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Evaluating the generalizability of clinical trials of directacting antivirals for HIV -hepatitis C virus coinfection

Regression of liver fibrosis after curing chronic hepatitis C with oral antivirals in patients with and without HIV coinfection

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Abstract

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Background:

Treatment with direct-acting antivirals (DAA) eradicates HCV from most chronic carriers.

Information on regression of liver fibrosis and the influence of HIV is scarce in cured patients.

Methods:

All consecutive HCV-infected individuals treated with DAA at our institution were examined. Hepatic elastography was performed at baseline and at the time of SVR12. Liver fibrosis regression was defined as a shift from advanced fibrosis (Metavir F3-F4) to nullmild fibrosis (F0-F2) and/or a reduction >30% KPa. APRI and FIB-4 scores were calculated in parallel.

Results:

A total of 260 patients were treated with DAA. All but 14 achieved SVR12 and represented the study population. HIV confection was present in 42%. At baseline, 57.2% had advanced liver fibrosis with a median of 11 kPa, FIB-4 of 2.4, and APRI of 0.95. At the time of SVR12, a median reduction of 2.1 kPa (p<0.001) was recognized using elastography. A significant fibrosis regression was seen in 40%, being more frequent in patients with baseline advanced fibrosis than in those with null-mild fibrosis (52.3% vs 22.5%; p <0.001). Even so, 41.2% of patients with baseline F3-F4 kept within cirrhotic scores. In multivariable analysis, only baseline stiffness was significantly associated with the extent of liver fibrosis regression.

Conclusions:

HCV cure with DAA is associated with regression of liver fibrosis in most patients treated with DAA, as measured using elastography, FIB-4 and APRI. This benefit is more pronounced in patients with baseline advanced fibrosis and cirrhosis. The dynamics of liver fibrosis regression are not influenced by HIV coinfection.

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