HEPATOLOGY FORUM

Volume 7, Issue 2, 2026 | 139–145 DOI: 10.14744/hf.2026.11444

ORIGINAL RESEARCH

Empagliflozin versus dapagliflozin in patients with liver cirrhosis: A comparative real-world study on hepatic decompensation

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Highlights & Insights

- **Scientific Gap:** Sodium-glucose cotransporter-2 inhibitors (SGLT2i) may benefit cirrhosis patients, but comparative real-world data on empagliflozin and dapagliflozin are limited. This study compares their outcomes.
- **Key Finding:** Empagliflozin and dapagliflozin showed similar five-year mortality and hospitalization rates. However, empagliflozin was associated with fewer hepatic decompensation events, such as hepatic encephalopathy and hepatorenal syndrome.
- **Clinical Impact:** Empagliflozin may offer additional hepatic benefits over dapagliflozin without increasing adverse events. Further research is needed to confirm these findings.

ABSTRACT

Background and Aim: Sodium-glucose cotransporter-2 inhibitors may improve outcomes in liver cirrhosis, but comparative evidence between individual agents is limited. We compared real-world outcomes of empagliflozin versus dapagliflozin in adults with liver cirrhosis.

Materials and Methods: We conducted a multicenter retrospective cohort study using the TriNetX US Collaborative Network (69 healthcare organizations). Adults with cirrhosis who were newly prescribed empagliflozin or dapagliflozin after the diagnosis of cirrhosis between March 1, 2013 and January 1, 2025, were included. Propensity score matching (1:1) was used to balance baseline characteristics. Outcomes were assessed from day 1 after the index prescription through 5 years. Primary outcomes were all-cause mortality and all-cause hospitalization. Secondary outcomes included hepatic decompensation events. Tertiary outcomes included prognostic hepatic and renal biomarkers. Safety outcomes included the incidence of acute kidney injury, urinary tract infection, and diabetic ketoacidosis.

Results: Before matching, 17,700 empagliflozin users and 8,619 dapagliflozin users were identified; 7,852 patients remained in each cohort after matching. Five-year mortality was similar between groups (12.7% vs. 12.6%; odds ratio [OR]: 1.012, 95% confidence interval [CI]: 0.921–1.112; p=0.8075), as was the risk of hospitalization (14.7% vs. 13.4%; OR: 1.105, 95% CI: 0.94–1.30; p=0.216). Empagliflozin was associated with lower rates of hepatic encephalopathy (4.0% vs. 4.7%; OR: 0.84; p=0.0295), hepatorenal syndrome (1.0% vs. 1.6%; OR: 0.614; p=0.0007), and paracentesis (2.9% vs. 3.7%; OR: 0.785; p=0.0083). Albumin levels were higher and bilirubin levels were lower with empagliflozin (p<0.01 for both). Safety outcomes were similar between groups.

Conclusion: In matched adults with cirrhosis, empagliflozin and dapagliflozin demonstrated comparable five-year mortality and hospitalization rates. However, empagliflozin was associated with fewer selected decompensation events without an increase in adverse events.

Keywords: Cirrhosis, empagliflozin, dapagliflozin.

How to cite this article: Ahmed H, Razzak IA, Abdulrazzak E, Gomaa S, Lauchner J. Empagliflozin versus dapagliflozin in patients with liver cirrhosis: A comparative real-world study on hepatic decompensation. Hepatology Forum 2026; 7(2):139–145.

Received: December 16, 2025; **Revised:** February 10, 2026; **Accepted:** February 16, 2026; **Available Online:** April 14, 2026

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Introduction

Liver cirrhosis remains a major global health burden and is associated with substantial morbidity and mortality driven by hepatic decompensation, hepatocellular carcinoma (HCC), and progression to end-stage liver disease.[1] Despite advances in understanding the pathophysiology of cirrhosis, disease-modifying therapies remain limited, particularly for patients with decompensated disease.[2] The emerging concept of recompensated cirrhosis—defined as the resolution of prior decompensating events with improvement in hepatic function—has been linked to better long-term outcomes and represents a potential therapeutic target.[3] Concurrently, the rising prevalence of metabolic dysfunction–associated steatotic liver disease (MASLD) and its inflammatory subtype, metabolic dysfunction–associated steatohepatitis (MASH), further underscores the need for novel strategies to improve outcomes in the cirrhosis population.[4]

