Case Report

Acquired long QT syndrome in a patient with human immunodeficiency virus and disseminated *Mycobacterium avium* complex

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ABSTRACT

Acquired long QT syndrome is usually seen secondary to metabolic abnormalities and medications, including macrolide antibiotics, anti-psychotics, and anti-arrhythmics. QT prolongation has also been seen in patients with human immunodeficiency virus (HIV), particularly those with poorly controlled HIV status, immunocompromised state, and electrolyte imbalances associated with chronic HIV infection. We present a case report of a young male with poorly controlled HIV (CD4 count 11 cells/uL, high viral load) who presented with fatigue, shortness of breath, and a mechanical fall, with an initial electrocardiogram showing a prolonged corrected QT (QTc) interval, and was eventually found to have disseminated *Mycobacterium avium* complex (MAC) infection upon bone marrow biopsy. The primary clinical dilemma involved starting initial macrolide-based antibiotic therapy for disseminated MAC, and in the presence of a QTc >500. The case highlights the complex challenge of managing critical illness in advanced HIV and balancing the pro-arrhythmic danger of treatment against the immediate mortality of untreated infection when widespread MAC necessitates the use of macrolides.

Key words: Disseminated *Mycobacterium avium* complex, Long QT syndrome, Macrolides, Wellens' syndrome

A prolonged corrected QT (QTc) in electrocardiography is the hallmark of long QT syndrome (LQTS), a condition affecting the heart's electrical circuit that results in delayed repolarization [1]. LQTS is linked to a higher incidence of polymorphic ventricular tachycardia (VT) and torsades de pointes (TdP), which is a potentially fatal arrhythmia, making it clinically relevant [2]. LQTS may be acquired or congenital. Romano–Ward syndrome and Jervell and Lange–Nielsen syndrome are the two forms of congenital LQTS. Although congenital variants of LQTS are extensively documented, acquired LQTS is much more prevalent in clinical practice and is typically brought on by drugs, metabolic problems, or systemic diseases [3]. Antibiotics (such as macrolides), anti-psychotics, antiarrhythmics, and anesthetics are some of the culprit medications that have been linked to QT interval prolongation [4]. A prolonged QTc interval is more likely to occur in persons living with human immunodeficiency virus (HIV). Some factors include simultaneous anti-HIV drug therapy, immunocompromised state, and electrolyte imbalances associated with chronic HIV infection [5]. HIV infection has been independently associated with a longer QTc interval and increased odds of QT prolongation compared to uninfected controls [6,7].

We report this case to draw attention to the complex risk of QT interval prolongation in advanced HIV infection. A young male with poorly controlled HIV was ultimately diagnosed with *Mycobacterium avium* complex (MAC) infection after experiencing palpitations and syncope-like episodes in the context of severe immunosuppression. This case is clinically significant because, instead of a single, independent cause, QT prolongation in this clinical setting most likely represented the combined effects of advanced HIV disease, systemic sickness, metabolic disorders, and exposure to QT-prolonging treatments.

CASE REPORT

A 27-year-old African-American male with past medical history of HIV, not compliant with antiretrovirals, syphilis treated in the past, esophageal candidiasis, and cryptosporidium gastroenteritis presented with chief complaints of generalised fatigue and shortness of breath for the last 3–4 weeks. The patient was brought in by the emergency medical services (EMS) after falling in the kitchen after he felt his legs give out. Shortness of breath was more associated with exertion and was associated

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with intermittent palpitations. A history was otherwise negative for any chest pain, headaches, orthopnea, paroxysmal nocturnal Dyspnea, fevers, abdominal pain, diarrhea, constipation, dysuria, hematuria, hematemesis, hemoptysis, blood in stools, melena, sick contacts, and/or any recent travel.

During the physical examination, the patient appeared malnourished, thin, pale, and ill-appearing. Head-and-neck examination showed pallor, scleral icterus, and cracked lips; however, mucous membranes were moist, and oropharynx showed white cotton-like exudates. Further examination demonstrates normal respiratory status with normal bilateral lung sounds and normal S1 and S2 heart sounds without any murmurs or gallops. Signs of bruising on the arms, elbows, thighs, and buttocks were present, indicating severe coagulopathy. Neurologically, he did not have any motor or sensory deficits, and all cranial nerves were intact with preserved cortical functions.

Initial laboratory investigations revealed acute kidney injury-on-chronic kidney disease with high anion gap metabolic acidosis and hyperkalemia, requiring immediate intervention with bicarbonate infusion and a hyperkalemia cocktail. Hematological studies showed pancytopenia with hemoglobin of 4.7 g/dL and platelet count of 3500/μL. The patient's antibody screen was also positive for auto-antibodies, including Immunoglobulin G, anti-K, and anti-M. The patient was subsequently diagnosed with HIV associated auto-immune hemolytic anemia. Over the entire course of hospitalization, he required a blood transfusion of 35 units of packed red blood cells and 11 units of platelets. His HIV viral load was 3.3 million copies/mL, and his initial CD4 count was 31 cells/μL, which rapidly dropped to 11 cells/μL.

