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ISCHEMIC HEART DISEASE AFTER VIRAL INFECTIONS: CURRENT INSIGHTS INTO PATHOGENESIS AND CLINICAL IMPLICATIONS

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Annotation

Ischemic heart disease (IHD) continues to represent a major global health burden and remains the leading cause of morbidity and mortality worldwide. According to contemporary epidemiological data, cardiovascular diseases account for more than one-third of all deaths globally, with IHD constituting the largest proportion[1]. Despite significant advances in prevention, diagnosis, and treatment, the incidence of IHD remains high, particularly in low- and middle-income countries, where the burden of cardiovascular risk factors continues to increase. In recent years, growing attention has been directed toward the role of infectious agents—particularly viral pathogens—in the initiation and progression of cardiovascular diseases [4]. Viral infections, including influenza, enteroviruses, and most notably severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), have been shown to exert both direct and indirect effects on the cardiovascular system. These effects range from acute myocardial injury and myocarditis to long-term complications such as endothelial dysfunction, accelerated atherosclerosis, and ischemic heart disease[5].Accumulating evidence suggests that viral infections may act as potent triggers of myocardial ischemia through a complex interplay of pathophysiological mechanisms. One of the central pathways involves systemic inflammation characterized by a cytokine-mediated response, often referred to as a “cytokine storm,” which contributes to endothelial damage, plaque instability, and increased thrombogenicity. In parallel, viral-induced endothelial dysfunction plays a critical role in impairing vascular homeostasis by reducing nitric oxide bioavailability, promoting vasoconstriction, and enhancing procoagulant activity[6].Another important mechanism is the dysregulation of neurohumoral systems, particularly activation of the sympathoadrenal system, which leads to increased circulating catecholamines. This results in elevated myocardial oxygen demand, coronary vasospasm, and exacerbation of ischemic processes.

Key words

Ischemic heart disease, viral infections, COVID-19, endothelial dysfunction, inflammation.

Abstract. Additionally, oxidative stress induced by viral infections contributes to lipid peroxidation, mitochondrial dysfunction, and further endothelial injury, thereby accelerating the progression of atherosclerotic disease. The COVID-19 pandemic has provided unprecedented insights into the cardiovascular consequences of viral infections. SARS-CoV-2 infection has been associated with a broad spectrum of cardiac manifestations, including acute coronary syndromes, microvascular dysfunction, thromboembolic complications, and persistent cardiovascular symptoms in the post-acute phase, commonly referred to as “long COVID.” Importantly, emerging data indicate that even individuals without prior cardiovascular disease may develop ischemic complications following COVID-19, underscoring the systemic nature of viral-induced vascular injury[2]. Furthermore, viral infections may exacerbate pre-existing cardiovascular conditions by destabilizing atherosclerotic plaques and triggering acute coronary

events. The interplay between immune activation, coagulation abnormalities, and endothelial dysfunction creates a proatherogenic and prothrombotic environment, significantly increasing the risk of adverse cardiovascular outcomes. Despite considerable progress in understanding these mechanisms, many aspects of the pathogenesis, clinical course, and long-term prognosis of post-viral ischemic heart disease remain insufficiently elucidated[7]. In particular, there is a need for further research into the role of immune dysregulation, neurohumoral activation, and oxidative stress in mediating these effects, as well as the development of targeted preventive and therapeutic strategies.

Material and methods. This study was conducted as a narrative literature review aimed at summarizing current evidence on the relationship between viral infections and the development of ischemic heart disease (IHD), with a particular focus on pathogenetic mechanisms and clinical implications. A comprehensive literature search was performed using major electronic databases, including. The search covered publications from, with emphasis on studies published after 2019 due to the emergence of COVID-19–related cardiovascular complications.

Results. Overview of Included Studies:The literature search identified a substantial body of evidence demonstrating a significant association between viral infections and the development of ischemic heart disease (IHD). A total of relevant studies, including observational cohorts, case-control studies, and systematic reviews, were analyzed. The majority of studies focused on respiratory viral infections, particularly influenza viruses and SARS-CoV-2, due to their global prevalence and well-documented cardiovascular complications.

Inflammatory and Immune-Mediated Mechanisms:A consistent finding across studies was the central role of systemic inflammation in the pathogenesis of post-viral IHD[8]. Viral infections were associated with elevated levels of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). These inflammatory mediators contribute to:

Destabilization of atherosclerotic plaques

Increased vascular permeability

Activation of immune cells within the vascular wall

In patients with COVID-19, a “cytokine storm” has been identified as a key factor accelerating myocardial ischemia and acute coronary events.

Endothelial Dysfunction

Endothelial injury emerged as a critical mechanism linking viral infections to IHD. Several studies demonstrated that viruses can directly or indirectly damage endothelial cells, leading to:

Impaired nitric oxide production

Increased oxidative stress

Enhanced expression of adhesion molecules

Endothelial dysfunction promotes vasoconstriction, inflammation, and thrombogenesis, thereby increasing the risk of coronary artery occlusion[3].

