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STUDY THE ROLE OF HEPATITIS C VIRUS INFECTION IN PATIENTS WITH CEREBRAL INFARCTION

By

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ABSTRACT

Background: Chronic hepatitis C virus (HCV) infection has been linked to numerous co-existing conditions including metabolic abnormalities and cardiovascular disease. Stroke is a major public health crisis world-wide. About 87% of strokes are ischemic. Between 25- 40% of ischemic stroke may be cryptogenic. HCV infection is associated with a wide spectrum of extra-hepatic manifestations, affecting different organ systems. Neurological complications occur in a large number of patients and range from peripheral neuropathy to cognitive impairment and/or cerebral infarctions (CI).

Objective: Studying the role of HCV infection in patients having CI.

Patients and Methods: After departmental ethics committee approval and patient consents were obtained, 60patients included in this work: 20 patients having CI and chronic hepatitis C (CHC), 20 patients having CI without CHC, and 20 patients having CHC without CI. All patients were subjected to history taking and clinical examination, complete blood count (CBC), evaluation of HCV antibodies, polymerase chain reaction (PCR) for HCV ribonucleic acid (RNA), hepatitis B surface antigen (HBs-Ag), liver function tests, renal function tests, lipid profile, antinuclear antibody (ANA), anticardiolipin antibodies immunoglobulin G (ACL-IgG), antineutrophil cytoplasmic antibodies (ANCA), cryoglobulin, fasting plasma glucose (FPG) and post-prandial plasma glucose (PPPG). In addition, ultrasound (U/S) abdomen, magnetic resonance imaging (MRI) brain, doppler study of the carotid arteries and echocardiography**.**

Results: Group A (CI and CHC) patients were significantly younger and females were predominant in these patients than those of either group B (CI without CHC) or C (CHC without CI). Group B patients were significantly overweighed compared to group A or C. Total cholesterol (TC), triglyceride (TG), FPG and low density lipoprotein (LDL) significantly decreased in group A and C compared with group B. Patients of group A showed significantly higher positive of HCV viremia, cryoglobulins, ANA, c-ANCA and ACL-IgG, compared with group C, while those of group B were negative for same parameters. Carotid atherosclerosis and ischemic heart disease (IHD) were significantly higher in patients of group A, compared with group (B and C), respectively.

Conclusion: Chronic HCV infection is a risk factor in development of CI by multiple mechanisms including atherosclerosis, thromboembolism and vasculitis. Also, it increases stroke risk and might be considered as an important and independent risk factor.

Keywords: Hepatitis C virus, ANA, c-ANCA.

INTRODUCTION

HCV infection is a common and chronic disorder with numerous extra-hepatic manifestations **(Acharya and Pacheco, 2008).** HCV infection has been found to be strikingly associated with autoimmune phenomenon with autoantibodies commonly seen **(Cojocaru et al., 2007).** HCV infection is a condition that has increased the risk of stroke **(Cojocaru et al., 2005).**

People with HCV infection appear to have a higher likelihood of dying from strokes than non-infected individuals**.** Ischemic stroke can occur when blood clots triggered by atherosclerosis especially build-up of plaque in the carotid arteries that supply the brain, break off and lodge in small blood vessels **(Lee et al., 2010).**

Neurological complications of HCV occur in a large number of patients and range from peripheral neuropathy to cognitive impairment and/or cerebral infarctions (CI). Pathogenetic mechanisms responsible for nervous system dysfunction are mainly related to production of autoantibodies**,** immune complexes, cryoglobulins**,** extra-hepatic replication of HCV in neural tissues and the effects of circulating inflammatory cytokines and chemokines **(Salvatore et al., 2012).**

PATIENTS AND METHODS

Patients:

Sixty patients were included in this study. They were divided into 3 equal groups:

- Group (A) having CI and CHC,
- Group (B) having CI without CHC,
- Group (C) having CHC without CI.

Patients were presented to Emergency Department of Sayed Galal Hospital, Al-Azhar University, and the outpatient clinic, and admitted to the Internal Medicine Department.

The study was carried out during the period from March 2013-June2016.

Exclusion criteria:

- Smokers or alcoholics or had any other special habits of significant importance.
- Those receiving IFN therapy.
- Diabetics, hypertensives or hyperlipidemics.
- Those having renal impairment, endocrine diseases, cardiac problems or collagen diseases.
- Those having history of contraceptive pills intake.
- Those having any other liver disease.

