Assessment of Risk Factors of Hepatic Steatosis Diagnosed by Vibration Controlled Transient Elastography (VCTE) in Chronic Hepatitis C Virus infected Patients.

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INTRODUCTION

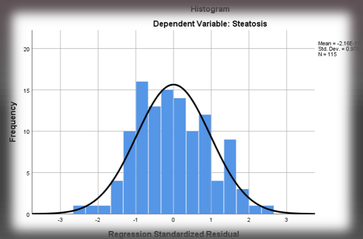
In chronic hepatitis C patients, the prevalence of steatosis ranges from 40% to 86%. Several hypotheses for the development of parenchymal steatosis in HCV infection have been proposed: host factors (alcohol consumption, obesity, diabetes mellitus (DM), hyperlipidemia, IL28 polymorphism) and viral factors (HCV genotype 3).

AIM

To evaluate the predictive factors of moderate/severe hepatic steatosis diagnosed by vibration controlled transient elastography (VCTE).

METHODOLOGY

This retrospective cross-sectional study included a cohort of 158 adult patients with suspected nonalcoholic fatty liver disease (NAFLD) evaluated in clinic. Patients with significant alcohol consumption, oral contraceptive use, hepatitis B, autoimmune hepatitis and primary biliary cirrhosis were excluded. Steatosis was categorized as S0-S1 (mild) and S2-S3 (moderate/severe) based on the controlled attenuation parameter (CAP) grade. Continuous variables were assessed using an unpaired t-test and categorical variables using Chi-Square with $p<0.05$ considered statistically significant. A multinominal logistic regression analysis was done to study the relationship between the CAP grade (dependent variable) and significant covariates (independent variables). The model was tested for goodness-of-fit and Pseudo R-square showed a Negelkerke value of 0.520.



Patient Characteristics	S0-S1(mild)	S2-S3(mode/severe)	Total	Chi-square	p-value
	Obesity				
Normal BMI: 18.5-24.9 kg/m²	20	5	25	36.369	0.0001*
Overweight 25-29.9 kg/m²	33	17	50		
Obese >30 kg/m²	12	49	61		
	Diabetic status				
DM	13	28	41	6.088	0.014*
Non-DM	52	43	95		
	HIV				
Absent	64	69	133	0.006	0.938
Present	1	2	3		
	Co-morbidities				
Hepatitis C	44	30	74	9.080	0.028*
Hepatitis C + DM	7	15	22		
DM	6	13	19		
No Hepatitis C or DM	8	13	21		

Likelihood Ratio of risk factors associated with moderate-severe hepatic steatosis by multivariate regression analysis

Patient characteristic	Model Fitting Criteria: -2 Log Likelihood of Reduced Model	Likelihood Ratio Tests: Chi-Square	df	Sig.
Intercept	113.377[a]	.000	0	.
Gender	118.582	5.206	3	.157
Obesity Class	165.619	52.242	12	.000
Hepatitis C	113.377[a]	.000	0	.
DM	114.202	.825	3	.843
Hepatitis C and DM	113.377[a]	.000	0	.

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model.
The reduced model is formed by omitting an effect from the final model.
The null hypothesis is that all parameters of that effect are 0.

This reduced model is equivalent to the final model because omitting the effect does not increase the degrees of freedom

RESULTS

Of the 158 patients, 136 patients met inclusion criteria. A moderate/severe steatosis score was associated with various risk factors: obesity ($p<0.05$), DM ($p<0.014$), metformin use ($p<0.0017$), fibrosis ($p<0.009$). A 4x2 chi-square table showed 40% patients with only hepatitis C, 68% with hepatitis C and DM, 68% with DM and 61% non-hepatitis C, non-DM patients (p-0.028) had moderate to severe hepatic steatosis. Regression analysis was used to predict the probabilities of the different possible outcomes on hepatic steatosis. The -2 log-likelihood of the reduced model in patients with hepatitis C and hepatitis C and DM was equivalent to the final model because omitting the effect did not increase the degrees of freedom (chi-Square value .000, df 0). Obesity had a significant association with steatosis (chi-square value 52, df 12). Interestingly, DM independently predicted a weak association with steatosis (Chi-square value 0.825, df 3).

CONCLUSIONS

This study highlights that hepatic steatosis is independently associated with metabolic parameters like obesity and DM. The chi-square analysis initially indicated that hepatitis C is associated with steatosis, but using multivariate analysis, we accounted for potential confounders, i.e., the most significant risk factor for steatosis in untreated hep C patients is their BMI. If the obesity class increased by 1 unit, the hepatic steatosis also increased by 50.8 units and vice versa. Thus, the management of obesity in patients with chronic hepatitis C may be necessary for reducing the risk of steatosis progression and improving their fibrosis score. It is unlikely that hepatitis C independently contributes to steatosis in non-genotype 3 patients. Larger studies may be needed to further evaluate this effect.