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EFFECT OF STATIN THERAPY ON LIVER FIBROSIS AND ITS ASSESSMENT IN PATIENTS WITH CHRONIC NON-ALCOHOLIC FATTY LIVER DISEASE AND CONCOMITANT ATHEROSCLEROSIS

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD), now commonly classified within metabolic dysfunction-associated steatotic liver disease (MASLD), frequently coexists with dyslipidemia and atherosclerotic cardiovascular disease. Fibrosis stage is a major determinant of liver-related prognosis, whereas cardiovascular disease remains a major source of morbidity and mortality. Statins are fundamental for prevention of ASCVD events, yet concerns about hepatotoxicity have historically contributed to undertreatment, while experimental and observational data have raised the separate possibility that statins may attenuate hepatic fibrogenesis. [7]

Objective: To evaluate the effects of statin therapy on liver-fibrosis progression and regression in patients with NAFLD/NASH and concomitant atherosclerosis, while synthesizing evidence on mechanisms, cardiovascular outcomes, hepatic safety and the optimal methods for measuring fibrosis change.

Methods: A narrative, non-systematic review was conducted with priority given to literature from approximately September 2016 through September 2026 and to seminal older studies. PubMed/MEDLINE, the Cochrane evidence base, major AASLD, EASL-EASD-EASO and ESC/EAS guidance, and clinical-trial registry information were searched for randomized trials, cohort and case-control studies, systematic reviews/meta-analyses, diagnostic-accuracy studies and mechanistic research. English-language evidence was prioritized, with relevant Russian and Uzbekistan-origin literature considered. No original pooled effect was calculated.

Results: Human observational evidence generally associates statin exposure with less advanced fibrosis or slower progression. In a biopsy-based multicenter cohort, statin use was associated with lower odds of F2–F4 fibrosis (adjusted OR 0.42, 95% CI 0.20–0.80). A nationwide Korean analysis reported an adjusted OR of 0.43 for a BARD-defined significant-fibrosis endpoint. A primary-care cohort found a hazard ratio of 0.60 for progression to FIB-4 ≥2.67. Most importantly for longitudinal imaging, a 16-center cohort of 7,988 patients with steatotic liver disease found statin exposure associated with less liver-stiffness progression both with and without compensated advanced chronic liver disease, but not with significantly more liver-stiffness regression. Meta-analysis has reported lower odds of “significant fibrosis,” but with very high heterogeneity, while the pooled change in fibrosis stage

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itself was not significant. A short randomized trial containing a rosuvastatin arm showed no significant MRE fibrosis change over 24 weeks. These data support a potential progression-preventing effect more strongly than true regression. [8]

Conclusions: Statins are appropriate and generally safe for ASCVD prevention in patients with NAFLD/MASLD, including compensated advanced liver disease when otherwise clinically indicated. Current evidence does not establish statins as an approved or proven antifibrotic treatment. Evidence is strongest for an association with slower fibrosis/liver-disease progression, whereas convincing demonstration of fibrosis regression remains lacking. Serial assessment should use a multimodal strategy: FIB-4 for triage, VCTE and/or ELF for secondary evaluation, and MRE or biopsy when greater precision is required. Because statins can alter aminotransferases, changes in FIB-4 alone should not be interpreted as proof of collagen regression. [9]

Keywords: statins; non-alcoholic fatty liver disease; NAFLD; non-alcoholic steatohepatitis; NASH; MASLD; MASH; liver fibrosis; atherosclerosis; cardiovascular disease; FIB-4; elastography; magnetic resonance elastography; liver biopsy.

