Acute myocardial infarction (AMI) in a patient with Human Immunodeficiency Virus infection

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ABSTRACT

Introduction: Recent studies have shown that in HIV patients, the incidence of acute myocardial infarction is up to two times higher than in people who are not infected with HIV. HIV patients presenting with the first episode of the acute coronary syndrome are, on average, a decade younger than the general population, more frequently in men and current smokers. Case Report: We report a case report of a 33-year-old man with a diagnosis of anteroseptal STEMI and recent HIV on first-line ART, a combination of zidovudine 300 mg and lamivudine 150 mg, plus efavirenz 600 mg. The patient had typical angina complaints and had risk factors, namely smoking and a history of dyslipidemia. Patients were treated according to acute coronary syndrome guidelines, given DAPT, heparinization for five days, statins, anti-ischemia, ACE-I, and DCA-PCI. The DCA-PCI result in this patient was CAD 1VD with 1 DES installed in the proximal LAD (complete revascularization). The patient was discharged from the hospital after seven days of treatment. Prognosis in these patients is still quite good, but preventing recurrent myocardial infarction needs risk factors and control for successful myocardial infarction therapy and ART. Conclusion: Special attention should be given to the risk of myocardial in HIV patients. Early diagnosis and prompt treatment could significantly lower mortality and improve patients’ quality of life.

Keywords: Acute Myocardial Infarction, HIV, STEMI, anti-retroviral therapy.


INTRODUCTION

In recent years, there has been an increase in knowledge about cardiovascular diseases associated with the human immunodeficiency virus (HIV). Recent studies have shown that in HIV patients, the incidence of acute myocardial infarction is up to two times higher than in people who are not infected with HIV.1,2 In fact, a cohort study presented the results that HIV patients who did not have risk factors for cardiovascular disease were twice as likely to be infected with HIV. Acute myocardial infarction occurs compared to patients with cardiovascular disease risk factors but without HIV infection.3 HIV patients presenting with the first episode of the acute coronary syndrome are, on average, a decade younger than the general population, more frequently in men and current smokers.4,5

Myocardial infarction is one of the causes of death in HIV patients. Atherosclerosis remains the main basis for the pathogenesis of coronary artery disease in HIV patients, which can lead to myocardial infarction. HIV infection itself plays a role in triggering atherosclerosis through inflammatory mechanisms, endothelial dysfunction and coagulation disorders, but this mechanism is not clear. HIV-associated atherosclerosis can be further complicated by antiretroviral therapy (ART), drug abuse, and traditional atherosclerosis risk factors (sedentary life, smoking, obesity, hypertension, dyslipidemia, chronic kidney disease and so on). The effects of certain ART can lead to dyslipidemia, insulin resistance, and endothelial dysfunction that may contribute to the development of atherosclerosis in HIV patients.6,7

Current data suggest that the management of myocardial infarction in HIV patients is similar to that of patients without HIV infection. Recent studies have shown no significant difference in the rate of revascularization of target vessels after stent implantation between HIV and non-HIV patients. However, further studies are needed to understand the critical pathways in the pathogenesis of atherosclerosis in HIV patients, the role of aggressive primary and secondary preventive measures for the disease, and the role of current antiretroviral therapy.5,8

CASE REPORT

A male patient with the initials DA, aged 33 years, came to the emergency department with complaints of left chest pain four days before being admitted to the hospital. Chest pain occurs suddenly when the patient rests for more than 20 minutes. Chest pain felt like a heavy load had been hit, radiated to the left arm, neck, and jaw and penetrated to the back. Pain does not improve with rest and is accompanied by cold sweats. At that time, the patient did not go to the hospital to seek first aid and just let the chest pain subside on its own. After that, the patient still felt intermittent chest pain with a duration of ± 5 minutes, but it subsided on its own. Four days later, the chest pain reappeared and got worse when the patient was active, so the patient
went to the emergency room for a check-up.

The patient has smoked since he was young, approximately one pack/day. The patient also has a habit of consuming junk food. The patient had a history of ART treatment for the last two years. After accidentally doing a health check, the patient tested positive for human immunodeficiency virus (HIV). The ART he is taking is a combination of zidovudine 300 mg and lamivudine 150 mg, plus efavirenz 600 mg. None of the patient’s family had a history of HIV, but his mother had a history of hypertension, and his grandfather had a history of heart disease.

