ORIGINAL ARTICLE

# Relationship between Chronic Hepatitis C Infection and Cardiovascular Calcification in Hemodialysis Patients

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Abstract

*Introduction:* ***Chronic Hepatitis C virus (HCV) infection is frequent in hemodialysis (HD) patients. Cardiovascular complications, including atherosclerosis and vascular calcification, are common in HD patients, especially in HCV-seropositive patients.***

*Aim:* ***To study the relationship between chronic hepatitis C infections and cardiovascular calcification in HD patients.***

*Patients and Methods* ***This cross-sectional study involved 70 HD patients from Al-Santa Hospital and the HD unit of Al-Zahraa University Hospital, divided into two groups: 35 HCV-seropositive and 35 HCV-seronegative patients, all on regular HD for more than six months. All patients were subjected to full Laboratory investigations and Radiological investigations, including Carotid duplex U/S and Echocardiography.***

*Results:* ***HCV seropositive hemodialysis patients exhibited a significantly higher frequency of cardiac calcification (37.1% vs. 11.4%, $p<0.05$) compared to HCV seronegative patients, while no significant difference was detected in EF m-mode ($p>0.05$). Additionally, there was a notable rise in CIMT and atheromatous plaque formation, along with a higher frequency of atheromatous plaque calcification in HCV seropositive patients ($p<0.05$). Right-sided atheromatous plaque formation was more common in both groups (34.3% vs. 11.4%), but no significant association was found between plaque site and PCR or Bone isomer ALP levels.***

*Conclusion:* ***Chronic HCV infection in HD patients significantly elevated the possibility of atherosclerosis and cardiovascular calcification, highlighting the importance of monitoring HCV PCR and bone isomer ALP as predictive markers.***

*Keywords:* Hepatitis C; Hemodialysis; Atherosclerosis; Cardiovascular Calcification

## 1. Introduction

Chronic kidney disease (CKD) is a crucial public health issue. The occurrence of chronic kidney disease in the general populations of different nations is ten to twelve percent, and this proportion considerably rises with age .[1, 2]

HD is a line to manage advanced renal failure that can help carry on an active lifestyle despite failing kidneys. In HD, a machine removes waste, salts, and fluids from the blood when the kidneys are no longer healthy enough to do this work effectively .[3, 4]

Vascular Calcification (VC) is a common complication of HD, with prolonged duration on HD correlated with increased severity of calcification .[5] Cardiovascular disease (CVD) is the primary etiology of early mortality in the setting of chronic kidney disease .[6] Cardiovascular events may result from an imbalance among mineralization promoters and inhibitors, leading to VC .[7]

Disturbances in the metabolism of minerals, including hypercalcemia and hyperphosphatemia, seem to contribute to progressive calcification. Certain calcium-regulatory proteins can act systemically or locally as inhibitors of calcification. Dysregulations of calcification inhibitors, such as osteoprotegerin, fetuin-A, matrix Gla protein (MGP), and pyrophosphates, can be pathophysiologically significant contributors to uremic extraosseous calcification among HD cases .[8]

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Infection with HCV often persists among patients undergoing long-term dialysis in both developed and less-developed countries .[9]

Chronic inflammation, immune dysregulation, and oxidative stress associated with HCV infection may contribute to the development of a procalcific setting in the vascular endothelium. Additionally, HCV may directly influence the mineral metabolism and VC process through viral proteins and interactions with cellular signaling pathways .[10]

This study aimed to study the relationship between chronic hepatitis C infections and cardiovascular calcification in HD patients.

## 2. Patients and methods

This cross-sectional descriptive research was performed on 70 HD cases and was collected from the HD unit of Al-Santa hospital, Gharbia Governorate, and the HD unit of Al-Zahraa University Hospital after their approval. Patients have been divided into two groups: Group I: Includes 35 HCV seropositive cases on regular HD for more than six months, and Group II: Includes 35 HCV seronegative cases on regular HD for more than six months.

Ethical statement: The Research Ethics Committee of Al-Azhar University, Cairo, Egypt, examined and approved the study protocol before progressing in the study. Informed consent has been attained from all cases prior to enrollment in this research. All data has been kept confidential. Participants had the right to withdraw from the research at any time without reason and will receive the usual management.

