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Effect of ursodeoxycholic (UDCA) acid on fibrogenolysis markers in alcoholic liver disease in man¹Rania Hamed and ²Hafez Ahmed¹Dubai Medical College for Girls, UAE²St. George's, University of London, UK

The fibrogenolysis markers laminin, collagen IV, Collagen VI and collagen XIV are important molecules involved in liver fibrosis that can be measured in the blood to assess liver fibrogenolysis. UDCA is a relatively hydrophilic bile acid that has been shown to be beneficial in cholestatic liver diseases like primary biliary cirrhosis. Alcoholic liver disease (ALD) has many cholestatic features and a beneficial effect for UDCA is therefore hypothesized. We aimed to assess the effect of 6 months UDCA therapy (10 mg/kg, n=24, Childs-Pugh score 21A, 3B) or placebo (n=24, Childs-Pugh score 21A, 3B) on the classic liver function tests and fibrogenolysis markers laminin, collagen IV, Collagen VI and collagen XIV in biopsy confirmed ALD patients. We used carbohydrate deficient transferrin to assess alcohol intake (>6% considered heavy drinking), and measured % UDCA enrichment in total serum bile acids using GC/MS. We observed a statistically significant reduction in γ GT (20% reduction) and ALT (10% reduction) from entry values, $p < 0.05$ in both enzymes with UDCA therapy, while ALP and bilirubin were not significantly different in UDCA treated or control groups over the study period. Mean baseline level of laminin was reduced from 26.5 ± 2.1 to 22.2 ± 2.5 ng/ml with UDCA treatment but the difference, which accounts for 16%, was not statistically significant. Collagen IV was reduced from a mean value of $464 + 39.3$ to $445 + 40.5$ ng/ml, but the change was not statistically significant. A reduction of 12% in collagen VI from $5.1 (+ 0.34)$ to $4.4 (+ 0.30)$ was also observed but was not statistically significant. Collagen XIV was largely unchanged at $25.1 (+ 2.3)$ and $26.0 (+ 2.0)$ before and after UDCA treatment. These preliminary results are difficult to interpret as the trend of reduction in three of the four fibrogenolysis markers tested was maintained at 3 and 6 months of UDCA therapy, but statistical significance for any of these differences was not obtained perhaps for the relatively small sample studied since the number determined by sample size calculation was difficult to recruit over the study period. A larger clinical trial is needed to further explore UDCA efficacy in the treatment of ALD and how to optimise the selected battery of the fibrogenolysis markers in the assessment of response to therapy, compliance and prognosis.

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