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Wellens' Syndrome in a HIV-positive Patient: A Case Report

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Abstract Patients with human immunodeficiency virus (HIV) are at higher risk for coronary artery disease, due to accelerated atherosclerosis resulting from chronic inflammation, the prevalence of cardiovascular risk factors and the side effects of highly active antiretroviral therapy (HAART). The Wellens' pattern is an electrocardiographic (ECG) finding that represents critical proximal left anterior descending (LAD) coronary artery stenosis that, that when is not promptly treated, can lead to extensive anterior wall myocardial infarction and death. Very few cases of Wellens' syndrome in HIV positive patients have been reported. We present a case of Wellens' syndrome in a 38-year-old male with HIV on HAART and hyperlipidemia, as his only traditional cardiovascular risk factor. Recognition of the characteristic biphasic T-waves in V2 and V3 on ECG in the setting of typical angina and elevated troponin levels directed the clinicians to proceed with an emergent cardiac catheterization and percutaneous coronary intervention with drug eluting stent placement in the proximal left anterior descending artery (LAD). Physicians should recognize Wellens' syndrome as it indicates critical LAD stenosis requiring intervention. HIV positive patients can present with Wellens' sign at a younger age, indicating premature coronary artery disease (CAD) in this population.

Keywords: *human immunodeficiency virus, HIV, Wellens syndrome, premature coronary artery disease, biphasic T-waves, chronic inflammation, critical left anterior descending stenosis, dyslipidemia*

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1. Introduction

Wellens' syndrome, also known as left anterior descending (LAD) coronary T-wave syndrome, is an electrocardiographic (ECG) pattern associated with critical, proximal LAD artery stenosis. On ECG these patients lack Q waves and significant ST-segment elevations and have normal precordial R wave progression however, demonstrate T-wave changes in the anterior chest leads with deeply inverted T-waves or biphasic T-waves. The T-wave abnormalities may persist for hours to weeks and be found in asymptomatic patients [1]. These changes, however, are associated with significant obstruction in the proximal LAD such that patients with Wellens' syndrome are at high risk for extensive anterior wall myocardial infarction and death within a few weeks after presentation [2]. Thus, identifying pathognomonic ECG changes and patients at risk for Wellens' syndrome is important because urgent coronary angiography is necessary to evaluate the utility of angioplasty or possibly coronary bypass surgery [3]. Even though total mortality in HIV infected patients has decreased in recent years, their cardiovascular disease (CVD) mortality has significantly increased over the same period [4]. HIV infected patients are at increased risk of acute myocardial infarction, perhaps due to chronic inflammation and antiretroviral therapy side effects [5,6]. Only a few cases of Wellens' syndrome have been reported in HIV infected patients [7,8,9]. We present a case of Wellens' syndrome in a 38 year old man with HIV.

2. Case Report

A 38-year -old male with a past medical history of syphilis, HIV on HAART and hyperlipidemia presented with retrosternal chest pain, severe in intensity and non-radiating, not related to chest palpation, change in position or respiration. The patient took 162 mg of aspirin after the onset of chest pain, which improved the chest pain. His blood pressure on presentation was 1369/91 mmHg and heart rate was 74 beats per minute. Chest was clear to auscultation, first and second heart sounds were heard, no murmurs were appreciated. Initial electrocardiogram (ECG) (Figure 1) revealed normal sinus rhythm and biphasic T wave in V2-V3 leads. His hemogram and electrolytes were within normal limits. Troponin was found to be

elevated at 0.038 ng/mL and a repeat troponin level six hours later revealed 0.191 ng/ml. An ECG obtained two hours from the first one revealed resolution of the biphasic T-waves in the V3 lead (Figure 2). Patient was loaded with aspirin 325 mg, ticagrelor 600 mg and was anticoagulated with enoxaparin. An urgent cardiac catheterization revealed hazy, ulcerated, eccentric, tubular 99% stenotic proximal left anterior descending artery lesion (Figure 3) to which balloon angioplasty followed by everolimus eluting stent was placed (Figure 4). Patient was discharged home, on dual antiplatelet therapy consisting of aspirin and ticagrelor, high intensity statin, and beta-blockers.