Inclusion criteria: Patients >18 years old of both sexes, CKD patients on regular HD for more than 6 months, including diabetes mellitus (DM) and hypertension (HTN) patients, and both HCV seropositive and seronegative.

Exclusion criteria: Patient's positive hepatitis other than HCV, malignancy, autoimmune diseases, active or chronic infection.

Methods

All cases have been exposed to the following:

Full medical history including: The history includes patient age, sex, duration of HD, comorbidities such as DM and HTN, and symptoms indicating other system involvement. A full clinical examination is conducted to assess overall health, including blood pressure (BP), Body mass index (BMI), hepatic function using the Child-Pugh classification, and signs of systemic impact.

Laboratory investigations include complete blood count (CBC), CRP titer, liver function tests (ALT, AST, bilirubin, serum albumin, prothrombin time (PT), prothrombin concentration (PC), and international normalized ratio (INR)), PCR for HCV, lipid profile (cholesterol, triglycerides (TG), HDL, LDL), renal function tests (blood urea, serum creatinine), intact parathormone (iPTH), and serum levels of phosphorus (P), calcium (Ca), potassium (K), uric acid (UA), sodium (Na), in addition to ALP and bone isomer ALP.

Biochemical parameters, including serum levels of Ca, P, albumin, serum creatinine, blood urea, cholesterol, LDL, HDL, TG, AST, ALT, Na, and K, have been determined utilizing an autoanalyzer (Olympus AU 800; Olympus Diagnostica GmbH, Hamburg, Germany). Parathyroid hormone (PTH) concentration has been measured using chemiluminescent immunoassay (Liaison Ntact; DiaSorin Inc, Stillwater, MN).

Imaging studies

Carotid duplex ultrasound was performed to evaluate intima-media thickness, the presence of atheromatous plaques, and calcification. Echocardiography was used to assess valvular calcification and myocardial function.

Statistical Methods

Data have been gathered, tabulated, and then analyzed utilizing IBM© SPSS© Statistics version 22 (IBM© Corp., Armonk, NY). Data that was normally distributed has been expressed as the mean and SD, whereas data that was skewed has been expressed as the median and the interquartile range. Qualitative data has been expressed as percentages and numbers. The unpaired Student's t-test has been applied to compare normally distributed numerical data. The Mann-Whitney U test has been utilized to compare skewed data. Categorical data were analyzed utilizing the chi-squared test or Fisher's exact test, as appropriate. A two-sided p-value of less than 0.05 has been deemed statistically significant.

## 3. Results

There was statistically insignificant difference as regard age, sex, BMI, and duration of hemodialysis in HCV seropositive on hemodialysis compared to HCV seronegative on hemodialysis patients (Table 1).

In hemodialysis patients, no significant differences were observed in hematological data between HCV seropositive and seronegative groups. HCV seropositive patients showed significantly higher intact PTH and serum creatinine levels, along with lower uric acid levels ($p<0.05$), while serum urea, calcium, sodium, potassium, and phosphorus levels showed no significant differences ($p>0.05$). Additionally, HCV seropositive patients had significantly elevated AST, ALT, bilirubin, and INR levels, and reduced serum albumin and prothrombin concentration (PC) compared to seronegative patients. However, serum ALP and bone-specific ALP levels did not differ significantly between the two groups ($p>0.05$). (Table 2)

24.3% of patients have valve calcification, with a mean EF of 56.97 ± 5.87. 40% have atheromatous plaques, and 22.8% have plaque calcification, with a mean CIMT of 1.42 ± 0. 49.. (Table 3)

patients with HCV seropositive on hemodialysis had a higher frequency of cardiac calcification formation (37.1% vs 11.4%), increased CIMT and atheromatous plaque formation ($p<0.05$), and a higher frequency of atheromatous plaque calcification compared to HCV seronegative patients. (Table 4)

Right sided atheromatous plaque formation was more frequent either in HCV seropositive on hemodialysis (34.3%) or HCV seronegative on hemodialysis subjects (11.4%) but there was no statistically significant association between atheromatous plaque site and PCR or Bone isomer ALP level in the studied groups. (Table 5)

