Diagnostic accuracy of non-invasive markers of fibrosis in HIV mono-infected patients with histologically confirmed NAFLD

J Maurice[1], E Tsochatzis[2], M Nelson[3], P Kelleher[3], L Garvey[4], M Thursz[1] and M Lemoine[1]

[1]Imperial College London, London, UK, [2]University College London, London, UK, [3]Chelsea and Westminster Hospital NHS Foundation Trust, London, UK, [4]Imperial College Healthcare NHS Foundation Trust, London, UK

Background: Non-alcoholic fatty liver disease (NAFLD) is a common cause of chronic liver disease. Only about 15% develop liver fibrosis, the main predictor of liver-related mortality. Therefore it is critical to risk stratify patients with fibrosis using non-invasive (NI) markers. NAFLD is at least as common in patients with HIV, but few studies have validated the performance of NI markers of fibrosis in this population.

Methods: Prospective cross-sectional study. Patients with HIV monoinfection, radiological evidence of hepatic steatosis and abnormal liver function tests (ALT>80) and/or transient elastography (TE, Fibroscan_)≥7.1 kPa), with no evidence of other chronic liver disease, were offered a liver biopsy. A fasted Fibroscan and blood tests were collected. Liver histology was reported using NASH CRN scoring system. Continuous variables are expressed as mean (SD) or median (IQR) as appropriate. The performance of NI markers was assessed using area under ROC curves (AUROC). Cut-off values were evaluated for sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ration (LR+) and negative likelihood ration (LR-).

Results: From April 2016-January 2018, 36 patients had a liver biopsy. The population characteristics were: age 46.0 (12.5) years, BMI 30.8 (4.5) kg/m^2, waist circumference 103.8 (11.5) cm, time since HIV diagnosis 9.5 (5.0–17.3) years, diabetes 16.7%. Median time between liver biopsy and NI tests was 1.0 (0.0–4.0) month. The histological diagnosis was NASH n=24 (67%), simple steatosis n=9 (25%), non-specific n=3 (8%). Significant (≥F2) and advanced (≥F3) fibrosis was present in n=16 (44%) and n=13 (36%). The performance of TE and serological markers using previously validated cut-offs for F3 fibrosis are in table 1. All markers performed poorly at diagnosing F2 fibrosis. Combining TE and FIB-4 increased the AUROC for F3 to 0.83 (0.68–0.98).

Conclusion: TE and FIB-4 combined are most effective to rule out F3 fibrosis, but performed poorly with F2 fibrosis. Further work with larger cohorts is required to validate optimal cut-offs and diagnostic algorithms in the HIV population.