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COMPARATIVE EFFECTS OF SGLT2 INHIBITORS VERSUS METFORMIN ON LIVER ENZYMES, GLYCEMIC CONTROL, AND BLOOD PRESSURE IN TYPE 2 DIABETES WITH MASLD

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ABSTRACT

Background: Comparative evidence on empagliflozin versus metformin in patients with type 2 diabetes mellitus (T2DM) and metabolic dysfunction-associated steatotic liver disease (MASLD) is limited in South Asian populations. This study compared their short-term effects on liver enzymes, glycemic control, and blood pressure.
Methods: This twelve-week randomized comparative study was conducted in Islamabad, Pakistan. Of 210 patients screened, 170 were randomized equally to empagliflozin 10 mg once daily or metformin 1000 mg/day; 126 participants completed follow-up and were included in the per-protocol analysis. MASLD was confirmed by baseline ultrasonography. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), glycated hemoglobin (HbA1c), systolic blood pressure (SBP), diastolic blood pressure (DBP), and adverse effects were assessed at baseline and week twelve. Adjusted between-group comparisons were performed using analysis of covariance.
Results: Both groups improved significantly in ALT, AST, HbA1c, SBP, and DBP at twelve weeks. Adjusted mean differences favored empagliflozin for ALT (−6.02 U/L; 95% CI −7.62 to −4.41), AST (−2.84 U/L; 95% CI −3.67 to −2.01), HbA1c (−0.19%; 95% CI −0.24 to −0.15), SBP (−1.80 mmHg; 95% CI −3.32 to −0.28), and DBP (−1.99 mmHg; 95% CI −3.07 to −0.91). Overall adverse-effect frequency did not differ significantly between groups.
Conclusion: Empagliflozin was associated with greater short-term improvement in hepatic biochemical markers, glycemic control, and blood pressure than metformin in patients with T2DM and baseline ultrasound-confirmed MASLD.

KEYWORDS: *Type 2 diabetes mellitus; Empagliflozin; Metformin; Metabolic dysfunction-associated steatotic liver disease; Liver enzymes; Glycated hemoglobin*

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major cause of morbidity and premature mortality, with its prevalence continuing to increase globally and imposing a substantial health burden in Pakistan. This growing burden is particularly important because T2DM frequently coexists with metabolic dysfunction-associated steatotic liver disease (MASLD), increasing both hepatic and cardiometabolic risk.

MASLD is highly prevalent among individuals with T2DM and is closely associated with insulin resistance, dyslipidemia, adipose-tissue dysfunction, and chronic low-grade inflammation. The burden of steatotic liver disease is also substantial in Pakistan, emphasizing the need for therapeutic strategies that can address metabolic and hepatic abnormalities using approaches feasible in routine clinical practice.

Metformin remains one of the most widely used glucose-lowering agents because of its established efficacy, safety, affordability, and accessibility. However, its role as a treatment directed specifically toward hepatic steatosis or fibrosis remains limited. Sodium-glucose cotransporter-2 (SGLT2) inhibitors, including empagliflozin, provide effective glucose lowering, while emerging evidence suggests favorable effects on hepatic biochemical parameters and liver fat in patients with T2DM and steatotic liver disease.

Although glucose-lowering therapies have been evaluated in Pakistani patients with coexisting T2DM and fatty liver disease, locally generated head-to-head evidence comparing empagliflozin with metformin remains limited. Such comparative data may be particularly useful in clinical settings where liver enzymes and routine metabolic parameters are more readily available than advanced hepatic assessment. Therefore, the present study aimed to compare the short-term effects of empagliflozin 10 mg and metformin 1000 mg on liver enzymes, glycemic control, and blood pressure in patients with T2DM and MASLD.

