



Proceedings

PS02.09 | ePoster Session II

Correlations Between Interstitial Stromal Fibrillary Network And Disease Progression In Hepatitis C

I.E. Plesea^{*1}, C.D. Uscatu¹, M.S. Serbanescu¹, M. Indries², R.M. Plesea¹

¹University of Medicine and Pharmacy of Craiova, Pathology, Craiova, Romania, ²University of Oradea, Infectious Diseases, Oradea, Romania

Introduction/ Background

Since the virus description in the 80s [1], [2], the infection with hepatitis virus C (HVC) became a real health problem because 50-80% of acute HVC infections evolve to chronic hepatitis, from which 4–20% of patients develop liver cirrhosis within 20 years and, finally, the risk of developing HCC of patients with liver cirrhosis is 1–5% per year [3]. The principal stage of pathological processes is the interstitial space and mainly the portal areas. Fibrillary network, one of the important components of the interstitial space, undergoes dramatic and highly variable changes, fibrosis representing one of the elements of the morphologic triad of the pathologic conflict within the liver parenchyma.

Aims

The aim of the study is to assess quantitatively the liver fibrosis on biopsy fragments of patients with virus C hepatitis (VCH) and to compare it with the fibrosis score described qualitatively in METAVIR system.

Methods

The studied material was represented by liver biopsies from 87 patients with VCH. Tissue fragments were processed following classical histological techniques (formalin fixation and paraffin embedment) and serial sections were stained, for each case, with Hematoxylin Eosin and Mason Trichrome. Tissue fragments images were acquired with a dedicated optical system, using the X10 objective and the portal and periportal areas were acquired using X20 objective. The fibrosis was firstly assessed using METAVIR qualitative score for fibrosis (MV-F1, MV-F2 and MV-F3). There were no cases with no fibrosis or with cirrhosis. The quantitative parameters determined were: total area of examined hepatic parenchyma, portal spaces area, total area of fibrosis and area of portal fibrosis. The quantitative parameters calculated were: the percentage of the total parenchymal area represented by fibrosis (TF/TA-HP), the percentage of the parenchymal area represented by the portal spaces (PS-A/TA-HP), the percentage of the portal spaces represented by the portal fibrosis (PS-F/PS-A). The measurements were made with two dedicated software programs, after preceding software calibration. For numerical parameters minimum (MIN), maximum (MAX) mean (AV) values and standard deviation (STDEV) were calculated. For comparison with METAVIR fibrosis score grades, the values of quantitative parameters calculated were stratified in classes. For statistical analysis of the correlation between the quantitative and qualitative assessment of fibrosis, t-test (2-sample, unequal variance), One-Way ANOVA test and χ^2 test were used.

Results

TF/TA-HP and PS-A/TA-HP correlated with the METAVIR degrees of fibrosis <Figure 1>. Both correlations were statistically validated at very high significance level. <Figure 2>. In turn, PS-F/PS-A didn't correlate with the METAVIR degrees of fibrosis, as statistical tests revealed <Figures 1, 2>. So, in VCH, one of the main morphological aspects is the constant enlargement of portal spaces but with a reduced extension of the



destructive and reparatory processes towards the lobule center. Collagen fibers production is not an accelerated process, being in a relative equilibrium with the reactive inflammatory cellular population as demonstrated by the relatively constant percentage of the portal spaces represented by the fibrillary structures.

