

## Vascular Wall: Another Victim of Hepatitis C Virus?

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### Editorial

Hepatitis C virus (HCV) infection is a major overall health challenge. It is estimated that more than 180 million people are chronically infected worldwide. Chronic hepatitis C (CHC) remains a serious problem of global concern because of its other long-term consequences. Patients with CHC infection may progress to cirrhosis and subsequently develop complications such as variceal bleeding, ascites, encephalopathy and hepato-cellular carcinoma [1]. Vascular bleeding is the third leading cause of mortality in patients with CHC. Nevertheless, whether CHC is associated with an increased risk of death from atherosclerotic cardio-vascular disease (CVD) is indefinite [2].

Previous studies have shown that, atherosclerosis results from injured vascular wall, not simply from the old theory of lipid accumulation at the vascular wall. The inability of traditional risk factors such as hypertension, hyperlipidemias, diabetes and smoking to clarify the incidence of atherosclerosis in about half of the cases provoked a search for additional assumed risk factors involved in the development of the disease. Infectious agents have long been suspected to initiate/contribute to the process. Moreover, it has been suggested that inflammation, either related to infectious agents or independent from infection, may mediate the atherogenic process [3,4]. Up to date, several studies propose that, certain chronic infections increase the risk for CVD and such infections considered modifiable risk factors. Chronic infection with various microorganisms, particularly, Cytomegalovirus, Chlamydia pneumoniae, H pylori and HCV infections may play a role in etiological factors, linking inflammation and atherogenesis [5].

Furthermore, HCV seems to be independent risk factor for CVD probably due to its association with insulin resistance, diabetes and steatosis. Currently, cardiovascular risk in CHC patients has been an interesting focus, and the relationship

between cardiovascular risk, cytokines, lipids, viral load and sub-clinical atherosclerosis in these patients is not fully explicated. It seems that, HCV infection represents an initial step in the chronic inflammatory cascade of atherosclerosis either subclinical or manifest, via increased levels of pro-atherogenic chemokines and cytokines and amplified systemic oxidative stress levels [6].

Adding to bleeding varices, many authors acknowledge vascular wall changes as another cause of morbidity and mortality in patients with CHC infection. Many studies have found that chronic HCV infection is a risk factor for carotid and coronary arteries plaques and abnormal aortic elasticity (stiffness) and significant mortality. However, the exact association between CHC and subclinical atherosclerosis is still unclear [7,8].

Therefore, the judgment of presence of subclinical or manifest atherosclerosis in HCV infected patients is necessary, because of its prognostic value and its influence on patient management. Detection of such factors along with their associated mechanisms of action would have insightful implications for the advance of new therapeutic strategies that would reduce the devastating impact of this disease. Published clinical studies examining the association between CHC and CVD are still conflicting. There are pros and cons about the link between HCV and vascular wall changes.

Maria et al. investigated whether HCV infection can facilitate asymptomatic carotid lesions. Their figures strongly supported that HCV infection facilitates the incidence of carotid atherosclerotic lesions [9]. Also, Liao et al. seek out to answer the question: Does Hepatitis C Virus Infection Increase Risk for Stroke? Their data exposed, Yes CHC infection increases stroke risk and should be considered an imperative and independent risk factor [10].

Oliveira et al. evaluated the direct effect of HCV on cardiovascular risk and correlate it with pro and anti-inflammatory cytokines in patients with HCV. They concluded

that, even in patients formerly expected to have low-risk to CVD, HCV seems to increase that risk, as the existence of the infection was interrelated to higher Framingham Risk Scoring System as well as elevated pro-anti-inflammatory cytokine profile [11].

One of the interesting articles in 2013 is the Reza et al. Meta-analysis study. 364,712 individuals underwent different researches for potential associations between HCV infection and CVD. Only 6 out of 31 reviewed studies reported a negative association between HCV infection and CVD. Such statistics suggests that HCV has a significant effect on the expansion of CVD in the general inhabitants, either in the coronary or carotid arteries levels [12].

Luigi et al. analyzed and discussed in their published article several studies concerning CHC and atherosclerosis. They fulfilled that, "based on the evidence discussed in the article, it is reasonable to recommend to screen for atherosclerosis noninvasively all patients with CHC" [13]. In a similar way, Pateria et al. proved that, genotype 1 infection is linked with increased carotid-intima thickness and greater endothelial dysfunction compared with non-genotype 1 infection in CHC subjects [14]. McKibben et al. [15] assessed 994 men for coronary plaque, using non-contrast coronary computed tomography. They reported elevated prevalence of subclinical coronary atherosclerosis among men with CHC, especially men with the highest HCV RNA levels. Moreover, recently, Ashraf et al. studied atherosclerosis in CHC patients with and without liver cirrhosis. Their data revealed carotid intima media thickness and increased epicardial fat thickness; especially in those with cirrhosis [16].

On the contrary Pateria et al. investigated the association between CHC, viral load and atherosclerosis of carotid artery and aortic stiffness in a case control study by non-invasive techniques. They concluded that, subclinical vascular disease is not greater in CHC subjects compared with controls [14]. This negative data was previously acknowledged in 2012 by Roed et al. [17] and recently in 2017 by Salvatoreta [18]. Roed et al. reported 2 remarks "i) Most of the results revealed a tendency towards a higher risk of CVD among patients with HCV infection. ii) As the majority of studies available on this topic are of deprived quality, firm conclusions are hard to reach. Moreover, none of the mentioned researches determined whether HCV infection duration or treatment influence CVD development.

With respect to the above mentioned controversial data, advanced studies are needed to clarify definitively the role of HCV infection in cardiovascular alterations, as well as the impact of viral eradication on cardiovascular changes outcomes. Researchers should make efforts to look at the magnitude of this association.

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