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COMPLICATIONS OF HIV ON CARDIOVASCULAR SYSTEM

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Abstract : Thanks to the development of highly active antiretroviral therapy, HIV infection has changed from a potentially fatal disease to a chronic but treatable sickness (HAART). Cardiovascular disease (CVD) has become a major source of morbidity and mortality for individuals living with HIV, despite their longer lifespans. An overview of the effects of HIV on the cardiovascular system and the underlying mechanisms that lead to this elevated risk is intended to be provided by this abstract. Evidence points to accelerated atherosclerosis, endothelial dysfunction, and an increased risk of myocardial infarction, stroke, and heart failure as consequences of HIV infection and some antiretroviral drugs. Immune activation, dyslipidemia, metabolic abnormalities, and chronic inflammation are important factors in the pathophysiology of HIV-related cardiovascular problems. Traditional cardiovascular risk factors that increase the risk of CVD include diabetes mellitus, hypertension, and smoking, all of which are common in people living with HIV. For comprehensive management techniques to work, it is imperative that the intricate interactions between HIV infection, antiretroviral therapy, and traditional cardiovascular risk factors are understood. In order to reduce the burden of CVD in this population, screening for cardiovascular risk factors, early detection of subclinical CVD, lifestyle changes, and aggressive management of modifiable risk factors are essential. Furthermore, further research is required to clarify new therapy targets and approaches that are specific to the requirements of people living with HIV. In this susceptible group, multidisciplinary cooperation between cardiologists, infectious disease experts, and primary care physicians is crucial to maximizing cardiovascular health outcomes.

IndexTerms **- Component,formatting,style,styling,insert.**

INTRODUCTION

1.1.HIV ASSOCIATED CARDIOMYOPATHY

1.1 HIV-associated cardiomyopathy is a disorder in which HIV infection weakens the heart muscle. Heart failure, arrhythmias, abnormal heartbeats, and other heart-related issues can result from cardiomyopathy.
1.2 HIV-associated cardiomyopathy develops as a result of multiple factors:
1.2.1 HIV's Direct Effects: HIV can infect cardiac cells directly, causing inflammation and heart muscle damage. Additionally, the virus has the ability to set off an autoimmune reaction, in which the body attacks its own cardiac tissue.
1.2.2 Chronic Inflammation: HIV infection causes the body to enter a condition of chronic inflammation, which over time may cause damage to the heart muscle. Cardiomyopathy develops and progresses as a result of inflammation.
1.2.3 Antiretroviral therapy (ART): Although ART has greatly improved the prognosis for those living with HIV, some antiretroviral medications have the potential to cause cardiomyopathy due to their cardiotoxic side effects. Furthermore, certain older HIV drugs might have been more cardiotoxic, especially those from the early stages of treatment.
1.2.4 Opportunistic Infections: Individuals living with HIV are more vulnerable to opportunistic infections such as toxoplasmosis and cytomegalovirus (CMV), which can have a direct impact on the heart. Inflammation and heart muscle damage may result from these infections.
1.2.5 Drug Abuse: People with HIV are more likely to abuse drugs, especially cocaine and methamphetamine. These medications have the potential to cause cardiomyopathy and have cardiotoxic side effects.
1.2.6 Coexisting disorders: Hypertension, diabetes, and hyperlipidemia are a few of the coexisting disorders that HIV is linked to. These conditions are risk factors for cardiovascular disease and can hasten the onset of cardiomyopathy.
1.2.7 Vascular Disease: HIV infection raises the risk of atherosclerosis and other vascular illnesses, which can impede the heart's ability to pump blood and hasten the onset of cardiomyopathy.
1.2.8 Immune dysregulation: HIV infection results in immune dysregulation, which can cause immunological cells essential for heart health to become out of balance and lead to cardiomyopathy.
1.2.9 Genetic variables: Although the precise mechanisms behind this association are still unclear, certain genetic variables may predispose HIV-positive individuals to develop cardiomyopathy.