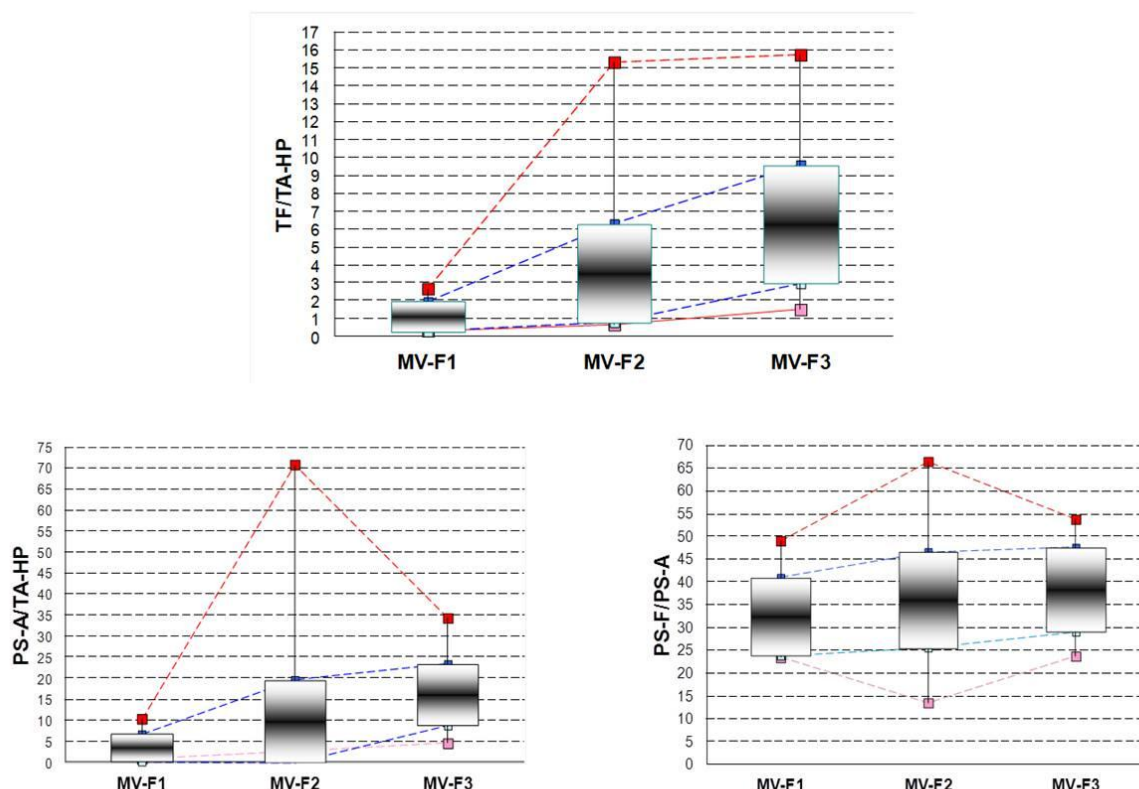


Figure 1.

Computed Parameter	METAVIR Score	Statistical test – P-value ($\alpha=0.05$)			
			t-test	ANOVA	χ^2 test
TF/TA-HP	MV-F1	<0.00001		<0.00001	<0.0001
	MV-F2		0.0009		
	MV-F3		0.00001		
PS-A/TA-HP	MV-F1	0.0005		<0.00028	<0.0001
	MV-F2		0.0026		
	MV-F3		0.00001		
PS-F/PS-A	MV-F1	0,237		0,265	0,364
	MV-F2		0,352		
	MV-F3		0,079		

Figure 2.

References:

- [1] Choo Q.L., Kuo G., Weiner A.J., Overby L.R., Bradley D.W., Houghton M., Isolation of a cDNA clone derived from a blood-borne non-A, non-B viral hepatitis genome, *Science* 1989, 244(4902):359-362.
- [2] Kuo G., Choo Q.L., Alter H.J., Gitnick G.L., Redeker A.G., Purcell R.H., Miyamura T., Dienstag J.L., Alter M.J., Stevens C.E., et al. An assay for circulating antibodies to a major etiologic virus of human non-A, non-B hepatitis, *Science* 1989, 244(4902):362-364.



[3] [*Brass V., Moradpour D., Blum H.E., Hepatitis C virus infection: In vivo and in vitro models, J Viral Hepat 2007, 14:64–67.*](#)