Methods:

- Thorough history taking and full clinical examination were done for all patients.
- Laboratory investigations for selected patients included assessment of HCV-Ab and HBs-Ag by using 3rdgeneration ELISA technique, detection of HCV-RNA by PCR (Cobas amplicor analyzer), viral load in international unit (IU) and degree of viremia, liver function tests evaluation including alanine transaminase (ALT), aspartate transaminase (AST), serum albumin, serum bilirubin, prothrombin time (PT), and international normalized ratio (INR) using (Hitachi, 911automatic analyzer), renal function tests assessment including blood urea and serum creatinine using (Hitachi, 911automatic analyzer), CBC including

hemoglobin(Hb), white blood cells (WBCs) and platelets (PLT), lipid profile assessment including TC, TG, LDL and high density lipo-protein (HDL), assessment of ANA, ANCA (anti-proteinase-3 antibody), ACL-IgG and cryoglobulinemia evaluation by simple blood test **(Ferri, 2008).**

- Abdominal ultrasonography to assess the chronic liver diseases and its complications.
- Echocardiography to detect criteria suggestive of ischemic heart diseases.
- Doppler study of the carotid system to assess carotid intima media thickening (C-IMT).
- MRI brain to diagnose cerebral infarction (CI).
- Informed consents were taken from all patients.

Statistical analysis of data by IBM computer using statistical program for social science (SPSS) version 20: Chi-square test to compare qualitative variables between groups, unpaired (Independent) t-test to compare quantitative variables between groups, and one way analysis of variance (ANOVA) followed by post hoc analysis (LSD test) to compare between more than two groups regarding quantitative data with parametric distribution.

RESULTS

Group (A) patients were significantly younger than those of groups (B) and (C). Females were predominant in group (A) patients, while males were predominant in group (B) patients, and both sexes were equally distributed in group (C) patients. Group (B) patients were significantly overweighed compared with either that of groups (A) or (C) (Table 1).

Table (1): Age, sex and BMI among the studied groups.

Groups / Parameters		Group A (CI+ CHC) No = 20	Group B (CI without CHC) No = 20	Group C (CHC without CI) No = 20	One way ANOVA F/X²*	P
Age (years)	M± SD	50.6 ± 3.65	59.4 ± 6.34	55.3 ± 4.41	15.951	0.000*
	Range	44 – 58	43 - 70	48 – 65		
Sex	Female	15 (75%)	6 (30%)	10 (50%)	8.142	0.017*
	Male	5 (25%)	14 (70%)	10 (50%)		
BMI (kg/m^2)	M ± SD	23.28 ± 3.86	27.95 ± 3.63	24.80 ± 5.38	8.222	0.001*
	Range	16 - 32	23 – 32	17 – 34		

Total cholesterol, TG, FPG and LDL significantly decreased in groups (A) and (C) compared with group (B). There was an insignificant difference between the 3 groups as regards 2-hr PPPG (Table 2).

Table (2): Results of FPG, 2hrs-PPPG, TC, TG, LDL & HDL in the 3 groups.

Groups / Parameters		Group (A)	Group (B)	Group (C)	One way ANOVA		Post hoc analysis		
		No= 20	No= 20	No= 20	F	P	P1	P2	P3
FPG (mg/d)	M±SD	92.55 ±21.28	106.40 ±15.60	92.45 ±20.59	3.450	0.039	0.043	0.214	0.002
	Range	60 - 134	60 - 134	80 - 145					
PPPG (mg/d)	M±SD	143.75 ±20.64	150.70 ±30.60	131.75 ±24.09	2.838	0.067	0.073	0.064	0.003
	Range	110 - 200	110 – 200	110 - 210					
TC (mg/d)	M±SD	162.65±21 .40	210.85 ±57.10	131.25 ±32.22	20.277	0.000	0.001	0.001	0.000
	Range	113- 200	113- 200	118 - 352					
TG (mg/d)	M±SD	104.55 ±25.81	163.50 ±52.79	88.20 ± 31.00	21.330	0.000	0.000	0.078	0.000
	Range	67 - 145	67 - 145	89 - 320					
LDL (mg/d)	M±SD	79.25 ± 19.08	105.00 ±26.21	70.75 ± 22.65	12.203	0.000	0.001	0.207	0.000
	Range	45 - 110	45 - 110	65 - 155					
HDL (mg/d)	M±SD	57.30 ± 17.35	49.90 ± 16.78	58.90 ± 17.79	1.539	0.223	0.178	0.775	0.108
	Range	29 - 100	29 - 100	20 – 87					

HCV-viremia was significantly higher in group (A) compared with group (C) (Table 3).

