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Original Article

Weight Loss and Change in FIB-4 Score Following Semaglutide Treatment in Overweight Adults with Metabolic Dysfunction–Associated Fatty Liver Disease.

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Abstract

Objective: To assess changes in body weight and fibrosis-4 (FIB-4) score over 24 weeks of semaglutide treatment in overweight adults with MAFLD.

Methodology: This observational cohort included patients from January to December 2025 at a hospital in Multan, Pakistan. Adults with overweight or obesity and ultrasonographically documented MAFLD received once-weekly subcutaneous semaglutide alongside lifestyle advice. We recorded body weight, aminotransferases, and platelet count before treatment and at 24 weeks. We also assessed treatment exposure, follow-up completion, and adverse effects.

Results: Of 98 patients screened, 80 began treatment and 72 completed paired assessment. Mean body weight decreased from 92.4 ± 13.6 to 84.7 ± 13.0 kg, a mean change of −7.7 kg (95% confidence interval −8.7 to −6.7; $p<0.001$). Forty-seven patients (65.3%) lost at least 5% of their initial weight, and 19 (26.4%) lost at least 10%. Median FIB-4 decreased from 1.58 to 1.29 ($p<0.001$), alongside reductions in aspartate and alanine aminotransferases. Nausea was recorded in 16 patients (20.0%), and five (6.3%) stopped treatment because of adverse effects.

Conclusion: Patients completing follow-up showed meaningful weight loss and lower FIB-4 scores during semaglutide treatment. The FIB-4 change reflected a change in a non-invasive risk score rather than confirmed regression of liver fibrosis.

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Introduction

Metabolic dysfunction–associated fatty liver disease (MAFLD) describes hepatic steatosis occurring alongside overweight or obesity, type 2 diabetes, or other evidence of metabolic dysregulation.[1] The later term, metabolic dysfunction–associated steatotic liver disease (MASLD), reflected a change in nomenclature and diagnostic criteria; the two definitions overlapped but were not identical.[2] In either framework, clinicians could not assess liver health without considering a patient's weight and broader metabolic health. Weight reduction remained central to management because it addressed a condition many patients recognized in daily life while also targeting metabolic risk.[3] Liver assessment needed a practical pathway from general medical care to specialist review, particularly when an abnormal result required further testing.[4] These needs were relevant to hospitals in Pakistan, where patients might receive care from general physicians, gastroenterologists, and nephrologists for closely related conditions. Semaglutide has shown marked effects on body weight in controlled trials. In STEP 1, once-weekly treatment produced greater weight loss than placebo among adults with overweight or obesity. [5] The STEP 5 trial showed that substantial weight loss could persist over two years of continued treatment.[6] Follow-up after STEP 1 also found considerable weight regain following withdrawal, making treatment duration and persistence important when interpreting a shorter course of care.[7]Evidence on liver outcomes has become more detailed but requires attention to what each study measured. A phase 2 trial found greater resolution of biopsy-confirmed steatohepatitis among participants receiving semaglutide than among those receiving placebo.[8] In an imaging trial, liver fat decreased, but the study did not show significant improvement in liver stiffness.[9] A trial involving patients with compensated cirrhosis likewise did not show significant improvement in fibrosis.[10] More recently, the ESSENCE trial reported favorable histological outcomes in patients with biopsy-confirmed steatohepatitis and stage F2–F3 fibrosis.[11] These carefully selected trial populations differed from patients identified through ultrasound and routine laboratory testing in general hospital settings. Smaller clinical studies added information about treatment in patients who also had type 2 diabetes. A 24-week pilot study of oral semaglutide reported changes in weight and liver-related measurements.[12] A subsequent multicenter observational study examined oral semaglutide in patients with diabetes and MASLD.[13] Another clinical study assessed vascular and liver measurements during semaglutide treatment, illustrating the range of outcomes considered in people with overlapping metabolic disease.[14] The fibrosis-4 index (FIB-4) used age, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count to support initial assessment of advanced fibrosis risk. It was inexpensive and readily calculated from common laboratory tests. However, a lower FIB-4 score could follow a change in aminotransferases without proving that existing liver fibrosis had regressed. Against this background, the present study assessed weight and FIB-4 over 24 weeks of semaglutide treatment in overweight adults with MAFLD at a hospital in Multan. We examined treatment exposure, follow-up completeness, and adverse effects to interpret the measured changes in clinical context.

