ing criteria for PTSD. Patient demographics varied significantly between sites, including ethnicity (p=.005), 5 year history of homelessness (p=0.005) drinking days per month (p=.05), and lower physical function as measured by the Hepatitis C Quality of Life measure (p=.05). Conclusions: The majority of current VA HCV clinic patients are considered "high risk" and demonstrate considerable active morbidity. Significant patient variation exists between sites. Implementation of active integrated care strategies into HCV Clinics appears warranted as a strategy for increasing antiviral treatment rates with the improved efficacy of new antiviral medications.

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FATIGUE BEFORE, DURING AND AFTER ANTIVIRAL THERAPY OF CHRONIC HEPATITIS C: ANALYSIS OF THE VIRAHEP-C TRIAL

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Background: Fatigue is the most frequent symptom of liver disease and has a major effect on quality of life in patients with chronic hepatitis C (CHC). However, the degree of fatigue varies considerably among patients and correlates poorly with disease severity. In the Virahep-C Trial similar numbers of African American (AA) and Caucasian American (CA) patients with CHC were treated with peginterferon alfa-2a and ribavirin for 48 weeks while undergoing careful assessment of viral kinetics and clinical features including symptoms. Aim: Analysis of the significance of fatigue in CHC and its change during therapy and with subsequent virologic response. Method: Fatigue measurements were done at baseline, regularly during treatment and again 24 weeks afterwards. Fatigue was assessed using a yes/no question ("do you have fatigue") and a visual analog scale which scored fatigue on a scale of 0 to 100 mm. Patients with and without fatigue and with different degrees of fatigue were compared for baseline clinical features, and serial results from during and after therapy were analyzed by sex, race, fibrosis scores, and sustained virologic response (SVR). Results: At screening, among 401 adults with chronic hepatitis C enrolled in the Virahep-C trial, 207 (52%) admitted to fatigue. Fatigue was more common among women than men (59% vs. 48%, p=0.02). In unadjusted analysis, there was no significant association between frequency of fatigue and age, body mass index, ALT or AST, HCV RNA level, necroinflammatory scores or fibrosis stage. In multivariable analysis, controlling for sex, race, baseline viral level, and Ishak score, older age and higher ALT was associated with the higher frequency of fatigue (p<0.05 for both), at screening. The proportion of patients reporting fatigue increased on treatment (52% before vs. 79% at week 24, p<0.0001), as did the median visual analog scale fatigue score (25 vs. 43 mm, p<0.0001). Importantly, fatigue scores increased more among patients who ultimately had an SVR compared to those with a non-response during the first 24 weeks of treatment. After treatment, fatigue scores returned to baseline. However, in patients with an SVR both the frequency and average severity of fatigue decreased between baseline and 24 weeks after therapy (53% vs. 33%, p<0.0001 and 27 vs 13 mm, p<0.0001). Conclusion: The Virahep-C trial provided a unique opportunity to assess fatigue in patients with CHC and the changes in fatigue with therapy and with ultimate clearance of HCV. An SVR was associated with the presence of greater degrees of fatigue on therapy and with ultimate decrease in levels and frequency of fatigue 24 weeks after stopping therapy.

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THE MINOR IL28B SNP RS8099917 ALLELE FREQUENCY IS INCREASED IN A COHORT OF CHRONIC LIVER DISEASE PATIENTS AS COMPARED TO THE GENERAL POPULATION

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The minor IL28b SNP rs8099917 has been associated with progression to chronic infection and failure to respond to antiviral therapy in HCV patients. Considerable variation in carriage frequencies (range, 2%–31%) have been reported in different ethnicities. The AIM was to study the allele frequency and genotype distribution of this SNP in our specific general population and in a cohort of chronic liver diseases patients. In this cross-sectional study rs8099917 genotyping was performed by PCR and SSCP analysis. A 91 bp nucleotide fragment, which included the SNP, was amplified. The genotypes identified by SSCP were confirmed by direct sequencing. A total of 174 subjects were studied. The control Group A consisted of 74 randomized and anonymized ethnic DNA bank samples (m/f=37/37); Group B-43 HCV patients (m/f=26/17); Group C–32 chronic HBV patients (m/f=21/11); Group D–25 patients with advanced liver disease and transplanted livers(Ci/Ltx) (m/f=19/6). HCV RNA, HBV DNA and genotyping was done by Roche TaqMan IVD FDA approved RT-PCR. RESULTS: The allele frequency in the control group reached Hardy Weinberg equilibrium;the study population did not. The frequencies of the rs8099917 of GG, GT and TT genotypes were as follows. In Group A: 0.05, 0.35, 0.60 (G=23%/T=77%); in Group B: 0.07, 0.54, 0.39 (G=33%/T=67%); in Group C: 0.09, 0.6, 0.41 (G=34%/T=66%) Group D: 0.12, 0.64. 0.24 (G=44%/=T56%), respectively. The difference of the allele frequencies was statistically significant only for Group A vs. combined (B+C+D), p= 0.0071 and for Group A vs. Group D, p=0.0062, two-tailed Fisher's exact test. Of the 43 HCV patients 28 (genotype G1=25, G2=1 G3=1 G1+3=1) received peg-interferon/ribavirin therapy. There was no correlation between the viral load and the G/T alleles in both HBV and HCV. Of those who completed 48 week therapy, 6 had SVR and 8 were non-responders. In the SVR group, G containing genotypes were significantly lower compared to the NR group Chi sq.=5.091, P= 0.012. None of the 8 transplanted patients had the major TT allele genotype. DISCUSSION: The frequency of rs8099917 in healthy individuals in our region is similar to that of Italians from Toscana. The prevalence of the minor/heterozygous genotype in HCV patients was higher as compared to the reported for Spanish, Australian and Swiss populations. As expected, minor allele frequency was greater

in the HCV therapy non-responder patients. The increased frequency of the minor allele in our Ci/Ltx group, as compared to the control group, was unexpected. It might be due to a more general relationship of IL28b with liver inflammation or due to a study population selection bias and needs further study.