Sodium-glucose cotransporter-2 inhibitors (SGLT2i), initially developed for the treatment of type 2 diabetes mellitus (T2DM), have demonstrated pleiotropic benefits extending beyond glycemic control, including cardioprotective, nephroprotective, and anti-inflammatory effects.[5,6] These agents are now routinely incorporated into the management of heart failure and chronic kidney disease (CKD), including in individuals without diabetes, supported by evidence from large clinical trials across diverse clinical settings.[7]

Increasing evidence suggests that SGLT2 inhibitors may also confer hepatoprotective effects. Preclinical and clinical studies have reported improvements in liver enzymes (alanine aminotransferase [ALT], aspartate aminotransferase [AST], and gamma-glutamyl transferase [GGT]), reductions in hepatic steatosis and fibrosis, and potential mitigation of cirrhosis-related complications such as ascites, portal hypertension, and hepatic encephalopathy.[8–11] Proposed mechanisms include attenuation of oxidative stress and suppression of pro-inflammatory cytokines (e.g., tumor necrosis factor alpha [TNF-α] and interleukin-6 [IL-6]), with downstream anti-inflammatory and antifibrotic effects that may counter pathways implicated in cirrhosis progression.[12–17]

Despite these promising observations, comparative real-world data evaluating individual SGLT2i agents in cirrhosis are limited. Accordingly, we used a large real-world electronic health record database (TriNetX) to compare outcomes among patients with liver cirrhosis initiated on empagliflozin versus dapagliflozin. Using propensity score matching, we evaluated mortality, hospitalization, hepatic decompensation events, and safety outcomes to determine whether medication-specific differences exist within the SGLT2i class in this high-risk population.

Materials and Methods

Study Design and Data Source

We conducted a multicenter retrospective cohort study using the TriNetX Research Network, a federated health research platform that aggregates real-time, de-identified electronic health record (EHR) data from more than 120 million patients across 69 healthcare organizations in the United States. This study used the TriNetX US Collaborative Network, which includes both structured and unstructured clinical data (e.g., diagnoses, medications, laboratory results, and procedures). Diagnoses were identified using the International Classification of Diseases, Tenth Revision (ICD-10) codes; medications using RxNorm codes; procedures using Current Procedural Terminology (CPT) codes; and laboratory tests using Logical Observation Identifiers Names and Codes (LOINC). All diagnostic, medication, procedure, laboratory, and encounter codes used for cohort construction and outcome ascertainment are provided in Supplementary Table 1A–E.

Study Population and Cohort Definition

Eligible patients were adults (aged ≥18 years) with a documented diagnosis of liver cirrhosis identified using ICD-10 codes. Patients were excluded if they had a history of liver transplantation, renal transplantation, dialysis, primary biliary cholangitis, or primary sclerosing cholangitis. Two exposure cohorts were defined among eligible patients: patients prescribed empagliflozin after cirrhosis diagnosis and patients prescribed dapagliflozin after cirrhosis diagnosis.

The index date was defined as the first prescription for empagliflozin or dapagliflozin occurring after the initial cirrhosis diagnosis. Patients who received both medications or who were exposed to other sodium-glucose cotransporter-2 inhibitors were excluded. Patients who experienced any study outcome prior to the index date were excluded from the analysis of that specific outcome but remained eligible for analyses of other outcomes. The study period extended from March 1, 2013 (initial U.S. Food and Drug Administration [FDA] approval of SGLT2i), through January 1, 2025.

Covariates and Baseline Characteristics

Baseline characteristics and covariates were assessed during the five years preceding each patient's index date. These included demographics (age, sex, race); comorbidities (type 2 diabetes mellitus, hypertension, heart failure, chronic kidney disease, and obesity); liver disease etiologies (e.g., alcoholic liver disease, nonalcoholic steatohepatitis, and chronic viral hepatitis); and medication exposures, including diuretics, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), statins, insulin, metformin, glucagon-like peptide-1 (GLP-1) receptor agonists, dipeptidyl peptidase-4 (DPP-4) inhibitors, and sulfonylureas. Procedural history, including transjugular intrahepatic portosystemic shunt (TIPS) placement, was also captured.

Laboratory parameters used to characterize baseline liver and renal function included aspartate aminotransferase, alanine aminotransferase, gamma-glutamyl transferase, total bilirubin, serum albumin, serum creatinine, estimated glomerular filtration rate (eGFR), international normalized ratio (INR), hemoglobin A1c (HbA1c), platelet count, serum sodium, and body mass index (BMI).