Introduction

NAFLD/NASH is closely linked to obesity, insulin resistance, type 2 diabetes, atherogenic dyslipidemia and ASCVD. Contemporary nomenclature defines MASLD as steatotic liver disease occurring with cardiometabolic risk factors and distinguishes the inflammatory form as MASH; the older NAFLD/NASH terminology remains embedded in most pivotal statin studies and is therefore retained when describing them. Fibrosis is the central liver-related prognostic variable: progression from mild fibrosis to bridging fibrosis and cirrhosis is associated with escalating risks of decompensation, hepatocellular carcinoma and liver-related death. [1]At the same time, treating ASCVD risk is indispensable. Current European lipid guidance continues to place statin-based LDL-C lowering at the center of risk-based prevention, particularly in patients with established atherosclerotic disease. In a patient who has both NAFLD/MASH and coronary, cerebrovascular or peripheral atherosclerosis, the relevant therapeutic question is therefore not merely whether statins change hepatic enzymes: it is whether cardiovascular benefit can be obtained safely while liver fibrosis is monitored appropriately, and whether the treatment may additionally influence the natural history of liver fibrosis. [1–3] [10]Historically, statin treatment was sometimes withheld when alanine aminotransferase (ALT) or aspartate aminotransferase (AST) was elevated. This approach is increasingly inconsistent with evidence. AASLD guidance identifies statins as safe for cardiovascular risk reduction in NAFLD, and a systematic review of 22 studies involving 2,345 NAFLD patients reported reductions rather than deterioration in ALT, AST and γ-glutamyl transferase after statin treatment. Contemporary randomized safety evidence across the much broader statin-treated population further supports a favorable benefit–risk profile. [1,13,18] [11]The more uncertain question is whether inhibition of 3-hydroxy-3-methylglutaryl-coenzyme A reductase changes fibrosis itself. Several biological mechanisms make such an effect plausible. Inhibition of the mevalonate pathway affects not only cholesterol synthesis but also production of isoprenoid intermediates required for prenylation of signaling molecules such as Rho family GTPases. Experimental work has

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linked statin exposure to reduced RhoA/Rho-kinase signaling, enhanced endothelial nitric-oxide synthase activity, induction of Kruppel-like factor 2 (KLF2), improved sinusoidal endothelial function and paracrine deactivation of hepatic stellate cells. These effects could theoretically reduce stellate-cell activation, extracellular-matrix deposition, inflammation and intrahepatic vascular resistance. [19,20] [12]Human clinical evidence, however, is methodologically more difficult. Statins are prescribed preferentially to patients with diabetes, dyslipidemia and ASCVD—features that themselves correlate with more severe MASLD. This creates confounding by indication. Conversely, statin users may receive more intensive medical surveillance and better cardiometabolic care, creating a "healthy-user" or care-intensity effect. Different studies have defined fibrosis using biopsy, BARD, FIB-4, elastography or clinical outcomes, so apparently similar endpoints are not necessarily equivalent. [13] A particularly important measurement issue is that some commonly used noninvasive fibrosis scores contain variables that statins can change independently of fibrosis. FIB-4 incorporates AST and ALT together with age and platelet count. Because statin therapy in NAFLD has repeatedly been associated with reductions in aminotransferase concentrations, a fall in FIB-4 during treatment may partly represent improvement in hepatocellular injury rather than remodeling of collagen. This is an inference from the structure of FIB-4 and the biochemical effects of statins, not proof that reported FIB-4 associations are spurious; it does, however, mean that biochemical scores should not be used alone to claim antifibrotic efficacy. [14] The purpose of this review is therefore to separate four different outcomes that are often conflated: lowering LDL-C and preventing ASCVD events; improving serum liver enzymes; preventing progression to more advanced fibrosis; and producing genuine regression of established fibrosis. These endpoints have different evidentiary standards and should not be treated as interchangeable.

Materials and Methods

This work was designed as a narrative, non-systematic review, consistent with the methodological style of the supplied PCSK9 exemplar rather than as a registered systematic review or original meta-analysis. The principal sources were PubMed/MEDLINE, the Cochrane evidence base, major professional guidelines and official/registry-derived clinical-trial information. Guideline sources were prioritized from the American Association for the Study of Liver Diseases (AASLD), European Association for the Study of the Liver together with EASD/EASO, and European Society of Cardiology/European Atherosclerosis Society. Priority was given to the preceding approximately 10 years, while older studies were retained when they were seminal for biopsy validation, mechanistic understanding, long-term histological observation or cardiovascular safety. [15]

The principal search concept combined terms equivalent to (statin OR atorvastatin OR rosuvastatin OR simvastatin OR HMG-CoA reductase inhibitor) AND (NAFLD OR NASH OR MASLD OR MASH) AND (fibrosis OR cirrhosis OR liver stiffness OR elastography OR FIB-4 OR NFS OR ELF OR biopsy OR histology) AND/OR (atherosclerosis OR ASCVD OR coronary disease OR cardiovascular outcomes).

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Additional targeted searches addressed statin safety, hepatic stellate-cell mechanisms, noninvasive-test diagnostic performance and ongoing trials.

