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THE INTERSECTION OF VIRAL INFECTIONS AND CARDIAC INFARCTION

Introduction

Viral infections have been implicated in cardiovascular complications, including cardiac infarction. This comparative analysis explores the associations between COVID-19, Cytomegalovirus (CMV), Influenza, and Human Immunodeficiency Virus (HIV) in the context of cardiac health. Understanding these relationships can provide insights into the mechanisms by which viral infections may contribute to cardiac events.

Goal

The primary goal of this study is to analyze the intersection of viral infections and cardiac infarction, examining the pathophysiological mechanisms, prevalence, and outcomes associated with COVID-19, CMV, Influenza, and HIV. A Comparative Analysis of COVID-19, CMV, Influenza, and HIV.

Material and methods of research

A literature review was conducted, focusing on peer-reviewed articles published between 2013 and 2023. Key databases such as PubMed, Scopus, and Google Scholar were utilized to identify relevant studies. Data on the incidence of cardiac infarction in patients with these viral infections were extracted and compared. Additionally, the analysis included the immune response mechanisms and inflammatory pathways activated by each virus.

The results of the research and their discussion

Cytomegalovirus Infection and Cardiac Cell Viability: Cytomegalovirus (CMV) is a common herpesvirus with a worldwide seroprevalence of up to 95%. While most infections are asymptomatic, they severely affect immunocompromised patients, leading to complications such as congenital disabilities and increased cardiovascular disease (CVD) risk (table 1) [1]. This study investigates the differential effects of CMV on cardiac cell death mechanisms specifically necroptosis and apoptosis in primary cardiomyocytes and cardiac fibroblasts. Results indicate that CMV infection inhibits TNF-induced necroptosis [3] in cardiomyocytes while prompting the opposite effect in fibroblasts. Additionally, CMV suppresses inflammation [2] and apoptosis [3] in cardiomyocytes, enhancing their mitochondrial viability.

Influenza infection and cardiovascular morbidity**,** emphasizing prevalent complications such as cardiovascular death [4], myocardial infarction (table 1), and heart failure hospitalization. The review discusses proposed mechanisms that include both direct effects of the influenza virus, such as cardiac infraction [5] and endothelial dysfunction, and systemic inflammatory responses leading to increased metabolic demand and plaque instability. While the risk of cardiovascular events rises during influenza infections, the direct and indirect roles of the virus in causing these complications remain uncertain. The systemic inflammatory responses and pro-thrombotic states significantly contribute to cardiovascular risks [6] particularly in patients with existing heart conditions.

HIV infection remains a global health crisis, with 34 million people affected, predominantly in developing nations. Despite advances in antiretroviral therapy (ART) that improve prognosis, chronic diseases, particularly cardiovascular complications, have emerged as significant concerns among the HIV population. These individuals typically present with double the cardiovascular disease risk (table 1) [7] compared to the general population, associated with traditional risk factors like smoking and diabetes. Additionally, ART contributes to abnormalities in lipid and glucose metabolism, hypertension, and an increased likelihood of acute myocardial infarction (MI) [8]. The intersection of HIV, ART, and co morbid conditions necessitates careful cardiovascular monitoring and management [9], highlighting the importance of understanding potential drug interactions and treatment considerations in this population.

The COVID-19 pandemic has rapidly evolved into a global health crisis, significantly stressing healthcare systems and leading to decreased hospital admissions for acute coronary syndromes, notably acute myocardial infarction [10]. Several factors contributed to this trend, including public lock-downs, a decline in outpatient services, and patients' reluctance to seek care due to fears of virus exposure. Reports indicate a 38% reduction in STEMI [11] admissions in the Usa early in the pandemic, with similar trends observed globally. Title: Myocardial Injury and Prognosis in COVID-19 Hospitalized Patients the risk factors and prognostic value of myocardial injury in hospitalized COVID-19 patients. Among 559 patients, 320 had troponin testing, with 91 exhibiting elevated levels. Key predictors for elevated troponin included age, female sex, low systolic blood pressure, and increased creatinine. Elevated troponin levels correlated with a higher risk of death (HR 4. 32) and adverse outcomes (HR 1. 96). The findings suggest that troponin levels may serve as an important tool for risk stratification in COVID-19 patients. Despite the overall incidence of acute MI remaining high. primary percutaneous coronary intervention remained the preferred treatment for STEMI; however, both STEMI and NSTEMI cases faced increased delays and reduced rates of treatment during the pandemic. The increase in late presentations has been linked to higher mortality [13] rates and complications. Moreover, COVID-19 infection is associated with an increased risk of acute MI [12], particularly within the initial weeks following infection.

Table 1 – Risk of Viral Infections

CONDITION	RISK INCREASE	TIME FRAME
COVID 19	21% long-term CVD risk	Up to 3 years
HIV	1.5-2× baseline risk	Chronic
INFLUENZA	6.16× acute risk	7 days post-infection
CMV	22%× acute risk	Long term (decades)

Conclusions

This comparative analysis highlights the complex interplay between viral infections and cardiac health. COVID-19, CMV, Influenza, and HIV each contribute to an elevated risk of cardiac infarction through various mechanisms, including inflammation and endothelial dysfunction. Future research should focus on targeted prevention strategies and the long-term cardiovascular effects of these viral infections in affected populations.

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