2.HIV ASSOCIATED VASCULOPATHY

2.1 A collection of vascular conditions linked to HIV infection is referred to as HIV-associated vasculopathy. It is well recognised that the HIV (Human Immunodeficiency Virus) can impact the body's systems, including the cardiovascular system. Blood vessel illnesses or anomalies are referred to as vasculopathy.

2.2 HIV-associated vasculopathy is caused by multiple processes, including immunological dysfunction, inflammation, direct viral impact, and antiretroviral medication side effects (ART).

2.3 HIV-associated vasculopathy develops as a result of several factors:

2.3.1 HIV Infection: Endothelial cells can be directly infected by the HIV virus, which can cause endothelial dysfunction and blood vessel damage. This may set off inflammatory reactions and aid in the onset of vasculopathy.

2.3.2 HIV infection suppresses the immune system by reducing CD4+ T cells, which are essential for controlling the immunological response. People who have a compromised immune system may be more vulnerable to opportunistic infections and inflammatory reactions that may damage blood vessels.

2.3.3 Antiretroviral therapy (ART): Although ART has greatly improved the prognosis for HIV infection, certain antiretroviral medications have been linked to metabolic side effects such insulin resistance and dyslipidemia, which raise the risk of vasculopathy and cardiovascular disease.

2.3.4 Coinfections: People living with HIV are frequently more vulnerable to contracting hepatitis B, hepatitis C, and cytomegalovirus (HBV, HCV, and CMV). These co-infections have the potential to cause vasculopathy directly or to worsen immunological dysregulation and inflammation in the blood vessels.

Conventional Risk Factors: Smoking, high blood pressure, diabetes, and dyslipidemia are examples of traditional cardiovascular risk factors that may be more common in HIV-positive people. These risk factors raise the possibility of vasculopathy and worsen endothelial dysfunction.

3.HIV ASSOCIATED PERICARDITIS

3.1 HIV-associated pericarditis is a disorder in which HIV infection causes inflammation of the pericardium, the thin sac that surrounds the heart. It is one of the symptoms of problems brought on by HIV. Any stage of HIV infection, from acute infection to advanced stages of AIDS, can result in pericarditis.

3.2 HIV-associated pericarditis develops as a result of multiple factors:

3.2.1 HIV Infection: The HIV virus compromises immunity, increasing a person's vulnerability to opportunistic infections, such as pericarditis. HIV can directly induce inflammation in the pericardium and other organs.

3.2.2 Opportunistic Infections: Due to their compromised immune systems, people living with HIV are more vulnerable to opportunistic infections. HIV-positive people can develop pericarditis from infections such as TB, cytomegalovirus (CMV), and Mycobacterium avium complex (MAC).

3.2.3 Immune Reconstitution Inflammatory Syndrome (IRIS): People living with HIV may experience immune system recovery and reactivation following the initiation of antiretroviral therapy (ART). Pericarditis is one of the inflammatory disorders that can occasionally result from this immune reaction.

3.2.4 Direct Viral Effects: Pericarditis and inflammation can result from HIV infection that enters the pericardium directly. In patients with advanced HIV disease, direct viral impacts can cause pericardial inflammation, though they are less prevalent.

4.HIV ASSOCIATED ENDOCARDITIS

4.1 Infectious endocarditis (IE) in HIV-positive patients is referred to as HIV-associated endocarditis. Bacteria that enter the bloodstream and attach themselves to damaged parts of the heart are the usual cause of endocarditis, an infection of the inner lining of the heart chambers and heart valves.

4.2 HIV-positive people are more susceptible to developing endocarditis for a number of reasons:

4.2.1 Immunosuppression: HIV infection, especially in its later stages, causes a steady decline in the immune system. An increased vulnerability to several infections, including endocarditis, results from this compromised immune response.

4.2.2 Intravenous Drug Use (IVDU): Regardless of HIV status, injection drug use poses a serious risk for infective endocarditis. Nonetheless, the danger is significantly increased in HIV-positive intravenous drug users because of shared needles, weakened immune systems, and other risky behaviours.