At cutoff point 452500, HCV PCR has 95.5% sensitivity and 86.7% specificity for detection of atheromatous plaque formation. (Table 6)

At cutoff point 531950, HCV PCR has 92.3% sensitivity and 81.8% specificity for detection of cardiac valve calcification. (Table 7)

*Table 1. Comparative analysis among examined groups as regard demographic data and dialysis duration*

| | | GI:HCV SEROPOSITIVE ON HEMODIALYSIS | GII:HCV SERONEGATIVE ON HEMODIALYSIS | T/X2 | P-VALUE | SIG. |
|---|---|---|---|---|---|---|
| SEX | Male | 22 (62.9%) | 21 (60%) | 0.060 | 0.806 | NS |
| | Female | 13 (37.1%) | 14 (40%) | | | |
| AGE (YEARS) | Mean ± SD | 48.89 ± 11.96 | 45.34 ± 11.73 | 1.251 | 0.215 | NS |
| | Range | 22-73 | 29-73 | | | |
| BMI | Mean ± SD | 28.34 ± 1.87 | 29.29 ± 3.68 | -1.363 | 0.177 | NS |
| | Range | 23.76-40.39 | 24.95-32.08 | | | |
| DURATION OF HEMODIALYSIS (YEARS) | Mean ± SD | 8.64 ± 4.68 | 9.37 ± 5.79 | -0.580 | 0.564 | NS |
| | Range | 4-15 | 2-14 | | | |

*Table 2. Comparative analysis among examined groups as regard laboratory findings*

| | GI: HCV SEROPOSITIVE ON HEMODIALYSIS | | GII:HCV SERONEGATIVE ON HEMODIALYSIS | | T | P-VALUE | SIG. |
|---|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | | |
| RBCS ($X10^6$) | 3.86 | 0.35 | 3.81 | 0.39 | 0.576 | 0.566 | NS |
| HB GM/DL | 9.64 | 1.42 | 9.74 | 1.61 | -0.268 | 0.790 | NS |
| WBCS($X10^3$) | 7.46 | 1.99 | 8.00 | 2.34 | -1.046 | 0.299 | NS |
| PLATELETS($X10^3$) | 273.22 | 74.53 | 262.57 | 71.50 | 0.617 | 0.540 | NS |
| CRP (MG/DL) | 3.57 | 1.17 | 3.49 | 1.27 | 0.294 | 0.770 | NS |
| S. UREA | 80.49 | 11.57 | 83.91 | 10.14 | -1.319 | 0.192 | NS |
| S. CREAT | 3.88 | 0.81 | 3.52 | 0.65 | 2.051 | 0.044 | S |
| P | 5.36 | 0.64 | 5.49 | 0.53 | -0.919 | 0.362 | NS |
| CA | 8.06 | 0.37 | 7.99 | 0.51 | 0.732 | 0.467 | NS |
| NA | 136.74 | 6.05 | 137.71 | 6.28 | -0.659 | 0.512 | NS |
| K | 5.93 | 0.76 | 5.78 | 0.73 | 0.79 | 0.432 | NS |
| IPTH | 757.83 | 286.39 | 582.46 | 188.03 | 3.028 | 0.003 | HS |
| UA | 4.12 | 0.22 | 4.92 | 0.94 | -4.913 | 0.000 | HS |
| AST | 78.17 | 26.11 | 21.14 | 7.86 | 12.373 | 0.000 | HS |
| ALT | 92.06 | 26.18 | 41.00 | 9.99 | 10.788 | 0.000 | HS |
| BILIRUBIN | 1.43 | 0.79 | 0.75 | 0.45 | 4.425 | 0.000 | HS |
| PC | 85.14 | 9.76 | 91.54 | 5.33 | 3.405 | 0.001 | HS |
| INR | 1.16 | 0.52 | 0.92 | 0.13 | 2.649 | 0.010 | S |
| S. ALBUMIN | 3.14 | 0.88 | 3.68 | 0.89 | -2.553 | 0.012 | S |
| ALP | 145.43 | 50.97 | 139.57 | 62.34 | 0.43 | 0.668 | NS |
| BONE ISOMER ALP | 92.29 | 20.65 | 98.89 | 31.88 | -1.028 | 0.308 | NS |