On physical examination, the general condition was moderately ill with comatosus consciousness. Height 180 cm, weight 77 kg, with a body mass index of 23.76 kg/m2. Blood pressure 120/70 mmHg, pulse rate 100 beats per minute, respiratory rate 20 breaths per minute, body temperature 36.7˚C, with oxygen saturation of 98% without oxygen supplementation. On examination of the head and neck, the conjunctiva was not anemic; the sclera was not icteric; the jugular venous pressure was 5+1 cm H2O. On thoracic examination, it looks symmetrical; the left chest wall movement is the same as the right. On physical examination of the lungs, the left fremitus was the same as the right; the left pulmonary resonant percussion was the same as the right. Auscultation of vesicular breath sounds in the left and right lungs; no rhonchi and wheezing were found. On physical examination of the heart, ictus cordis is visible at the fifth intercostal space, the left midclavicular line, the right heart border is in the right sternal border, and the left heart border is in the left midclavicular line. On auscultation, the first and second heart sounds were normal, with a frequency of 100 beats per minute, regular. There are no murmurs or gallops. On abdominal examination, inspection looks flat, and auscultation reveals normal bowel sounds, gentle palpation, and no liver or spleen enlargement. The extremities were warm, the capillary refill time was less than 2 seconds, and no edema was found in both legs.

On the ECG examination (Appendix 1), it was found sinus rhythm, heart rate 84 beats per minute, axis -7°, normal P wave, PR interval 0.16 seconds, QRS wave width 0.12 seconds, pathological Q wave in V1- V3, ST-segment elevation of 1 mm in V1, 2 mm in V2, and 1.5 mm in V3, and a biphasic T wave in V4. The conclusion of the ECG examination in this patient was sinus rhythm, normo-axis, and the impression of anteroseptal myocardial infarction.

In laboratory examination on July 29, 2021, hemoglobin levels were 14.6 g/dL, leukocytes 4,000/μL, platelets 217,000/μL, hematocrit 42.1%, SGOT 52 U/L, SGPT 42 U/L, urea 23 mg/dL, creatinine 0.8 mg/dL, sodium 139 mEq/L, potassium 4.91 mEq/L, chloride 100.8 mEq/L, CKMB 66 U/L, creatinine 120 mg/dL, SGOT 78 U/L, SGPT 42 U/L, PT 12.1 seconds, aPPT 28 seconds and INR 0.89 seconds. The patient had a chest X-ray (Appendix 2), and the results of his expertise by a radiology specialist were that the heart and lungs were within normal limits.

Based on the history, physical examination, EKG, and laboratory tests, the patient was hospitalized with a working diagnosis of recent ST elevation Myocardial Infarction (STEMI), Anteroseptal Thrombolysis in Myocardial Infarction (TIMI) 2/14 Killip I onset four days. At the beginning of hospitalization, the patient was given fondaparinux therapy 1 x 2.5 mg subcutaneously for five days, acetylsalicylic acid 320 mg followed by 1 x 80 mg orally, clopidogrel 300 mg followed by 1 x 75 mg orally, atorvastatin 1 x 40 mg orally, bisoprolol 1 x 1.25 mg, glycercyl trinitrate 2x2.5 mg orally, captopril 3x6.25 mg orally, lansoprazole 2x30 mg orally, and lactulose syrup 2x10 cc orally. The patient was planned for diagnostic coronary angiography, percutaneous coronary intervention (DCA stands by PCI), and echocardiography.

Laboratory examinations on July 30, 2021, obtained results, namely hemoglobin 14.7 g/dL, leukocytes 4,700/μL, platelets 173,000/μL, hematocrit 44.7%, magnesium 1.94 mg/dL, calcium 9.25 mg/dL, fasting blood sugar 71 mg/dL, HbA1C 5.1%, uric acid 5.6 mg/dL, albumin 4.17 g/dL, total cholesterol 179 mg/dL, triglycerides 326 mg/dL, high density lipoprotein (HDL) 42 mg/dL.

Figure 1. Results of the patient’s early percutaneous coronary intervention.
25 mg/dL, low density lipoprotein (LDL) 89 mg/dL, and D-Dimmer 0.37 g/mL. On the second day of treatment, there was a change in therapy, namely captopril was replaced with ramipril 1x2.5 mg orally.