Table (3): Comparison between groups (A) and (C) as regards HCV-viremia.

HCV-RNA	Group (A)	Group (C)	P
	No = 20	No = 20	
Mean ± SD	2446152.25 ± 2338691.00	365870.45 ± 289148.80	0.000
Range	150321 - 9805500	12069.00 - 930100.00	

Cryoglobulinemia significantly increased in group A (50%), compared to either group C (10%) or group B (0%). Patients of group (A) showed significantly higher positive ANA (55%), c-ANCA (65%) and ACL-IgG(30%), compared with those of group C (20%), (15%), (5%) respectively, while those of group (B) were negative for these parameters (Table 4).

Table (4): Cryoglobulins, ANA, c-ANCA and ACL-IgG in the studied groups.

Parameters \ Groups		Group (A)		Group (B)		Group (C)		Chi-Square tests	
		No	%	No	%	No	%	X^2	P-Value
Cryoglobulin	Negative	10	50%	20	100%	18	90%	17.500	0.000*
	Positive	10	50%	0	0%	2	10%		
ANA	Negative	9	45%	20	100%	16	80%	17.883	0.007
	Positive	11	55%	0	0%	4	20%		
c-ANCA	Negative	7	35%	20	100%	17	85%	23.693	0.000*
	Positive	13	65%	0	0%	3	15%		
ACL-IgG	Negative	14	70%	20	100%	19	95%	11.170	0.043*
	Positive	6	30%	0	0%	1	5%		

Carotid atherosclerosis and IHD were significantly higher in group A (70%, 45%), compared with group B (30%, 20%) and group C (10%, 10%), respectively (Table 5).

Table (5): Carotid Doppler and Echo finding among the studied groups**.**

Parameters \ Groups		Group A	Group B	Group C	One way ANOVA	
		No = 20	No = 20	No =20	F/X^2	P
Carotid IMT	Negative	6 (30%)	14 (70%)	18(90)	21.073	0.000*
	Mild	7 (35%)	6 (30%)	1 (5%)		
	Moderate	7 (35%)	0 (0%)	1 (5%)		
IHD	Negative	11 (55%)	16 (80%)	18(90)	6.933	0.031*
	Positive	9 (45%)	4 (20%)	2(10%)		

DISCUSSION

The obtained results in the current study showed that the female predominance was evident in group A compared with groups B and group C. This difference was highly significant between the 3 groups. Also, the mean age of group (A) was younger than groups (B and C), this difference was highly significant between the 3 groups. So, the CI associated with HCV was more in females particularly the younger women.

This result was not in agreement with **Chen and Morgan (2006)** who found that male predominance was evident in the infected group compared with other groups, and the rate of chronicity in HCV infection appears to be lower in women particularly younger women. The reason of the difference was because in our study we discussed HCV with CI, while previous study discussed the chronicity of HCV only. So, we found that HCV infection with CI more in females suggested theory of vasculitis which was more common in females as a contributing factor in occurrence of CI in these patients. Moreover, stroke occurred at a younger age in HCV-infected patients as well as the negative prognostic impact of HCV-RNA serum levels on outcome, as opposed to a more limited role for gender and classic predisposing conditions **(Zampino et al., 2013 and Adinolfi et al., 2014).**

In this study, non-infected HCV group (B) was overweighed more than infected HCV groups (A and C), with a significant difference between the 3 groups regarding BMI. On the other hand, **Aks?z et al. (2008)** found that BMI is not statistically different between patients with HCV infection and healthy subjects. So, increased BMI is not a risk factor for development of cerebral infarctions in CHC patients.