During the admission, the patient's Electrocardiogram (EKG) was noted to have a prolonged QTc of 605 ms, even before starting the QT prolonging antibiotics and or antifungals (Fig. 1). Subsequently, he underwent echocardiography, which revealed mildly reduced heart failure with mildly reduced ejection fraction (HFmrEF) of 40–45%, Grade I diastolic dysfunction, and a trace pericardial effusion.

On further investigations during his hospital course, the workup for pancytopenia was done simultaneously with guidance from the hematology and oncology service. The patient underwent a bone marrow biopsy. The procedure yielded a dry tap, but the core biopsy results revealed a paradoxical finding of 95% hypercellular bone marrow consisting of a densely packed population of histiocytes containing acid-fast bacilli. Tissue cultures eventually confirmed disseminated MAC in the bone marrow, liver, and gastrointestinal tract.

The treatment of his widespread MAC was a risky therapeutic challenge. He was started on a multidrug regimen consisting of rifabutin 300 mg, ethambutol 1200 mg, moxifloxacin 400 mg IV, and azithromycin 500 mg IV to treat the high-burden illness. His CD4 count of 11 and ongoing MAC bacteremia required this polypharmacy, which made his already prolonged QTc even more difficult to manage. The patient remained at high risk of transitioning to TdP despite aggressive electrolyte supplementation (maintaining K >4.0 and Mg >2.0) and cautious titration of his MAC therapy.

During the subsequent course of treatment, the patient's follow-up EKGs showed deep T-wave inversion (TWI) in V1 and V2, concerning for Wellen's syndrome (Fig. 2). The patient was then upgraded to the cardiac care unit (CCU) for ischemia workup and treatment of suspected Wellen's Syndrome because of the combination of HFmrEF, new apical wall motion abnormalities, and EKG results suggesting TWI. In the CCU, management was complicated with persistent QTc prolongation. The patient's heart rate remained in the range of 90–100/min. The team decided to use coronary computed tomography angiography (cCTA) as severe thrombocytopenia continued to prevent invasive angiography. The coronary artery calcium score on the cCTA was zero, and there was no evidence of obstructive coronary artery disease. A follow-up cardiac MRI was then performed, which demonstrated prominent apical hypokinesis but was notably negative for late gadolinium enhancement or T2-signal edema. In the context of a clear cCTA, these results were indicative of Takotsubo cardiomyopathy. The etiology was attributed to the overwhelming systemic burden of his HIV/acquired immune-deficiency syndrome (AIDS) and disseminated MAC infection.

DISCUSSION

LQTS is found to be associated with HIV, especially in patients with advanced HIV disease (high viral load)

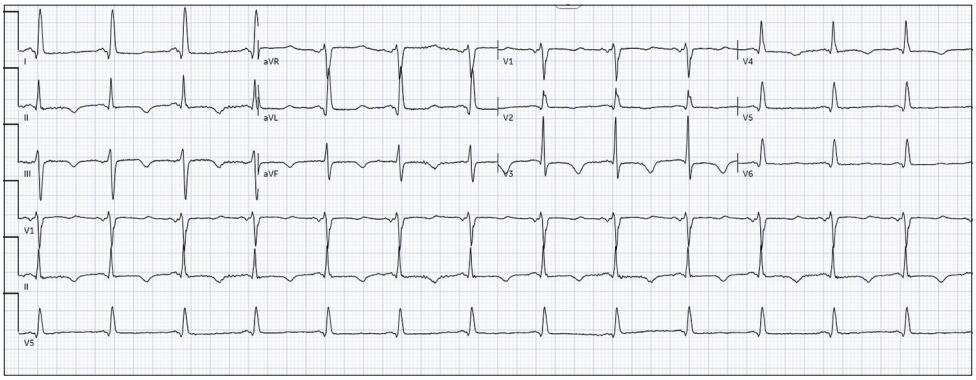

Figure 1: Initial electrocardiogram with T wave inversion V3 and inferior leads and corrected QT of 605 ms