Table 1. Hepatic and prognostic laboratory markers in the empagliflozin and dapagliflozin cohorts at five-year follow-up

Laboratory marker	Empagliflozin (n=7,852) Mean±SD	Dapagliflozin (n=7,852) Mean±SD	p
Aspartate aminotransferase (AST)	57.86±336.33	57.37±227.51	0.9266
Alanine aminotransferase (ALT)	39.51±135.45	41.37±141.22	0.4636
Gamma-glutamyl transferase (GGT)	164.89±274.91	141.66±231.12	0.1013
Total bilirubin	1.22±2.36	1.34±2.83	0.0094
Serum albumin	3.69±0.72	3.61±0.74	<0.0001
International normalized ratio (INR)	1.37±0.67	1.39±0.68	0.1429
Platelets	181.43±92.25	179.72±91.88	0.302
Creatinine	1.26±0.94	1.29±0.83	0.0681
Glomerular filtration rate (eGFR)	66.88±31.75	66.37±32.34	0.3654
Serum sodium	137.93±4.08	137.76±4.16	0.02

Values are presented as mean±standard deviation (SD).

Study Outcomes

The primary outcomes were all-cause mortality and all-cause hospitalization during follow-up. Secondary outcomes were hepatic decompensation events, including ascites, need for peritoneal drainage, spontaneous bacterial peritonitis (SBP), jaundice, esophageal variceal bleeding, hepatic encephalopathy, hepatorenal syndrome (HRS), and hepatocellular carcinoma. Tertiary outcomes included laboratory markers reflecting hepatic and renal function (e.g., AST, ALT, INR, serum albumin, serum creatinine, and eGFR). Safety outcomes included acute kidney injury (AKI), urinary tract infections (UTIs), and diabetic ketoacidosis (DKA).

Statistical Analysis

To reduce confounding, 1:1 propensity score matching (PSM) was performed using greedy nearest-neighbor matching without replacement and a caliper width of 0.1 pooled standard deviations within the TriNetX Analytics Platform (TriNetX LLC, Cambridge, MA, USA). Propensity scores were estimated via logistic regression including all prespecified covariates. Balance after matching was evaluated using standardized mean differences (SMDs), with an absolute SMD<0.1 indicating acceptable balance.

All analyses were conducted within the TriNetX analytics platform using its built-in modules. For binary outcomes within the follow-up window, TriNetX "Measure of Association" analyses were used to compute risk, risk difference, risk ratio, and odds ratios (OR), with corresponding 95% confidence intervals (CI). Time-to-event outcomes were evaluated using TriNetX Kaplan–Meier survival analyses with log-rank testing; censoring was applied according to the platform rule that patients are censored after the last recorded clinical fact in their EHR. For laboratory outcomes, TriNetX laboratory analyses were used, which incorporated only the most recent laboratory value within the specified time window; group means were compared using t-tests. All tests were two-sided, and p-values <0.05 were considered statistically significant.

Ethical Approval/Waiver

This study was reviewed by a Phoenixville Hospital Institutional Review Board and determined to be exempt (No. 2025-109, September 10, 2025) because it involved the secondary analysis of de-identified data obtained through the TriNetX platform. Our article was written in accordance with the Helsinki declaration.

Results

Cohort Selection and Baseline Characteristics

Among 720,923 adults with cirrhosis, 604,728 remained after exclusions. We identified 17,700 empagliflozin users (Group A) and 8,619 dapagliflozin users (Group B). After 1:1 propensity score matching, 7,852 patients remained in each cohort. Baseline characteristics were well balanced after matching (absolute SMD<0.1 across covariates), with a small residual imbalance in serum albumin (SMD=0.102). Full baseline characteristics are shown in Supplementary Table 1.

Primary Outcomes: Mortality and Hospitalization

At five years, all-cause mortality did not differ significantly between cohorts (12.7% vs. 12.6%; OR: 1.012; 95% CI: 0.921–1.112; p=0.8075). Kaplan–Meier analysis similarly showed no significant difference in time to death between empagliflozin and dapagliflozin (hazard ratio: 0.970; 95% CI: 0.888–1.060; log-rank p=0.500) (Fig. 1). The risk of all-cause hospitalization was also similar (14.7% vs. 13.4%; OR: 1.105; 95% CI: 0.94–1.30; p=0.216).