*Table3. Echocardiography and Carotid duplex findings of studied groups*

| | | N=70 |
|---|---|---|
| EF MMODE | Range | 48-67 |
| | Mean ± SD | 56.97 ± 5.87 |
| VALVE CALCIFICATION | Yes | 17 (24.3%) |
| | No | 53 (75.8%) |
| VALVE CALCIFICATION SITE | Mitral valve | 5 (7.1%) |
| | Aortic valve | 8 (11.4%) |
| | Both mitral & Aorta | 4 (5.7%) |
| CIMT | Range | 0.95-2 |
| | Mean ± SD | 1.42 ± 0.49 |
| ATHEROMATOUS PLAQUE | Yes | 28 (40%) |
| | No | 42 (60%) |
| ATHEROMATOUS PLAQUE SITE | Right | 16 (22.8%) |
| | Left | 6 (8.6%) |
| | Bilateral | 6 (8.6%) |
| ATHEROMATOUS PLAQUE CALCIFICATION | Yes | 16 (22.8%) |

*EF: Ejection fraction

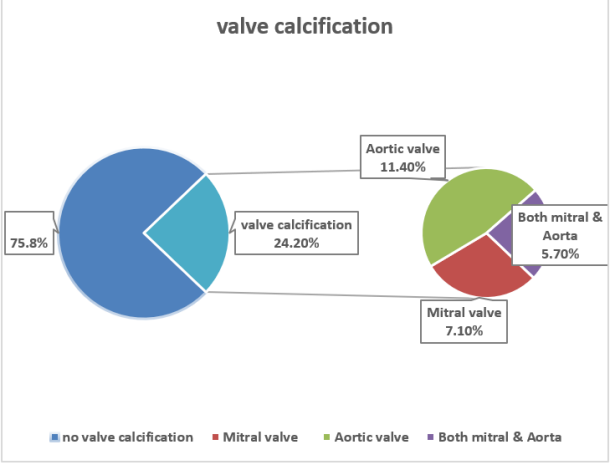


Figure 1. Valve calcification and its site among the studied groups.

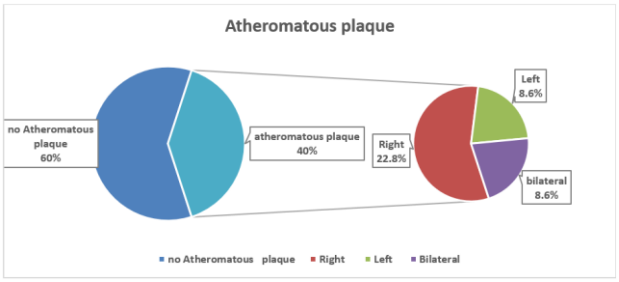


Figure 2. The incidence of atheromatous plaques and its site among the studied groups.

Table 4: Comparison between studied groups as regard Echocardiography and Carotid duplex findings.

| | | GI:HCV SEROPOSITIVE ON HEMODIALYSIS | GII:HCV SERONEGATIVE ON HEMODIALYSIS | T | P-VALUE | SIG. |
|---|---|---|---|---|---|---|
| EF MMODE | Range | 48-67 | 50-65 | -1.425 | 0.159 | NS |
| | Mean ± SD | 55.97 ± 7.14 | 57.97 ± 4.24 | | | |
| VALVE CALCIFICATION | Yes | 13 (37.1%) | 4 (11.4%) | 6.293 | 0.012 | S |
| | No | 22 (62.9%) | 31 (88.6%) | | | |
| CIMT | Range | 1.1-2 | 0.95-1.76 | 4.777 | 0.0001 | HS |
| | Mean ± SD | 1.59 ± 0.29 | 1.27 ± 0.26 | | | |
| ATHEROMATOUS PLAQUE | Yes | 20 (57.1%) | 8 (22.9%) | 8.571 | 0.003 | HS |
| | No | 15 (42.9%) | 27 (77.1%) | | | |
| ATHEROMATOUS PLAQUE CALCIFICATION | Yes | 14 (70%) | 2 (25%) | 4.725 | 0.030 | S |
| | No | 6 (30%) | 6 (75%) | | | |