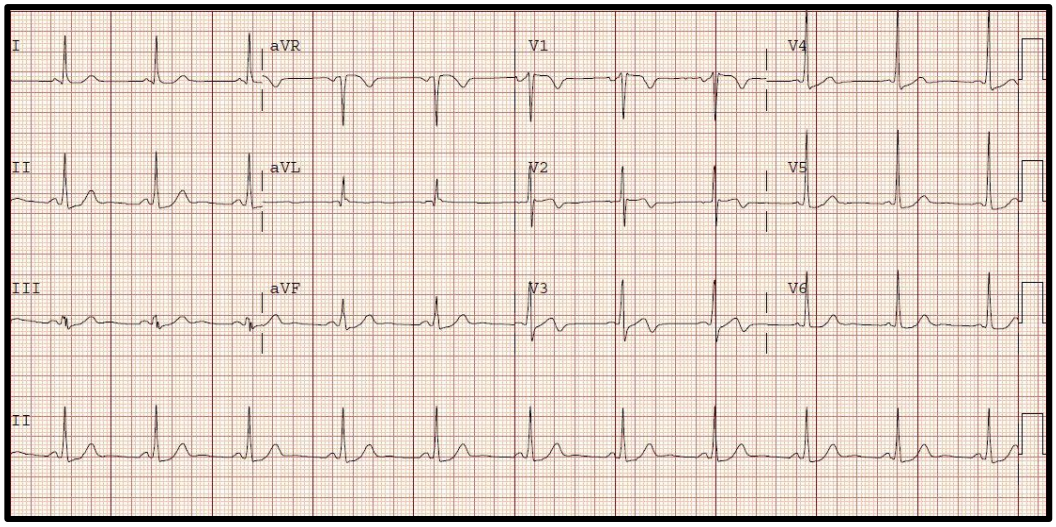

Figure 1. ECG at presentation showing normal sinus rhythm and biphasic T wave in V2-V3 leads

