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Perspective

Short Note on HIV and Cardiovascular Diseases

Michelle Morone*

Division of Cardiology, University of Alberta and Mazankowski Alberta Heart Institute, Edmonton, Canada

INTRODUCTION

Human immunodeficiency virus (HIV) infections have spread to all four corners of the world and are one of the most deadly infections of the last century. Currently, it is estimated that about 34 million people worldwide live with HIV, 90% of whom live in developing countries.

With advances in the treatment of HIV, people with HIV are now living longer but unfortunately experience heart disease and its complications at faster rates than people without HIV infection.

A large cohort of studies found that individuals infected with HIV had a significantly higher risk of myocardial infarction (MI) compared to uninfected controls. When adjusting for atherosclerotic cardiovascular disease (ASCVD) risk factors, HIV-infected (HIV+) persons have a 1.5-fold to twofold increased risk of having CAD. Furthermore, compared with uninfected persons, HIV+ persons presenting with a first episode of acute coronary syndromes (ACS) are, on average, a decade younger.

Antiretroviral therapy (ART) is so effective in controlling HIV infection that it has revolutionized the prognosis of people living with HIV and has significantly extended survival. However, people infected with HIV live much longer in ART and are increasingly exposed to emerging health problems, especially chronic illnesses. Cardiovascular complications are now the leading contributor to morbidity and mortality in HIV-infected persons, especially in developed countries.

SOME HEART DISEASES RELATED TO HIV

Many studies suggest that an increased risk of acute myocardial infarction (AMI) in HIV-positive people is likely to be associated with a co-existing burden of disease, including HIV, ART, and conventional risk factors. HIV patients have a 50% increased risk of AMI and increased cardiac adverse events after discharge. The incidence of AMI is 3.5 per 1000 patients / year. Possible mechanisms may include inflammation, decreased CD4 cell count, altered coagulation, dyslipidemia, impaired arterial elasticity, and impaired endothelial dysfunction. Anti-retroviral therapy is associated with metabolic changes and abnormal fat distribution, which are associated with insulin resistance, diabetes and dyslipidemia. Although HIV and ART are associated with AMI risk, SMART study results show that continuous HIV virus suppression lowers the risk of cardiovascular disease than drug interruption, suggesting that the virus may play a direct role.

Since the introduction of ART, the cardiovascular manifestations of HIV have changed. On the one hand, ART has significantly changed the course of HIV disease, prolonging survival and improving the quality of life of HIV-infected patients. Early data, on the other hand, raise concerns that ART is associated with increased peripheral and coronary artery disease. However, this treatment is also associated with the reduction of other heart diseases such as pericardium, myocardium, and valvular heart disease.

Cardiovascular involvement in HIV-infected persons can take the form of nonspecific electrocardiographic abnormalities. Arrhythmias are observed and may be associated with tumor involvement or cardiomyopathy, but may be secondary to therapeutic agents.

Pulmonary hypertension (PH) is a rare complication of HIV, but it occurs more frequently in the HIV-infected population than in the general population.

PREVENTION

Prevention of cardiovascular disease has become an integral part of the daily care of people living with HIV. Cardiovascular risk factors should be reduced in all possible conditions. Current strategies for reducing CHD risk include early start of the ART regimen with the least metabolic syndrome and careful management of traditional cardiovascular risk factors with both non-medical and medical treatments. Future strategies for preventing CHD in HIV-infected individuals include the use of HIV-tailored cardiovascular risk-prediction paradigms and and the administration of therapies alongside ART that could further decrease proatherogenic HIV-specific immune activation. Many ongoing studies evaluating the benefits of modulating immune activation and the associated vascular risk using methotrexate, canakinumab (interleukin 1β monoclonal antibody), tocilizumab (interleukin 6 monoclonal antibody), probiotics, etc.

CONCLUSION

HIV-related cardiovascular disease is caused by a complex network of viruses, hosts, and ART-related factors. Appropriate treatments weigh the risks and benefits of individual treatments and are based on knowledge of the relevant pathophysiological mechanisms. Over the next few years, the aging of this particular population will continue to increase both the risk and prevalence of HIV-related cardiovascular disease. Therefore, precautionary measures need to be strengthened to reduce cardiovascular risk.

Correspondence to: Michelle Morron, Division of Cardiology, University of Alberta and Mazankowski Alberta Heart Institute, Edmonton, Canada, E-mail: michellemorone@ualberta.ca

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