



## Original Article

# Prevalence of Metabolic-Associated Steatotic Liver Disease in Patients with Type 2 Diabetes with and without HIV: Retrospective Multicenter Study

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## ABSTRACT

**Introduction:** Metabolic dysfunction-associated steatotic liver disease (MASLD) is a frequent complication in patients with Type 2 Diabetes (T2D). This study aims to evaluate the impact of HIV status on the prevalence of MASLD in patients with T2D.

**Methods:** We utilized the TriNetX global federated health research network to perform a comparative analysis of two cohorts: T2D patients with HIV (Cohort 1) and T2D patients without HIV (Cohort 2). Propensity score matching controlled for confounders such as age, gender, Hemoglobin A1c, LDL, HDL, total cholesterol, triglycerides, BMI, and hypertension. The study was exempt from IRB review as it did not involve direct human subjects, per the University at Buffalo Institutional Review Board.

**Results:** Initial data included 168,428 patients in Cohort 1 and 9,040,558 in Cohort 2. After matching, each cohort consisted of 166,803 patients. MASLD prevalence was 7.1% in HIV-positive T2D patients and 6.7% in HIV-negative T2D patients, with a significant risk difference (RD = 0.004, 95% CI: 0.002 to 0.006,  $p < 0.0001$ ). The risk ratio (RR) was 1.062 (95% CI: 1.036 to 1.089), and the odds ratio (OR) was 1.067 (95% CI: 1.039 to 1.096).

**Conclusion:** HIV-positive T2D patients exhibit a slightly higher risk of developing MASLD than their HIV-negative counterparts. These results underscore the need for specialized screening and management of MASLD in patients with T2D, particularly those living with HIV.

## 1. Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) and Type 2 diabetes mellitus (T2D) represent two of the most significant metabolic disorders that often co-occur, reflecting a complex interplay of lifestyle, genetic predisposition, and systemic insulin resistance. T2D is characterized by chronic hyperglycemia and insulin resistance, factors that are strongly linked to the development of serious complications such as cardiovascular diseases and nephropathy. Similarly, MASLD, which is defined by the accumulation of fat in the liver in the absence of excessive alcohol consumption, ranges from simple steatosis to more severe forms like steatohepatitis, which can progress to fibrosis and cirrhosis [1]. The prevalence of MASLD among individuals with HIV has risen, necessitating a deeper understanding of the synergistic effects of these conditions on liver health. This is particularly crucial as the mechanisms driving liver disease progression in HIV-positive individuals may differ, thereby requiring tailored management

strategies to effectively address the unique challenges posed by this combination of conditions [2, 3].

Research exploring the intersection of MASLD and HIV has garnered considerable attention, particularly among people living with HIV (PLWH). Understanding the risk factors and developing effective management strategies for MASLD in this population is critical to improving their overall health outcomes. A systematic review and meta-analysis were conducted to ascertain the prevalence of MASLD and significant fibrosis within the population of PLWH, as well as to delineate the associated risk factors. Analyzing data from studies published over the period 2009 to 2022, which encompassed a cumulative sample of 6,326 PLWH, the meta-analysis calculated a pooled prevalence of 38% for MASLD and 13% for significant fibrosis. Notable regional and economic variations were observed, and key risk factors were identified as elevated body mass index (BMI), increased triglyceride levels, and dyslipidemia, all of which were significantly correlated with heightened risk-adjusted odds of developing MASLD in PLWH [4]. Furthermore, specific predictors such as increased BMI and certain ART regimens, like Tenofovir Alafenamide (TAF), have been linked to the development of MASLD, indicating the need for targeted monitoring and management strategies in this vulnerable group [5].

