxicity is one of the most relevant adverse effect of antiretroviral therapy (ART)[8]. Although there are many other causes of liver disease in these patiens the most common are viral co-infections, drugs and narcotics, which are most prominent upon treatment with highly active anti-retroviral therapy (HAART). Most patients with hepatic alterations are asymptomatic, and liver disease may unexpectedly be diagnosed during the evaluation of unexplained persistent elevations of serum liver enzymes. In this case, and once other known aetiologies have been ruled out, imaging procedures often suggest fatty infiltration of the liver parenchyma. Since most of these patients are indolent, fatty liver seems to be a benign and stable disease. Nevertheless, sensitizing the liver may lead to acute-on-chronic liver failure or cryptogenic cirrhosis with the necessity of liver transplantation. Consequently, it is of major clinical importance to assess stage and prognosis of liver disease in the HIV-positive patient[9].

CONCLUSION

In summary, this is a case of anemia and hepatitis with newly diagnosed HIV infection for which he was advised with ART therapy for further treatment. ART therapy has a greater risk of causing liver damage, besides patient also has alcohol addiction due to which the risk will be increased furthermore. So, the patient has to be properly counseled about further complications and adverse effects of ART therapy and consumption of alcohol increases mortality in HIV patients. If the hepatitis condition worsens, it can lead to further liver damage such as cirrhosis, permanent liver damage. Patient has to visit physicians frequently, to undergo regular liver function tests which helps in detecting the extent of liver damage. Liver biopsy should be performed in any patient with acquired immunodeficiency syndrome who has an unexplained fever, hepatomegaly or abnormal results of serum biochemical liver tests, and all specimens should be stained and cultured for mycobacteria and fungi.

Abbreviations used:

NAFLD: Non –Alcoholic fatty liver disease; HAART: Highly Active Antiretroviral Therapy; AIDS: Acquired immune deficiency syndrome; ROS: Reactive oxygen species; HIV: Human immunodeficiency virus; GALT: Gut- Associated Lymphoid Tissue

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Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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