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Original Research Article

The status of liver enzymes in patients of newly diagnosed human immuno-deficiency virus and their correlation with CD4 counts in a tertiary centre of North Eastern region of India

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ABSTRACT

Background: Liver dysfunction is a common complication in human immunodeficiency virus (HIV)-infected individuals due to factors like co-infections, antiretroviral therapy (ART) toxicity, and immune dysregulation. Evaluating liver enzyme abnormalities can help predict disease progression and guide therapy.
Methods: A cross-sectional study was conducted on 100 newly diagnosed HIV-positive individuals. Liver function was assessed using serum glutamate-oxaloacetate transaminase (SGOT) and serum glutamate-pyruvate transaminase (SGPT) levels, and immune status was measured by CD4 counts. The correlation between liver enzymes and CD4 levels was analyzed statistically.
Results: The study population had a male predominance (70%) with a mean age of 37.29±13.01 years. A weak positive correlation was found between CD4 count and both SGOT and SGPT levels. Liver enzyme derangements were more prominent in patients with lower CD4 counts, indicating advancing immunosuppression.
Conclusion: This study highlights the universally raised liver enzymes in all the patients of newly diagnosed HIV indicating hepatic injury by the virus itself. Medical literature regarding the probable mechanism is all together lacking. There was minimal positive correlation found between CD4 count and liver enzymes.

Keywords: Newly diagnosed HIV, Liver enzymes, CD4 count

INTRODUCTION

Liver enzyme elevations are frequently observed in individuals living with human immunodeficiency virus (HIV), posing a significant clinical challenge due to their multifactorial etiology. While co-infections with hepatitis B (HBV) and hepatitis C (HCV) viruses are well-known contributors to liver damage in this population, abnormal liver chemistries, including elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT), are common even in the absence of viral hepatitis.[1] The direct impact of HIV infection itself on hepatocyte function, coupled with the potential hepatotoxic effects of antiretroviral therapy (ART), plays a crucial role in these enzyme abnormalities.[2,3] Furthermore, the complex interplay between chronic inflammation, immune dysregulation, and metabolic comorbidities inherent to HIV disease can also contribute to liver injury.[4]

The relationship between liver enzyme levels and CD4+T-cell counts, a key indicator of immune status in HIV, is of particular interest. Studies have suggested an inverse correlation, where lower CD4+ counts (indicating more advanced immunosuppression) may be associated with a higher prevalence or severity of liver enzyme elevations.[5] This highlights the potential for HIV-associated liver damage even in ART-naïve patients, and underscores the

importance of monitoring liver function to prevent chronic liver disease progression in people living with HIV.[4,6]

METHODS

Study type

Prospective observational based study was done in this study.

Study place

This study was conducted at Swaroop Rani Nehru Hospital, Prayagraj from 01 March 2024 to 01 March 2025.

Inclusion criteria

Newly diagnosed HIV cases aged ≥18 years were included in the study.

Exclusion criteria

Subjects who were already suffering from Liver diseases and those on any hepatotoxic drugs were excluded. Subjects younger than 18 years of age were excluded.

Study procedure

After obtaining informed written consent from each participant, newly diagnosed HIV-positive patients were systematically enrolled into the study. A detailed history was recorded, focusing on the presence of any known thyroid disorder, current pregnancy or lactation status, and any prior exposure to antiretroviral therapy (ART). This was followed by a comprehensive physical examination and a full general and systemic examination to assess the clinical status of the patient.

Relevant laboratory investigations were conducted, including thyroid function tests (free T3, free T4, and serum TSH), HIV 1 and 2 confirmation, CD4 lymphocyte count, complete blood count parameters (hemoglobin, total leukocyte count, platelet count, MCH, MCHC, MCV), liver function tests (SGOT/AST, SGPT/ALT), and renal function tests (serum urea and creatinine). All clinical and biochemical findings were meticulously documented to evaluate the baseline health profile of the participants and to facilitate follow-up analysis of thyroid function after the initiation of ART.

