











## Myasthenia And HIV Infection: An Unusual Combination, One Case Report In Abidjan, Ivory Coast

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### ABSTRACT

**Introduction:** The co-occurrence of autoimmune myasthenia gravis and HIV infection is poorly documented. Existing cases of combination myasthenia gravis and HIV are often associated with immune reconstitution inflammatory syndrome (IRIS), whereas the onset of myasthenia gravis prior to HIV diagnosis is exceptional. **Observation:** We report the case of a 43-year-old woman with seronegative generalized myasthenia gravis since 2002, 23 years before presentation, diagnosed with HIV-1 infection in 2009, 16 years before presentation. She was virologically stable on triple antiretroviral therapy (CD4 = 993/mm<sup>3</sup>, undetectable viral load). Electroneuromyography showed a 25% decremental response on repetitive nerve stimulation. Anti-AChR antibodies were negative; MuSK and LRP4 were not tested. During a myasthenic crisis with respiratory distress and muscarinic features, pyridostigmine was stopped and resuscitation measures were undertaken. Subsequent corticosteroid and azathioprine therapy led to marked improvement (Garches score 55 → 85 after 8 months). **Conclusion:** This rare case of myasthenia preceding HIV diagnosis illustrates an association independent of IRIS. It suggests that immunosuppressive therapy can be used safely in well-controlled HIV infection under close virological and immunological monitoring within a multidisciplinary framework.

**Keywords:** Myasthenia gravis, HIV, Immunosuppressive therapy, Antiretroviral therapy, Ivory Coast

### INTRODUCTION

Myasthenia gravis (MG) is a rare neuromuscular joint autoimmune disease characterized by impaired neuromuscular transmission linked to autoantibodies directed against postsynaptic receptors, most commonly acetylcholine receptors (AChR). Its overall average incidence is 5.3 per million person-years [1]. Depending on geographical location, the prevalence of MG varies between 2.19 and 36.7 cases per 100,000 inhabitants worldwide [1,2], but is probably underestimated in Africa due to underdiagnosis. The association between HIV infection and myasthenia gravis is rare. In the majority of cases described, myasthenia gravis

occurs after HIV diagnosis, often in the context of immune reconstitution inflammatory syndrome (IRIS) or following the initiation of antiretroviral therapy [3,4]. Only a few cases have been reported where myasthenia gravis preceded HIV diagnosis, representing less than 10% of the published associations [5–7]. We report here the case of a patient living with HIV whose myasthenic symptoms were present several years before the diagnosis of infection, which makes this case unique, followed by an emphasis of the diagnostic and therapeutic implications in the light of international and African literature.

## CASE REPORT

A 43-year-old female patient from Côte d'Ivoire, infected with HIV-1 and undergoing antiretroviral treatment since 2009 with ongoing triple antiretroviral therapy consisting of Tenofovir 300mg/ Lamivudine 300mg/day + Efavirenz 400mg/day, reinstated due to intolerance to the highly active molecules introduced a few years earlier, consulted for phonation disorders such as a nasal voice and swallowing disorders marked by frequent choking, initially with solids and then with liquids. The patient's history included several similar episodes since 2002, the last of which occurred five months before her consultation, with progressive fatigue on exertion and fluctuating bilateral ptosis with blurred vision. Myasthenic symptoms first appeared in 2002, characterised by fluctuating ptosis and fatigue. HIV-1 infection was diagnosed in 2009. The patient experienced several relapses, with the most recent myasthenic crisis occurring in March 2023 and follow-up extending to November 2023. Electroneuromyography showed a 25% decremental response on repetitive nerve stimulation of facial and limb muscles. Single-fibre EMG was not performed. Anti-AChR antibodies were negative; MuSK and LRP4 antibodies were not tested. Chest CT scan (contrast-enhanced, 1 mm slices, dedicated

