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FibroScan–aspartate aminotransferase score in an Asian cohort of non-alcoholic fatty liver disease and its utility in predicting histological resolution with bariatric surgery

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Abstract

Background and Aim: The FibroScan–aspartate aminotransferase (FAST) score was developed for identifying patients with non-alcoholic steatohepatitis, who also have an elevated non-alcoholic fatty liver disease (NAFLD) activity score (NAS) ≥ 4 and significant fibrosis (F ≥ 2). We aimed to validate it in our NAFLD cohort and assess if it correlates with the histological changes after bariatric surgery.

Methods: Patients with NAFLD, including those undergoing bariatric surgery, were included. The FAST score was calculated using liver stiffness measure, controlled attenuation parameter, and aspartate aminotransferase. Calibration and discrimination of the model were assessed by calibration plots and area under the receiver operating characteristic curve, respectively. Sensitivity and specificity were assessed at the rule-out and rule-in cutoffs (≤0.35 and ≥0.67), respectively. Changes in the NAS and FAST scores were compared in the bariatric cohort 1 year after surgery.

Results: The cohort composed of 309 patients, of which 48 patients underwent repeat liver biopsy at 1 year. The model showed good discrimination with area under the receiver operating characteristic curve of 0.79 (0.74–0.84); however, it was not satisfactorily calibrated (Hosmer–Lemeshow test, $P = 0.008$). The sensitivity and specificity at the rule-out and rule-in cutoffs were 0.90 and 0.84, respectively. A significant correlation was seen between the 1-year reduction in the NAS and FAST scores ($r = 0.38$, $P = 0.009$). A significant reduction in the median FAST score was seen in patients who had ≥2-point reduction in NAS after bariatric surgery.

Conclusion: FibroScan–aspartate aminotransferase score demonstrated good discrimination for fibrotic non-alcoholic steatohepatitis in our cohort. However, a miscalibration resulted in overprediction. The score correlated well with the histological response to interventions for NAFLD.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a growing epidemic worldwide, with an estimated prevalence varying between 10% and 40% in various geographical areas of the world.[1] The global prevalence of cirrhosis has also increased worldwide, mostly due to a parallel increase in non-alcoholic steatohepatitis (NASH).[2] Liver biopsy remains the gold standard for the diagnosis of NASH,[3] and most therapeutic drug trials include patients with biopsy-proven NASH.[4,5] Prompt detection of early fibrosis is important, thereby giving us a therapeutic window where early interventions may prevent the development of cirrhosis. Multiple serum biomarkers and imaging modalities, alone or in combination, have been developed and validated for determining liver fibrosis,[6,7] precluding the need for liver biopsy. The NAFLD fibrosis score (NFS), fibrosis-4 (FIB-4) score, and aspartate aminotransferase (AST)-to-platelet ratio index (APRI) have been extensively used for assessing fibrosis.[8–10] Magnetic resonance elastography and magnetic resonance imaging-estimated proton density fat fraction have shown promising results in estimating histological response after therapeutic drug trials in patients with NASH.[11,12] However, there is an unmet need to develop models to determine ongoing liver cell injury and inflammation and non-invasively assess the histological response to drugs.[13]

The FibroScan–AST (FAST) score[13] was developed to predict the presence of active fibrotic NASH, with a non-alcoholic fatty