Echocardiographic examination (Appendix 3) on August 2, 2021, obtained results, namely normal cardiac chamber dimensions, no left ventricular hypertrophy, adequate left ventricular systolic function, 48% ejection fraction, hypokinetic left ventricular segmental analysis mid anteroseptal, anterior, apico-septal, apico-anterior other segments normo-kinetic, mild mitral valve regurgitation, other heart valves within normal limits, normal right ventricular contractility, tricuspid annular plane systolic excursion (TAPSE) 2.0 cm, inferior vena cava diameter 1.6 cm, collapsibility > 50%, and an estimated right atrial pressure of 3 mmHg.

On August 3, 2021, the patient underwent DCA stand-by PCI. The results of DCA stand by PCI that has been done are 80-90% tubular stenosis in the proximal left anterior descending (LAD), while the other coronary arteries are normal. The conclusion of this action is coronary artery disease (CAD) 1 vessel disease (VD) with one drug-eluting stent (DES) installed in the proximal LAD.

On August 5, 2021, a postoperative DCA-PCI laboratory examination was carried out. The results obtained hemoglobin levels of 14.7 g/dL, leukocytes 4,400/μL, platelets 169,000/μL, hematocrit 45.9%, urea 23 mg/dL, creatinine 0.9 mg/dL, sodium 139 mEq/L, potassium 4.57 mEq/L, chloride 98.4 mEq/L. The patient also underwent a 6-minute walk test with 8.07 METs. The patient was then discharged from the hospital after seven days of treatment and received outpatient therapy, namely acetylsalicylic acid 1x80 mg orally, clopidogrel 1x75 mg orally, lansoprazole 2x30 mg orally.

DISCUSSION

HIV-infected patients have an increased risk of cardiovascular disease. Several observational cohort studies have demonstrated a twofold increase in the incidence of acute myocardial infarction in HIV-infected patients compared to HIV-uninfected patients.4 HIV patients experience the first episode of acute myocardial infarction, an average of a decade younger than the general population. Most occur in men, active smokers with low HDL, and single vessel coronary heart disease. STEMI is the most common acute coronary syndrome in HIV patients.5 In this case, a 33-year-old male patient, an active smoker, has a low HDL of 25 mg/dL, with a recent diagnosis of anteroseptal STEMI and coronary angiography results indicating single vessel disease, namely LAD.

Atherosclerosis-related cardiovascular disease, including myocardial infarction and stroke, is currently one of the leading causes of death in HIV patients. Evidence suggests that HIV infection and subsequent inflammatory processes in humans accelerate atherosclerosis. However, the mechanism remains unclear.5,9 The etiopathogenesis of atherosclerosis in HIV patients is secondary to a highly complex interaction between many factors. Over the years, it has been well recognized that there is an increasing prevalence of traditional risk factors in HIV patients. In addition to traditional risk factors (age, family history, dyslipidemia, hypertension, smoking), other factors contribute to the development of atherosclerosis in HIV patients such as direct HIV-induced damage to the endothelium, chronic inflammation associated with long-term immune system activity, and even possible direct effect of ART.5,10 In this case, the patient had been taking ART for two years, the patient was an active smoker since age, but there was no history of HIV in his family.

The immune system has an important role in the development and progression of atherosclerosis in HIV patients. Higher levels of inflammatory markers, such as c-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor (TNF), were found in HIV patients compared to the normal population. The risk of myocardial infarction increases more than 4-fold in HIV patients with elevated CRP levels compared to the normal population.11,12

HIV infection has been postulated to alter the balance of immune cell subsets, such as intermediate monocytes and CD4 helper T cells, in a proinflammatory and pro-atherosclerotic manner.13 Even patients on ART routinely with complete virological suppression still have the residual risk for coronary heart disease due to inflammation and activation; the immune system is not completely suppressed.14 Neutrophil extracellular traps, which are chromatin structures secreted from neutrophils, have been identified as important in clearing pathogens from the circulation. However, the extracellular trapping of neutrophils has now also been shown to induce proatherogenic and prothrombotic effects and may promote atherosclerosis.15 Endothelial dysfunction, which is known to be associated with atherosclerosis, has been described in many HIV patients.5 There is a complicated interaction between endothelial function and inflammation; for example, markers of endothelial activation, such as intercellular adhesion molecule-1 and vascular cell adhesion molecule-1, both regulated by inflammatory cytokines, are elevated early after HIV infection.16