Materials and Methods

The single-center observational cohort covered 01 January 2025 to 31 December 2025 at Bakhtawar Amin Hospital in Multan, Pakistan. Patients entered the cohort between January and June, allowing a 24-week assessment before the end of December. We screened adults attending general medicine and gastroenterology clinics on a consecutive basis. Eligibility required age 18–65 years, a body mass index (BMI) of at least 25 kg/m², hepatic steatosis documented by ultrasonography, and overweight or obesity meeting the stated MAFLD definition. Baseline weight, AST, ALT, and platelet count had to be available before starting semaglutide. Patients with known cirrhosis, another significant liver disease, substantial alcohol consumption, pregnancy, previous bariatric surgery, or recent use of another weight-loss medicine were excluded. Acute illness at baseline blood sampling was also an exclusion criterion because it could affect aminotransferases or platelet count. Patients with advanced chronic kidney disease or a previously documented serious reaction to a glucagon-like peptide-1 receptor agonist were not enrolled. Semaglutide was given by once-weekly subcutaneous injection. Treatment began at 0.25 mg weekly for

four weeks and increased to 0.5 mg weekly. An increase to 1.0 mg depended on tolerability and the treating clinician's assessment. At enrolment, patients received advice on portion size, everyday physical activity, and reducing sugar-sweetened drinks. We recorded dose increases, missed injections, treatment interruptions, and discontinuations during follow-up visits approximately every four to eight weeks. We measured weight in light clothing on a clinic scale and calculated BMI using recorded height. Laboratory measurements included AST, ALT, platelet count, and serum creatinine. We recorded glycated hemoglobin for patients with diabetes when available. For the week-24 assessment, we accepted measurements taken within two weeks of the scheduled visit; we matched blood results to the clinical visit within a 14-day interval. We described kidney function using estimated glomerular filtration rate (eGFR). Nausea, vomiting, constipation, and other symptoms recorded at visits were reviewed, together with reasons for stopping treatment. The two principal outcomes were the paired change in body weight and the paired change in FIB-4 between baseline and week 24. Percentage weight change used baseline weight as the denominator. We also calculated the proportions losing at least 5% and at least 10% of baseline weight. FIB-4 was obtained from age in years, AST and ALT in U/L, and platelet count in \(10^9\)/L using the formula: age × AST ÷ [platelet count × √ALT]. We reported its laboratory components separately to clarify changes in the calculated score. For descriptive purposes, we grouped FIB-4 results as below 1.3, 1.3–2.67, and above 2.67. These categories supported risk assessment; we did not treat them as measured fibrosis stages. Patients with a concerning result required interpretation in the context of their broader clinical findings and consideration of secondary assessment, such as transient elastography. Table 1 sets out the timing and purpose of the study measurements.

Table 1. Assessment schedule and outcome measures

Measure	Baseline	During treatment	Week 24	Purpose
Weight and BMI	Recorded	Weight checked	Repeated	Absolute and percentage weight change
AST, ALT and platelet count	Measured	As clinically indicated	Repeated	Laboratory change and FIB-4 calculation
FIB-4	Calculated	—	Calculated	Change in fibrosis risk score
Semaglutide exposure	Starting dose	Escalation and interruptions recorded	Highest tolerated dose recorded	Interpretation of treatment course
Kidney function	Creatinine and eGFR	Clinical review	Available results recorded	Description of kidney status
Adverse effects	Relevant history	Symptoms recorded	Records reviewed	Tolerability and discontinuation

We analyzed data using SPSS version 26.0. We reported continuous variables as mean ± standard deviation or median (IQR), and categorical variables as frequency and percentage. We compared paired weight and FIB-4 values using the paired *t-test* and Wilcoxon signed-rank test, respectively. We reported mean weight change with a 95% confidence interval; *we considered* $p<0.05$ significant.

Results

Of 98 adults screened, 18 were excluded: seven lacked complete baseline laboratory results, five had another significant liver condition, three had recent exposure to a different weight-loss medicine, and three met another exclusion criterion. Semaglutide was started in 80 patients. Eight did not complete the week-24 assessment, leaving 72 with paired weight and FIB-4 measurements. Table 2 presents baseline characteristics. Type 2 diabetes and hypertension were common in the treatment cohort.