Statistical analysis

Pearson correlation was used to assess associations between SGOT, SGPT and CD4 count.

This prospective observational study was conducted at the Department of Medicine, M.L.N. Medical College, Prayagraj, including 100 newly diagnosed HIV-positive patients aged ≥18 years. Baseline liver function tests (SGOT and SGPT) and CD4 counts were measured prior to ART initiation. Statistical analysis was performed using Pearson correlation used to assess associations between SGOT, SGPT and CD4 count.

RESULTS

Total 100 subjects newly diagnosed with HIV were included in the study. The majority of those subjects were young males (70%) (Table 1); with mean age of study subjects was 37.29 years (±13.01) (Table 2).

Table 1: Gender-wise distribution of enrolled patients.

Gender distribution	N	%
Female	30	30.00
Male	70	70.00

Table 2: Age-wise distribution of participants.

Age distribution (years)	N	%
<20	3	3.00
20-29	34	34.00
30-39	25	25.00
40-49	19	19.00
50-59	11	11.00
60-70	8	8.00
Mean±SD	37.29±13.01	

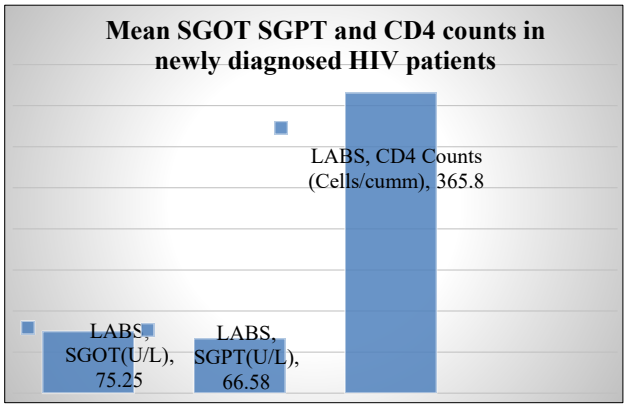

Figure 1: Graphical representations of mean SGOT, SGPT and CD4 counts.

As the subjects were yet not started on ART so the derangement of SGOT and SGPT were solely attributable to HIV itself. Surprisingly all the subjects had abnormal SGOT i.e. more than 40 IU (≤40 IU/l was the reference range) and all had abnormal SGPT levels i.e. more than 40 IU (≤40 IU/l was the reference range). Mean value of SGPT was 66.58 and SGOT was 75.25. Mean CD4 count was 365.8 (Figure 1). Karl Pearson correlation coefficient was 0.1379 when CD4 count was correlated with SGPT and was 0.152694 when CD4 count was correlated with SGOT. The graph depicts the bar diagram showing age wise distribution abnormal liver enzymes (Figure 2).

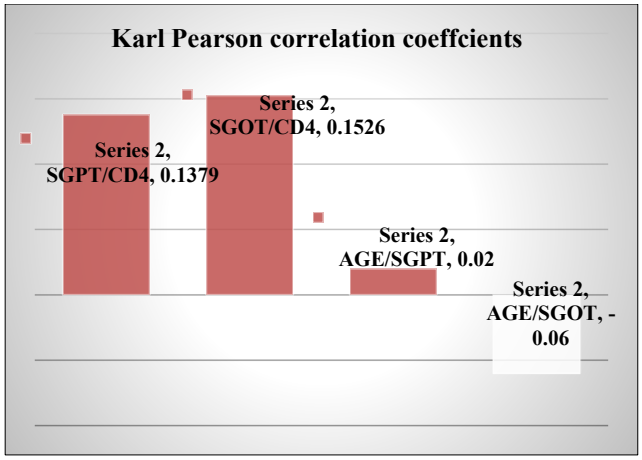

Figure 2: Karl Pearson coefficient with SGOT and SGPT.