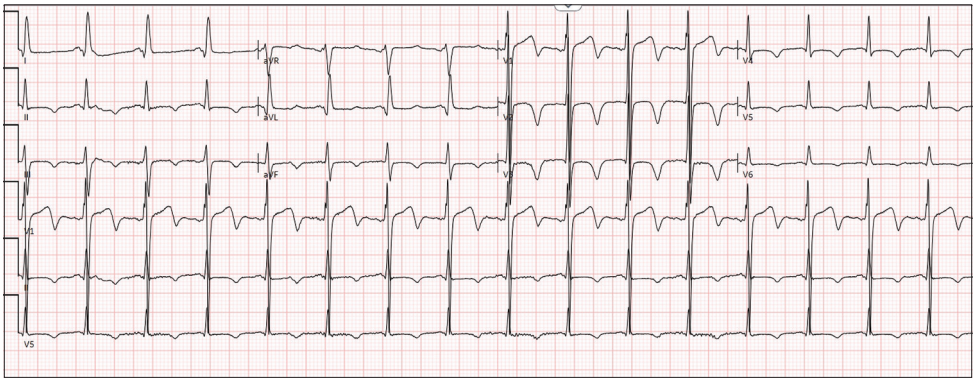

Figure 2: Electrocardiogram showing biphasic T waves in V1 and deep T wave inversion in V2 and V3, concerning Type B Wellen's syndrome

and low CD4 counts [8]. A study conducted in 802 HIV-positive patients to explore the link between HIV seropositivity and long QT interval revealed that HIV-positive individuals had a 20% higher rate of QT interval prolongation compared to those without HIV [9]. In another study from Nigeria, 100 consecutive AIDS patients at the Jos University Teaching Hospital were assessed for cardiovascular symptoms and underwent a 12-lead EKG for the determination of QTc. It was found that 45% of AIDS patients had a prolonged QTc interval, compared to 28% in HIV-positive controls and 10% in HIV-negative controls [10].

As mentioned before, it has been thought that low CD4 counts behave like an independent factor for QT prolongation. A study conducted in 2017 with 351 patients at two primary prevention clinics in Italy found that a lower CD4 cell count (below 200 cells/μL) was consistently linked to a prolonged QT interval. In addition, patients with prolonged QT intervals tended to be older on average compared to those with normal QT intervals [11]. In addition, another study at Bronx Lebanon Hospital, including the African-American population, was done, which aimed at finding the frequency of prolonged QT interval in African-American patients with HIV and its correlation with VT and CD4 count. 5,892 HIV-positive patients (mean age 50) were examined retrospectively between 2009 and 2014. A prolonged QT interval was present in 17% of the patients (1,009 people), with no discernible sex differences. The mean CD4 count was 124, and a longer QT interval was closely associated with a lower CD4 level. Furthermore, VTs were found to occur in 2% of patients with a prolonged QT interval; the prevalence was higher in AIDS patients (2.7%) than in HIV patients without AIDS [12]. The results emphasize how crucial it is to keep a careful eye on QT intervals, especially in African-American patients with AIDS who are HIV positive and have low CD4 levels, as was the case in our patient. We also looked for any ethnic predisposition of HIV related QT prolongation; however, contrary to our expectations, ethnicity seems to influence the probability of QTc prolongation irrespective of the ethnic background. QTc prolongation was almost twice as likely among Asian participants (12%) as in other racial groups (7%), including African-American participants, in the massive reprieve experiment, which included 7,720 HIV-positive individuals from various racial backgrounds [13]. On the other hand, the Heart Rhythm Society reports that there are few cases of hereditary LQTS among African Americans, indicating potential ethnic variations in genetic susceptibility to the inherited LQTS [14].

Current evidence suggests the role of cellular and molecular level mechanisms affected in response to HIV, which can cause alteration in the current flow in the myocardiocytes. The α-subunit of voltage-gated potassium channels (Kv11.1), which conduct the rapid delayed rectifier potassium current (IKr), encoded by the hERG gene, has special gating characteristics, especially fast voltage-dependent inactivation combined with slow deactivation, which enable them to stay open during the action potential's plateau period and significantly aid in phase 3 repolarization. Inhibition of hERG channels reduces IKr, delays repolarization, and prolongs action potential duration, which translates as QT interval prolongation on the EKG [15]. HIV virus transactivator protein (Tat) has been identified to play an important role in the pathophysiology. Two different mechanisms have been found that explain the inhibition of hERG currents by the HIV Tat protein: acute sequestration of phosphatidylinositol-(4,5)-bisphosphate (PIP_2) and persistent reduction of hERG protein production. The amplitude of the hERG current is considerably decreased after cells are exposed to HIV Tat protein. Both the inhibition time and the recovery time after inhibition of hERG channels are markedly altered by this long-term exposure, and as a result, the pool of accessible channels for conducting current during repolarization is decreased [16,17]. A study by Brailoiu *et al*. examined the effects of Tat proteins on rats. In HIV-1 infection, the signal of Tat-mediated pathways increased with the concentration of HIV Tat protein. It was found that bradycardia and QT interval lengthening were promoted by higher HIV Tat protein concentrations (Fig. 3) [18].