MATERIALS AND METHODS

Study Design, Setting and Duration

This twelve-week open-label randomized comparative study was conducted at the Department of Pharmacology and Therapeutics, Shaheed Zulfiqar Ali Bhutto Medical University (SZABMU), Islamabad, in collaboration with the Medical Outpatient Department of Pakistan Institute of Medical Sciences (PIMS), Islamabad. Participant recruitment and clinical follow-up were performed in the outpatient diabetic care setting, while laboratory investigations were conducted at the diagnostic laboratory of PIMS. The overall study was conducted over ten months from September 2025 to April 2026, including participant recruitment, baseline assessment, intervention, follow-up, and data analysis.

Ethical approval was obtained from the Ethical Review Board of SZABMU (Approval No. 1451; dated 03-07-2025) and the Advanced Study and Research Board of SZABMU (Approval No. 2025-2129; dated 04-08-2025). Written informed consent was obtained from all participants before enrollment.

Study Population and Eligibility Criteria

Adult patients with T2DM attending the PIMS outpatient department were screened consecutively for eligibility. Patients aged 30–70 years with established T2DM, MASLD identified on abdominal ultrasonography (USG), and baseline glycated hemoglobin (HbA1c) <7.5% were eligible for inclusion.

Patients were excluded if they were pregnant or lactating; had an estimated glomerular filtration rate <30 mL/min/1.73 m^2; active malignancy or recent hospitalization; active or recurrent urinary tract infection during the preceding six months; or another recognized cause of chronic liver disease, including viral hepatitis, autoimmune hepatitis, or primary biliary cholangitis.

Sample Size and Randomization

The minimum sample size was calculated using the WHO sample size calculator for comparison of two independent means. Using a two-sided significance level of 5%, statistical power of 80%, population standard deviation of 1.0, and anticipated mean HbA1c values of 7.8% and 7.34% in the metformin and empagliflozin groups, respectively, a minimum of 63 participants per group was required, giving an analyzable sample of 126 participants.

Eligible participants who provided written informed consent were randomly allocated in a 1:1 ratio to the two treatment groups using a random-number table. Group allocation was completed before baseline testing. Baseline clinical and laboratory assessments were subsequently performed at week 0 before initiation of the allocated treatment.

Intervention

Participants allocated to the Empagliflozin group received empagliflozin 10 mg orally once daily. Participants allocated to the Metformin group received metformin at a total oral dose of 1000 mg/day, as recommended by the treating medical specialist and specified in the study protocol. Both treatments were continued for twelve weeks. The study was open label; therefore, participants and treating clinicians were aware of treatment allocation.

Data Collection and Outcome Assessment

A structured study proforma was used to record demographic and clinical information, including age, sex, duration of T2DM, baseline USG grade of hepatic steatosis, and comorbidities. MASLD was identified by abdominal USG in participants with T2DM after exclusion of recognized secondary causes of chronic liver disease. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were evaluated as hepatic biochemical outcomes and were not used as diagnostic criteria for MASLD.

Clinical and laboratory assessments were performed at baseline and after twelve weeks of treatment. Outcomes included ALT, AST, HbA1c, systolic blood pressure (SBP), diastolic blood pressure (DBP), and adverse effects. Blood pressure was measured using a calibrated sphygmomanometer and recorded as the mean of three readings obtained five minutes apart.

Adverse effects documented during follow-up included genital infection, urinary tract infection, dizziness, metallic taste, and gastrointestinal intolerance. The occurrence of adverse effects and whether any event resulted in treatment discontinuation were recorded on the study proforma.

Statistical Analysis

Data was analyzed using IBM SPSS Statistics version 27.0. Continuous variables were presented as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. Between-group comparisons of continuous variables were performed using the independent-samples t-test, while categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Within-group changes from baseline to week twelve were assessed using the paired-samples t-test.

Adjusted between-group comparisons at week twelve were performed using analysis of covariance (ANCOVA). Treatment group, sex, baseline USG grade, dyslipidemia, and hypertension were entered as categorical factors, while the corresponding baseline outcome value, age, and duration of T2DM were included as covariates. Adjusted mean differences with 95% confidence intervals (CI) were estimated for ALT, AST, HbA1c, SBP, and DBP.