Secondary Outcomes: Hepatic Decompensation Events

Composite hepatic decompensation was similar between groups (11% vs. 10.6%; OR: 1.004; 95% CI: 0.886–1.137; p=0.9516). However, empagliflozin was associated with lower rates of hepatic

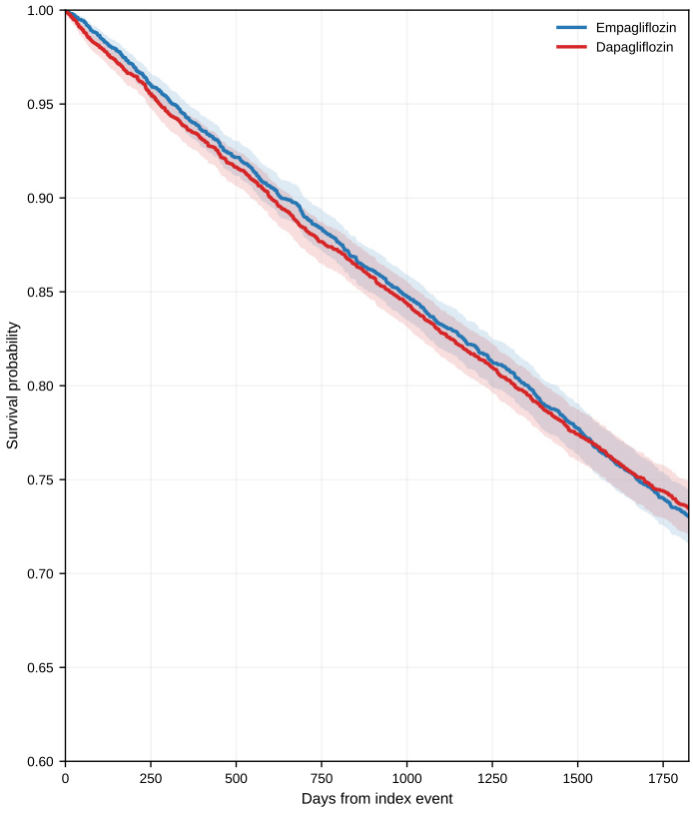


Figure 1. Kaplan–Meier survival curves for all-cause mortality after initiation of empagliflozin versus dapagliflozin in propensity score–matched adults with cirrhosis. Shaded areas indicate 95% confidence intervals (CI). Groups were compared using the log-rank test (χ^2=0.455; p=0.500); hazard ratio of 0.970 (95% CI: 0.888–1.060). Note: The y-axis is truncated (0.60–1.00) to improve visualization of between-group differences.

encephalopathy (4.0% vs. 4.7%; OR: 0.84; 95% CI: 0.717–0.983; p=0.0295), hepatorenal syndrome (1.0% vs. 1.6%; OR: 0.614; 95% CI: 0.462–0.816; p=0.0007), and paracentesis (2.9% vs. 3.7%; OR: 0.785; 95% CI: 0.656–0.94; p=0.0083). Other hepatic outcomes were not significantly different (Table 2). These associations are summarized in Figure 2.

Tertiary Outcomes: Prognostic Markers

At five years, empagliflozin users had higher albumin levels (3.69±0.72 vs. 3.61±0.74 g/dL; p<0.0001) and lower total bilirubin levels (1.22±2.36 vs. 1.34±2.83 mg/dL; p=0.0094). Serum sodium was also statistically, though modestly, higher with empagliflozin (137.93±4.08 vs. 137.76±4.16 mmol/L; p=0.02). Other laboratory markers were similar (Table 1).

Adverse Events

Safety outcomes were comparable between cohorts: AKI (12.8% vs. 13.6%; OR: 0.927; 95% CI: 0.823–1.045; p=0.214), UTI (6.5% vs. 6.9%; OR: 0.95; 95% CI: 0.825–1.095; p=0.4783), and DKA (0.8% vs. 0.8%; OR: 0.966; 95% CI: 0.674–1.383; p=0.849).