DISCUSSION

As viral illnesses are known to elevate liver enzymes and marked rise in SGOT and SGPT is seen in viral hepatitis A, B, C, D, E viruses besides variable rise seen in dengue. This study is unique as all the newly diagnosed HIV patients had raised liver enzymes. In this study all the subjects had abnormal SGOT i.e. more than 40 IU and all had abnormal SGPT levels i.e. more than 40 IU. Mean value of SGPT was 66.58 and SGOT was 75.25. Mean CD4 count was 365.8.

Is HIV causing direct liver injury?

Several studies align with the notion that HIV has a pleotropy towards liver and causes direct liver injury. Dusingize et al determined the associations of HIV infection/CD4 count with markers of hepatocellular damage (elevated AST and ALT) and liver synthetic function (decreased albumin) in HIV-infected antiretroviral therapy-naive and uninfected Rwandan women. Findings suggest that HIV-associated liver damage may occur in ART-naive patients. Although liver abnormality prevalences in this cohort of HIV-infected Rwandan women are less than reported in developed countries, caution is needed for risk assessment measures to monitor and screen HIV-infected patients pre- and post-ART initiation in African clinical settings to curtail potential risks associated with HIV infection.[7] Shiferaw et al In his study total of 164 HAART experienced and 164 HAART naïve patients were studied. Blood specimen was collected to determine alanine aminotransferase (ALT) and aspartate aminotransferase (AST), CD4 count, and viral hepatitis. The prevalence of liver enzyme abnormality was 20.1% and 22.0% among HAART experienced and HAART naïve patients, respectively.

Liver disease has emerged as the most common non-AIDS-related cause of death in HIV patients.[8] Mata-Marín et al suggest that there is an association between HIV viral load and aminotransferases as markers of hepatic damage; we should improve recognition, diagnosis and potential therapy of hepatic damage in HIV infected patients. There was a moderately strong, positive correlation between AST serum levels and HIV viral load ($r=0.439$, $p<0.001$); and a weak correlation between ALT serum levels and HIV viral load ($r=0.276$, $p=0.034$); after adjusting the confounders in lineal regression model the correlation remained significant. Abnormalities in liver function tests could be produced exclusively by direct inflammation in hepatocytes, caused by the HIV. Mechanisms by which HIV causes hepatic damage are still unknown.[9]

Multifactorial etiology of liver dysfunction

The liver is highly susceptible to injury from various causes in HIV-infected individuals, making a simple linear relationship with CD4 counts unlikely. These causes include the following.

Co-infections

Hepatitis B virus (HBV) and hepatitis C virus (HCV) co-infections are highly prevalent among HIV patients and are a leading cause of chronic liver disease and elevated transaminases, often independent of CD4 count progression.[5,10]

Opportunistic infections

Certain opportunistic infections, particularly in more advanced HIV disease, can affect the liver and cause enzyme elevations.

Alcohol and drug use

Substance abuse, especially alcohol, is a significant contributor to liver damage in this population.

Non-alcoholic fatty liver disease (NAFLD)

Growing recognition of NAFLD in HIV patients, potentially linked to metabolic changes, can also contribute to transaminase elevations.[5]

Drug-induced liver injury (DILI)

While our data is from "newly diagnosed" patients, implying they may be ART-naive, it is crucial to acknowledge that once antiretroviral therapy (ART) is initiated, it becomes a major factor influencing liver enzyme levels, sometimes causing elevations that are unrelated to CD4 count changes.[11] Even newer regimens, while generally safer for the liver, can still have some impact.[5]

Correlation of liver enzymes with CD4 counts

In the present study Pearson correlation coefficient between SGOT and CD4 count is 0.1527 and Pearson

correlation coefficient between SGPT and CD4 count is 0.1379.

However, since the values are indeed numerically positive, even a slight positive trend might imply and how it relates to existing literature, while maintaining scientific accuracy about the strength of this correlation.