PLWH with MASLD demonstrates accelerated fibrosis progression, marked by higher fibrosis stages despite lower disease activity and BMI compared to HIV-negative counterparts, implicating HIV-specific factors [6, 7]. These patients also face an elevated

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incidence of major adverse cardiovascular events, with atherogenic dyslipidemia—characterized by increased triglycerides and reduced HDL cholesterol—further heightening cardiovascular risk [7, 8]. Additionally, HIV-associated chronic inflammation and TAF-induced weight gain contribute to metabolic dysfunction, thereby increasing the risk of metabolic syndrome, T2D, and subsequent fibrosis progression [9, 10]. Specific ART regimens, such as those including TAF and Integrase Strand Transfer Inhibitors (INSTIs), have been linked to metabolic disturbances that may exacerbate MASLD, although rilpivirine appears to offer protective effects against liver fibrosis [11, 12]. Concomitantly, alterations in gut microbiota and persistent immune activation with mitochondrial dysfunction further promote hepatic inflammation and metabolic injury [10, 13]. Finally, MASLD in PLHIV is emerging as an independent risk factor for hepatocellular carcinoma—even in the absence of cirrhosis—underscoring the need for intensified liver screening protocols to facilitate early detection [7, 8].

This study aims to explore how HIV status affects the prevalence of MASLD in people with T2D. Using the TriNetX global health research network, it compares T2D patients with and without HIV, adjusting for variables like age, gender, and clinical factors through propensity score matching. This approach helps identify important differences in MASLD prevalence, informing targeted screening strategies for patients with T2D living with HIV. This study provides novel insights into the prevalence of MASLD in patients with T2D with and without HIV, an underexplored population. Utilizing the TriNetX global federated health research network, it draws from a vast dataset across 122 healthcare organizations, making it one of the most comprehensive investigations on this topic.

## 2. Methods

### 2.1. Study Design and Participants

This retrospective cohort study utilized the TriNetX global federated health research network, which provides access to anonymized electronic medical records from 122 healthcare organizations globally. The study aimed to evaluate the impact of HIV status on the prevalence of MASLD among patients with T2D.

### 2.2. Cohort Definition

For this analysis, we established two distinct cohorts based on their medical diagnoses: Cohort 1, consisting of patients with both T2D and HIV, was identified using the ICD-10 codes E11 for T2D, B20 for HIV disease, and Z21 for asymptomatic HIV infection status. Cohort 2 comprised patients diagnosed with T2D but without any recorded HIV infection; this group explicitly excluded patients identified by the ICD-10 codes B20 or Z21. All participants in both cohorts were required to be at least 18 years old at the time of their most recent diagnosis.

### 2.3. Data Collection

Data extracted from the TriNetX network included demographics, clinical diagnoses, laboratory values, and medication prescriptions. This data spanned the entire period the patients were active within the healthcare system, up to the index event defined below.

### 2.4. Index Event and Time Window

The index event for each cohort was defined as the first recorded diagnosis of T2D. The analysis time window spanned from 1-day post-index event up to five years post-index event. Patients whose index event occurred more than 20 years before the analysis date were excluded to ensure the relevance and accuracy of the medical data.

### 2.5. Propensity Score Matching

Propensity score matching was employed to balance the two cohorts based on age, gender, and key clinical parameters such as hemoglobin A1c, LDL cholesterol, triglycerides, HDL cholesterol, total cholesterol, BMI, and the presence of hypertensive diseases. Matching was performed using a nearest neighbor matching algorithm without replacement, ensuring a 1:1 ratio between the matched cohorts.

### 2.6. Statistical Analysis

The primary outcome assessed was the prevalence of MASLD, identified by the ICD-10 code K76.0. Statistical measures calculated included the risk difference, risk ratio, and odds ratio between the two cohorts. The significance of differences in MASLD prevalence between cohorts was determined using chi-squared tests for proportions. A p-value of less than 0.05 was considered statistically significant.

### 2.7. Ethical Considerations

The study protocol was reviewed by the University at Buffalo Institutional Review Board (UBIRB) and determined to be not research involving human subjects as per IRB ID: STUDY00008312, thus IRB review and approval were not required.