4.2.3 Previous Valve Damage: The HIV virus itself has the potential to inflame and harm heart valves, especially the mitral valve. This injury may provide the right environment for endocarditis to develop.

4.2.4 Compliance with Antiretroviral Therapy (ART): Failure to follow ART regimens can result in unchecked HIV replication and the development of AIDS. The danger of opportunistic infections, such as endocarditis, rises with weakened immunity.

4.2.5 High-Risk Sexual Behaviour: Having intercourse with several partners without protection or other high-risk behaviours raises the chance of contracting STIs, some of which can result in endocarditis.

5.HIV ASSOCIATED MYOCARDITIS

5.1 HIV-associated myocarditis is the term used to describe inflammation of the heart's muscle tissue, or myocardium, in people who have HIV infection. Numerous conditions, such as autoimmune diseases, poisons, drug responses, and viral infections, can result in myocarditis. When it comes to HIV, the virus itself has the ability to either directly infect the heart or cause an inflammatory immunological response.

5.2 HIV-associated myocarditis, or HIV-related inflammation of the heart muscle, can result from a number of things:

5.2.1 Direct viral effects: Heart tissue damage and inflammation can result from HIV's direct infection of heart muscle cells. Myocarditis may occur as a result of this direct viral impact.

5.2.2 Immunological dysfunction: One of the hallmarks of HIV infection is immunological dysfunction, which includes a reduction in CD4+ T cell counts and functions, which are essential for organising the immune response. This malfunction may result in insufficient regulation of viral replication and heightened vulnerability to opportunistic infections, such as heart-related ones.

5.2.3 Co-existing conditions: Individuals with HIV frequently experience co-occurring conditions that increase their risk of myocarditis. These conditions include drug misuse, viral hepatitis, and other STDs.
5.2.4 HIV infection is associated with long-term inflammation and immunological activation, both of which increase the risk of cardiovascular problems, such as myocarditis. Over time, the heart muscle may sustain continuous injury due to persistent immunological activity.

6.HIV ASSOCIATED PAH

6.1 The term "HIV-associated pulmonary artery hypertension" (PAH) describes a type of PAH that affects people with HIV infection. High blood pressure in the arteries supplying blood to the lungs is a characteristic of PAH. In people with HIV infection, PAH can arise from a number of the virus's own causes as well as other aggravating variables like immunological compromise, inflammation, and opportunistic infections.
6.2 In HIV patients, a number of variables can lead to the development of PAH:
6.2.1HIV Infection: As a prelude to pulmonary hypertension, HIV infection itself can cause inflammation and endothelial dysfunction. Pulmonary arterial hypertension and vascular remodelling may be influenced by HIV-related chronic inflammation.
6.2.2 Antiretroviral Therapy (ART): Although ART has greatly enhanced HIV patients' prognosis and life expectancy, certain antiretroviral medications have been linked to a higher risk of pulmonary hypertension. Specifically, medications such as nucleoside reverse transcriptase inhibitors (NRTIs) and protease inhibitors have been connected to pulmonary hypertension.
6.2.3 Co-Infections: People with HIV frequently have opportunistic infections like pneumonia, TB, and cytomegalovirus (CMV). Pulmonary hypertension may worsen as a result of these infections' direct effects on the lungs and pulmonary vasculature.
6.2.4 Vasoactive chemicals: Endothelin-1 and thromboxane are two examples of vasoactive chemicals that may be more prevalent in HIV-positive people. These compounds can cause vasoconstriction and vascular remodelling in the lungs, which can result in PAH.
6.2.5 HIV-Associated Lung Disorders: By reducing lung function and raising pulmonary vascular resistance, HIV-associated lung disorders such as pneumocystis pneumonia (PCP), pulmonary fibrosis, and chronic obstructive pulmonary disease (COPD) can lead to the development of pulmonary hypertension.