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CHRONIC HEPATITIS C IS ASSOCIATED WITH ADRENAL TUMORS

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Objective: Adrenal tumors have been reported in a series of patients with CHC (1); accordingly, the aim of this study was to explore further a potential association between CHC and adrenal tumors. Increased serum levels of insulin growth factor (IGF) and insulin resistance have been reported in patients with chronic hepatitis C (CHC). Insulin and IGF-1 have also been associated with increased growth of adrenal cells in cell culture. These observations suggest that both IGF and insulin can trigger adrenal tumorogenesis. Methods: Patients with a diagnosis of adrenal tumors over the past ten years were identified and their records reviewed for evidence of CHC. Descriptive statistics were used to describe the study group. Results: Twenty three patients with adrenal tumors were identified; of these patients, fifteen (65.2%) had hepatitis C antibody positive in serum, and eleven (47.8%) had hepatitis C RNA, consistent with CHC. The mean age of these fifteen patients was 60 years (range 44-81). Ten (66.7%) were women. Seven (46.7%) patients were African American and the rest (53.3%), was of Hispanic descent. The risk factor for viral hepatitis was intravenous drug use in four patients (26.7%) and blood transfusions in eight (53.3%). Ten patients (66.7%) had hypertension, four (26.7%) of them had documented hypokalemia, and five (33.3%) had type II diabetes mellitus. One patient was co-infected with the hepatitis B virus and three with the human immunodeficiency virus. One patient had documented high serum aldosterone levels and low plasma renin activity consistent with hyperaldosteronism; eleven patients had no documented work up for adrenal tumors at all. Conclusions: These results support an association between CHC and adrenal tumors. The fact that this association has not been reported until recently (1), may be due to the low level of enthusiasm to investigate further the relationship between adrenal tumors and hypertension, as has been interpreted to explain the paucity of work up for hyperaldosteronism (2), which has been attributed to "clinical ignorance and indifference"(2). Adrenal tumors may be an extrahepatic manifestation of CHC. Attention to subtle signs of hyperaldosteronism (e.g. hypertension, hypokalemia) will increase the recognition of adrenal tumors in patients with CHC and will surely improve patient care. References: (1) Gastroenterology & Hepatology 2010; 6: 385 (2) Science 2011; 331(6018): 685

Key Words: chronic hepatitis C, adrenal adenoma, hyperaldosteronism, extrahepatic manifestation.

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DO PATIENTS WITH CHRONIC HEPATITIS C AND ANTIBODIES TO HEPATITIS B CORE ANTIGEN (HBCAB) HAVE AN INCREASED RATE OF PROGRESSION OF HEPATIC FIBROSIS?

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Background: There has been conflicting data as to whether HBcAb positivity (HBcAb+) in patients with chronic hepatitis C (CHC) predisposes them to a higher risk of progression of fibrosis. While some European studies have found an association between occult hepatitis B infection (OBI) in CHC and progression of fibrosis, other European and US studies with paired liver biopsies have not. Purpose: To determine whether patients with CHC and concurrent HBcAb+ have an increased rate of progression of fibrosis, based on paired liver biopsies. Methods: We conducted a retrospective chart review of 500 pateints with CHC seen at the academic hepatology practice who have undergone at least 2 liver biopsies between 1992 and 2010, at least 1 year apart. Liver transplant recipients and those with coexisting liver disease (including steatohepatitis), HIV, malignancy, missing HBcAb data and baseline fibrosis score of stage 3-4/4 (Scheuer classification) were excluded. Univariate and multivariate analysis including chi-square testing, t-test and logistic regression modeling using SAS 9.2 were done to assess for association between HBcAb+ and progression of fibrosis. Results: Data on 198 patients were analyzed after exclusion criteria were applied. 70 patients were HBcAb+ (69% of which were also HBsAb+). Baseline characteristics were similar between both groups (BMI 26 vs 25, alcohol use 60% vs 68%, smoking 48% vs 38%, genotype 1 83% vs 88%, stage of fibrosis 1.15 vs 1.24). Mean progression of fibrosis between biopsies was <1 for both groups (0.39 in HBcAb+ vs 0.41 in HBcAb-); several patients did not progress between biopsies (mean time 4.6 vs 5.2 years). Both univariate and multivariate analysis found no association between HBcAb+ and progression of fibrosis (aOR 1.11, CI 0.46, 2.69 and aOR 1.12, CI 0.35, 3.58, respectively). In multivariate analysis, higher baseline stage of fibrosis was associated with lower likelihood of progression of fibrosis (aOR 0.13, CI 0.06, 0.32). Conclusions: Our study found no difference in progression of fibrosis among CHC patients with HBcAb+ compared to those HBcAb-. Well-designed prospective studies with a larger number of patients with paired liver biopsies looking at serum and liver HBV DNA and liver tissue immunostaining for HBcAg/HBsAg are required to determine the role of OBI in affecting the rate of progression of fibrosis in CHC patients.

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