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Relevance

In the world today, about 3–4 million people are newly infected every year by Hepatitis C Virus (HCV), and 350,000 patients die every year due to HCV-related disorders [1]. Chronic hepatitis C (CHC) infection is known to cause liver diseases, but it also causes other extrahepatic comorbidities, like cryoglobulinemia, autoimmune and/or lymphoproliferative diseases, cardiovascular diseases (CVD), renal diseases, diabetes mellitus (DM) and insulin resistance [2]. Many researches, dating as far back as 1995, have been conducted to show the link between chronic hepatitis C and cardiovascular diseases. Several studies have shown that HCV has direct and indirect mechanism of causing atherosclerosis, which is a major factor for the development of cardiovascular diseases. The ways through which HCV causes increased atherosclerosis includes chronic inflammation, endothelial dysfunction, increased insulin resistance, direct vascular invasion. A research conducted by Chew et al. showed that patients who have achieved sustained virologic response (SVR) had decreased levels of SICAM-1 (non-hepatic producer marker of endothelial dysfunction and inflammation) and sCD163 (a marker of monocyte/macrophage activation associated with the presence or burden of atherosclerotic plaques and arterial wall inflammation).

Purpose of the study

To determine the link between chronic hepatitis C and cardiovascular diseases, reviewing different researches done on this topic. Furthermore, to determine if HCV causes CVD as an independent factor or not.

Materials and research methods

Reviews, analysis and data processing of scientific literature on cardiovascular diseases and its risk in patients with chronic hepatitis C. In addition, the materials are based on effects of HCV subclinical and clinical outcomes. Subclinical CVD are evidences of atherosclerotic diseases such as Carotid intima media thickness (CIMT), Flow-mediated dilation (FMD) and Pulse wave velocity (PWV), while clinical CVD is any clinical CVD event such as Coronary artery disease (CAD), Myocardial infarction (MI), Angina, Congestive heart failure (CHF).

Results and discussion

HCV role in subclinical CVD was shown in different researches. In this review, subclinical CVD is measured by CIMT, PWV & FMD. A research conducted by Ebtissam *et al.* [3] showed a positive association between HCV infection and CIMT. The research was conducted with 100 patients, 50 had active HCV infection and 50 belonged to the control group. Result of the research showed significantly statistical difference between both HCV patients and HCV negative control as regarding the common carotid artery intima media thickness (CIMT) (0.93 ± 0.4 vs 0.67 ± 0.10 ; $p < 0.001$). Another research which was done by Petta *et al.* [4] included 174 patients and control groups, it was revealed that patients with G1 CHC had a higher prevalence of carotid atherosclerosis compared with a control population (1.04 ± 0.21 vs. 0.90 ± 0.16 , $p < 0.001$), and identified older age and the presence of severe hepatic fibrosis as two factors independently associated with the presence of carotid plaques. Subsequently, a study done by Fukui *et al.* [5] showed the presence of plaques and median score ($p < 0.001$) and mean IMT ($p = 0.004$) significantly higher in HCV+ vs control. Also, it was said that HCV seropositivity was an independent risk factor for atherosclerosis after adjustment for RF ($p < 0.01$). A review done by Vespasiani-Gentilucci *et al.* showed a direct role of HCV proteins, which, for example, can enhance oxidative stress and increase the concentration of soluble intracellular adhesion molecules at the atherosclerotic plaque level.

Pulse wave velocity (PWV) is a surrogate marker of arterial stiffness, and increase in PWV has been associated with CVD and mortality. A research conducted by Tomiyama *et al.* [6] which included 7514 subjects, the PWV was determined and the result showed that seropositivity for hepatitis C virus was a significant variable for PWV independent from atherosclerotic risk factors. Thus, hepatitis C virus seropositivity was associated with increased PWV.

On the other hand, there were some studies which found no link between CHC and carotid artery atherosclerosis. Bilora *et al.* examined the same cohort of patients with CHC infection in 2001 and 2006 and in both instances found a lower prevalence of carotid IMT and plaques in patients with chronic viral hepatitis compared to uninfected controls.

HCV has also shown its link in clinical CVD. In this review, clinical CVD is defined by the following outcomes: CAD, MI, Unstable Angina and CHF. A research conducted by Tsui *et al.* [7] showed HCV seropositivity to be an independent associated risk for heart failure (HR = 2.13; 95 % CI: 1.19–3.80). This shows HCV link with cardiomyopathy and its ischemic effects on myocardium. In 2004, a research was carried out in Italy by Vassalle *et al.* [8] and it included 491 subjects, with 195 of them being in the control group. There was a report of increased rate of HCV seropositivity in CAD subjects vs. controls (6.3 vs 2; $p = 0.05$), which increased with the number of vessels affected ($p < 0.05$). It was also established that HCV seropositivity was an independent predictor of CAD. A large meta-analysis conducted to evaluate the impact of CHC on CAD by Ambrosino *et al.* [9] included 273,219 HCV-infected and 473,928 HCV-uninfected patients showed a significantly increased risk of CAD associated with HCV positivity (OR: 1.382, 95% CI: 1.103–1.732; $p = 0.005$).

An association between HCV infection and cardiovascular mortality was also established as seen in retrospective cohort conducted by Lee *et al.* [10] between 1991–2008, and was published in 2012, which included 23,820 participants. Increase in both hepatic and extrahepatic mortality when compared to seronegative controls and an increased risk of death from CVD based on diagnosis reported in the Taiwanese National Death Certification Registry. Additionally, mortality from CVD was significantly higher among patients who had detectable HCV RNA levels compared to those with undetectable HCV RNA but positive anti-HCV antibodies, suggesting antiviral therapy may have a role in decreasing HCV related CVD mortality.

Conclusions

According to the researches reviewed, it has been shown that HCV infection has as a link to cardiovascular diseases. Increase in factors like CIMT, PWV in CHC patients and coronary artery disease, hepatic & extrahepatic mortality in HCV seropositive patients, shows a connection between Hepatitis C virus infection and cardiovascular diseases.

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UDC 616.832.21-002-036.21

IMMUNIZATION ACTIVITY AND ERADICATION PROGRESS OF POLIOMYELITIS IN ENDEMIC COUNTRIES

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Relevance

Poliomyelitis (polio) is a highly infectious viral disease, which mainly affects young children. The virus transmitted by person-to-person spread mainly through the faecal-oral route or, less fre-