**CASE REPORT**

# Bilateral Lower Motor Neuron Type Facial Nerve Palsy as the First Presentation of Seroconversion of HIV: A Case Report

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## Abstract

Unilateral involvement is the commonest presentation of facial nerve palsy. Bilateral lower motor neuron (LMN) facial nerve palsy is uncommon, indicating an underlying systemic pathology. Guillain-Barré syndrome and its variant Miller-Fisher syndrome is the commonest aetiology with an underlying demyelinating pathology. Sarcoidosis, leprosy, infectious mononucleosis, Lyme disease, and tuberculous meningitis are associated with bilateral facial nerve palsy. Human immunodeficiency virus (HIV) can cause facial nerve palsy at any stage of the disease, but rarely during the phase of acute seroconversion. We report a 41-year-old female who presented with fever, arthralgia, and myalgia, followed by the development of bilateral LMN facial nerve palsy during the fifth day of her illness, associated with non-tender cervical lymphadenopathy, generalised maculo-papular rash without any organomegaly. Neurological examination was unremarkable except for bilateral lower motor neuron facial nerve weakness. HIV-1 antibodies were detected using an enzyme-linked immunosorbent assay (ELISA), and a CD4 count confirmed the diagnosis. This case highlights the importance of considering HIV as a cause for bilateral facial nerve palsy in patients presenting with an acute febrile illness.

**Keywords:** Bilateral facial nerve palsy, lower motor neuron facial nerve palsy, HIV, seroconversion

## Introduction

Facial nerve palsy commonly manifests as unilateral weakness. Bilateral LMN facial palsy is rare-representing <1% of all cases – and usually indicates an underlying systemic disorder.[1] Recognised causes include Guillain-Barré syndrome and its variant Miller-Fisher syndrome, chronic inflammatory demyelinating polyneuropathy (CIDP), sarcoidosis, haematological malignancies such as lymphomas and leukaemias, small-vessel vasculitis, and infections including Lyme disease, leprosy, syphilis, tuberculous meningitis, infectious mononucleosis, and HIV.[1,2]

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Neurological manifestations can occur at any stage of HIV infection. During acute seroconversion, transient neurological syndromes can arise due to immune activation or direct viral neurotropism.[3] Bilateral LMN facial nerve palsy as the first manifestation of HIV infection is exceedingly rare, with only a few cases described globally.[4-6]

We describe a patient who presented with bilateral LMN facial palsy secondary to HIV seroconversion, underscoring the need to consider HIV in the evaluation of acute bilateral facial paralysis.

## Case presentation

A 41-year-old previously healthy female presented with a history of low-grade fever, arthralgia, and myalgia for three days. She denied headache, photophobia, cough, gastrointestinal, or urinary symptoms. There was no history of recent travel or insect bites. She was not on long-term medications.

On admission, she was febrile but haemodynamically stable. The general examination revealed non-tender cervical lymphadenopathy and a generalised non-pruritic maculopapular rash, sparing the head, neck, and mucosae. Cardiovascular, respiratory, abdominal, and neurological examinations were otherwise unremarkable.

On day five of illness, she developed progressive bilateral facial weakness. Examination revealed features of LMN facial nerve palsy. Deep tendon reflexes were normal, and there was no limb weakness, sensory deficit, or signs of meningeal irritation. Examination of the optic fundi was unremarkable.

Routine blood investigations, including full blood count, renal and liver profiles, were within normal limits. Erythrocyte sedimentation rate (19 mm/1$^{st}$ hour) and C-reactive protein (12 mg/dL) were mildly elevated. Chest X-ray was normal. Cerebrospinal fluid analysis showed no pleocytosis or elevated protein. Autoimmune screening (ANA, ANCA) was negative. HbA1c level was 5.5%. Contrast-enhanced MRI brain yielded no abnormalities.

HIV testing was positive for HIV-1, by ELISA and confirmed by Western blot. CD4 count was 430 cells/µL, which is within the expected range for seroconversion illness. Serological studies for Epstein-Barr virus, cytomegalovirus, scrub typhus, and syphilis were negative.