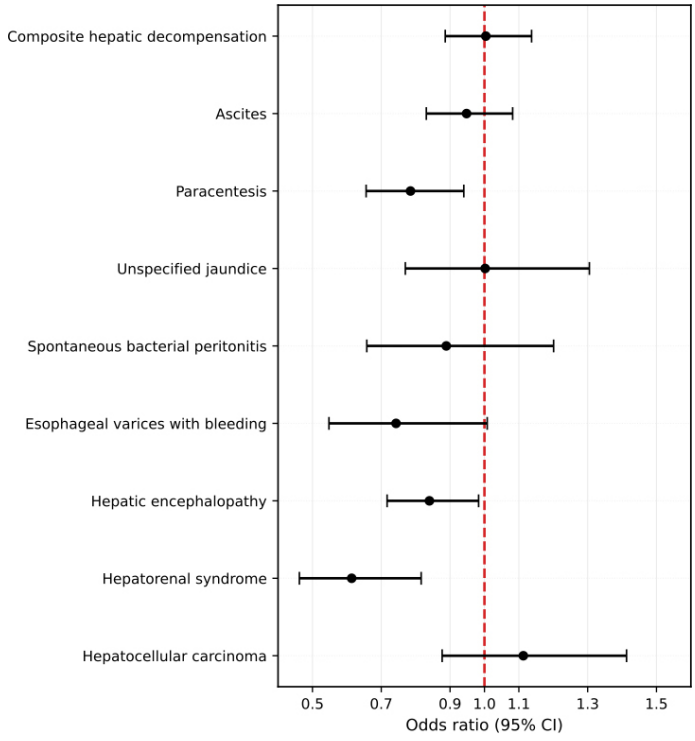


Figure 2. Forest plot of hepatic decompensation outcomes comparing empagliflozin and dapagliflozin. Odds ratios (OR) with 95% confidence intervals are shown for five-year outcomes after propensity score matching. The vertical reference line indicates OR=1.0.

Discussion

In this real-world retrospective cohort study, empagliflozin and dapagliflozin were associated with comparable five-year risks of mortality and all-cause hospitalization in matched adults with cirrhosis. However, empagliflozin was associated with fewer selected hepatic decompensation events—particularly hepatic encephalopathy, hepatorenal syndrome, and paracentesis—along with modest differences in albumin and bilirubin levels, without an observed increase in AKI, UTI, or DKA. To our knowledge, this is the first large-scale real-world comparison of these two agents in liver cirrhosis.

In the cirrhosis literature, empagliflozin has been most consistently linked to improved ascites control and reduced need for therapeutic drainage, aligning with the lower paracentesis rates observed in our cohort.[18–20] A case report described resolution of refractory ascites and hepatic hydrothorax after empagliflozin initiation in primary biliary cirrhosis;[18] a pilot study showed improved natriuresis and circulatory, cardiac, and renal parameters in cirrhotic patients with refractory ascites;[19] and a randomized trial demonstrated reduced large-volume paracentesis requirements and complete ascites resolution in a subset of patients when empagliflozin was added to standard care.[20] Safety data in cirrhosis, though limited, support feasibility and are consistent with our similar rates of AKI, UTI, and DKA between cohorts.[21]

Table 2. Five-year hepatic outcomes in the empagliflozin and dapagliflozin cohorts after propensity score matching

Secondary outcome	Empagliflozin (n=7,852) n (%)	Dapagliflozin (n=7,852) n (%)	OR	95% CI	p
Composite hepatic decompensation	564 (11)	555 (10.6)	1.004	(0.886, 1.137)	0.9516
Ascites	466 (7.9)	489 (8.3)	0.948	(0.831, 1.082)	0.4311
Paracentesis	221 (2.9)	277 (3.7)	0.785	(0.656, 0.94)	0.0083
Unspecified jaundice	112 (1.5)	112 (1.5)	1.002	(0.77, 1.305)	0.988
Spontaneous bacterial peritonitis	81 (1.0)	91 (1.2)	0.889	(0.658, 1.201)	0.4417
Esophageal varices with bleeding	73 (0.9)	98 (1.3)	0.743	(0.548, 1.008)	0.0554
Hepatic encephalopathy	299 (4.0)	353 (4.7)	0.84	(0.717, 0.983)	0.0295
Hepatorenal syndrome	78 (1.0)	126 (1.6)	0.614	(0.462, 0.816)	0.0007
Hepatocellular carcinoma	145 (1.9)	131 (1.7)	1.113	(0.877, 1.413)	0.3779

Values are presented as n (%). OR: Odds ratio; CI, Confidence interval.

Beyond cirrhosis, randomized trials in metabolic dysfunction-associated steatotic liver disease populations show that empagliflozin reduces hepatic fat content and improves liver-related biomarkers, providing a broader hepatometabolic rationale for differences in liver-related outcomes.[22–25] Across E-LIFT (Effect of Empagliflozin on Liver Fat in Patients with Type 2 Diabetes Mellitus) and subsequent placebo-controlled studies in type 2 diabetes, mixed diabetic and nondiabetic populations, and nondiabetic MASLD, empagliflozin consistently reduced liver fat on imaging and was associated with favorable metabolic changes.[22–25]

Taken together, existing evidence most directly supports empagliflozin's role in ascites and volume-related management and hepatic steatosis reduction, which is directionally consistent with our findings of reduced paracentesis and modestly more favorable liver synthetic and cholestatic markers. The observed differences in hepatic encephalopathy and hepatorenal syndrome warrant prospective validation, as empagliflozin-specific data for these endpoints remain limited.

