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ORIGINAL

Observational Study

Steatotic liver disease in patients with chronic hepatitis C



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SLD group was predominantly male (62.8%), in contrast to the non-SLD group, where women predominated. Patients with SLD were significantly older ($P = 0.0007$), had a higher body mass index ($P < 0.0001$), and more frequently pre-sented with diabetes ($P = 0.01$), obesity ($P < 0.0001$), hyperlipidemia ($P = 0.004$), and a history of alcohol abuse ($P < 0.0001$). They also had more advanced liver disease as indicated by a higher rate of cirrhosis (35.5% vs 12% in the non-SLD group, $P < 0.0001$), elevated aminotransferase activity ($P < 0.0001$), bilirubin concentration ($P < 0.0001$), and international normalized ratio values ($P = 0.0001$), and lower albumin concentration ($P = 0.0028$). While most patients in both groups completed treatment as planned, adverse events, including severe events and deaths, were more frequent in the SLD group. A sustained virologic response was achieved in 97.6% of the overall population but was significantly lower among patients with SLD compared to the non-SLD group (95.6% vs 99.0%, $P = 0.0081$). However, logistic regression analysis did not identify SLD as an independent predictor of treatment failure.

CONCLUSION

Comorbid SLD was common among CHC patients treated with DAAs and was associated with adverse baseline characteristics, including older age, higher body mass index, greater comorbidity burden, and more advanced liver disease. Although SLD patients achieved slightly lower rates of sustained virologic response, SLD itself was not an independent predictor of treatment failure. These findings suggest that poorer treatment outcomes in this subgroup are largely attributable to coexisting risk factors rather than SLD per se, highlighting the need for comprehensive management of metabolic and liver-related comorbidities to optimize antiviral therapy outcomes.

CONCLUSION

We documented a 42% rate of SLD comorbidity in a real-world population of HCV-infected patients treated with DAAs. Patients with liver steatosis were older, had a higher BMI, a higher burden of comorbidities, more frequent GT3 infection, and more advanced liver disease compared to those without SLD. Due to these negative predictors, the effectiveness of DAA treatment was significantly lower than in patients without SLD. These findings underscore the importance of systematically assessing SLD in HCV-infected individuals, not only as a comorbidity but also as a clinically relevant modifier of treatment response and prognosis. Integrating non-invasive tools for steatosis evaluation into routine HCV management could help identify high-risk patients, guide closer monitoring, and inform adjunctive interventions aimed at reducing metabolic risk factors. Given the ageing population and the rising prevalence of obesity and metabolic disorders, SLD is likely to play an increasingly important role in determining long-term liver health in those with past or ongoing HCV infection. Addressing this challenge will require both improved clinical pathways for risk stratification and broader public health strategies to mitigate the growing burden of metabolic disease.

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Association of Metabolic Comorbidities With Fibrosis Severity and Fibrosis Regression in Patients With Chronic Hepatitis B

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The presence of metabolic comorbidities is associated with a higher risk of liver-related events in chronic hepatitis B (CHB) patients. However, the association between presence of metabolic comorbidities and the severity of biopsy-proven liver fibrosis is yet unknown.

Data from CHB patients from 2 tertiary clinics and 8 clinical trials was analyzed. We studied the association between presence of metabolic comorbidities with severity of liver fibrosis in untreated patients, and with fibrosis regression or progression in biopsies taken after initiation of antiviral therapy.

We analyzed biopsies from 3179 untreated CHB patients. Median age was 37 years, 57.6% were hepatitis B e antigen positive, with median hepatitis B virus DNA of 7.30 logIU/mL. Overweight (29.4% vs 19.0%; $P < .001$), hypertension (40.7% vs 23.2%; $P < .001$), diabetes (42.2% vs 23.6%; $P < .001$), and dyslipidemia (42.9 vs 23.6%; $P < .001$) were associated with a higher risk of advanced fibrosis, with the highest risk observed in patients with multiple comorbidities. Findings were consistent in multivariable analysis (1 comorbidity: adjusted odds ratio [aOR], 1.115; ≥ 2 comorbidities: aOR, 1.627; $P = .006$). Regression to nonadvanced fibrosis, after treatment initiation, was more often observed in patients without metabolic comorbidities (43.1%), compared with patients with 1 (31.6%) or ≥ 2 comorbidities (17.0%) ($P = .005$). Findings were consistent in multivariable analysis (1 comorbidity: aOR, 0.792; ≥ 2 comorbidities: aOR, 0.260; $P = .025$). The risk of progression to advanced fibrosis was highest in patients with ≥ 2 comorbidities (14.3% vs 4.6%; $P = .001$).

Presence of metabolic comorbidities in untreated CHB patients is associated with more severe liver fibrosis and, after initiation of antiviral therapy, with less fibrosis regression and a higher risk of fibrosis progression.

What You Need to Know

Background

The presence of metabolic comorbidities increases the risk of liver-related events in patients with chronic hepatitis B (CHB). The association between presence of metabolic comorbidities with severity of liver fibrosis before and during antiviral therapy is currently unclear.