**Table. Differential diagnoses of bilateral LMN facial palsy**

| *Category* | *Recognised causes* | *Distinguishing features* |
|---|---|---|
| **Infections** | Lyme disease, leprosy, HIV, infectious mononucleosis, syphilis, tuberculous meningitis | Fever, rash, lymphadenopathy, CSF changes, serological tests |
| **Demyelinating** | Guillain-Barré syndrome | Limb weakness, areflexia, cyto-protein dissociation in CSF |
| | Miller-Fisher variant, CIDP | |
| **Granulomatous** | Sarcoidosis | Pulmonary and or systemic features, abnormal imaging, raised Angiotensin Converting Enzyme (ACE) level and non-caseating granulomas in tissue biopsy samples |
| **Infiltrative/ Neoplastic** | Lymphoma, leukaemia | Systemic symptoms, cytopenia, abnormal blood picture and bone marrow biopsy |
| | Primary CNS tumours and metastatic neoplasms | Features of increased intra-cranial pressure, CNS imaging |
| **Autoimmune / Vasculitis** | Systemic lupus erythematosus<br>Polyarteritis nodosa | Multisystem involvement<br>Positive autoantibodies |
| **Miscellaneous** | Diabetes mellitus | Depends on the underlying condition |
| | Head trauma | |

The patient was commenced on antiretroviral therapy after appropriate counselling. Supportive eye care was given. At two months of follow-up, there was complete improvement of facial weakness.

## Discussion

Bilateral LMN facial nerve palsy is rare and often indicates a systemic pathology. Unlike unilateral Bell's palsy, which is usually idiopathic, bilateral involvement necessitates urgent evaluation for underlying infectious, autoimmune, demyelinating, granulomatous, or neoplastic causes.[1]

Our patient developed isolated bilateral LMN facial palsy in the context of a short febrile illness with cervical lymphadenopathy, consistent with acute retroviral syndrome, which was confirmed by positive HIV-1 serology and compatible CD4 counts. Absence of limb weakness, areflexia, and normal CSF analysis results excluded Guillain-Barré syndrome. No radiological or haematological evidence supported sarcoidosis or malignancy, and autoimmune markers were negative, excluding alternative causes.[1]

HIV can involve the nervous system at all stages of the disease. During acute infection, viral replication and immune activation may lead to lymphocytic meningitis, aseptic encephalitis, or cranial neuropathies.[3,4] Facial nerve involvement, particularly bilateral LMN type, is unusual but recognised.[5]

### Pathophysiological mechanisms linking HIV seroconversion and LMN facial nerve palsy

The facial nerve can be affected during acute HIV infection through two mechanisms. Direct viral neurotropism allows HIV to enter neural tissue via infected monocytes and macrophages, leading to inflammation and oedema within the facial canal of the temporal bone.[3,6] Immune-mediated demyelination results from molecular mimicry and immune complex deposition triggered by abrupt seroconversion.[7,9] These processes may transiently disrupt nerve conduction without irreversible axonal loss, explaining the good prognosis and full recovery after initiation of antiretroviral therapy (ART).

A recent systematic review noted that cranial neuropathies, although rare, can occur during all phases of HIV infection, with the facial nerve being the most frequently involved.[9] Early recognition enables prompt ART, which may reduce recurrence and prevent further neurological sequelae.

This case highlights the importance of testing for HIV in patients presenting with bilateral facial palsy, even in the absence of other risk factors or advanced immunosuppression. Early diagnosis is the key to commencing antiretroviral therapy in a timely manner, which improves the prognosis and minimises the transmission.

## Conclusion

Bilateral LMN facial nerve palsy should prompt clinicians to investigate for an underlying systemic cause. HIV infection during any stage of its illness, particularly during seroconversion, should be considered in the differential diagnosis. Clinical awareness and vigilance regarding bilateral facial palsy facilitates early diagnosis and treatment of HIV during seroconversion.

## Authors' Contributions

Concept and design: DP, KT

Literature review: DP

Compilation of manuscript: DP

Manuscript editing and proofreading: DP, KT

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