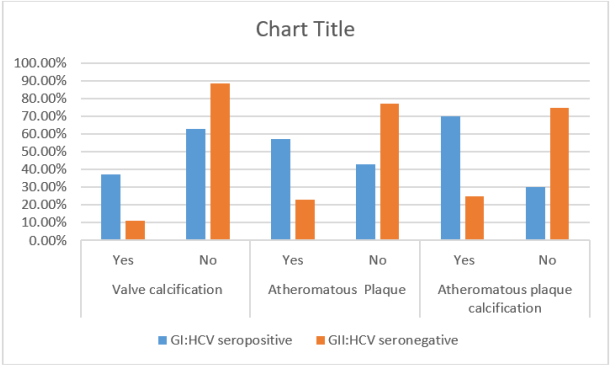


Figure 3: Cardiac calcification and Atheromatous plaque calcification among the studied groups.

*Table 5. The relation of Atheromatous plaque site with both (PCR and bone isomer ALP) in the studied groups*

| | | ATHEROMATOUS PLAQUE SITE | | | F/X2 | P-VALUE | SIG. |
|---|---|---|---|---|---|---|---|
| | | Right | Left | Bilateral | | | |
| HCV | HCV seropositive on hemodialysis | 12 (34.3%) | 4 (11.4%) | 4 (11.4%) | 2.800 | 0.247 | NS |
| | HCV seronegative on hemodialysis | 4 (11.4%) | 2 (5.7%) | 2 (5.7%) | | | |
| PCR IN GROUP I | Mean ± SD | 607025 ± 52691.6 | 616575 ± 208072.3 | 573775 ± 49018.3 | 1.087 | 0.581 | NS |
| BONE ISOMER ALP | Mean ± SD | 99.13 ± 20.71 | 106.83 ± 32.04 | 90.83 ± 12.12 | 0.781 | 0.469 | NS |

*Table 6. ROC curve of HCV PCR for detection of Atheromatous plaque formation*

| | AREA UNDER CURVE | CUTOFF VALUE | SENSITIVITY% | SPECIFICITY% | 95% CONFIDENCE INTERVAL | |
|---|---|---|---|---|---|---|
| | | | | | Lower Bound | Upper Bound |
| HCV PCR | 0.940 | 452500 | 95.5% | 86.7% | 0.837 | 1.000 |

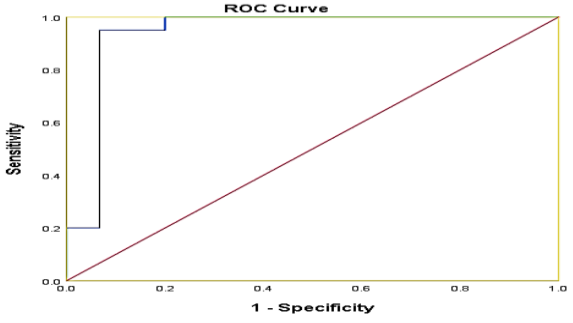


Figure 4. ROC curve for sensitivity and specificity of HCV PCR for detection of atheromatous plaque formation.

*Table 7. ROC curve of HCV PCR for detection of cardiac valve calcification*

| | AREA UNDER CURVE | CUTOFF VALUE | SENSITIVITY% | SPECIFICITY% | 95% CONFIDENCE INTERVAL | |
|---|---|---|---|---|---|---|
| | | | | | Lower Bound | Upper Bound |
| HCV PCR | 0.937 | 531950 | 92.3% | 81.8% | 0.855 | 1.000 |

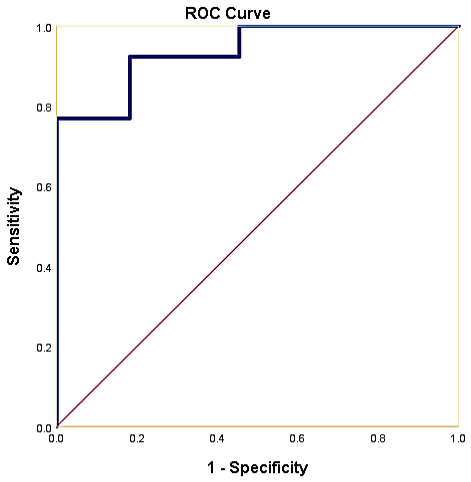


Figure 5. ROC curve for sensitivity and specificity of HCV PCR for detection of cardiac calcification.