7.HIV ASSOCIATED MYOCARDIAL INFRACTION (MI)

7.1 Heart attacks, or myocardial infarctions (MI), can be caused by a number of conditions, the most prevalent of which is coronary artery disease (CAD). Through a number of ways, HIV, the virus that causes AIDS, might indirectly raise the risk of myocardial infarction:
7.2 Chronic inflammation is brought on by HIV infection and affects every part of the body, including the blood vessels. An important risk factor for myocardial infarction is atherosclerosis, or the accumulation of plaque in the arteries, which might be facilitated by this persistent inflammation.
7.3 Effects on metabolism: dyslipidemia, or abnormal blood lipid levels, and insulin resistance are two metabolic side effects of several antiretroviral drugs used to treat HIV. These conditions can raise the risk of atherosclerosis and myocardial infarction.
7.4 Co-illnesses: HIV infection raises the chance of contracting hepatitis C virus (HCV) and cytomegalovirus (CMV), two infections that can also aggravate cardiovascular disease.

8.CONCLUSION

8.1 HIV-related cardiovascular problems have been a major worry ever since the epidemic began. The following are some significant findings from investigations and clinical trials on this subject:
8.1.1 Increased Risk of Cardiovascular Disease (CVD): Compared to the general population, HIV-positive people are more likely to develop cardiovascular conditions like peripheral arterial disease, myocardial infarction, atherosclerosis, and coronary artery disease.
8.1.2 Contributing Factors: The HIV virus itself, co-infections (like hepatitis C), immune activation, side effects of antiretroviral therapy (ART), chronic inflammation, immunological activation, and traditional cardiovascular risk factors (like smoking, dyslipidemia, hypertension, and diabetes) all raise the risk of cardiovascular disease (CVD) in people living with HIV.
8.1.3 Chronic inflammation and endothelial dysfunction are brought on by HIV infection and are important factors in the onset and advancement of atherosclerosis and other cardiovascular diseases.
8.1.4 Impact of Antiretroviral Therapy (ART): Although ART has extended the survival time of HIV-positive individuals, some antiretroviral drugs, especially older generation nucleoside reverse transcriptase inhibitors and protease inhibitors, have been linked to metabolic disorders like lipodystrophy, insulin resistance, and dyslipidemia, which raise the risk of cardiovascular disease (CVD).
8.1.5 Early Detection and Management: To reduce the risk of cardiovascular problems in HIV-positive persons, early detection and proactive management of traditional cardiovascular risk factors are crucial. This involves managing dyslipidemia, hypertension, and diabetes; regular cardiovascular risk assessment; and lifestyle changes (such as quitting smoking, maintaining a balanced diet, and engaging in regular exercise).
8.1.6 Screening Guidelines: Guidelines suggest routinely evaluating cardiovascular risk in HIV-positive persons, which includes blood pressure, glucose metabolism, lipid screening, and other cardiovascular risk factors. Furthermore, in some high-risk populations, screening for subclinical atherosclerosis with methods such as coronary artery calcium scoring or carotid intima-media thickness measurement may be appropriate.
8.1.7 Integrated Care: To effectively address both HIV-related and conventional cardiovascular risk factors, optimal management of cardiovascular complications in HIV-infected individuals frequently necessitates an integrated approach involving infectious disease specialists, cardiologists, and primary care providers.
8.1.8 Research That's Still Going On: Studies into the mechanisms causing cardiovascular problems in HIV-positive people as well as the creation of cutting-edge treatment plans to lower cardiovascular risk in this population are still going on.
8.1.9 To sum up, cardiovascular problems are still a major worry for people living with HIV. To lessen the burden of cardiovascular disease in this population, thorough risk assessment, early detection, and proactive therapy of modifiable risk factors are required.