Coagulation abnormalities are common in HIV infection. These include elevated levels of D-dimer, fibrinogen, factor VII, von Willebrand factor, soluble thrombomodulin, and tissue factor.17 Another mechanism by which HIV infection can alter the coagulation pathway is the activation of tissue factors. Tissue factor levels, measured four months before a cardiovascular disease event, were elevated compared with tissue factor levels in HIV patients who did not have cardiovascular disease events.18 Thrombophilia is more common in HIV patients with lower protein C levels and higher factor VII levels and those with higher anticardiolipin and anti-thrombin antibodies.19 HIV also causes chronic platelet activation, which can increase atherogenesis and the risk of thrombosis.5

Atherosclerosis-associated coronary heart disease in HIV patients is complicated by an increase in traditional cardiovascular risk factors, or HIV infection itself, and the side effects of antiretroviral therapy.” The relative incidence of myocardial infarction in HIV patients receiving antiretroviral therapy increased by 26% per year in
the study. Data Collection on Adverse Events of Anti-HIV Drugs. ART-induced lipid abnormalities are characterized by elevated levels of total cholesterol, LDL, VLDL, triglycerides, and apolipoprotein B and are accompanied by low HDL levels. This usually occurs three months after starting ART.20

Many studies have evaluated whether therapy with nucleoside reverse transcriptase inhibitors (NRTIs) is associated with an increased risk of myocardial infarction. The Data Collection study on Adverse Events of Anti-HIV Drugs reported an increased risk of myocardial infarction in patients treated with abacavir or exposed to this drug in the previous six months. However, no significant increase in the incidence of myocardial infarction was observed in HIV patients treated with zidovudine, stavudine, or lamivudine.20 The incidence of cardiovascular disease was increased in HIV patients on abacavir-lamivudine combination therapy compared with HIV patients on tenofovir-emtricitabine combination therapy in the STEAL study.21

NRTI-induced lipid abnormalities were reported to be lower than protease inhibitor-induced lipid abnormalities. The combination of tenofovir with emtricitabine or lamivudine was associated with reduced total cholesterol, triglycerides, LDL, HDL, and non-HDL compared to other NRTI combinations.20,22 Increased LDL was reported in patients treated with didanosine and lamivudine, whereas the highest triglyceride levels were found in patients treated with stavudine and lamivudine. Lipodystrophy may occur as a result of NRTI therapy. Inhibition of mitochondrial DNA replication and transcription has been found in HIV patients on NRTI therapy.20


A report from the Data Collection on Adverse Events of Anti-HIV Drugs shows that treatment with NNRTIs causes a significant increase in triglycerides. Higher total cholesterol and triglyceride levels were reported in treatment with efavirenz than with nevirapine. However, NNRTIs can also increase HDL. A greater increase in HDL and a decrease in the triglyceride-HDL ratio was reported in patients treated with NNRTIs compared to protease inhibitors. Previous studies reported that efavirenz and nevirapine showed an HDL-enhancing effect.20 Analysis of the Data Collection on Adverse Events of Anti-HIV Drugs study analysis reported an increase in total cholesterol in patients treated with an NNRTI in combination with an NRTI, a protease inhibitor, or both compared with patients who did not treated.20,21

In this case, information was obtained that the patient had been an active smoker since he was young; the patient only found out that he was infected with HIV 2 years ago and immediately started ART. This information cannot be concluded with certainty when the patient was infected with HIV for the first time. The ART used by the patient was a combination of zidovudine and lamivudine plus efavirenz. Zidovudine and efavirenz have increased triglyceride and LDL levels, whereas lamivudine has a neutral effect on lipids. These three drugs do not affect glucose metabolism.5,24 Dyslipidemia in these patients could be influenced by the habit of consuming junk food, HIV infection, and the effects of antiretroviral therapy. Endothelial dysfunction that occurs in patients can be caused by HIV infection, the effects of antiretroviral therapy, and the negative impact of smoking from a young age. A myocardial infarction that occurred in this patient can be assumed to be multifactorial, in the form of traditional risk factors for myocardial infarction (smoking, dyslipidemia), the impact of HIV infection, and the negative effects of ART.5

Most protease inhibitors inhibit statin metabolism and can significantly increase statin levels, thereby increasing the risk of toxicity.5 Atorvastatin is less lipophilic, reducing its need for extensive hepatic metabolism. A significant increase in the area under the curve (AUC) of atorvastatin was observed after PI administration.