## 4. Discussion

HCV is a transmissible disease that varies in severity from mild disease lasting several weeks to severe, chronic illness affecting the liver, typically transmitted by contact with the blood of an infected individual.[11]

The main results of our study were as follows:

Regarding demographic data, the present research showed that most cases were males (61.4%) with a mean age of 46.93 ± 11.82 years, and a mean length of dialysis was 8.87±4.92 years. Moreover, the research demonstrated that there was no statistically significant variance between seropositive and seronegative patients with regard to age, sex, BMI, and duration of HD.

In agreement with the current study, Fayed et al.[12]revealed that most HD patients were males (57%), the

The study showed no significant difference between HD seronegative and hepatitis C positive patients with regard to age, sex, and BMI.

Also, Abd El Raouf & Alghazaly[13] revealed that most patients with ESRD who underwent HD were males (70%), and no significant variations were found among cases with and without HCV infection with regard to age, sex, and BMI.

The current research showed that a statistically insignificant distinction has been observed among seropositive and seronegative patients with regard to hematological data.

In contrast to the current study, AbouSeif et al.[14] showed that the HD patients with HCV infection have significantly higher CRP and lower Hb compared to those with HCV seronegative patients ($p<0.05$ for all).

Regarding renal functions, the current study demonstrated that there were highly statistically significant increased levels of iPTH and serum creatinine in HCV seropositive patients on HD compared to HCV seronegative patients on HD. Also, there was a statistically significant lower level of UR in HCV seropositive patients on HD compared to HCV seronegative patients on HD.

However, there was no statistically significant difference regarding serum urea, Ca, Na, K, and P in HCV seropositive on HD compared to HCV seronegative on HD patients.

In agreement with the current study, Abd El Raouf & Alghazaly[13] showed that serum concentration of iPTH was significantly higher in chronic hepatitis C virus positive cases undergoing HD compared to HCV seronegative patients, and insignificant variance has been observed among the studied groups in urea and BUN. But in contrast with the current study, they found insignificant variation in serum creatinine among the studied groups.

The correlation between HCV positivity and serum creatinine could represent either a consequence of an immunocompromised state in chronic kidney disease or a causal relationship, as HCV infection can induce glomerular damage through extrahepatic manifestations such as membranoproliferative glomerulonephritis, cryoglobulinemia, focal segmental glomerulosclerosis, renal thrombotic microangiopathy, and membranous nephropathy.[15]

Regarding liver functions, the present research exhibited that there were highly significant increased levels of AST, ALT, and bilirubin in HCV seropositive patients on HD compared to HCV seronegative patients on HD. Also, there were statistically significantly higher levels of INR and lower levels of serum albumin and PC in HCV seropositive on HD compared to HCV seronegative on HD patients.

In agreement with the current study, Abd El Raouf & Alghazaly[13] showed that serum levels of AST and ALT were significantly higher in HCV-positive cases undergoing HD than in HCV seronegative patients; however, there was no significant difference between the studied groups in albumin, INR, and bilirubin.

Regarding lipid profile, the present research exhibited that there was a statistically significantly higher level of Cholesterol, TG, and LDL in HCV seropositive patients on HD compared to HCV seronegative patients on HD. There was a statistically significant lower level of HDL in HCV seropositive patients on HD

compared to HCV seronegative patients on HD.

In agreement with the current study, Abd El Raouf & Alghazaly[13] showed that LDL level was significantly higher in HCV-positive cases undergoing HD compared to HCV seronegative patients; however, in contrast to the present cases, there was insignificant variance among the examined groups in HDL, cholesterol, and TG.

Regarding echocardiography findings, the present research revealed that there was a statistically significantly higher frequency of cardiac calcification formation (37.1% vs 11.4%) in HCV seropositive patients on HD compared to HCV seronegative patients on HD. There was a statistically insignificant difference regarding EF m-mode in HCV seropositive patients on HD compared to HCV seronegative patients on HD.