In this study, there was a highly significant difference between the 3 groups regarding TC, TG, LDL and FPG. Infected HCV groups (A and C) have low lipid profile, while infected HCV group (B) has a higher level. These findings were in agreement with those obtained by **Dai et al. (2008)** who found that TC and TG levels were significantly lower in anti-HCV positive patients with high HCV-RNA than in anti-HCV positive patients with HCV-RNA negative. This could be explained by the binding of HCV particles to HDL, LDL and VLDL, the impaired hepatocyte assembly of VLDL and the entry of HCV into hepatocytes through the LDL receptor. Thus, chronic HCV can impair the synthesis of TC and TG **(Andre´ et al., 2002 and Perlemuter et al., 2002).**

In this study, HCV-RNA levels were higher (high viremia) in group A compared with group (C) with a significant difference. An elevation of serum HCV-RNA level was associated with an increased risk of cerebrovascular death, suggesting that individuals with an active HCV infection may trigger a stronger inflammation response by host-virus interaction, leading to atherothrombosis **(Werner et al., 2007).** Also, localization of HCV-RNA in human carotid plaques provides strong evidence for an association between HCV infection and atherosclerosis **(Boddi et al., 2010).**

In the current study, the immunological markers, ANA, c-ANCA and ACL-IgG in all the studied groups revealed high

positivity in infected HCV group (A) with CI, 55%, 65% and 30% respectively, while in infected HCV group (C) without CI, were 20%, 15% and 5% and non-infected group (B), were negative. This result was highly significant which suggested theory of vasculitis in developing of cerebral infarctions in HCV patients.

This result was in agreement with **Cojocaru et al. (2007)** who reported that c-ANCA were positive in 58 % patients with ischemic stroke and HCV infection. All sera with ANCA showed c-ANCA patterns and contained anti-PR3 specificity. HCV patients with ANCA showed a higher prevalence of cerebral vasculitis**.**

In addition**, Cojocaru et al. (2005)** reported that the mean levels of serum IgG-aCL antibodies were significantly higher among the HCV patients with acute ischemic stroke than those with acute ischemic stroke without HCV. So, c-ANCA and anticardiolipin antibodies associated with HCV may be an important marker for acute ischemic stroke.

In this study, cryoglobulinemia was demonstrated in 10 patients of group (A), in 2 patients of group (C), and no patients of group (B). This difference was highly significant. High level of cryoglobulinemia in group (A) suggested the role of cryoglobulins inducing CNS vasculitis. This result was supported by the result of **Giordano et al. (2007)** who reported that HCV infection is associated with an increased risk of cryoglobulinemia**,** which is thought to participate in the formation of immune complexes precipitating in vessel walls then leads to vasculitis**.**

Occlusive cerebral vascular diseases can also occur in the context of HCV-related vasculitis such as mixed cryoglobulinemia, antiphospholipid syndrome and ANCA-associated vasculitis **(Castro et al., 2014).** In addition**, Gulliaet al. (2016)** reported higher percentage of ANA was found in CGs HCV-patients, suggesting that the production of ANA seems strictly related to CGs production.

C-IMT in the current study showed a highly significant difference between the 3 groups, group (A) had carotid atherosclerosis (70%) comparing with group (B) (30%), while group C had 10% regardless the gender. These results were in agreement with those reported by **Aslam et al. (2010) and Boddi et al. (2010)** who found positive correlations between HCV infection and carotid IMT, plaque and stroke.

In this study, the stroke groups (A and B), HCV positive patients showed a higher prevalence of IHD, while group (C) non-stroke has only 10%. This result was in agreement with that seen by **Forde et al. (2012)** who reported a higher prevalence of past ischemic cardiovascular events in HCV positive subjects. However, such previous cardiovascular event was not associated with an increased risk of cerebral ischemic stroke in studied population who HCV negative subjects**.**

In patients having CI and HCV (group A), there were high HC viremia, increased vasculitis markers as cryoglobulinemia (70%), ANA (55%), c-ANCA (65%) and ACL-IgG (30%). These were in addition to more IHD affection (45%) and carotid atherosclerosis as indicated by increased IMT (70%). These results supported the suggestion that HCV infection may cause

CI through multiple mechanisms including vasculitis and development of early atherosclerosis as an extra-hepatic manifestations.

