Accelerated atherosclerosis in a human immunodeficiency virus infected patient not on highly active anti-retroviral therapy: An autopsy case report

R. Kalyani, M. J. Thej, K. Prabhakar1, J. Kiran2

Departments of Pathology, 1Medicine, 2Forensic Medicine, Sri Devaraj Urs Medical College, Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India

Address for correspondence: Dr. Kalyani. R., Professor of Pathology, Sri Devaraj Urs Medical College, Sri Devaraj Academy of Higher Education and Research, Kolar – 563 101, Karnataka, India.
E-mail: drkalyanir@rediffmail.com

ABSTRACT

The pandemic spread of human immunodeficiency virus (HIV) has been the greatest challenge to public health in modern times. However today, people infected with HIV are living longer due to highly active antiretroviral therapy (HAART). This has resulted in age related complications like cardiovascular diseases, causing increased morbidity and mortality. The relative contributions of HIV infection versus potential adverse effects of HAART to coronary heart disease risk remains unclear. Recent reports implicate both HIV infection per se and HAART therapy to cause metabolic derangements which are pro-atherogenic. Here, we report a case of HIV infected young patient never exposed to HAART, presenting with accelerated atherosclerosis in aorta, coronary and carotid arteries.

Key words: Accelerated atherosclerosis, autopsy, highly active anti-retroviral therapy, human immunodeficiency virus

INTRODUCTION

Human immunodeficiency virus (HIV) infected individuals have an increased risk of coronary heart disease (CHD).[1-3] The cause might be the viral infection itself, the use of highly active antiretroviral therapy (HAART) or altered immune responses seen in these patients.[1] The risk is influenced by traditional factors such as age, smoking, diabetes and dyslipidemia as well as non-traditional factors like local and systemic inflammation. Clinical presentation of CHD in HIV infected patients tends to be different from CHD due to traditional risk factors in that they present at an age which is almost a decade younger, with a mean age of 50 years, compared with non-infected controls.[3]

Early diagnosis of CHD is of paramount importance in the present day scenario where HIV infection has become a manageable but not yet curable, chronic condition.

CASE REPORT

A 36 year old man, who met with a road traffic accident, was admitted to the hospital with severe head injuries. During his stay in the hospital he was diagnosed to be infected with HIV. He was a smoker, non alcoholic, non-diabetic and had no past history of CHD symptoms or family history of CHD. He died on the 8th day of admission and an autopsy was conducted. Grossly, the aorta showed extensive areas of atherosclerosis [Figure 1]. Coronary [Figure 2] and carotid arteries showed atherosclerotic plaques and narrowing of the lumen. Microscopy and histopathological examination with hematoxylin and eosin staining showed well formed atherosclerotic plaques in the aorta [Figures 3 and 4], circumferential involvement of all the
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Discussion

With advancements in medicine and HAART therapy, people with HIV infection are now living longer and an understanding of its relationship with development of atherosclerosis is the need of the hour. Whether HIV infection per se is an independent CHD risk factor, or it is solely due to HAART remains controversial.[4] Clinical and epidemiological studies have shown relation between HIV infection and increased risk of CHD across large cohorts.[1] People with HIV infection have 1.5 to 2 fold higher incidence of cardiovascular events reported, compared with uninfected individuals.[1-4] When first reports of myocardial infarction in young HIV infected patients surfaced, the initial focus was primarily on the

Three coronary vessels [Figures 5 and 6] and carotid arteries with large lipid cores, intense inflammation, abundant foam cells, speckled calcification, thin cap atheroma and lack of normal vessel wall between the atheromas.

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The effects of HIV on non-traditional risk factors like local and systemic inflammation and endothelial function have gained importance in recent times. Chronic inflammation plays a central role in the development of atherosclerosis. Increased vascular inflammation seen in HIV infection might be due to direct viral infection or due to the associated metabolic defects. Vascular inflammation causes endothelial cell activation leading to an increased expression of adhesion molecules and also causes activation of the monocytes. This in turn leads to an increased migration of monocytes into the atherogenic lesion, maturation into macrophages and conversion into lipid rich foam cells. Activated macrophages are a major source of cytokines and chemokines that direct monocytes into the vascular lesions, creating a positive feedback loop. More recently, HIV infection has been shown to interfere with the cholesterol efflux from the macrophages resulting in accumulation of many foam cells, subsequent necrosis and apoptosis of these cells leading to the formation of the lipid core of the atheroma. HIV infection impairs reverse cholesterol transport whereas HAART affects mostly forward cholesterol transport (increasing cholesterol delivery to cells).

The present case highlights the role of traditional risk factors like male sex and smoking in the causation of atherosclerosis which seems to be accelerated by the HIV infection induced inflammation. Since the patient was never exposed to HAART nor did he have a positive family history of CHD, the accelerated atherosclerosis was most probably caused by smoking and HIV infection. Histopathological examination showed circumferential atheromas with complication in all the three coronary vessels, carotid arteries and aorta. Lesions with large lipid core and thin cap carry a higher risk of rupture and thrombosis.

**CONCLUSION**

HIV related cardiovascular disease is an under-recognized cause for symptomatic illness. A high degree of suspicion and early screening may allow appropriate intervention and improved quality of life in those affected. Observations in the present case highlight the fact that traditional factors such as smoking need to be controlled for reducing CHD. These patients should be prescribed statins and platelet anti-aggregants along with antiretroviral regimens. Strategies to decrease cardiovascular risk in HIV infected patients will be important in the public health perspective.

**REFERENCES**