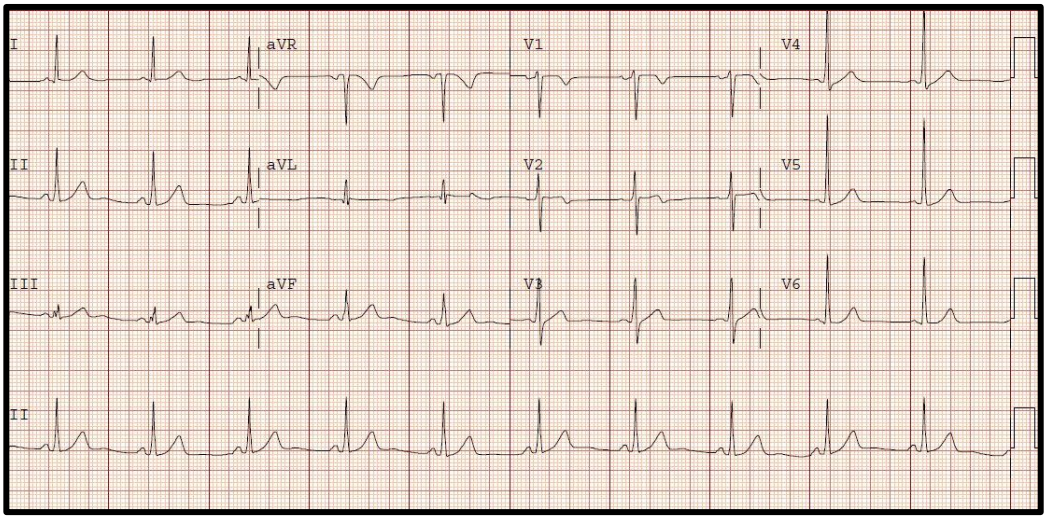

Figure 2. Repeat ECG after 2 hours showing resolution of biphasic T-waves in V3

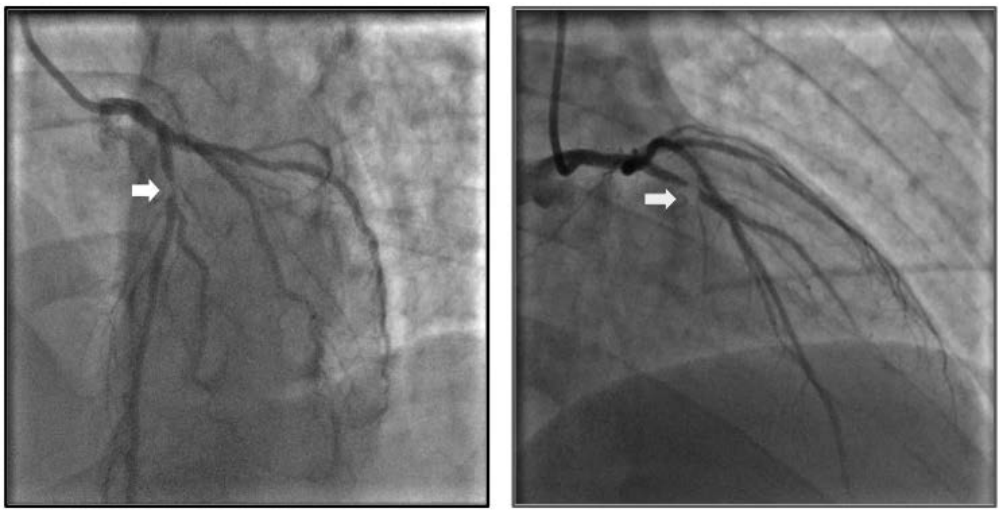

Figure 3. Coronary angiogram showing 99% stenosis of proximal left anterior descending coronary artery (white arrow)

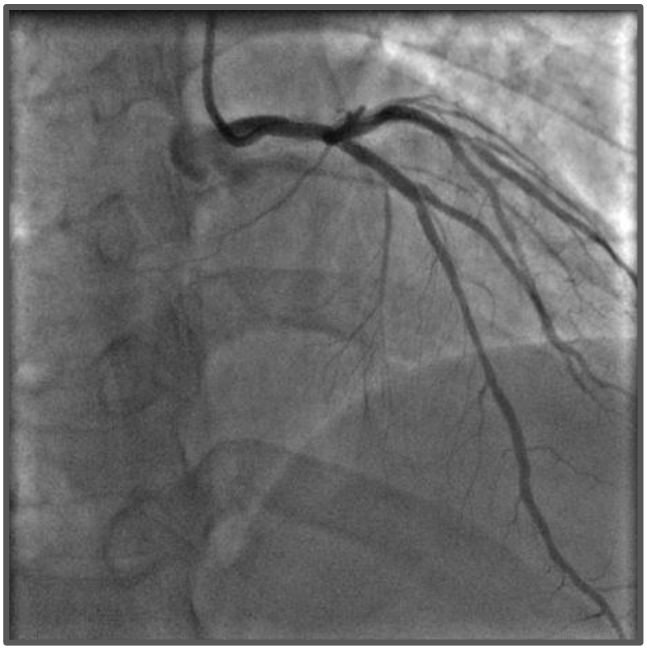

Figure 4. Coronary angiogram showing normal coronary flow in LAD following everolimus eluting stent placement

3. Discussion

HIV is an independent risk factor for major adverse cardiac events including acute myocardial infarctions. Although some risk factors are more common among HIV infected individuals, such as smoking history, these patients are more likely to have non-calcified arterial plaques after adjusting for age, race, and other CAD risk factors [10]. Proposed mechanisms for increased cardiovascular risk in this population are chronic inflammation secondary to viremia, dyslipidemia secondary to antiretroviral therapy, increased macrophage activation leading to plaque erosions, immune dysfunction, and endothelial dysfunction; all these factors increase the risk of atherosclerosis/thrombosis, and the prevalence of traditional cardiovascular risk factors [11,12,13]. Protease inhibitors, commonly prescribed as part of HAART, can also cause dyslipidemia: elevated triglycerides, decreased levels of high-density lipoprotein cholesterol levels, and insulin resistance [14]. Low CD4 may also correlate inversely with acute myocardial infarction (AMI) risk, with one analysis of the North American AIDS Cohort Collaboration on Research and Design finding that patients with a CD4 count less than 100 carried a 2-fold increase in AMI incidence rate compared to those with a CD4 count greater than 500 [15].

Recognition of the subtle ECG changes in Wellens' syndrome is vital to initiating early, urgent intervention to preserve anterior wall myocardium and prevent death, as patients may initially be asymptomatic. While the patient in this case presented with typical angina and was treated successfully with stenting and pharmacological management, seropositive HIV patients have been found to suffer from disparities in standard-of-care management strategies for AMI when compared to seronegative patients [16]. The combination of potentially subclinical, high mortality stenosis and suboptimal care in a population whose CVD risk is underestimated by the traditional risk calculators, allows for delay diagnosis and worse outcomes in these patients. The aging HIV population, burdened less with end-stage AIDS-defining illnesses due to HAART, requires special consideration by clinicians as to how this chronic condition modulates cardiovascular risk profile [17]. In our patient, the Wellens' syndrome partially resolved likely due to endogenous thrombolysis [18] and antiplatelets that were provided at presentation to the emergency department

4. Conclusion

HIV positive patients can present with premature CAD. The detection of a Wellens's pattern in patients with typical chest pain and cardiovascular risk factors demands prompt attention and early intervention as severe proximal LAD stenosis is likely to be present.

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