9.REFRENCES

1.Palella FJ Jr, Delaney KM, Moorman AC, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. N Engl J Med. 1998;338:853-860.
2.Koutkia P, Grinspoon S. HIV-associated lipodystrophy: pathogenesis, prognosis, treatment, and controversies. Annu Rev Med. 2004;55:303-317.
3.Mocroft A, Ledergerber B, Katlama C, et al. Decline in the AIDS and death rates in the EuroSIDA study: an observational study. Lancet. 2003;362:22-29.
4.Sepkowitz KA. AIDS-the first 20 years. N Engl J Med. 2001;344:1764-1772.
5.Manfredi R, Sabbatani S, Agostini D. Trend of mortality observed in a cohort of drug addicts of the metropolitan area of Bologna, North-Eastern Italy, during a 25-year-period. Coll Antropol. 2006;30:479-488.
6.U.S. Public Health Service Task Force. Recommendations for use of antiretroviral drugs in pregnant HIV-1-infected women for maternal health and interventions to reduce perinatal HIV-1 transmission in the United States.
7.Wong MD, Shapiro MF, Boscardin WJ, et al. Contribution of major diseases to disparities in mortality. N Engl J Med. 2002;347:1585-1592.
8.El-Sadr WM, Lundgren JD, Neaton JD, et al. CD4+ count-guided interruption of antiretroviral treatment. N Engl J Med. 2006;355:2283-2296.
9.Emery S, and the SMART Study Group and INSIGHT. Major clinical outcomes in patients not treated with antiretroviral therapy (ART) at baseline in SMART: a rationale for a trial to examine early treatment of HIV disease. Presented at: 4th IAS Conference on HIV Pathogenesis, Treatment, and Prevention; 2007; Sydney, Australia. Abstract.
10.Choi AI, Rodriguez RA, Bacchetti P, et al. The impact of HIV on chronic kidney disease outcomes. Kidney Int. 2007;72:1380-1387.
11.Hsue, P. Y., & Waters, D. D. (2009). HIV infection and coronary heart disease: mechanisms and management. Nature Reviews Cardiology, 6(12), 33-42.
12.Triant, V. A., Lee, H., Hadigan, C., Grinspoon, S. K. (2007). Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. Journal of Clinical Endocrinology & Metabolism, 92(7), 2506-2512.
13.Paisible, A. L., Chang, C. C., So-Armah, K. A., et al. (2015). HIV infection, cardiovascular disease risk factor profile, and risk for acute myocardial infarction. Journal of Acquired Immune Deficiency Syndromes, 68(2), 209-216.