Table 2. Baseline characteristics of patients who began semaglutide

Characteristic	Treatment cohort (\(n=80\))
Age, years, mean ± SD	44.8 ± 10.2
Women, n (%)	48 (60.0)
Weight, kg, mean ± SD	92.6 ± 13.8

BMI, kg/m², mean ± SD	32.7 ± 4.1
Type 2 diabetes, n (%)	43 (53.8)
Hypertension, n (%)	33 (41.3)
Chronic kidney disease, n (%)	9 (11.3)
AST, U/L, median (IQR)	40 (32–49)
ALT, U/L, median (IQR)	57 (44–72)
Platelet count, \(10^9\)/L, mean ± SD	244 ± 58
FIB-4, median (IQR)	1.57 (1.10–2.02)
eGFR, mL/min/1.73 m², mean ± SD	82.1 ± 17.5

SD, standard deviation; IQR, interquartile range.

Among the 72 patients who completed paired assessment, mean weight fell from 92.4 ± 13.6 kg to 84.7 ± 13.0 kg. The paired mean change was −7.7 kg (95% CI −8.7 to −6.7; $p<0.001$), equivalent to an 8.3% reduction from baseline. Table 3 shows the weight-loss thresholds and treatment exposure. Forty-seven patients reached at least 5% weight loss, while 19 reached at least 10%. Eleven patients had at least one treatment interruption.

Table 3. Weight outcomes and semaglutide exposure

Measure	Result
Patients with paired weights, n	72
Baseline weight, kg, mean ± SD	92.4 ± 13.6
Week-24 weight, kg, mean ± SD	84.7 ± 13.0
Paired change, kg, mean (95% CI)	−7.7 (−8.7 to −6.7)
Percentage weight change, mean ± SD	−8.3 ± 4.4
At least 5% weight loss, n/N (%)	47/72 (65.3)
At least 10% weight loss, n/N (%)	19/72 (26.4)
Highest tolerated dose of 0.5 mg, n/N (%)	24/80 (30.0)
Highest tolerated dose of 1.0 mg, n/N (%)	56/80 (70.0)
At least one treatment interruption, n/N (%)	11/80 (13.8)

Median FIB-4 decreased from 1.58 at baseline to 1.29 at week 24 ($p<0.001$). AST and ALT also decreased, while mean platelet count changed only slightly. Table 4 presents the laboratory components alongside the calculated score. Fifteen patients whose baseline FIB-4 was at least 1.3 had a score below 1.3 at follow-up. Movement between these categories described a change in risk classification; no paired liver biopsy or stiffness measurement established a change in fibrosis.

Table 4. Paired liver-related measurements at baseline and week 24 (\(n=72\))

Measure	Baseline	Week 24	Comparison
AST, U/L, median (IQR)	41 (33–50)	32 (26–40)	$p<0.001$
ALT, U/L, median (IQR)	58 (45–73)	43 (34–54)	$p<0.001$
Platelet count, \(10^9\)/L, mean ± SD	245 ± 57	252 ± 55	$p=0.14$
FIB-4, median (IQR)	1.58 (1.12–2.04)	1.29 (0.94–1.70)	$p<0.001$
FIB-4 <1.3, n (%)	35 (48.6)	46 (63.9)	Descriptive
FIB-4 1.3–2.67, n (%)	32 (44.4)	24 (33.3)	Descriptive
FIB-4 >2.67, n (%)	5 (6.9)	2 (2.8)	Descriptive

Table 5 shows kidney measurements, follow-up completeness, and adverse effects. Paired eGFR was available for 68 patients and showed little change over 24 weeks. Nausea was the most frequently recorded symptom. Five of the 80 patients who began treatment stopped it because of an adverse effect. We excluded the eight patients without a complete week-24 assessment from paired outcome calculations, but they remained in the treatment-cohort denominator used to describe follow-up.