While these molecular mechanisms establish a baseline vulnerability in the HIV-infected myocardium, the clinical challenge becomes exponentially more complex when opportunistic infections necessitate the

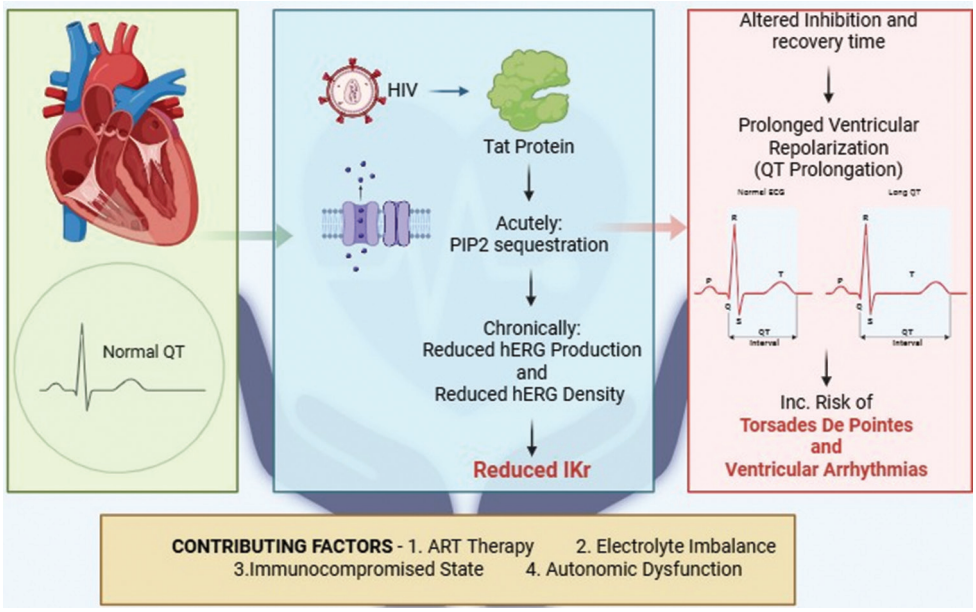

Figure 3: Mechanism of action of human immunodeficiency virus induced QT prolongation

use of medications that further antagonize these already compromised pathways. As our patient was diagnosed with disseminated MAC following a bone marrow biopsy, the management of this infection presented a profound clinical paradox. The gold-standard treatment for disseminated MAC, a macrolide-based regimen, acts as a secondary "hit" to the cardiac conduction system. The rapidly activating delayed rectifier potassium channel is strongly inhibited by macrolides, especially clarithromycin, which further lowers the repolarization current that the HIV Tat protein has already reduced (first hit) [8]. This clinical conundrum is further complicated by the fact that significant pancytopenia caused by MAC infiltration of the bone marrow results in systemic inflammatory stress and persistent anemia, both of which are independent risk factors that might further contribute to prolonged QT [19]. Consequently, the clinician is forced to navigate a precarious "risk-risk" trade-off: withholding life-saving antimycobacterial therapy versus risking a fatal arrhythmia in a patient whose hERG channel pool is already depleted by chronic viral exposure. The threshold for TdP is considerably lowered in such high-stakes situations, requiring a strategy of aggressive electrolyte optimization, specifically, maintaining potassium (>4.0 mmol/L) and magnesium (>2.0 mg/dL) and careful ECG monitoring during the induction phase of therapy [20].

Our patient's QTc interval continued to be chronically prolonged even after rigorous electrolyte replenishment. This delayed electrophysiological recovery emphasizes how severe and perhaps long-lasting HIV-associated "inflammatory channelopathy" is. The persistence of a prolonged QT interval in the face of normalized potassium and magnesium levels indicates that the molecular hit from the HIV-Tat protein may not be immediately reversible upon starting treatment, specifically the persistent reduction in hERG protein production and the sequestration of PIP_2 [17]. This possibly suggests that long-term cardiac vigilance and avoidance of all secondary QT-prolonging agents are required because the "repolarization reserve" in advanced, multi-organ HIV disease may be so severely depleted that standard corrective measures are insufficient.

CONCLUSION

LQTS is found to be associated with HIV, especially in patients with advanced HIV disease (high viral load) and low CD4 counts. There is a specific association between a person's ethnic background and HIV-related QT prolongation. However, few cases have documented hereditary LQTS in the African-American population. HIV Tat protein has been found to be one of the initial hits causing QT prolongation. In addition, other co-morbid conditions related to HIV-AIDS, like disseminated MAC and medication-related QT prolongation, add to the second hit, causing persistent QT prolongations. Although long-term cardiac vigilance is essential throughout the treatment plans for patients with HIV-AIDS, balancing the risks associated with certain medications becomes increasingly challenging.

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