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Antiretroviral</th>
<th>Effects on lipids*</th>
<th>Effects on glucose*</th>
<th>Impact on coronary artery disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nucleoside reverse</td>
<td>Abacavir</td>
<td>TC↑↑ LDL↑↑</td>
<td>No effect</td>
<td>Recent exposure associated with</td>
</tr>
<tr>
<td>reverse transcriptase</td>
<td></td>
<td></td>
<td></td>
<td>increased risk for MI (controversial)</td>
</tr>
<tr>
<td>inhibitors</td>
<td>Azidothymidine</td>
<td>TC↑↑ LDL↑↑</td>
<td>Neutral effect</td>
<td>No association with increased risk for MI</td>
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<tr>
<td>Emtricitabine</td>
<td>Lamivudine</td>
<td>Neutral effect</td>
<td>No effect</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>Stavudine</td>
<td>Tenofovir</td>
<td>Dyslipidemia+</td>
<td>Insulin resistance+</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>Non-nucleoside reverse</td>
<td>Efavirenz</td>
<td>TC↑↑ LDL↑↑</td>
<td>No effect</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>reverse transcriptase</td>
<td>Nevirapine</td>
<td>HDL↑↑</td>
<td>Neutral effect</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>inhibitors</td>
<td>Rilpivirine</td>
<td></td>
<td></td>
<td>No data available (not enough patients exposed)</td>
</tr>
<tr>
<td>Protease inhibitors</td>
<td>Amprenavir +</td>
<td>Dyslipidemia+++</td>
<td>Insulin resistance+</td>
<td>Cumulative exposure independently increased risk for MI</td>
</tr>
<tr>
<td>Ritonavir</td>
<td>Atazanavir +</td>
<td>Dyslipidemia+++</td>
<td>Insulin resistance+</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>Ritonavir</td>
<td>Darunavir +</td>
<td>Dyslipidemia+++</td>
<td>Insulin resistance+</td>
<td>No data available (not enough patients exposed)</td>
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<tr>
<td>Ritonavir</td>
<td>Indinavir</td>
<td>Dyslipidemia+++</td>
<td>Insulin resistance+++</td>
<td>Cumulative exposure independently increased risk for MI</td>
</tr>
<tr>
<td>Lopinavir + Ritonavir</td>
<td>Nelfinavir</td>
<td>Dyslipidemia++++</td>
<td>Insulin resistance++</td>
<td>No association with increased risk for MI</td>
</tr>
<tr>
<td>Saquinavir</td>
<td>Tipranavir +</td>
<td>Dyslipidemia++++</td>
<td>Insulin resistance++</td>
<td>No data available (not enough patients exposed)</td>
</tr>
<tr>
<td>Ritonavir</td>
<td>Integrase inhibitors</td>
<td>Neutral effect</td>
<td>No effect</td>
<td>No data available (not enough patients exposed)</td>
</tr>
<tr>
<td>Eltandefavir/tuboxacin</td>
<td>Maraviroc</td>
<td>Neutral effect</td>
<td>No effect</td>
<td>No data available (not enough patients exposed)</td>
</tr>
<tr>
<td>Raltegravir</td>
<td></td>
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</table>

*Dyslipidemia defined as increased total cholesterol (TC), low-density lipoprotein cholesterol (LDL), triglycerides and decreased high-density lipoprotein cholesterol (HDL). +, weak effect; ++, moderate effect; ++++, important effect; #: increase; ↓, decrease. MI, myocardial infarction.

Figure 2. Major classes of ARVs and their impact on lipids, glucose metabolism and coronary artery disease.
Atorvastatin should be started at the lowest dose and titrated to achieve target lipid levels. Doses of atorvastatin exceeding 20 mg warrant special attention in HIV patients treated with protease inhibitors. In contrast, CYP3A4 inducers, e.g., efavirenz, may decrease the serum concentration of atorvastatin. Serum lipid concentrations should be evaluated before starting ART, at 3–6 months after initiation, and then annually in the absence of abnormalities. In addition to lipids, blood pressure control and glucose metabolism are also needed. A reduction in systolic blood pressure of 10 mmHg, a reduction in total cholesterol every one mmol/L (39 mg/dL), and the use of acetylsalicylic acid each reduced the risk of ischemic heart disease by 20–25%. Low-dose acetylsalicylic acid in HIV patients has been shown to increase platelet activation and immune activation in HIV patients on ART. It is attenuated after one week of acetylsalicylic acid therapy. Smoking cessation is strongly recommended because the life expectancy of HIV-infected smokers is, on average, eight years less compared to smokers who are not infected with HIV.