The literature on dapagliflozin in cirrhosis is more limited and mixed. A small randomized trial in cirrhotic patients with ascites reported improved natriuresis and ascites control but no survival or disease severity benefit, along with higher rates of AKI and infections,[26] whereas a retrospective study in cirrhotic patients with diabetes found better ascites control, reduced diuretic requirements, and fewer complications, including hepatic encephalopathy, variceal bleeding, and infections.[27] These data suggest that dapagliflozin may provide hepatic benefits in selected populations, but its safety profile in cirrhosis requires further clarification.

In our analysis, empagliflozin was associated with slightly more favorable hepatic laboratory parameters, including higher serum albumin and lower bilirubin levels, suggesting better preservation of hepatic synthetic function and fluid homeostasis. This pattern is consistent with reports of reduced liver fat and improved metabolic and nutritional parameters with empagliflozin in cirrhosis and MASLD.[19,24] Overall, our findings add comparative effectiveness evidence and support the possibility that empagliflozin may confer hepatic benefits beyond glycemic and cardiorenal effects, particularly with respect to hepatic decompensation events and selected hepatic biomarkers.

Limitations

This study has several limitations. First, its retrospective design and reliance on EHR-based administrative coding introduce the possibility of residual confounding and misclassification. Although propensity score matching balanced measured covariates, unmeasured factors—such as cirrhosis severity (e.g., Model for End-Stage Liver Disease [MELD] or Child-Pugh scores), medication adherence, and alcohol consumption patterns—could not be assessed. Second, due to platform limitations, we could not ascertain cause-specific mortality or indications for hospitalization, nor could we determine the timing of SGLT2i discontinuation. Finally, generalizability may be limited to the healthcare systems represented in TriNetX.

Conclusion

In this large, real-world multicenter cohort study of patients with liver cirrhosis, empagliflozin and dapagliflozin were associated with comparable five-year risks of all-cause mortality and hospitalization after propensity score matching. However, empagliflozin use was associated with a lower risk of key hepatic decompensation events—particularly hepatic encephalopathy and hepatorenal syndrome—and a reduced need for paracentesis, along with modestly more favorable liver function markers (higher albumin and lower bilirubin levels). Adverse event rates were similar between groups, supporting comparable short-term safety in this population. Collectively, these findings provide novel comparative effectiveness evidence suggesting that empagliflozin may confer additional hepatic benefits within the SGLT2 inhibitor class in patients with cirrhosis. Prospective randomized studies are needed to confirm these observations, clarify mechanisms, and guide optimal SGLT2 inhibitor selection in patients with advanced liver disease.

Online Appendix Link: https://hepatologyforum.org/storage/upload/files/1775572025-appendix-en.pdf

Ethics Committee Approval: This study was reviewed by a Phoenixville Hospital Institutional Review Board and determined to be exempt (No. 2025-109, September 10, 2025) because it involved the secondary analysis of de-identified data obtained through the TriNetX platform.

Conflict of Interest: The authors declare no potential conflicts of interest.

Financial Disclosure: No financial support was received for the preparation of this research article.

Use of AI for Writing Assistance: The authors declare that no artificial intelligence (AI)–assisted technologies, including large language models, chatbots, or image-generation tools, were used in the conception, writing, analysis, or preparation of this manuscript.

Author Contributions: Concept: HA; Design: HA; Supervision: SG, JL; Funding: HA, SG; Materials: HA, IAR; Data Collection and/or Processing: HA, IAR, EA, SG; Analysis and/or Interpretation: HA, IAR, EA, SG; Literature Review: HA, IAR, EA, SG; Writing: HA; Critical Review: IAR, EA, SG, JL.

Acknowledgement: A preliminary abstract based on this work was submitted to the American College of Gastroenterology (ACG) Annual Meeting in October 2025 (Arizona) and was accepted as a poster presentation. The abstract was subsequently published by the American College of Gastroenterology (DOI: 10.14309/01.ajg.0001139060.76824.1f). The full manuscript has not been published previously and is not under consideration by any other journal.

Peer-review: Externally peer-reviewed.

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