The efficacy analysis was performed on a per-protocol basis and included participants with both baseline and week-twelve measurements. Participants without week-twelve outcome data were excluded from the efficacy analysis. A sensitivity analysis was performed using the same ANCOVA models after excluding participants with congestive heart failure. Overall adverse-effect frequency was compared using the chi-square test, while individual adverse effects with sparse cell counts were evaluated using Fisher's exact test. All statistical tests were two-sided, and a p-value <0.05 was considered statistically significant.

RESULTS

Participant Flow and Baseline Characteristics

A total of 210 patients were screened for eligibility. Of these, 18 did not meet the inclusion criteria and 22 declined participation. The remaining 170 participants were randomized equally to the empagliflozin and metformin groups. During follow-up, 22 participants in each group did not return for the week-twelve assessment. Consequently, 126 participants (63 in each group) completed follow-up and were included in the per-protocol analysis.

Baseline demographic and clinical characteristics are summarized in Table 1. Participants in the empagliflozin group were older and had a longer duration of T2DM than those in the metformin group. Dyslipidemia was more frequent in the metformin group, whereas congestive heart failure (CHF) was present only in the empagliflozin group. Sex distribution, USG grade of hepatic steatosis, and prevalence of hypertension did not differ significantly between groups.

Table: *Baseline demographic and clinical characteristics of participants.*

Characteristic	Empagliflozin 10 mg (n = 63)	Metformin 1000 mg (n = 63)	p-value
Female, n (%)	33 (52%)	37 (59%)	0.473
Male, n (%)	30 (48%)	26 (41%)	—
Age, years, Mean ± SD	55.95 ± 4.79	53.56 ± 5.71	0.012
Duration of T2DM, years, Mean ± SD	5.27 ± 1.83	3.89 ± 1.38	<0.001
USG grade I, n (%)	35 (56%)	26 (41%)	0.109
USG grade II, n (%)	28 (44%)	37 (59%)	—
No comorbidity, n (%)	15 (24%)	12 (19%)	0.515
Any comorbidity, n (%)	48 (76%)	51 (81%)	—
Dyslipidemia, n (%)	10 (16%)	33 (52%)	<0.001
Hypertension, n (%)	32 (51%)	41 (65%)	0.104
Congestive Heart Failure, n (%)	29 (46%)	0 (0%)	<0.001

Treatment Outcomes

Both treatment groups showed significant reductions in ALT, AST, HbA1c, SBP, and DBP from baseline to week twelve (all p<0.001). The magnitude of reduction was consistently greater in the empagliflozin group (Table).

After adjustment for baseline outcome values and relevant demographic and clinical covariates, the between-group differences remained statistically significant for ALT, AST, HbA1c, SBP, and DBP, with all adjusted estimates favoring empagliflozin. Sensitivity analysis excluding participants with CHF produced comparable effect estimates, indicating that the principal findings were not materially altered by the baseline imbalance in CHF.

Table: *Within-group changes in ALT, AST, HbA1c, SBP, and DBP from baseline to week twelve.*

Treatment Group	Timepoint / Analysis	ALT (U/L)	AST (U/L)	HbA1c (%)	SBP (mmHg)	DBP (mmHg)
Empagliflozin (n = 63)	Baseline	81.41 ± 15.39	45.49 ± 2.94	7.30 ± 0.35	138.22 ± 8.89	85.97 ± 2.99
Empagliflozin (n = 63)	Week Twelve	71.51 ± 15.76	40.15 ± 3.28	6.82 ± 0.36	132.67 ± 8.56	81.52 ± 3.50
Metformin (n = 63)	Baseline	85.23 ± 16.30	45.56 ± 2.62	7.22 ± 0.34	140.10 ± 8.82	85.75 ± 3.46
Metformin (n = 63)	Week Twelve	82.04 ± 16.46	43.19 ± 2.92	6.96 ± 0.33	137.05 ± 8.04	83.59 ± 3.45
Adjusted Mean Difference (95% CI)	p-value	−6.02 (−7.62 to −4.41); <0.001	−2.84 (−3.67 to −2.01); <0.001	−0.19 (−0.24 to −0.15); <0.001	−1.80 (−3.32 to −0.28); 0.021	−1.99 (−3.07 to −0.91);