14.Freiberg, M. S., Chang, C. C., Kuller, L. H., & Skanderson, M. (2013). HIV infection and the risk of acute myocardial infarction. JAMA Internal Medicine, 173(8), 614-622.
15.Baker, J. V., Lundgren, J. D., & Sharma, S. (2020). Cardiovascular implications from untreated human immunodeficiency virus infection. European Heart Journal, 41(35), 3418-3429.
16.Post, W. S., Budoff, M., & Kingsley, L. (2003). Associations between HIV infection and subclinical coronary atherosclerosis. Annals of Internal Medicine, 158(11), 815-824.
17.Yarchoan R, Klecker RW, Weinhold KJ, Markham PD, Lyerly HK, Durack DT, et al. Administration of 3'-azido-3'-deoxythymidine, an inhibitor of HTLV-III/LAV replication, to patients with AIDS or AIDS-related complex. Lancet. 1986;1(8481):575-80.
18.Srinivasa S, Grinspoon SK. Metabolic and body composition effects of newer antiretrovirals in HIV-infected patients. Eur J Endocrinol. 2014;170(5):185-202.
19.Non LR, Escota GV, Powderly WG. HIV and its relationship to insulin resistance and lipid abnormalities. Transl Res. 2017 May;183:41-56.
20.Antiretroviral Therapy Cohort Colaborattion. Causes of death in HIV-1-infected patients treated with antiretroviral therapy, 1996-2006: collaborative analysis of 13 HIV cohort studies. Clin Infect Dis. 2010;15(10):1387-96.
21.Worm SW, De Wit S, Weber R, Sabin CA, Reiss P, El-Sadr W, et al. Diabetes mellitus, preexisting coronary heart disease, and the risk of subsequent coronary heart disease events in patients infected with human immunodeficiency virus: the Data Collection on Adverse Events of Anti-HIV Drugs (D:A:D Study). Circulation. 2009;119(6):805-11.
22.Vidrine DJ, Kypriotakis G, Li L, Arduino RC, Fletcher FE, Tami-Maury I, et al. Mediators of a smoking cessation intervention for persons living with HIV/AIDS. Drug Alcohol Depend. 2015 Feb 1;147:76-80.
23.da Silva CM, Mendoza-Sassi RA, da Mota LD, Nader MM, de Martinez AM. Alcohol use disorders among people living with HIV/AIDS in Southern Brazil: prevalence, risk factors and biological markers outcomes. BMC Infect Dis. 2017;17(1):263.
24.Dube MP, Lipshultz SE, Fichtenbaum CJ, Greenberg R, Schecter AD, Fisher SD, et al. Effects of HIV infection and antiretroviral therapy on the heart and vasculature. Circulation. 2008;118(2):e36-40.
25.Grunfeld C, Delaney JA, Wanke C, Currier JS, Scherzer R, Biggs ML, et al. Preclinical atherosclerosis due to HIV infection: carotid intima-medial thickness measurements from the FRAM study. AIDS. 2009;23(14):1841-9.
26.Kazemi T, Sharifzadeh GR, Zarban A, Fesharakinia A, Rezvani MR, Moezy SA. Risk factors for premature myocardial infarction: a matched case-control study. J. Res. Health Sci. 2011;11(2):77-82. Accessed April 17, 2023. https://pubmed.ncbi.nlm.nih.gov/22911956/
27.MOHAMMADIAN-HAFSHEJANI A, SALEHINIYA H, KHAZAEI S. Some Facts about Case Fatality of Acute Myocardial Infarction in Iran. Iran. J. Public Health. 2015;44(12):1718-1719. Accessed April 17, 2023. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4724752/
28.Mohammadreza Asgari, Fatemeh Alhani, Monireh Anoosheh. Risk Factors in Patients with Myocardial Infraction Hospitalized in Fatemieh Hospital in Semnan. Iran. J. Nurs. Res.. 2010;23(64):8-16. Accessed April 17, 2023. https://ijn.iums.ac.ir/browse.php?a_id=788&slc_lang=en&sid=1&printcase=1&hbnr=1&hmb=1
29.Kazemi T, Mohseni J, Maleki M, Beydokhti H. A systematic review on the prevalence of acute myocardial infarction in Iran. Heart Views. 2017;18(4):125. Doi:https://doi.org/10.4103/heartviews.heartviews_71_17