Prothrombotic State and Coagulation Abnormalities

A procoagulant state was frequently reported in patients following viral infections. Elevated levels of D-dimer, fibrinogen, and other coagulation markers were observed, particularly in COVID-19 patients. This hypercoagulable state contributes to:

Microvascular thrombosis

Coronary artery thrombosis

Increased incidence of myocardial infarction

Several studies highlighted that thrombotic complications may persist even after the acute phase of infection, indicating long-term cardiovascular risk.[9]

Neurohumoral Dysregulation

Emerging evidence suggests that viral infections may disrupt neurohumoral regulation, including activation of the sympathetic nervous system and the renin–angiotensin–aldosterone system (RAAS). Increased catecholamine levels can:
Elevate myocardial oxygen demand
Induce coronary vasospasm [10]
Promote oxidative stress and myocardial injury
This mechanism is particularly relevant in post-COVID conditions, where persistent autonomic imbalance has been reported.
Clinical Manifestations and Outcomes
Clinical data indicate an increased incidence of:
Acute coronary syndromes (ACS)
Myocardial infarction (both type 1 and type 2) [11]
Silent myocardial ischemia]
Patients with pre-existing cardiovascular disease were found to be at higher risk of adverse outcomes. Moreover, several longitudinal studies demonstrated that the risk of IHD remains elevated for weeks to months following viral infection.
Long-Term Cardiovascular Consequences
Long-term follow-up studies revealed that post-viral patients may develop:
Accelerated atherosclerosis
Chronic endothelial dysfunction
Persistent low-grade inflammation[12]
These changes contribute to an increased burden of chronic ischemic heart disease and may have significant public health implications.

Discussion. The present review synthesizes current evidence on the association between viral infections and ischemic heart disease (IHD), highlighting the multifactorial nature of post-viral cardiovascular pathology. The findings demonstrate that viral infections are not only acute triggers of myocardial ischemia but may also contribute to the long-term progression of atherosclerotic disease.One of the central observations is the pivotal role of systemic inflammation in mediating cardiovascular complications. Viral infections, particularly those caused by respiratory pathogens such as influenza and SARS-CoV-2, induce a pronounced inflammatory response characterized by elevated cytokine levels. This inflammatory milieu promotes destabilization of atherosclerotic plaques, thereby increasing the risk of acute coronary events. These findings are consistent with the concept of inflammation as a key driver of atherothrombosis. Another important mechanism identified in this review is endothelial dysfunction**,** which serves as a critical link between infection and vascular pathology. Endothelial injury leads to impaired vasodilation, increased oxidative stress, and enhanced leukocyte adhesion, all of which contribute to the progression of coronary artery disease. In the context of COVID-19, direct viral invasion of endothelial cells has been proposed, further exacerbating vascular damage and thrombogenicity. The results also emphasize the significance of a prothrombotic state following viral infections. Hypercoagulability, reflected by increased levels of D-dimer and fibrinogen, plays a central role in the development of both macrovascular and microvascular thrombosis. This mechanism is particularly relevant in explaining the increased incidence of myocardial infarction observed during and after viral illnesses. Importantly, thrombotic risk may persist beyond the acute phase of infection, suggesting the need for extended monitoring and preventive strategies. In addition to inflammatory and vascular mechanisms, neurohumoral dysregulation appears to be an emerging contributor to post-viral IHD. Activation of the sympathetic nervous system and alterations in the renin–angiotensin–aldosterone system may lead to increased myocardial oxygen demand, coronary vasospasm, and

oxidative stress. These findings align with broader evidence demonstrating the interplay between neurohumoral pathways and immune responses in cardiovascular disease, and they may be particularly relevant in patients with post-COVID syndrome. From a clinical perspective, the reviewed data indicate that patients with pre-existing cardiovascular risk factors are especially vulnerable to adverse outcomes following viral infections. However, even individuals without prior cardiovascular disease may develop ischemic complications, underscoring the systemic impact of viral pathogens. These observations highlight the importance of early cardiovascular risk assessment and targeted management in patients recovering from viral infections.The long-term implications of post-viral cardiovascular involvement are of particular concern. Persistent inflammation, endothelial dysfunction, and metabolic disturbances may contribute to accelerated atherosclerosis and chronic ischemic heart disease. This suggests that viral infections should be considered not only as transient insults but also as potential modifiers of long-term cardiovascular risk.Despite the growing body of evidence, several limitations should be acknowledged. The heterogeneity of study designs, populations, and outcome measures limits the ability to draw definitive causal conclusions. In addition, most available data are derived from observational studies, which may be subject to confounding factors. Further prospective and mechanistic studies are needed to better elucidate the pathways linking viral infections to ischemic heart disease.

Conclusion. In summary, viral infections play a significant role in the initiation and progression of ischemic heart disease through complex interactions involving inflammation, endothelial dysfunction, thrombosis, and neurohumoral imbalance. Understanding these mechanisms is essential for improving prevention, early diagnosis, and management of cardiovascular complications in the post-viral setting.

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