Management of acute myocardial infarction in HIV patients is similar to treatment in patients not infected with HIV. Still, it is important to be aware of the potential interactions of cardiovascular drugs with antiretroviral therapy. Antiplatelets are usually used in patients with coronary heart disease. CYP2C19 and CYP2C9 significantly metabolize clopidogrel, so there is an interaction with ART. Prasugrel can be used as an alternative but should not be combined with ritonavir as this will decrease prasugrel activity. Ticagrelor, predominantly metabolized by the CYP3A4/5 isoenzyme, should not be used in patients receiving protease inhibitors therapy. HIV patients also often have coagulopathy and thrombocytopenia, which increase the risk of bleeding, so the administration of antithrombotics should be more careful.

Recent studies have shown no significant difference in the rate of revascularization of target vessels after stent implantation between HIV patients and non-HIV patients. DES appears to be equally safe and efficacious in patients with and without HIV. Several studies have shown a higher incidence of stent thrombosis in HIV patients than in patients without HIV infection. Coronary artery bypass grafts (CABG) were feasible, and perioperative complications were no different compared to patients without HIV, even when CD4 cell count <500 cells/mm3.

In this case, DCA-PCI results were obtained: single vessel disease with 80-90% tubular stenosis in proximal LAD, and 1 DES was installed. Based on the explanation above, there is no difference in revascularization in HIV patients with myocardial infarction and those without HIV infection. The patient received double antiplatelet therapy (DAPT) with acetylsalicylic acid 1x80 mg orally and clopidogrel 1x75 mg orally. Efavirenz has been shown to reduce the effects of clopidogrel, while acetylsalicylic acid is safe and has no interaction with ART. Other therapies in this patient were atorvastatin 1x40 mg orally, bisoprolol 1x1.25 mg orally, ramipril 1x2.5 mg orally, and lansoprazole 2x30 mg orally. Efavirenz can cause a decrease in serum atorvastatin concentrations, so close monitoring of lipid levels is necessary for these patients. Efavirenz may also reduce the effects of bisoprolol, while all types of angiotensin-converting enzyme inhibitors (ACE-I), including ramipril, have no interaction with ART and can be used at standard doses in HIV patients.

As HIV infection becomes a chronic disease, the risk of death from acquired immunodeficiency syndrome (AIDS) is also decreased. However, the risk of death from cardiovascular disease increase in HIV-infected adults. Important risk factors for death in HIV patients were chronic kidney disease, NRTI-sparing therapy, low ejection fraction, and CD4 count <200 cells/mm3. Recurrent myocardial infarction after acute coronary syndrome was higher in HIV patients than in patients without HIV infection. In this case, the patient did not have comorbid chronic kidney disease, the ejection fraction was still quite good, the patient had been on ART for two years, and had a medical examination. CD4 levels with results still in the normal range. So far, the prognosis in this patient is still quite good, but in the future, the patient needs to control risk factors, such as quitting smoking, a low-fat diet, and adequate physical activity. Patients also need to be monitored for the success of myocardial infarction therapy and ART, such as evaluation of complaints, lipid profile, blood sugar, blood pressure, echocardiographic evaluation every six months, and CD4 levels.

CONCLUSION

This article reported a case of a 33-year-old man diagnosed with anteroseptal STEMI and HIV who was recently on first-line ART, i.e., a combination of zidovudine 300 mg and lamivudine 150 mg, plus efavirenz 600 mg. The patient had typical angina complaints and had risk factors, namely smoking and a history of dyslipidemia. The patient was treated according to acute coronary syndrome guidelines, given DAPT, heparinization for five days, statins, anti ischemia, ACE-I, and DCA-PCI were performed. The DCA-PCI result was CAD 1 VD with 1 DES at proximal LAD (complete revascularization). The patient was discharged from the hospital after seven days of treatment. The prognosis in this patient is still quite good. Still, it is necessary to control risk factors and monitor for successful myocardial infarction therapy and ART to prevent recurrent myocardial infarction.

CONFLICT OF INTEREST

All authors declared that there is no conflict of interest regarding this article.

ETHICS APPROVAL

Permission has been obtained from the patient, the cardiology department head, and Prof. Dr. R. D. Kandou Manado general hospital regarding the required data for this case report.

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None

AUTHOR CONTRIBUTION

All authors contributed equally to the writing of this article.
REFERENCES

Appendix 1. A. Patient’s ECG when admitted to the hospital; B. Patient’s ECG at the seventh day after therapy.
Appendix 2. Patient's Chest X-Ray performed at admission
Appendix 3. The result of patient’ echocardiography