30.Firoozabadi MD, Kazemi T. A memorandum of "World Heart Day 2013" – Stroke mortality among women in Birjand, East of Iran. Iran. J. Nurs. Midwifery Res.. 2014;19(2):215. Accessed April 17, 2023. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4020034/
31.Kazemi T, Sharifzadeh G, Hosseinaii F. Epidemiology of Trend of Acute Myocardial Infraction in Birjand between 2002-2006 years. IJE. 2009;4(3):35-41. Accessed April 17, 2023. https://irje.tums.ac.ir/article-1-131-en.html
32.Kasiolkheir S, Ahmadizadeh C, Hosseinzadeh Mogbeli H. Studying MiR-133 influencing the process of apoptosis of Cardiomyocytes floating in blood serum of patients suffering from acute Myocardial Infarction. Dev. Biol.. 2019;11(3):37-46. Accessed April 17, 2023. https://jdb.ntb.iau.ir/article_667655.html?lang=en
33.Mensah GA, Roth GA, Fuster V. The Global Burden of Cardiovascular Diseases and Risk Factors. JACC. 2019;74(20):2529-2532. Doi:https://doi.org/10.1016/j.jacc.2019.10.009
34.Barbaro, G. 1999. Dilated cardiomyopathy in the acquired immunodeficiency syndrome. Eur. Heart J. 20: 629–630
35.Lipshultz, S.E. 1998. Dilated cardiomyopathy in HIV-infected patients. N. Engl. J. Med. 339: 1153–1155
36.Yunis, N.A. &V.E. Stone. 1998. Cardiac manifestations of HIV/AIDS: a review of disease spectrum and clinical management. J. AIDS Hum. Retrovirol.
37.Kasten-Sportes, C. & C. Weinstein. 1998. Molecular mechanisms of HIV cardiovascular disease. In Cardiology in AIDS. S.E. Lipshultz, Ed.: 265–282. Chapman & Hall. New York.
38.Hajjar LA, Calderaro D, Yu PC, Guiliano I, de Oliviera LEM, Barbaro G, Caramelli B: Cardiovascular manifestations in patients infected with the human immunodeficiency virus. Arq Bras Cardiol. 2005, 85: 363-377. 10.1590/S0066-782X2005001800013
39.Sani MU: Myocardial disease in human immunodeficiency virus (HIV) infection: a review. Wien Klin Wochenschr. 2008, 120: 77-87. 10.1007/s00508-008-0935-3.
40.Calabarese LH, Proffitt MR, Yen-Lieberman B, Hobbs RE, Ratliff NB: Congestive cardiomyopathy and illness related to the acquired immunodeficiency syndrome (AIDS) associated with isolation of retrovirus from myocardium. Ann Intern Med. 1989, 107: 691-692. 10.7326/0003-4819-107-5-691.
41.Fantoni M, Autore C, Del Borgo C: Drugs and cardiotoxicity in HIV and AIDS. Ann NY Acad Sci. 2001, 946: 179-199. 10.1111/j.1749-6632.2001.tb03912.x.
42.Hoffman M, Lipshultz SE, Miller TL: Malnutrition and cardiac abnormalities in the HIV-infected patients. Nutritional aspects of HIV infection. Edited by: Miller TL, Gorbach S. 1999, Arnold, London, 33-
43.Herskowitz A, Willoughby SB, Vlahov D, Baughman KL, Ansari AA: Dilated heart muscle disease associated with HIV infection. Eur Heart J. 1995, 16: 50-55. 10.1093/eurheartj/16.suppl_O.50.
44.Frustaci A, Chimenti C, Calabrese F, Pieroni M, Thiene G, Maseri A: Immunosuppressive therapy for active lymphocytic myocarditis: virological and immunologic profile of responders versus nonresponders. Circulation. 2003, 107: 857-863. 10.1161/01.CIR.0000048147.15962.31.
45.Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, Fu M, Heliö T, Heymans S, Jahns R, Klingel K, Linhart A, Maisch B, McKenna W, Mogensen J, Pinto YM, Ristic A, Schultheiss HP, Seggewiss H, Tavazzi L, Thiene G, Yilmaz A, Charron P, Elliott PM, European Society of Cardiology Working Group on Myocardial and Pericardial Diseases: Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013, 34 (33): 2636-2648. 10.1093/eurheartj/eht210.
46.Kühl U, Lassner D, von Schlippenbach J, Poller W, Schultheiss HP: Interferon-Beta improves survival in enterovirus-associated cardiomyopathy. J Am Coll Cardiol. 2012, 60: 1295-1296. 10.1016/j.jacc.2012.06.026.
47.Drucker NA, Colan SD, Lewis AB, Beiser AS, Wessel DL, Takahashi M, Baker AL, Perez-Atayde AR, Newburger JW: Gamma-globulin treatment of acute myocarditis in the pediatric population. Circulation. 1994, 89: 252-257. 10.1161/01.CIR.89.1.252.
48.Schultheiss HP, Kühl U, Cooper LT: The management of myocarditis. Eur Heart J. 2011, 32: 2616-2625. 10.1093/eurheartj/ehr165.
49.UNAIDS. The Report on the Global HIV/AIDS Epidemic: Joint United Nations programme on HIV/AIDS [online]. Available from URL: http://www.unaids.org [Accessed 2002 Jul 4]
50.Centers for Disease Control and Prevention. HIV AIDS Surveill Rep 1999; 11(1): 143. Available from URL: http://www.cdc.gov/hiv/pubs/facts.htm [Accessed 2002 Jul 4]
51.McKinney Jr RE. Shooting at a moving target: natural history studies and the rapidly improving state of anti-HIV treatment. J Pediatr 2002; 141(3): 301–2
52.Lipshultz SE, Fisher SD, Lai W, et al. Cardiac monitoring and therapy for HIV-infected patients. Ann N Y Acad Sci 2001; 946: 236–73
53.Lipshultz SE, Fisher SD, Lai W, et al. Cardiac monitoring and therapy for HIV-infected patients. Ann N Y Acad Sci 2001; 946: 236–73
54.Heidenreich PA, Eisenberg MJ, Kee LL, et al. Pericardial effusion in AIDS: incidence and survival. Circulation 1995; 92: 3229
55.Carrillo-Jimenez R, Lamas GA, Henneken CH. Plasma levels of brain natriuretic peptide: a potential marker for HIV-related cardiomyopathy. J Cardiovasc Pharmacol Ther 2002; 7(3): 135–7
56.Freiberg, M. S., Chang, C. C. H., Kuller, L. H., Skanderson, M., Lowy, E., Kraemer, K. L., … Justice, A. C. (2013). HIV infection and the risk of acute myocardial infarction. JAMA Internal Medicine, 173(8), 614–622. https://doi.org/10.1001/jamainternmed.2013.3728
57.Triant, V. A., Lee, H., Hadigan, C., & Grinspoon, S. K. (2007). Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. The Journal of Clinical Endocrinology & Metabolism, 92(7), 2506–2512. https://doi.org/10.1210/jc.2006-2190
58.Hsue, P. Y., Lo, J. C., Franklin, A., Bolger, A. F., Martin, J. N., Deeks, S. G., & Waters, D. D. (2004). Progression of atherosclerosis as assessed by carotid intima-media thickness in patients with HIV infection. Circulation, 109(13), 1603–1608. https://doi.org/10.1161/01.CIR.0000124480.32233.8A
59.Freiberg, M. S., & Feinstein, M. J. (2018). HIV and cardiovascular disease: We need a mechanism, and we need a plan. Journal of the American Heart Association, 7(9), e010762. https://doi.org/10.1161/JAHA.118.010762