Table 5. Kidney measurements, follow-up and recorded adverse effects

Measure	Result
Patients with paired eGFR, n	68
Baseline eGFR, mean ± SD	82.4 ± 17.1
Week-24 eGFR, mean ± SD	82.9 ± 16.8
Nausea, n/N (%)	16/80 (20.0)
Vomiting, n/N (%)	7/80 (8.8)
Constipation, n/N (%)	9/80 (11.3)
Stopped treatment because of an adverse effect, n/N (%)	5/80 (6.3)
No complete week-24 assessment, n/N (%)	8/80 (10.0)

Discussion

Weight fell by an average of 7.7 kg among patients who completed 24 weeks of follow-up, and nearly two-thirds achieved at least 5% weight loss. FIB-4 also decreased, together with AST and ALT. The findings described a clinically recognizable treatment course: patients reached different tolerated doses, some interrupted treatment, and a smaller group did not complete assessment. These details mattered when judging how closely an observed outcome resembled results achieved under controlled trial conditions. A clinical study in patients with obesity or type 2 diabetes and fatty liver disease also examined changes in weight and liver-related measurements during semaglutide treatment.[15] Across trials included in a systematic review, semaglutide showed favorable effects on weight and several steatohepatitis outcomes, although the evidence for improvement in fibrosis stage was less certain.[16] The present 24-week findings were therefore compatible with the broader pattern of weight reduction, but they did not establish a liver tissue response. The achieved doses were also below the 2.4 mg weekly regimen used in several major obesity trials, limiting a direct comparison of weight-loss magnitude. The distinction between a lower FIB-4 score and less fibrosis was especially important. FIB-4 was calculated from laboratory measurements that could change during treatment. Here, both AST and ALT fell; that shift helped explain the score's movement without demonstrating that liver scar tissue had regressed. Fifteen patients moved below the 1.3 threshold, but clinicians still needed to consider age, persistent abnormalities, and the broader clinical picture. Patients whose scores remained concerning required consideration of a second assessment rather than reassurance from the cohort's average result. The clinical value of weight management also extended beyond the liver. A cardiovascular outcomes trial involving adults with overweight or obesity and established cardiovascular disease found fewer major adverse cardiovascular events with semaglutide than with placebo.[17] In patients with type 2 diabetes and chronic kidney disease, the FLOW trial addressed major kidney outcomes over a much longer period than this cohort's follow-up.[18] A SELECT analysis assessed kidney outcomes in adults with overweight or obesity and cardiovascular disease who did not have diabetes.[19] These distinct trial populations showed why comorbid conditions belonged in the assessment, while the near-unchanged eGFR in Table 5 was only a short-term observation. A further FLOW analysis examined whether kidney findings differed with concomitant sodium-glucose cotransporter-2 inhibitor use, reinforcing the need to account for other treatment when interpreting routine clinical measurements.[20] Consecutive patient screening, paired weight and laboratory measurements, and documentation of semaglutide dose, treatment interruptions, and follow-up strengthened the study. Reporting AST, ALT, and platelet count alongside FIB-4 made the change in the score easier to interpret.

Conclusion

Over 24 weeks of semaglutide treatment, patients who completed follow-up experienced meaningful weight loss and a reduction in FIB-4 score. Nearly two-thirds lost at least 5% of their starting weight, while AST and ALT levels also decreased. These findings support monitoring

body weight and routine liver-related measurements together when caring for overweight patients with MAFLD.

Ethical Approval

The Hospital Research and Ethics Committee (IREB) approved this study under Document No. **BAMCM/Ref/874/2022** before the study commenced.

Informed Consent

We obtained written informed consent from all participants before enrolment. We informed participants and their parents or legal guardians about the study objectives, procedures, and the voluntary nature of participation. Participation was entirely voluntary.

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This study received no external funding and used available institutional resources.

Conflict of Interest

The authors declare no competing interests or conflicts of interest related to this study.

Authors' Contributions

Maryam Khawar, Nadeem Ullah: Contributed to conceptualization, study design, data collection, interpretation of findings, manuscript drafting, critical revision, and final approval. He also accepts accountability for all aspects of the work.

Muhammad Muzammil, Hassaan Yousaf: Contributed to study design, data collection, data curation, literature review, data interpretation, manuscript revision, and final approval. They also accept accountability for all aspects of the work.

Muhammad Mumtaz Ather, Syed Sarmad Ali Naqvi: Contributed to conceptualization, study supervision, clinical assessment, interpretation of findings, and critical revision of the manuscript for important intellectual content. She also approved the final version and accepts accountability for all aspects of the work.

All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work in accordance with the ICMJE authorship criteria.

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