Eligible evidence included adult randomized clinical trials, prospective or retrospective cohort studies, case-control studies, systematic reviews and meta-analyses, guideline statements, diagnostic-accuracy studies comparing noninvasive fibrosis tests with biopsy, and mechanistic studies capable of explaining observed clinical associations. Studies were excluded from the main efficacy synthesis when they were pediatric, focused predominantly on another liver etiology without relevance to the mechanism/safety question, lacked a relevant liver or cardiovascular endpoint, or provided insufficient methodological information.

English-language sources were prioritized. A Russian-language clinical recommendation on adult NAFLD was included as regional context. A targeted search also identified Uzbekistan-origin literature on NAFLD assessment, including FibroScan-based and diagnostic modeling research, but no Uzbekistan-origin comparative study of statin therapy with a validated fibrosis endpoint was identified; therefore these local publications were not used to estimate the treatment effect of statins. [26,27] [16]

The evidence was interpreted hierarchically. Randomized evidence was given the greatest weight for causal treatment effects, followed by biopsy- or elastography-based longitudinal cohorts, large adjusted administrative cohorts and cross-sectional evidence. Changes in ALT/AST were treated as biochemical rather than fibrosis outcomes. Likewise, a lower prevalence of high FIB-4/BARD scores was not equated automatically with histological fibrosis regression.

.Results

Mechanistic basis for a possible antifibrotic effect. Statins inhibit HMG-CoA reductase and reduce mevalonate production. The downstream consequence is not limited to lower hepatic cholesterol synthesis; reduced generation of farnesyl and geranylgeranyl intermediates can alter prenylation-dependent signaling. Experimental cirrhosis models indicate that atorvastatin can suppress RhoA/Rho-kinase activity and improve endothelial nitric-oxide signaling, while work in sinusoidal endothelial cells has demonstrated KLF2-dependent endothelial protection and paracrine deactivation of hepatic stellate cells after statin exposure. Because activated stellate cells are central producers of extracellular matrix during hepatic fibrogenesis, these mechanisms provide a credible biological pathway from statin exposure to reduced fibrogenic activity. [19,20] [12]

A second plausible pathway is reduction of hepatic cholesterol-related lipotoxicity and inflammatory signaling. In the biopsy cohort of Dongiovanni and colleagues, the biological hypothesis was that excess hepatic free cholesterol contributes to NASH pathogenesis; statin exposure was associated not only with less advanced fibrosis but also with lower prevalence of steatosis and steatohepatitis. Genetic heterogeneity may modify this relationship: the PNPLA3 I148M variant attenuated some of the observed association with

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steatohepatitis, illustrating why a uniform hepatic response to statins should not be assumed. [4] [17]

These mechanisms remain supportive rather than determinative. A pharmacological pathway can explain why a clinical effect might occur, but it cannot establish that established human fibrosis regresses. That question requires longitudinal human measurements with adequate controls.

Clinical evidence for fibrosis progression and regression. One of the earliest human histological observations came from Ekstedt and colleagues. Sixty-eight NAFLD patients underwent repeat liver biopsy after approximately 10.3–16.3 years; 17 were receiving statins at follow-up. Despite having a metabolically higher-risk phenotype, statin-treated patients showed reduced steatosis, and only four progressed in fibrosis stage. The study was small and nonrandomized and does not establish causality, but it was important in demonstrating that long-term statin exposure in NAFLD was not accompanied by obvious histological liver toxicity. [5] [18]

Dongiovanni et al. subsequently analyzed 1,201 European patients who underwent liver biopsy for suspected NASH. Statins were used by 107 patients. In propensity-matched analysis, exposure was associated with lower odds of steatosis (OR 0.09), NASH (OR 0.25) and F2–F4 fibrosis (OR 0.42, 95% CI 0.20–0.80). A dose-dependent association was reported. The use of histology is an important strength, but the design was observational and largely cross-sectional with respect to the fibrosis endpoint; therefore the result is evidence of an association with less severe histological disease, not direct demonstration that statins reversed pre-existing fibrosis. [4] [17]

A nationwide Korean study substantially expanded statistical power. Among more than 11 million participants, investigators constructed a NAFLD cohort using the fatty liver index and defined significant fibrosis using BARD ≥2. Among 712,262 subjects classified as having NAFLD, statin use was associated with lower odds of the fibrosis endpoint (adjusted OR 0.43, 95% CI 0.42–0.44), including analyses stratified by diabetes. The scale and adjustment are strengths, but neither NAFLD nor fibrosis was determined histologically, and BARD is a surrogate score influenced by metabolic and biochemical variables. [6] [19]