60.Barbaro, G., Di Lorenzo, G., Grisorio, B., Barbarini, G. (2003). Cardiac involvement in the acquired immunodeficiency syndrome: A multicenter clinical-pathological study. AIDS Research and Human Retroviruses, 19(4), 319–325. https://doi.org/10.1089/088922203321280800
61.Lewis, W., Kohler, J. J., Hosseini, S. H., Haase, C. P., Copeland, W. C., Bienstock, R. J., & Ludaway, T. (2006). Antiretroviral nucleosides, deoxynucleotide carrier and mitochondrial DNA: Evidence supporting the DNA pol γ hypothesis. AIDS, 20(5), 675–684. https://doi.org/10.1097/01.aids.0000216366.39732.2d
62.Twagirumukiza, M., Nkeramihigo, E., Seminega, B., Gasakure, E., Boccara, F., & Barbaro, G. (2010). Prevalence of dilated cardiomyopathy in HIV-infected African patients not receiving HAART: A multicenter, observational, prospective, cohort study in Rwanda. Current HIV Research, 8(7), 504–509. https://doi.org/10.2174/157016210793499176
63.Levy, A., Schiller, N. B., & Schwartz, R. S. (1996). Valvular heart disease in the acquired immunodeficiency syndrome. The American Journal of Cardiology, 77(14), 1184–1187. https://doi.org/10.1016/S0002-9149(96)00120-3
64.Steffens, F. T., Aurora, L., Menasche, P., Liss, H., Kierzek, G., & Slama, M. A. (2003). Valvular heart disease in patients infected with human immunodeficiency virus. The American Journal of Cardiology, 92(2), 292–295. https://doi.org/10.1016/S0002-9149(03)00615-1
65.Nkomo, V. T., Gardin, J. M., Skelton, T. N., Gottdiener, J. S., Scott, C. G., & Enriquez-Sarano, M. (2006). Burden of valvular heart diseases: A population-based study. The Lancet, 368(9540), 1005–1011. https://doi.org/10.1016/S0140-6736(06)69208-8