A slightly positive correlation of SGOT and SGPT with CD4 counts in newly diagnosed HIV patients.

The analysis of the study data reveals a very weak positive linear correlation between SGOT (0.1527) and SGPT (0.1379) levels with CD4 counts in newly diagnosed HIV patients. This finding, though quantitatively weak, prompts a discussion within the broader context of HIV pathophysiology and existing research.

Interpreting a slightly positive trend

In the highly dynamic environment of HIV infection, the relationship between immune status (CD4 count) and liver health (SGOT/SGPT) is complex and influenced by numerous factors. A very weak positive correlation could theoretically suggest the following.

Early immune activation/inflammation

In the early stages of HIV infection, even before severe immunosuppression sets in, there is immune activation and chronic inflammation. This systemic inflammation could potentially have minor, early effects on various organs, including the liver, leading to subtle elevations in liver enzymes. If these inflammatory processes are somehow linked to initial immune responses that might also correlate with a still-robust, or only slightly declining, CD4 count in newly diagnosed individuals, a very minor positive trend could theoretically emerge. However, this is largely speculative given the negligible correlation strength.

Co-occurring factors

It's more plausible that such minor positive correlations, if truly reflective of an underlying biological process, might be driven by co-occurring factors that independently influence both parameters. For instance, certain lifestyle factors, nutritional states, or very early, subclinical co-infections (e.g., undiagnosed viral hepatitis) could simultaneously lead to minor liver enzyme elevations and also reflect a general health status that has not yet significantly impacted CD4 counts in newly diagnosed patients.

Most research investigating the relationship between liver enzymes and CD4 counts in HIV tends to find either weak correlation or more complex, indirect associations, when confounding factors are considered especially when confounding factors are considered.

Absence of strong direct correlation

Several studies align with the general notion that there isn't a strong direct linear correlation between baseline liver enzymes and CD4 counts. For example, Devaraj et al a cross-sectional study in Western India found no statistically significant correlation between deranged liver function tests (LFTs) and CD4 values.[12] Similarly Priya et al research exploring the effect of antiretroviral treatment regimens on liver enzymes in HIV patients concluded that there was no correlation between age and CD4 count with liver enzymes.[13]

Given these numerous potential confounders, isolating a direct, independent, and clinically meaningful linear correlation between baseline CD4 count and liver enzymes in newly diagnosed patients becomes challenging. The very weak positive correlations observed in our data are more likely reflections of the inherent biological noise and the complex interplay of these multiple factors with a strong, direct biological linkage where higher CD4 counts directly lead to higher (or lower) liver enzymes or vice versa.

Limitations

The study was conducted with a relatively small sample size of 100 participants, which limits the generalizability of the findings to a larger population, the assessment of thyroid function was only done after 3 months of ART, which may not be sufficient to observe long-term effects or trends in thyroid dysfunction, other potential confounding variables, such as nutrition, lifestyle factors, and comorbidities, were not systematically controlled for, which may have influenced thyroid function independently of ART, and although the study evaluated changes pre- and post-ART, it did not provide a longitudinal analysis of thyroid function over an extended period, limiting insights into the long-term effects of ART on thyroid health.

CONCLUSION

This study is unique as all the newly diagnosed HIV subjects had raised liver enzymes. Our data indicates a positive linear correlation between SGOT/SGPT with CD4 counts in newly diagnosed HIV patients, though the correlation was very mild. This finding is largely consistent with the latest studies showing positive pleotropy of HIV virus towards liver. The multifactorial nature of liver enzyme elevations in HIV, driven by co-infections, lifestyle, other comorbidities, and eventually ART, typically obscures the simple linear relationship with CD4 count, making it a challenging task. Continuous monitoring of both immunological status and liver function remains paramount for comprehensive management of HIV patients, regardless of a direct strong linear correlation between these specific markers.

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