## 3. Results

The database records 1,823,448 patients under the ICD-10-CM code K76.0 for NAFLD, 535,733 patients under code B20 for HIV disease, and 227,396 patients under code Z21 for asymptomatic HIV infection status. Additionally, there are 9,439,663 patients listed under code E11 for T2D. These codes are commonly used in clinical settings to diagnose these conditions. In this comparative analysis using the TriNetX platform, we evaluated the prevalence of MASLD in two cohorts of patients with T2D: those with HIV (Cohort 1) and those without HIV (Cohort 2). Initially, Cohort 1 included 168,428 patients, which was matched down to 166,803 patients after propensity score matching to align with Cohort 2, which was reduced from 9,040,558 to the same number for consistency in comparison (Table 1).

The prevalence of MASLD in Cohort 1 (patients with T2D and HIV) was found to be 7.1%, with 11,806 patients diagnosed with the condition. In contrast, Cohort 2 (patients with T2D without HIV) showed a slightly lower prevalence of 6.7%, involving 11,112 patients. The marginal difference between the cohorts was statistically significant, with a risk difference of 0.004 (95% CI: 0.002 to 0.006), and a p-value of less than 0.0001, indicating that HIV-positive patients with T2D are at a marginally higher risk of developing MASLD compared to their HIV-negative counterparts (Table 2).

Furthermore, the analysis yielded a risk ratio of 1.062 (95% CI: 1.036 to 1.089) and an odds ratio of 1.067 (95% CI: 1.039 to 1.096), both reinforcing the increased risk among the HIV-positive cohort.

Propensity score matching revealed significant differences between the groups. Cohort 2 (patients with T2D without HIV) showed a metabolic profile with higher LDL, BMI, cholesterol, and A1c, as well as lower HDL levels, factors that typically favor an increased risk of MASLD. Despite this, our analysis demonstrated that the MASLD risk was actually higher in Cohort 1 (patients with T2D and HIV) (7.1% vs. 6.7%, with a risk difference of 0.004 [95% CI: 0.002, 0.006] and  $p < 0.0001$ ). This suggests that while the unfavorable metabolic parameters in the T2D without HIV group would ordinarily predispose them to a greater MASLD risk, HIV

**Table 1:** Characteristics of Cohort 1 (T2D with HIV) and Cohort 2 (T2D without HIV) before and after propensity score matching