						<0.001
CHF-Excluded Sensitivity Difference	p-value	−5.48 (−7.37 to −3.59); <0.001	−2.54 (−3.51 to −1.56); <0.001	−0.19 (−0.24 to −0.14); <0.001	−2.35 (−4.10 to −0.61); 0.009	−2.41 (−3.68 to −1.14); <0.001

Adverse Effects

The overall frequency of adverse effects did not differ significantly between the empagliflozin and metformin groups (79% vs. 65%; p=0.111). Genital infection, urinary tract infection, and mild dizziness were observed in the empagliflozin group, whereas metallic taste and gastrointestinal upset occurred in the metformin group. All recorded adverse effects were mild, and none resulted in treatment discontinuation.

Table: *Adverse effects recorded over twelve weeks of treatment in the empagliflozin and metformin groups.*

Adverse Effect	Empagliflozin (No)	Empagliflozin (Yes)	Metformin (No)	Metformin (Yes)	p-value
Any adverse effect	13 (21%)	50 (79%)	22 (35%)	41 (65%)	0.111
Genital Infection	47 (75%)	16 (25%)	63 (100%)	0 (0%)	<0.001
Urinary Tract Infection	41 (65%)	22 (35%)	63 (100%)	0 (0%)	<0.001
Mild Dizziness	51 (81%)	12 (19%)	63 (100%)	0 (0%)	<0.001
Metallic Taste	63 (100%)	0 (0%)	40 (64%)	23 (36%)	<0.001
Gastrointestinal Upset	63 (100%)	0 (0%)	45 (71%)	18 (29%)	<0.001

DISCUSSION

The present study showed that both empagliflozin and metformin were associated with significant short-term improvements in hepatic biochemical markers, glycemic control, and blood pressure in patients with T2DM and MASLD. However, reductions in ALT, AST, HbA1c, SBP, and DBP were consistently greater with empagliflozin. These differences persisted after adjustment for baseline demographic and clinical characteristics and remained consistent in sensitivity analysis excluding participants with CHF.

The greater reductions in ALT and AST observed with empagliflozin are consistent with international evidence suggesting beneficial hepatic effects of SGLT2 inhibitors. Meta-analyses by Zhou et al. and Ahmed et al. reported improvements in liver enzymes, hepatic steatosis, and other non-invasive hepatic parameters among patients receiving SGLT2 inhibitors. More recent evidence has similarly demonstrated reductions in hepatic fat content and favorable clinical outcomes among patients with coexisting T2DM and MASLD. Evidence from Pakistan has also reported favorable liver-related effects of SGLT2 inhibitor therapy in patients with T2DM, supporting the relevance of these findings in the local population. Proposed mechanisms include urinary glucose and caloric loss, improved insulin sensitivity, and reduced hepatic lipid accumulation and metabolic inflammation.

In contrast, metformin remains an established and accessible glucose-lowering therapy but appears to have a more limited effect on liver-specific outcomes. Previous reviews and meta-analyses have reported modest reductions in aminotransferase levels with metformin, while consistent improvement in hepatic steatosis or fibrosis has not been established. The present findings therefore add comparative evidence suggesting a greater short-term hepatic biochemical response with empagliflozin. However, because liver imaging was not repeated at week twelve, reductions in ALT and AST should not be interpreted as evidence of regression of hepatic steatosis or fibrosis.

Empagliflozin also produced a greater reduction in HbA1c than metformin, although the adjusted between-group difference was modest. This finding should be interpreted in the context of the study population, in which baseline HbA1c was restricted to <7.5%. Current diabetes management recommendations recognize SGLT2 inhibitors as glucose-lowering therapies with additional cardiorenal and metabolic benefits beyond glycemic control. The present results are therefore consistent with the broader metabolic profile of this drug class, while the relatively small HbA1c difference indicates that the principal distinction between treatments was not limited to glycemic efficacy alone.