Findings

The presence of metabolic comorbidities is associated with more severe liver fibrosis in untreated CHB patients. Furthermore, the presence of metabolic comorbidities was associated with less fibrosis regression, and even fibrosis progression, despite antiviral treatment.

Implications for patient care

These findings support thorough metabolic assessment in patients with CHB. Furthermore, our findings suggest that presence of metabolic comorbidities should be taken into consideration when interpreting the effects of antiviral therapy on liver fibrosis over time.

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US-05-174-105 did not specify AOD as an exclusion criterion, we performed a sensitivity analysis excluding patients from these studies, which showed consistent results (Supplementary Figures 9 and 10). Finally, as the decision to perform liver biopsies is never entirely random, and as trial participants have to fulfill specific criteria, the study population may not entirely reflect the patient population. Of note, the cohort used in this study was different from the other previously published studies on the SONIC-B database, as data from patients enrolled from mainland China for the Peginterferon-2a trials could not be included due to changes in legislature.

In conclusion, the presence of metabolic comorbidities in patients with CHB is associated with more severe liver fibrosis in untreated patients. Furthermore, the presence of metabolic comorbidities is associated with less fibrosis regression, and even a risk of fibrosis progression in patients on AVT.



More on the myths and perceived magic of corticosteroids

Do corticosteroids prevent reactions to infusions of contrast, monoclonal antibodies, or chemotherapy?

KEY POINTS

Rates of infusion-related reactions vary by medication class and the individual medication, and so do the data and recommendations about corticosteroid pretreatment, necessitating a medication- and patient-specific approach.

For radiocontrast media, it remains reasonable to consider pretreatment in patients with a history of high-grade reactions, multiple comorbidities, or other factors associated with elevated risk.

For monoclonal antibodies, it may be reasonable to give corticosteroids before the first infusion of agents associated with high rates of reactions (ie, cetuximab, trastuzumab, rituximab, and infliximab). Less evidence supports giving them before subsequent doses of these agents (except for rituximab given for B-cell malignancy) or before monoclonal antibodies with low rates of reactions.

For chemotherapy, corticosteroids are recommended before infusions of taxanes but not platinum or pegaspargase.



Serial Liver Stiffness Measurement and Serum Biomarkers Are Not Strong Predictors of the Regression of Fibrosis among Chronic Hepatitis B Patients Receiving Antiviral Therapy Based on Triple Liver Biopsies

Background/Aims: Noninvasive indexes can be used to diagnose and stage liver fibrosis caused by chronic hepatitis B (CHB). We aimed to evaluate whether changes in the liver stiffness measurement (LSM) and serum biomarkers can predict liver fibrosis regression in CHB patients based on triple liver biopsies.

Methods: This multicenter cohort study was based on triple liver biopsies and lasted for 260 weeks. Liver fibrosis regression was defined as Ishak decreased ≥ 1 stage or predominantly regressive by P-I-R classification with stable Ishak stage. Twelve noninvasive models were validated externally and yielded area under the receiver operating characteristic curve (AUROC) values ≥ 0.700 for predicting significant fibrosis in the training set.

Results: A total of 175 CHB patients were included (median age: 38 years, 76.6% male). A total of 69.2% (117/169) and 79.6% (78/98) patients achieved liver fibrosis regression at week 78 and week 260, respectively. The mixed effects model revealed significant group \times time interactions between the regression and non-regression groups for aminotransferase to platelet ratio index (APRI; $p=0.041$), new algorithm attributed to age, alanine aminotransferase-gamma-glutamyl transferase algorithm ($n=0.022$) and King's score ($n=0.016$) from baseline to week 78 as well as for APRI ($n=0.046$).

however, none demonstrated superior performance. This study also had several limitations. The included noninvasive models involved overlapping biochemical markers. However, these models were established based on different cohorts, and have not been verified in the same cohort to determine their performance in predictive of liver fibrosis regression. In addition, we could not validate certain noninvasive models, including FibroStage, FibroBox, GIVPR, because our study was unable to measure and calculate unique serological markers required for these models.

In conclusion, although significant $\text{time} \times \text{group}$ interactions were observed across several noninvasive models, such as APRI, AAG, and King's, these changes did not optimally evaluate liver fibrosis regression. Future research should focus on developing a dynamic noninvasive model for predicting liver fibrosis regression, offering a viable alternative to liver biopsy.

FDA green-lights bone scan measurements in osteoporosis trials

The U.S. Food and Drug Administration (FDA) has cleared the use of bone scan measurements as endpoints in clinical trials -- a move that has major implications for the development of new drugs for treating osteoporosis.

The “landmark” decision will now enable trials to determine if a drug is effective based on bone mineral density (BMD) changes measured by dual-energy x-ray absorptiometry (DEXA) rather than whether the drug prevents fractures, expert Mary Bouxsein, PhD, of Harvard Medical School in Cambridge, MA, explained to *AuntMinnie*. Bouxsein is an orthopedic surgeon at Beth Israel Deaconess Medical

Latest in Digital X-Ray

AI predicts osteoporosis from lumbar spine

x-rays

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FDA Qualifies Total Hip Bone Mineral Density (BMD) as Surrogate Endpoint for Osteoporosis Drug Development

THE END