| Variable       | Cohort     | Before Propensity Score Matching |           |             |         | After Propensity Score Matching |          |             |         |
|----------------|------------|----------------------------------|-----------|-------------|---------|---------------------------------|----------|-------------|---------|
|                |            | Mean $\pm$ SD                    | Patients  | % of Cohort | P-value | Mean $\pm$ SD                   | Patients | % of Cohort | P-value |
| Age at Index   | T2D w HIV  | 50.8 $\pm$ 18.3                  | 166,803   | 100%        | <0.001  | 50.8 $\pm$ 18.3                 | 166,803  | 100%        | 0.934   |
|                | T2D wo HIV | 60.7 $\pm$ 15.5                  | 8,743,382 | 100%        | <0.001  | 50.8 $\pm$ 18.3                 | 166,803  | 100%        | 0.934   |
| Female         | T2D w HIV  | –                                | 90,793    | 54.4%       | <0.001  | –                               | 90,793   | 54.4%       | 0.981   |
|                | T2D wo HIV | –                                | 4,220,146 | 48.3%       | <0.001  | –                               | 90,800   | 54.4%       | 0.981   |
| Hypertension   | T2D w HIV  | –                                | 81,881    | 49.1%       | <0.001  | –                               | 81,881   | 49.1%       | 0.986   |
|                | T2D wo HIV | –                                | 1,701,545 | 19.5%       | <0.001  | –                               | 81,876   | 49.1%       | 0.986   |
| Hemoglobin A1c | T2D w HIV  | 6.3 $\pm$ 1.7                    | 78,509    | 47.1%       | <0.001  | 6.3 $\pm$ 1.7                   | 78,509   | 47.1%       | <0.001  |
|                | T2D wo HIV | 7.1 $\pm$ 1.9                    | 1,778,427 | 20.3%       | <0.001  | 6.8 $\pm$ 1.9                   | 78,623   | 47.1%       | <0.001  |
| LDL            | T2D w HIV  | 97.3 $\pm$ 37.5                  | 80,292    | 48.1%       | <0.001  | 97.3 $\pm$ 37.5                 | 80,292   | 48.1%       | <0.001  |
|                | T2D wo HIV | 99.7 $\pm$ 38.4                  | 1,681,948 | 19.2%       | <0.001  | 100.7 $\pm$ 38.4                | 80,315   | 48.1%       | <0.001  |
| BMI            | T2D w HIV  | 30.1 $\pm$ 7.8                   | 127,021   | 76.2%       | <0.001  | 30.1 $\pm$ 7.8                  | 127,021  | 76.2%       | <0.001  |
|                | T2D wo HIV | 32.3 $\pm$ 8.1                   | 2,690,096 | 30.8%       | <0.001  | 33.1 $\pm$ 8.9                  | 126,895  | 76.1%       | <0.001  |
| Triglyceride   | T2D w HIV  | 157.5 $\pm$ 145.2                | 81,086    | 48.6%       | <0.001  | 157.5 $\pm$ 145.2               | 81,086   | 48.6%       | <0.001  |
|                | T2D wo HIV | 163.3 $\pm$ 167.7                | 1,723,725 | 19.7%       | <0.001  | 161.7 $\pm$ 156.6               | 81,121   | 48.6%       | <0.001  |
| HDL            | T2D w HIV  | 48.5 $\pm$ 17.4                  | 80,974    | 48.5%       | <0.001  | 48.5 $\pm$ 17.4                 | 80,974   | 48.5%       | <0.001  |
|                | T2D wo HIV | 44.6 $\pm$ 18.0                  | 1,703,359 | 19.5%       | <0.001  | 45.3 $\pm$ 18.0                 | 80,918   | 48.5%       | <0.001  |
| Cholesterol    | T2D w HIV  | 173.1 $\pm$ 49.2                 | 81,245    | 48.7%       | <0.001  | 173.1 $\pm$ 49.2                | 81,245   | 48.7%       | <0.001  |
|                | T2D wo HIV | 176.2 $\pm$ 48.5                 | 1,705,346 | 19.5%       | <0.001  | 176.9 $\pm$ 48.2                | 81,325   | 48.8%       | <0.001  |

T2D, Type 2 Diabetes; HIV, Human Immunodeficiency Virus; LDL, Low-Density Lipoprotein; BMI, Body Mass Index; HDL, High-Density Lipoprotein; w, with; wo, without

**Table 2:** Risk Analysis of MASLD in Type 2 Diabetes Patients With and Without HIV

| Comparison | Cohort     | Sample Size (n) | People with Outcome | Risk  | Risk Difference (95% CI) | P-value |
|------------|------------|-----------------|---------------------|-------|--------------------------|---------|
| MASLD      | T2D w HIV  | 166,803         | 11,806              | 0.071 | 0.004 (0.002, 0.006)     | <0.0001 |
| MASLD      | T2D wo HIV | 166,803         | 11,112              | 0.067 | 0.004 (0.002, 0.006)     | <0.0001 |

MASLD, Metabolic dysfunction-associated steatotic liver disease; T2D, Type 2 Diabetes; HIV, Human Immunodeficiency Virus; n, Sample Size; CI, Confidence Interval; w, with; wo, without; P-value, Probability Value

status itself appears to contribute an additional, independent risk for MASLD.

#### 4. Discussion

On the management front, nutritional and lifestyle modifications are recommended as primary interventions for MASLD in PLWH. A recent study highlighted the pivotal role of dietary and physical activity interventions in effectively managing MASLD, while also illuminating the unique obstacles faced by PLWH in implementing these lifestyle modifications [14]. Additionally, the presence of MASLD in PLWH has been associated with an increased risk of metabolic comorbidities such as diabetes and dyslipidemia, with one study pointing out that MASLD significantly predicts the development of these conditions in HIV-monoinfected patients [15]. These findings advocate for comprehensive, tailored interventions that address both the lifestyle and medical aspects of managing MASLD in HIV-infected individuals.

