Relation Between Serum Eotaxin and Carotid Intima-Media Thickness in Obese Patients With Non Alcoholic Fatty Liver and Hepatitis C Virus Patients.

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Introduction

Chronic hepatitis C (CHC) is a leading cause of chronic hepatitis, cirrhosis, liver failure, and hepatocellular carcinoma world wide. Aim of the Work: Is to evaluate the relation between serum eotaxin and carotid intima-media thickness hn obese patients with nonalcoholic fatty liver and hepatitis c virus patients. Patient and Methods: This prospective study was carried out in department of internal medicine, Minia university hospital, 80 persons were studied. The study protocol was approved by the institutional ethics committee of school of medicine, minia university. Results: The study included 80 subjects subdivided into: Twenty obese patients considered as group-1. Twenty patients with chronic hepatitis C virus (without treatment by combined interferon and ribayirin); considered as group-2. Twenty patients with chronic hepatitis C virus who was treated by combined interferon and ribavirin and had-ve per for HCV RNA; considered as group-3. **Discussion:** Hepatitis C virus (Hcv) is a leading cause of chronic hepatitis, cirrhosis, liver failure, and hepatocellular carcinoma worldwide. Conclusion & Recommendation: our study suggests that serum eotaxin level could be a good predictor for NAFLD; and response for anti-HCV .treatment. Also, our study recommends further studying of serum eotaxin levels and other chemokines, to clarify their role in CHC and NAFLD.

Keywords: alanine aminotransferase, bovine serum albumin, intima media thickness

Introduction

Chronic hepatitis C(CHC) IS A LEADING CAUSE OF CHRONIC hepatitis, liver failure, and hepatocellular carcinoma worldwide. CHC is a serious global medical problem necessitating effective treatment. Finding effective predictive markers of HCV

Virological response is one of the major challenges to increasing the effectiveness of HCV antiviral therapy.

Chemokines play a role in regulating leucocyte and trafficking, acting primarily as chemoattractants and activators of specific types of leucocyte such as T cells (memory and activated T lymphocytes), which infiltrate the liver during chronic HCV infection. furthermore, chemokines may also be involved in liver regeneration, fibrosis and malignant transformation, which can be induced by the persistence of inflammation.

Nonalcoholic fatty liver disease (NAFLD) is emerging as the most common chronic liver condition in the western world . it is associated with insulin resistance and frequently occurs with features of the metabolic syndrome. Disease presentation ranges form asymptomatic elevated liver enzyme levels to cirrhosis with complications of liver failure and hepatocellular carcinoma. NAFLD and insulin resistance are interrelated in a complex fashion and may be synergistic to some degree. cooccurrence of NAFLD with hepatitis C or HIV worsens their prognosis. NAFLD is reported to be an independent risk factor for cardiovascular disease. This may reflect similar risk factors such as dyslipidemia or immune dvsregulation NAFLD is associated with increased all-cause mortality and increased liver-related mortality.

Atherosclerosis (AS) is a major risk factor for cardiovascular diseases (CVD), INFLAMMATION plays a prominent role in AS. The traditional view of atherosclerosis as a lipid storage disease crumbles in the face of extensive and growing evidence that inflammation participates centrally in all stages of this disease, from the initial lesion to the end-stage thrombotic complications.

Chemokines have been defined as small cytokines involved in the migration and activation of cells, such as lymphocytes and phagocytic cells, and playing a central role in inflammation eotaxin plays a central role in eosinophil trafficking and is mediated by the CCR-3 receptor, which is expressed on eosinophils, basophils, the helper, and T-cells.

Aim of the work

Is to evaluate the relation between serum eotaxin and carotid intima-media thicknees in obese patients with nonalcoholic fatty liver and hepatitis c virus patients.

Patient and methods

This prospective study was carried out in dwparment of internal medicine, mini university hospital, 80 persons were studied. The study protocol was approved by the institutional ethics committee of school of medicine minia university, Egypt all persons gave informed consent to participate in this study. The study was conducted in accordance with the echical guidelines of the 1975 declaration of Helsinki and international conference on harmonization guidelines for good clinical practice. design of the study included 80 subjects subdivided into:

Twenty obese patients considered as group-1.

Twenty patients with chronic hepatitis C virus (without treatment by combined

interferon and ribavirin); considered as group-2.

Twenty patients with chronic hepatitis C virus who was treated by combined interferon and ribavirin and had-ve PCR for HCVRNA; considered as group-3.

Twenty subject as control group; considered as group-4.

Inclusion criteria for diagnosis: NAFLD

Obesity was established on the basis of BMI cut-off point equal or above 25.

Obese patients were diagnosed as having NAFLD if they have hyper-echogenicity, (the so-called "bright liver" in relation to renal cortex and echogenicity of the spleen). The hyperechogenicity is graded from 0-3 (no fatty infiltration "grade 0" ranges till diffuse bright echogenic liver with no fat spared areas "grade3". A reliable sign detects moderate to severe fatty (Erickson 2009).

Criteria for diagnosis of chronic hepatitis c (chc) Eligible patients were previously untreated adults 20 years of age or older who had CHC based on the presence of anti-HCV and detectable serum HCV RNA for 6 months or more.

Results

The study included 80 subjects subdivided into:

Twenty obese patients considered as group-1. Twenty patients with chronic hepatitis C virus (without treatment by combined interferon and ribavirin); considered as group-2.