A 2024 primary-care MASLD cohort provided longitudinal evidence. Among 1,238 patients initially below the high-risk FIB-4 threshold, 47% received a statin and 18% progressed to FIB-4 ≥2.67 over mean follow-up of 3.3 years. Statin prescription was associated with a lower adjusted risk of progression (HR 0.60, 95% CI 0.45–0.80), with similar estimates for moderate- and high-intensity therapy. This supports prevention of progression in a real-world setting but shares the fundamental limitation that FIB-4 is partly determined by aminotransferase concentrations. [7] [20]

The strongest large-scale longitudinal elastography evidence is the international VCTE-Prognosis cohort published in *Gut*. It included 7,988 patients with MASLD from 16 tertiary centers, 40.5% of whom were using statins, and followed them for a median of 4.6 years. Statin use was associated with less liver-stiffness progression in patients with compensated advanced chronic liver disease (adjusted HR approximately 0.54) and without

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it (adjusted HR approximately 0.45). Importantly, however, statin use was **not** significantly associated with liver-stiffness regression (adjusted HR 0.914, 95% CI 0.778–1.074). The same study reported lower liver-related clinical-event and all-cause mortality risks among statin users. This distinction—less progression without demonstrably more regression—is highly relevant to interpretation of the total evidence. [8] [21]

A Swedish histopathology-linked cohort provides complementary outcome evidence. Among matched statin users and nonusers with noncirrhotic chronic liver disease, statin exposure was associated with a lower risk of severe liver disease overall (HR 0.60). In the NAFLD subgroup the estimate was HR 0.68 (95% CI 0.45–1.00), and statin exposure was also associated with lower progression to cirrhosis, hepatocellular carcinoma and liver-related mortality in the overall chronic-liver-disease cohort. These outcomes strengthen the possibility of disease modification but still do not prove histological fibrosis regression. [9] [22]

Not all observational analyses are positive. A propensity-score-matched study among US veterans with diabetes matched 34,102 statin users with 34,102 nonusers and found a composite NAFLD-progression endpoint in 8.8% versus 8.6% (OR 1.02, 95% CI 0.97–1.08), indicating no statistically significant overall association. A post-hoc comparison suggested more recorded progression with more intensive LDL-C reduction, but the nonrandomized design, composite coding-based outcome and possibility of residual confounding preclude interpreting that finding as evidence that intensive statin therapy causes fibrosis. [10] [23]

Meta-analytic findings reflect the same uncertainty. Fatima et al. synthesized 14 studies totaling approximately 1.25 million participants and reported lower odds of "significant fibrosis" among statin users (OR 0.20), but heterogeneity for this endpoint was extremely high (I^2 97%). More tellingly, the pooled change in **fibrosis stage** was not significant (WMD 0.07, 95% CI −0.05 to 0.20). Improvements were clearer for liver enzymes, steatosis and NAFLD activity score than for established fibrosis. [11] [24]

A separate 2022 meta-analysis likewise found improvements in biochemical parameters, steatosis grade and NAFLD activity score, but methodological heterogeneity was high. More recent synthesis through 2025 continues to support improvements in aminotransferases and lipids while leaving direct antifibrotic efficacy less certain. [12,17] [25]

Randomized evidence remains a major weakness. The older Cochrane review identified only very limited, high-risk-of-bias randomized evidence and could not establish histological benefit. In the ESSENTIAL randomized trial, 70 NAFLD patients received rosuvastatin 5 mg either alone or combined with ezetimibe for 24 weeks. Both groups showed reduced MRI-measured liver fat, but MRE-derived fibrosis change was not significant. Because both groups received rosuvastatin, the trial cannot estimate statin-versus-placebo antifibrotic efficacy, and 24 weeks is short for observing meaningful collagen remodeling. [14,16] [26]

A dedicated phase-2 atorvastatin study in NASH with significant fibrosis has also been registered, using histologically relevant improvement criteria. This ongoing evidence

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gap is important: the central antifibrotic hypothesis is still awaiting a properly powered randomized answer rather than merely another observational association. [27]