Small but statistically significant reductions in SBP and DBP also favored empagliflozin. These findings are consistent with the established hemodynamic effects of SGLT2 inhibition, which include natriuresis, osmotic diuresis, and reductions in circulating volume. Although the absolute between-group blood pressure differences were modest, concurrent improvement in glycemic and hemodynamic parameters may be relevant in patients with T2DM and MASLD, who commonly have multiple cardiometabolic risk factors.

The adverse-effect profiles were consistent with the known pharmacological characteristics of the two therapies. Genitourinary adverse effects were observed with empagliflozin, whereas gastrointestinal intolerance and metallic

taste occurred with metformin. Despite these differences in pattern, overall adverse-effect frequency did not differ significantly between groups, and no recorded event resulted in treatment discontinuation during the twelve-week follow-up.

Clinical Significance, Strengths and Limitations

An important contribution of this study is the direct comparison of empagliflozin and metformin among Pakistani patients with T2DM and ultrasonographically identified MASLD. Diabetes and steatotic liver disease represent substantial and increasingly overlapping health burdens in Pakistan, while locally generated comparative therapeutic evidence remains limited. The present study therefore provides clinically relevant short-term data using liver enzymes, HbA1c, and blood pressure measurements that are readily available in routine tertiary-care practice.

The study has several strengths, including randomized treatment allocation, direct comparison of two commonly used glucose-lowering therapies, assessment of multiple metabolic and hepatic biochemical outcomes, adjustment for important baseline differences, and sensitivity analysis addressing the marked baseline imbalance in CHF. However, several limitations should be considered. The open-label design may have introduced performance or detection bias. Baseline differences in age, duration of diabetes, dyslipidemia, and CHF persisted despite randomization; although these were addressed statistically and the sensitivity analysis produced consistent findings, residual confounding cannot be completely excluded. The final efficacy analysis was per-protocol because week-twelve outcome data were unavailable for participants lost to follow-up, which may have introduced attrition bias. In addition, the twelve-week follow-up was insufficient to determine longer-term hepatic or cardiovascular effects, and liver USG was not repeated at follow-up. Consequently, the hepatic findings represent biochemical rather than structural improvement. Finally, the results apply specifically to empagliflozin 10 mg and metformin 1000 mg/day and should not be generalized to other SGLT2 inhibitors or different dosing regimens.

Within these limitations, the consistency of the findings across adjusted and sensitivity analyses supports an association between empagliflozin and greater short-term hepatic biochemical, glycemic, and hemodynamic improvement than metformin in this study population. Longer-duration studies incorporating repeat liver imaging and validated non-invasive fibrosis assessments are warranted to determine whether these biochemical responses translate into sustained improvement in hepatic disease.

CONCLUSIONS

Both empagliflozin and metformin improved hepatic biochemical markers, glycemic control, and blood pressure over twelve weeks in patients with T2DM and MASLD. Empagliflozin was associated with greater reductions in ALT, AST, HbA1c, SBP, and DBP after adjustment for baseline demographic and clinical differences. Overall adverse-effect frequency was comparable between the groups, although the pattern of adverse effects differed between treatments. These findings suggest that empagliflozin may provide greater short-term biochemical, glycemic, and hemodynamic benefit than metformin in this study population. As liver imaging was not repeated at follow-up, the hepatic findings should be interpreted as biochemical improvement rather than evidence of structural regression of MASLD.

DECLARATIONS

Authors' Contributions: Anam Nasir: Conceptualization, Methodology, Investigation, Formal analysis, Writing – original draft. Moosa Khan: Conceptualization, Supervision, Resources, Validation, Writing – review and editing. Sara Tariq Abbasi: Methodology, Data curation, Investigation, Formal analysis, Writing – review and editing. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Competing Interests: The authors declare no conflict of interest.

Consent to Participate: Written informed consent was obtained from all participants before enrollment.

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Data Availability: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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