Carotid ultrasound is a widely utilized technique for evaluating atherosclerosis and is increasingly utilized in cases with end-stage renal disease and chronic kidney disease in general. CIMT, the number of plaques, and indicators of vascular remodeling are considered primary US indications for the staging of atherosclerosis.[16]

Regarding CIMT, the current study showed that there was a highly significant increase in CIMT in HCV seropositive patients on HD compared to HCV seronegative patients on HD. There was a statistically significant increase in frequency of atheromatous plaque formation (57.1% vs 22.9%) in HCV seropositive patients on HD compared to HCV seronegative patients on HD. Also, there was a statistically significant increased frequency of atheromatous plaque calcification (70% vs 25%) in HCV seropositive patients on HD compared to HCV seronegative patients on HD.

In agreement with the current study, Fayed et al.[12] revealed that there was a significant association between HCV infection and arterial calcification among HD patients.

Regarding the relation between atheromatous plaque formation and PCR in the seropositive group, the current study showed that the PCR level is statistically significantly elevated in patients with atheromatous plaque formation than in those without.

Regarding the association between atheromatous plaque site and PCR in the studied groups, the current study revealed that the right-sided atheromatous plaque formation was more frequent either in HCV seropositive on HD (34.3%) or HCV seronegative on HD patients (11.4%), but there was no statistically significant association between atheromatous plaque site and PCR level in the studied groups.

Also, the current study showed that Valve calcification is more frequent in HCV seropositive on HD than HCV seronegative on HD patients, but there was no statistically significant association between Valve calcification site and PCR level in the studied groups. These results need to be confirmed by larger studies.

However, Fayed et al.[12] found a significant association between HCV PCR level and site of calcification and calcification score. Also, they found a significant difference between HD patients with and without HCV infection regarding the site of calcification.

ROC curve analysis was performed to test the accuracy of HCV PCR for the detection of atherosclerotic plaque formation and cardiac valve calcification, and it was revealed that at a cutoff point of 452500, HCV PCR has 95.5% sensitivity and 86.7% specificity for the detection of atherosclerotic plaque formation. Also, at the cutoff point 531950, HCV PCR has 92.3% sensitivity and 81.8% specificity for the detection of cardiac valve calcification.

The above results showed that HCV PCR was a reliable marker for the detection of atheromatous plaque formation and cardiac valve calcification, but these results need to be confirmed in larger studies.

ROC curve analysis of bone isomer ALP for the detection of atherosclerotic plaque formation showed that at a cutoff point of 93.5, bone isomer ALP has 57.1% sensitivity and 57.1% specificity for the detection of atherosclerotic plaque formation.

Also, for the detection of cardiac valve calcification at a cutoff point of 93.5, bone isomer ALP has 76.5% sensitivity and 67.9% specificity for the detection of cardiac valve calcification.

The above results showed that bone isomer ALP has better diagnostic accuracy for the detection of cardiac valve calcification compared to its ability for the detection of atheromatous plaque formation.

Several studies have revealed the role of ALP, particularly Bone ALP isoforms, in the calcification of soft and vascular tissues .[17, 18]

In randomized research involving 137 HD cases, Shantouf et al.[19] established that ALP was the only biochemical marker significantly correlated with coronary artery calcification; ALP levels over 120 U/L were related to an elevated risk of coronary calcifications.

Also, this was supported by Guo et al.[20] who showed that serum ALP level is significantly associated with cardiac valve calcification in a study of 145 patients with maintenance HD. ALP is suggested to be a promising interventional target for CVC in maintenance HD patients.

As well, Hwang et al.[21] showed that high serum ALP amplified the risk associated with VC in ESKD patients starting dialysis.

*4. Conclusion*

In conclusion, the current study showed that chronic hepatitis C virus infection in HD cases increases the probability of atherosclerosis, as proved by carotid duplex. High rate of HCV in HD patients in Al-Santa hospital. HCV infection and viral load in HD were associated with impaired renal and liver functions, as well as poor lipid profiles. HCV polymerase chain reaction (PCR) and bone isomer alkaline phosphatase (ALP) were found to be reliable markers for the detection of atheromatous plaque formation and cardiac valve calcification.

## Disclosure

The authors have no financial interest to declare in relation to the content of this article.

## Authorship

All authors have a substantial contribution to the article

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## Conflicts of interest

There are no conflicts of interest.

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