Discussion

The total evidence produces a clinically coherent but scientifically nuanced conclusion. Statins are strongly indicated for cardiovascular risk management in appropriately selected patients with NAFLD/MASH and atherosclerosis; they are not yet proven antifibrotic drugs. This distinction is essential because the standard of evidence required to prescribe a therapy for secondary ASCVD prevention is already satisfied, whereas the standard required to claim reversal of MASH fibrosis has not been satisfied for statins. [10]

The fibrosis data are nevertheless more encouraging than a simple "neutral" conclusion would suggest. Three different types of evidence point in the same direction: biopsy-based observational work shows less severe fibrosis among users; administrative and primary-care cohorts show lower incidence of high fibrosis-risk scores; and longitudinal VCTE data show less progression of liver stiffness. [4,6–8] The fact that the large VCTE cohort did not show significantly more regression is particularly informative. It suggests that any clinically relevant hepatic effect of statins may be better conceptualized as slowing or preventing progression rather than rapidly removing established scar tissue. [44]

This interpretation also reconciles the apparently conflicting meta-analytic outcomes. A lower frequency of "significant fibrosis" in statin users can coexist with a nonsignificant mean change in fibrosis stage if statins primarily reduce transition from lower to higher fibrosis stages, if follow-up is too short to demonstrate reversal, or if observational classification is influenced by changes in enzymes and metabolic status. The high heterogeneity in pooled fibrosis results is an additional reason to avoid converting these associations into a treatment recommendation for fibrosis itself. [24]

The measurement problem deserves particular emphasis. A drop in ALT is generally desirable but it is not a measurement of extracellular collagen removal. When AST and ALT fall, FIB-4 can change even if the architecture of the liver does not. Therefore studies in which the primary "fibrosis progression" outcome is defined by FIB-4 deserve less weight for antifibrotic causality than studies with serial elastography or biopsy. The same principle explains why the 7,988-patient serial-VCTE cohort provides especially useful information despite remaining observational. [45]

The cardiovascular side of the balance is much less ambiguous. In a patient with established atherosclerosis, not prescribing an indicated statin because of uncomplicated fatty liver can expose the patient to preventable myocardial infarction or ischemic stroke without a corresponding proven liver benefit. GREACE is particularly relevant because abnormal liver tests did not negate cardiovascular benefit and generally improved during therapy. Current liver and cardiovascular guidelines are therefore concordant:

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cardiovascular risk should be actively treated, and NAFLD itself is not a general contraindication to statin therapy. [1–3,15] [46]

This conclusion does not mean that all liver disease should be treated identically. Compensated MASLD without synthetic dysfunction differs substantially from decompensated cirrhosis. Advanced cirrhosis can alter drug handling, muscle vulnerability, nutritional state and competing risk. Evidence in this population is smaller, and individualized treatment and monitoring are warranted. The randomized atorvastatin cirrhosis trial is reassuring that carefully selected patients can receive therapy, but it did not establish liver-disease efficacy and should not be generalized indiscriminately to unstable decompensation. [25] [35]

Guideline implications are straightforward. AASLD guidance endorses statins for cardiovascular risk reduction in NAFLD, while EASL-EASD-EASO emphasizes active treatment of metabolic and cardiovascular comorbidities and recommends stepwise fibrosis case-finding with serum scores followed by elastography. The 2025 ESC/EAS focused update maintains intensive risk-based LDL management. The practical consequence is that hepatology and cardiology goals are complementary rather than competing: treat ASCVD appropriately while staging the liver independently. [1–3] [15]

The Russian adult NAFLD recommendations similarly emphasize integrated management rather than a liver-only strategy and provide useful regional context. No high-quality statin-specific fibrosis outcome study from Uzbekistan matching the inclusion criteria was identified in the targeted search. Uzbekistan-origin publications do, however, document active work on FibroScan and noninvasive NAFLD monitoring, indicating that the diagnostic infrastructure relevant to future local prospective studies is developing. [26,27] [16]

Several limitations affect this review. First, it is narrative rather than systematically registered, so study capture cannot be guaranteed to be exhaustive. Second, much of the statin-fibrosis literature is observational and vulnerable to confounding by indication, healthy-user effects and time-dependent exposure bias. Third, statin agents and doses vary considerably between studies, precluding confident statements that atorvastatin, rosuvastatin, simvastatin or another individual compound is uniquely antifibrotic. Fourth, NAFLD diagnostic definitions have changed over time and do not map perfectly onto current MASLD criteria. Fifth, fibrosis endpoints range from biopsy to BARD, FIB-4, VCTE and clinical outcomes; these cannot legitimately be pooled conceptually as though they represent the same biological construct. [47]