Twenty patients with chronic hepatitis C virus who was treated by combined interferon and ribavirin and had- ve PCR for HCV RNA; considered as group-3.

Twenty subjects as control group; considered as group -4.

Fatty liver						
group	Ivs II	Ivs III	Ivs IV	IIvs III	IIvs IV	IIIvs IV
P value	0.046*	0.005*	<0.001*	0.757	0.001*	0.004*
Carotid IMT						
group	Ivs II	Ivs III	Ivs IV	IIvs III	IIvs IV	IIIvs IV
P value	1	0.950	0.505	0.973	0.439	0.222

Table: Correlation between fatty liver infiltration in different groups:

Discussion

Hepatitis Cvirus (HCV) IS A LEADING CAUSE of chronic hepatitis, cirrhosis, liver failure, and hepatocellular carcinoma worldwide. CHC is a serious global medical problem necessitateing effective treatment. Over time, 20% will develop cirrhosis and its related complications, (kaser et al.2005).

Non –alcoholic fatty liver disease (NAFLD) is a condition defined by excessive fat accumulation in the form of triglycerides. NAFLD is a complex disease with many interacting metabolic pathways that appear to be regulated by the interplay of genetic predisposition and environmental factors, in the majority of patients, NAFLD is associated with metabolic risk factos such as obesity, diabetes mellitus, and dyslipidemia (lazo et al., 2008).

Chemokines play a role in regulating leucocyte recruitment and trafficking acting primarily as chemoattactants and activators of specific types of leucocyte such as T cells (memory and activated T lymphocytes). Eotaxin is a chemokine that is selectively attract eosinophils by activeting CCR3 receptors .it has been linked to various human disorders such as allergic asthma, eosinophil-associated gastrointestinal diseases, pulmonary fibrosis and atherosclerosis. (umemura and kiyosawa, 2007).

Death receptor- mediated apoptosis is critical for HCV and NAFLD – associated liver injury (davis et al., 2008).

Our study aims to use serum eotaxin levels as a simple noninvasive method to predict patients with CHC (pre and post treatment) and NAFLD.

We used 80 people usb divided into four groups; twenty obese patients, the degree of obesity was established on the basis of BMI cut-off point equal or above 30 "group I". Twenty patients with chronic hepatitis C virus (without treatment by combined interferon and ribavirin) "group II".

Conclusion & Recommendation

our study suggests that serum eotaxin leve could be a good predictor for NAFLD, and response for anti-hcv treatment. also, our study recommends further studying of serum eotaxin levels and other chemokines, to clarify their role in CHC and NAFLD.

References

- 1. Andrieux-meyer I, cohn J, de araujo ES, hamid SS.disparity in market prices for hepatitis C virus directacting drugs. Lancent glob health. 2015:3(11):676-677.
- 2. Angula p. noaalcoholic fatty liver disease. N engl Med 200;346:1221-31.
- 3. Angulo, P; J. M. HUI, G .marchesini, Ebigianesi, J. George, G. C Farrell, F. enders, S. Saksesa, A. D. burt, J. P. Bida, et al., the NAFLD fibrosis score: a noninvasive system that identifies liver fibrosismin patients with NAFLD. hepatology.2007;45:846-854.
- 4. Askarieh G, Alsio A, Pugnale P, et al., systemic and intrahepatic interferongamma-inducible protein 10 kDa predicts the first-phase decline in hepatitis Cvirus RNA and overall viral response to therapy in chronic hepatitis C . hepatology 2010; 51:1523-1530.
- 5. Aygun, C., O kocaman, T. Sahin, uraz, AT. Eminler, A. celebi, O.. Senturk, and S hulager. Evaluation of metabolic syndrome frequency and carotid artery

- intima-media thickness as risk factors for atherosclerosis in patients with nonalcoholic fatty liver disease. Dig. dis. sci. 2008; 53: 1352-1357.
- 6. Bason k, baggiolini M, broxmeyer H, horuk R, lindley I, mantovani Amatsushima K, murphy P,nomiyama H, Oppenheim J, et al.., Chemokine/ Chemokine receptor nomenclature. J leukoc boil 2001;70:465-466.
- 7. Benova L mohamoud Y A, calvert C, abu-raddad L J. Vertical transmission of hepatitis C virus: systematic review and meta-analysis . clin infect dis. 2014;59(6):765-773.
- 8. Bisset LR, schmid- grendelmeier P. chemokines and their receptors in the pathogenesis in the pathogenesis of allergic asthma: progress and perspective. Curr. Opin. Pulm. med. 2005; 11: 35-42.
- 9. Black S pak I, ingravallo P McM onagle P, chase R, Shaughnessy M, et

- al., editors. resistance analysis of virologic failures in hepatitis C genotype 1-infected patients treated with grazoprevir +elbasvir+_ ribavirin: the C- Worthy study. In: 2015 international liver congress: 50th annual meeting of the European association for the study of the liver (EASL). Vienna, 22-26 april 2015.
- 10. Bonora E targher G, alberiche M, et al., homeostasis model assessment closely mirros the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various degress of glucose to lerance and insulin sensitivity. diabetes care 2000; 23:57-63.
- 11. Browning J, szczepaniak l, dobbins R, Nuremberg P,Horton J, cohen J, et al., prevalence of hepatic steatosis in an urban population in the united states: impact of ethnicity. Hepatology 2004; 40:1387-95.