The long-term use of ART has been a game-changer in improving the life expectancy of PLWH; however, it also brings with it several metabolic challenges, including an increased risk of developing MASLD. Specific ART drugs such as TAF and INSTIs have

been pinpointed as independent predictors for the development of steatosis, with studies demonstrating a significant escalation in MASLD risk among patients using these drugs. Conversely, Tenofovir Disoproxil Fumarate (TDF) appears to offer a protective effect against weight gain and steatosis progression [5]. Furthermore, Protease Inhibitors (PIs), particularly Atazanavir/Ritonavir (ATV/r), are associated with a higher prevalence of metabolic syndrome and MASLD compared to treatments involving non-nucleoside reverse transcriptase inhibitors (NNRTIs) or in ART-naïve patients, highlighting the varied impacts of different ART regimens on metabolic health [16].

The duration of ART use plays a critical role in the development of MASLD, with long-term Highly Active Antiretroviral Therapy (HAART) usage linked to an increased prevalence of this liver disease. Factors such as elevated plasma glucose levels, increased waist circumference, and elevated serum triglycerides are significant risk factors emerging from prolonged HAART use, underscoring the need for ongoing monitoring and management of these metabolic risks in the treatment of HIV [17]. Additionally, the mechanisms connecting ART to MASLD involve both drug-induced effects and HIV-associated complications such as lipodystrophy, which promotes abnormal fat distribution and exacerbates

MASLD risk. This relationship is further complicated by chronic HIV-related inflammation and immune activation that contribute to hepatic steatosis and the progression toward more severe liver conditions like MASLD [14].

The study's strengths lie in its large sample size and the use of TriNetX, a global federated health research network that provides a robust dataset for examining the prevalence of MASLD in patients with T2D, both with and without HIV. The detailed stratification into specific cohorts based on HIV status allowed for a nuanced analysis of MASLD's prevalence across these distinct groups, enhancing the relevance and specificity of the findings. Additionally, the use of propensity score matching to control for confounders adds to the credibility of the results.

However, this study also has several limitations. Its retrospective nature inherently restricts the ability to establish causality, and there may be residual confounding factors that are not accounted for or measured such as smoking, alcohol consumption, other comorbidities, or ART regimens used by patients. The reliance on electronic medical records and diagnostic codes might lead to misclassification or underreporting of outcomes like MASLD. Furthermore, the study's setting within a specific network of healthcare organizations might limit the generalizability of the findings to broader populations. Although the data were adjusted for several known confounders, unknown or unmeasured variables could still influence the results. Lastly, the observational design cannot match the rigor of randomized controlled trials in determining the causal relationships and safety, making it crucial to approach the conclusions with an understanding of these contextual limitations [18].

## 5. Conclusions

This study's findings reveal that HIV-positive patients with T2D exhibit a marginally higher prevalence of MASLD compared to their HIV-negative cohort, underscoring the need for targeted screening and proactive management of MASLD in this subgroup. Given the unique interplay between HIV infection and metabolic health, these results highlight the importance of integrated healthcare strategies to address the increased risk of MASLD in patients living with HIV, thereby improving health outcomes and quality of life for this vulnerable population.

## Conflicts of Interest

The authors declare no conflicts of interest.

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## Institutional Review Board (IRB) approval

The Institutional Review Board at the Jacobs School of Medicine and Biomedical Sciences, University at Buffalo, NY, USA, approved the study protocol under IRB approval number (STUDY00008312).

## Large Language Model

No generative artificial intelligence or large language model tools were used in the preparation of this manuscript.

## Authors Contribution

HA, MA, and AM conceptualized and designed the study. HA and MA performed the data collection, statistical analysis, and wrote the initial manuscript draft. HA and ON contributed to data collection and validation. MA assisted with data interpretation and literature review. AM supervised the project, provided clinical expertise, and critically revised the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript for publication.

## Data Availability

All used data is available within the TriNetX database platform.

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