thymic protocol) showed no evidence of thymic hyperplasia or thymoma. The examination revealed myasthenic syndrome with a Garches score of 55. On admission, the Garches score, a clinical scale assessing the severity and functional impact of myasthenia gravis (ranging from 0 = severe disability to 100 = normal function), was 55 and improved to 85 after treatment. Her CD4 count was 993 and her viral load was undetectable. The patient received 180 mg of pyridostigmine divided into three daily doses for three weeks before experiencing a worsening of her symptoms, including respiratory failure as part of a myasthenic crisis. She developed muscarinic effects such as hypersalivation, hyperhidrosis and vomiting, leading to discontinuation of pyridostigmine and initiation of airway protection and oxygen therapy. No intravenous immunoglobulin or plasma exchange was required. Specifically, she received prednisolone at 1 mg/kg/day tapered over six months and azathioprine at 2 mg/kg/day, associated with a proton pump inhibitor and *Pneumocystis jirovecii* prophylaxis (trimethoprim-sulfamethoxazole). Potential drug-drug interactions with antiretroviral therapy were monitored. The patient also underwent motor physiotherapy sessions.

## DISCUSSION

The association between HIV infection and autoimmune myasthenia gravis is rare, but it raises important pathophysiological and therapeutic questions. Our patient had several clinical features that are worth noting: first, the possible presence of an autoimmune disease prior to the detection of acquired immunodeficiency, then the onset of a myasthenic crisis, whereas myasthenia associated with HIV is generally described as mild to moderate and very few cases of myasthenic crisis have been reported; and finally, immunosuppression is not absolutely contraindicated in HIV-positive individuals. In most cases described, myasthenia occurs after the initial treatment of HIV, often in the context of immune reconstitution syndrome or autoimmune stimulation linked to viral infection and antiretroviral drugs [5-7]. The particularity of our observation lies in the fact that the myasthenic symptoms preceded the diagnosis of HIV, suggesting coincidental coexistence rather than as an immune reconstitution associated with autoimmune disease. This chronology of symptoms makes IRIS less likely, although diagnostic overlap cannot be entirely excluded.

[7,8]. In Africa, the main reported cases of myasthenia gravis and HIV come from South Africa, where Heckman *et al.* reported several cases of autoimmune disorders, including myasthenia gravis, occurring in HIV-positive patients, confirming that despite immunosuppression, persistent autoimmune activity can occur [3]. Internationally, sporadic cases confirm the complexity of this association, while Vincent *et al.* and Wirtz *et al.* emphasized the broad spectrum of autoimmune diseases encountered in HIV-positive patients [8,9]. Comparison of our observation with these data shows that the clinical chronology (myasthenia preceding HIV) is an original element, reinforcing the idea of a fortuitous rather than etiopathogenic association. Beyond the diagnostic originality, our case highlights important therapeutic implications. The management of this association proves to be particularly complex. Pyridostigmine, the first-line treatment, may be poorly tolerated at moderate doses, as in our case. There is no single, universally accepted treatment regimen. Corticosteroids form the basis of immunosuppressive treatment in patients with

mild myasthenia gravis to induce remission. Immunosuppressive treatments, such as azathioprine, are prescribed in addition to, or even instead of, corticosteroids, depending on the presentation of the disease when comorbidities exclude or limit the use of steroids. The complexity in this patient lay in the particularity of using both corticosteroids and azathioprine in the presence of HIV infection, requiring a switch to corticosteroid therapy or immunosuppressants. In HIV-positive patients, such treatments must be introduced with caution, given the risk of infection and interactions with antiretroviral drugs [10]. Furthermore, several observations confirm the feasibility and efficacy of these strategies under close monitoring. Firstly, it is possible to take antiretroviral therapy when myasthenia gravis is diagnosed but it requires increased vigilance with regard to drug interactions (particularly between protease inhibitors and immunosuppressants). Secondly, corticosteroid therapy, although potentially immunosuppressive, remains an effective therapeutic lever under regular virological and immunological monitoring. In practical terms, special attention should be paid to drug–drug interactions, particularly between protease inhibitors or calcineurin inhibitors and immunosuppressants such as corticosteroids or azathioprine, which may require dose adjustment or therapeutic drug monitoring. Infection prophylaxis (e.g., trimethoprim-sulfamethoxazole when CD4 count <200/mm<sup>3</sup>) and vaccination status (including