CONCLUSION

Chronic HCV infection is a risk factor in the development of CI by multiple mechanisms including atherosclerosis, thromboembolic and vasculitis. Also, it increases stroke risk and might be considered as an important and independent risk factor**.**

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دراسة دور الإصابة بالفيروس الكبدى (سى) فى مرضى الجلطة الدماغية

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خلفية البحث : يصاحب الإلتهاب الكبدى المزمن " سى" بالعديد من الأمراض منها التمثيل الغذائى الغير طبيعى وأمراض الأوعية الدموية والقلب . وتعتبر الجلطات الدماغيه من أكثر الأمراض شيوعاً فى أنحاء العالم . ومعظم الجلطات الدماغية تحدث بسبب قطع الإمداد الدموى للمخ فى 87% من الحالات غير أن حوالي 25% - 40% من حالات الجلطات المخية غير معروفة السبب . وتؤدى العدوى بالإلتهاب الكبدى المزمن " سى" إلى أعراض خارج الكبد تؤثر على أعضاء أخرى بالجسم وتحدث مضاعفات مختلفة للجهاز العصبى لكثير من مرضى فيروس" سى" بداية من إلتهاب الاعصاب الطرفية والإضطرابات النفسية إلى حدوث جلطات بالمخ.

الهدف من البحث : دراسة دور الإصابة بفيروس الكبد "سى" المزمنة فى حدوث الجلطة المخية.

المرضى وطرق البحث: أجريت هذه الدراسة على 60 مريضاً بعد أخذ الموافقة منهم وتم تقسيمهم إلي ثلاث مجموعات متساوية : مجموعه (أ) مصابون بالإلتهاب الكبدى الفيروسى المزمن " سى" والجلطات الدماغية، ومجموعه (ب) مصابون بجلطات دماغية وغير مصابون بالإلتهاب الكبدى الفيروسى المزمن " سى"، ومجموعه (ج) مصابون بالإلتهاب الكبدى الفيروسى المزمن " سى" وغير مصابون بجلطات دماغية.

وقد تم عمل الآتى لكل المرضى : أخذ التاريخ المرضى الكامل وفحص اكلينيكى شامل وعمل تحاليل (صورة دم كاملة وقياس الأجسام المضادة لفيروس "سى" وعمل (PCR) الكمى وقياس دلائل الإصابة بفيروس الكبد المزمن "بي" ، ووظائف الكبد و الكلى ، وقياس نسبة كثافة الدهون بالدم ، وقياس مستوى السكر الصائم وبعد الأكل بساعتين ، وقياس نسبة الكريوجلوبيولين بالدم ، وقياس دلائل مناعية (c-ANCA, ACL-IgG , ANA) ، وعمل موجات صوتية على البطن ودوبلرملون على الشرايين الثباتية بالرقبة ورنين مغناطيسى على المخ وموجات صوتية على القلب.

النتائج :

- مرضى المجموعة (أ) اصغر عمراً ومعظمهم إناث مقارنة بمرضى المجموعتين (ب) و(ج) ، كما وجد زيادة ذات دلالة إحصائية فى متوسط وزن مرضى المجموعة (ب) مقارنة بالمجموعتين (أ) و(ج).
- وجود نقص ذو دلالة إحصائية فى الكوليسترول والدهون الثلاثية والسكر الصائم والدهون قليلة الكثافة بالدم لدى مرضى المجموعتين (أ) و(ج) مقارنة بمرضى المجموعة (ب).
- وجود زيادة ذات دلالة إحصائية فى نسبة الفيروس الكمي والكريوجلوبيولين والدلالات المناعية في الدم لدى مرضى المجموعة (أ) مقارنة بمرضى المجموعة (ج) ، بينما مرضى المجموعة (ب) سلبية لهذه الدلالات.
- وجود زيادة ذات دلالة إحصائية لتصلب الشرايين الثباتية و قصور الشرايين التاجى للقلب لدى مرضى المجموعة (أ) مقارنة بمرضى المجموعة (ب) و (ج).

الاستنتاج : تعتبر الإصابة بفيروس الكبد المزمن "سى" عامل خطورة منفصل لحدوث الجلطات الدماغية سواءً عن طريق تصلب الشرايين أو تكوين جلطة أو سدادة دموية أو عن طريق إلتهاب الأوعية الدموية المخية المناعى ، وكذلك تزيد من خطورة حدوث الجلطات الدماغية .