Application of LIVERFASt biomarkers to evaluate longitudinal hepatic fibrosis and inflammation after HCV cure

Mati Ullah Dad Ullah1, Arjun P. Kelaia1, Rohullah Raskhi2, Medhi Sakka2, Rana Alkouri2, Mukarram Jamat Ali1, Hao Wei Chen1, Iman Waheed Khan1, Mina Choudhry1, Muhammad Ashar Ali1, Maxime Deregnaucourt1, Dominique Bornefont-Rousselot1-3, Mona Munteanu1, Ronald Quaambao4 and Dany T. Lau4

1 Beth Israel Deaconess Medical Center, Harvard Medical School, USA, 2 Metabolic Biochemistry Department, Pitie-Salpetriere Hospital, Public Assistance Paris Hospitals,Aphe Surbonne University, France, 3 Pharmacy Training and Research Unit (UFR), Paris Cité University; Cnrs, Inserm, Uibes, France, 4 Fibronostics US Inc, USA

INTRODUCTION

• Hepatitis C virus (HCV) infection is a common cause of both liver cirrhosis and hepatocellular carcinoma
• Successful Direct antiviral agents (DAA) therapy is associated with >95% sustained virological response
• Although the goal of HCV treatment is to achieve SVR, patients with advanced fibrosis and cirrhosis still have a high risk of developing HCC
• It is important to identify patients who are at risk for liver disease complications after achieving SVR

AIMS

• To evaluate the prognostic values of LIVERFASt as a noninvasive biomarker in evaluating changes in fibrosis and inflammation post-DAA therapy
• To identify risks associated with HCC and liver disease progression after HCV cure post-DAA

METHOD

• Retrospective cohort study in a single tertiary liver center
• Patients achieved DAA-induced HCV cure with follow-up >1 year who had pretreatment and follow-up sera in the biorepository were included
• Medical record review was performed to record HCC, hepatic decompensations, and comorbid conditions
• APR1 and FIB-4 scores were calculated and correlated with the LIVERFASt Fibrosis scores at baseline.
• LIVERFASt™ is a blood-based diagnostic test that combines 10 biomarkers and algorithm technology to determine the fibrosis, inflammatory activity, and steatosis of the liver.

RESULTS

Hepatic fibrosis changes post-DAA
• At follow-up: 23 (58%) had ≥ 1 stage fibrosis regression
• 3 (7.5%) had progression of fibrosis
• 14 (35%) had no changes; 4 had F0-F1 at baseline

HCC post-DAA
• 9 developed HCC between 2.1 and 5.8 years after HCV cure, and 8 had advanced fibrosis prior to DAA.
• 5 (56%) had unchanged F3-4 stage
• 1 (11%) progressed from F2 to F4 fibrosis and had active inflammation
• 3 (33%) had fibrosis regression. All had a history of hepatic decompensation prior to DAA

Correlation of pretreatment APR1 and LIVERFASt

Correlation of pretreatment FIB-4 and LIVERFASt

CONCLUSIONS

• LIVERFASt has prognostic values by monitoring changes in hepatic fibrosis and inflammation after HCV cure
• Patients with advanced fibrosis and hepatic decompensation remain at high risk for HCC after HCV cure and require ongoing HCC Surveillance
• Patients with persistent hepatic inflammation despite SVR are at risk for liver disease progression and complications
• Other causes of liver disease must be evaluated for patients with hepatic inflammation or progressive fibrosis after HCV cure