influenza, pneumococcal, and hepatitis B vaccines) should be systematically reviewed before initiating immunosuppression. Regular multidisciplinary follow-up - ideally every 3 to 6 months - by neurology and infectious disease teams is essential to adjust treatment, prevent opportunistic infections, and optimize long-term outcomes and quality of life.

## CONCLUSION

Our observation illustrates a rare but instructive clinical situation. It supports, in line with African and international data, that the coexistence of myasthenia gravis and HIV, although complex, can be effectively managed through an integrated and cautious approach. Treatment must be individualized in older patients according to specific comorbidities. The uniqueness of this case, where myasthenia precedes HIV infection, enriches literature and highlights the importance of considering this association in clinical contexts of fatigue and atypical ear, nose and throat manifestations. Clinicians should remain alert to myasthenia gravis in HIV-positive patients presenting with bulbar symptoms and ensure close monitoring of immunosuppressive therapy to balance efficacy and infection risk.

*Ethics statement:* The patient provided written informed consent for publication. The report was conducted in accordance with institutional ethical standards and CARE guidelines, and patient identity was anonymised.

## REFERENCES

1. Bubuioc AM, Kudebayeva A, Turuspekova S, Lisnic V, Leone MA. The epidemiology of myasthenia gravis. *J Med Life*. 2021;14(1):7-16. doi:10.25122/jml-2020-0145.
2. Gilhus NE, Verschuuren JJ. Myasthenia gravis: subgroup classification and therapeutic strategies. *Lancet Neurol*. 2015;14(10):1023-36. doi:10.1016/S1474-4422(15)00141-0.
3. Heckmann JM, Marais S. Management issues in myasthenia gravis patients living with HIV: a case series and literature review. *Front Neurol*. 2020; 11:775. doi:10.3389/fneur.2020.00775.
4. Sherpa M, Metai RK, Kumar V, Hirachan T, Ahmed KU, Atkinson SJ. Comorbid human immunodeficiency virus (HIV) and muscle-specific kinase (MuSK) myasthenia gravis: a case report and literature review. *Am J Case Rep*. 2017;18:427-30. doi:10.12659/AJCR.902831.
5. Ragnathan K, Pathak B, Dahal K. MuSK myasthenia gravis as a manifestation of immune restoration disease in an HIV-positive patient. *J Neurol*. 2015;262(3):777-8. doi:10.1007/s00415-014-7582-5.
6. Choudhary A, Kumar N, Gupta S, Anand R, Verma R, Sharma P, et al. Myasthenia gravis in HIV-infected patients: report of two cases and review of the literature. *J Clin Neuromuscul Dis*. 2014;15(4):159-64. doi:10.1097/CND.0000000000000041.
7. O'Connor KC, Sriram S, Vincent A. Autoimmune neuromuscular disorders in HIV-positive patients: emerging evidence. *Curr Opin Neurol*. 2019;32(5):775-85. doi:10.1097/WCO.0000000000000740.
8. Wirtz PW. Dysphagia and respiratory failure in an HIV patient: MuSK myasthenia gravis. *Acta Neurol Belg*. 2020;120(6):1483-4. doi:10.1007/s13760-020-01461-9.
9. Gilhus NE, Tzartos S, Evoli A, Palace J, Burns TM, Verschuuren JJ. Myasthenia gravis. *Nat Rev Dis Primers*. 2019;5:30. doi:10.1038/s41572-019-0085-1.
10. Farrugia ME, Goodfellow JA. A practical approach to managing patients with myasthenia gravis: opinions and a review of the literature. *Front Neurol*. 2020;11:604. doi:10.3389/fneur.2020.00604.