A further limitation is treatment co-intervention. Modern MASLD patients increasingly receive GLP-1 receptor agonists, dual incretin therapies, SGLT2 inhibitors, intensive weight-management programs and, in selected jurisdictions and fibrosis stages, liver-targeted therapy. These interventions can change body weight, glycemia, steatosis, inflammatory activity and potentially fibrosis. Future statin studies therefore need to document concomitant metabolic therapies meticulously rather than attributing all

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longitudinal liver improvement to lipid lowering. The evolving EASL and AASLD therapeutic landscape makes this particularly important. [48]

For clinical practice, the evidence supports the following integrated interpretation. A patient with imaging-confirmed fatty liver, established coronary atherosclerosis and mildly elevated stable ALT should receive the statin intensity indicated by ASCVD risk unless an actual contraindication exists. Baseline assessment should determine whether advanced fibrosis is likely, starting with FIB-4 and moving to VCTE/ELF when indicated. A fall in LDL-C demonstrates lipid response; a fall in ALT indicates biochemical improvement; neither proves fibrosis regression. Fibrosis trajectory should be assessed using the same validated liver-specific pathway used for MASLD generally. [7]

For research, several priorities emerge. A definitive trial should enroll patients with well-characterized MASH/MASLD and preferably F2–F3 fibrosis who also have a clinically appropriate reason for lipid lowering, randomly allocate a clearly defined statin exposure when ethically possible, and maintain equivalent background metabolic care. Endpoints should distinguish NASH/MASH resolution without fibrosis worsening, ≥1-stage fibrosis improvement without worsening of steatohepatitis, and prevention of fibrosis progression. Serial MRE/VCTE and ELF could provide intermediate continuous measures, while blinded paired histology would remain valuable for a regulatory-level fibrosis claim.

Trials also need adequate duration. Collagen remodeling is substantially slower than LDL-C lowering and often slower than reduction in aminotransferases or hepatic fat. A 12- or 24-week study can demonstrate lipid and biochemical effects but is poorly suited to disprove an antifibrotic effect. Conversely, a long observational association cannot completely remove confounding. Multi-year randomized or carefully emulated target-trial designs are therefore needed.

Subgroup biology also deserves attention. PNPLA3 and other genetic variants, diabetes, obesity severity, baseline fibrosis, sex, age and statin intensity may influence liver response. The Dongiovanni findings concerning PNPLA3 interaction and newer mechanistic observations on statin-associated hepatic lipid-regulatory pathways suggest that the effect may not be uniform across all metabolic phenotypes. [49]

Finally, clinically meaningful outcomes should not be sacrificed for surrogate precision. Avoiding cirrhosis, decompensation, hepatocellular carcinoma, cardiovascular events and death matters more than changing a score by a small numerical amount. The large longitudinal cohorts associating statins with fewer severe liver outcomes are therefore important hypothesis-supporting evidence, even though they do not substitute for randomized causal proof. [50]

Conclusion

Statin therapy should generally be regarded as essential cardiovascular treatment rather than contraindicated therapy in patients with NAFLD/MASH and concomitant

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atherosclerosis. Current evidence demonstrates a favorable hepatic safety profile in compensated disease and suggests that statin exposure may reduce or delay progression toward advanced fibrosis and severe liver disease. However, convincing evidence that statins cause regression of established human liver fibrosis remains insufficient. [51]

Fibrosis should therefore be assessed independently of the lipid response. FIB-4 is appropriate for first-line stratification, followed when indicated by VCTE and/or ELF; MRE provides greater diagnostic precision for specialist or research use, and biopsy remains important when histological confirmation will alter management or is required for a clinical trial. A fall in aminotransferases or FIB-4 during statin therapy should not by itself be described as fibrosis regression. [5]

The most accurate current conclusion is therefore that statins have a proven cardiovascular benefit, a well-supported safety profile, and a probable but not yet proven disease-modifying effect on fibrosis progression in NAFLD/MASH. Dedicated long-term randomized trials with histology and complementary quantitative fibrosis measurements are required before statins can be considered an antifibrotic therapy in their own right.

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