Case Report

Human immunodeficiency virus neuropathy: A new mimicker of leprous neuropathy

Ankita Srivastava
Department of Skin and VD, JNU Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India

Address for correspondence:
Dr. Ankita Srivastava, Department of Skin and VD, JNU Institute for Medical Sciences and Research Centre, Jaipur, Rajasthan, India.
E-mail: ankitarnt@gmail.com

Abstract

Peripheral neuropathy is usually the domain of the physician or neurologist. Still, many times patients land up in dermatology department with complaints such as sensory loss, paresthesia, and trophic ulcers. Usually, these patients are evaluated for leprosy and then referred to departments of internal medicine or neurology. We report one such patient who was initially seen by a dermatologist but was ultimately found to be suffering from human immunodeficiency virus neuropathy.

Key words: Distal symmetric polyneuropathy, human immunodeficiency virus neuropathy, leprous neuropathy, small fiber sensory neuropathy

INTRODUCTION

Patients with peripheral neuropathy often present to dermatologists, where they are evaluated to rule out Hansen's disease and then referred to physician. Here, we report a case who presented with sensory loss and paresthesia over legs but was later found to be suffering from human immunodeficiency virus (HIV) neuropathy.

CASE REPORT

A 46-year-old male patient presented to the dermatology outpatient department with 1-year history of sensory loss and paresthesia over bilateral lower legs. He was admitted and evaluated for Hansen's disease. On examination, there was no cutaneous lesion suggestive of leprosy. Peripheral nerve trunks were not thickened. Sensory loss to touch, pain, and temperature was present over the distal part of legs and feet bilaterally. Ankle jerk was diminished bilaterally. No trophic ulcer was present. Higher mental function and cranial nerve examination were within normal limits. There was no sign of meningeal irritation. Motor examination did not reveal weakness in any major muscle groups. Slit skin smear taken from earlobes did not demonstrate any acid-fast bacilli. A skin biopsy was taken from hypoesthetic skin of the right leg and sent for histopathology. Initial investigations including complete blood count, liver and renal function tests, fasting plasma glucose, and urine examination did not reveal any abnormality. In such a clinical scenario, the possibility of Hansen's disease was remote, and therefore, a neurologist's opinion was sought. The patient was then advised to go for HIV serology and to our surprise found to be reactive for HIV-1 with CD4 count of 160 cells/mm^3. On retrospective questioning, the patient gave a history of on-and-off fever, fatigue, weight loss, and recurrent pyodermas. He also had multiple high-risk sexual exposures several years back. He was diagnosed as a case of HIV neuropathy-distal symmetric polyneuropathy (DSP) type and started on antiretroviral therapy (ART).

Access this article online

Quick Response Code:

Website: www.ijstd.org

DOI: 10.4103/ijstd.IJSTD_90_16

How to cite this article: Srivastava A. Human immunodeficiency virus neuropathy: A new mimicker of leprous neuropathy. Indian J Sex Transm Dis 2018;39:59-60.

DISCUSSION

Neurological manifestations are common in patients with HIV-AIDS. Of these, peripheral neuropathy is the most common one, owing to the efficacy of ART, which resulted in a decline in Central Nervous System opportunistic infections and HIV dementia.[1,2] Peripheral neuropathy in HIV-AIDS can result due to direct neurotoxicity of HIV, other associated viral infections such as *Cytomegalovirus*, and as an adverse effect of certain antiretroviral drugs (stavudine, didanosine, and zalcitabine).[3]

HIV-associated peripheral neuropathy presents in six major forms [Table 1].[3] Of these, DSP is the most common form and has been estimated to affect as many as 50% of all individuals infected with HIV.[3-5] It is a predominantly distal, symmetric, and sensory neuropathy clinically characterized by decreased sensation in the distal extremities along with paresthesias, dysesthesias, and pain in a symmetric stocking-glove distribution. Clinical signs include diminished or absent ankle jerks and decreased pinprick and vibration sensation involving the distal lower extremities. Weakness and muscle atrophy are rare. These manifestations are generally static or slowly progressive over time, but in advanced cases, more proximal areas and upper extremities can also be involved.[4]

HIV-DSP is predominantly a small fiber neuropathy, and nerve biopsy shows a length-dependent axonal degeneration. Nerve conduction studies may reveal slowed conduction velocities and reduced sensory nerve action potentials.[2-4]

Our patient too presented with sensory symptoms suggestive of small fiber neuropathy. However, the absence of characteristic skin lesions and nerve thickening made leprosy an unlikely possibility. Further, preservation of deep tendon reflexes in lepromatous leprosy is a useful sign, which helps to differentiate leprosy from other causes of neuropathy.[6] This patient had diminished ankle jerk, which also pointed against leprosy. Certain useful clues such as fever and weight loss, too, were missed initially. Therefore, this case is being reported to draw the attention of dermatologists toward HIV as a cause of sensory neuropathy.

Table 1: Major presentations of human immunodeficiency virus-associated peripheral neuropathy

DSP
Inflammatory demyelinating polyneuropathy (including Guillain-Barre syndrome and chronic inflammatory demyelinating polyradiculoneuropathy)
Multiple mononeuropathies (e.g., vasculitis, CMV related)
Polyradiculopathy (usually CMV related)
Autonomic neuropathy
Sensory ganglionitis

DSP=Distal symmetric polyneuropathy; CMV=Cytomegalovirus

CONCLUSION

In the present era of HIV-AIDS, one must always keep HIV neuropathy as a potential differential diagnosis while dealing with any case of peripheral neuropathy. Dermatologists should especially focus on HIV as a cause of neuropathy, apart from leprosy, as they are more likely to deal with patients suffering from sexually transmitted diseases including HIV.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Mangus LM, Dorsey JL, Laast VA, Ringkamp M, Ebenezer GJ, Hauer P, *et al.* Unraveling the pathogenesis of HIV peripheral neuropathy: Insights from a simian immunodeficiency virus macaque model. ILAR J 2014;54:296-303.
2. Amruth G, Praveen SK, Nataraju B, Nagaraja BS. HIV associated sensory neuropathy. J Clin Diagn Res 2014;8:MC04-7.
3. Amato AA, Barohn RJ. Peripheral neuropathy. In: Kasper DL, Hauser SL, Jameson JL, Fauci AS, Longo DL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 19th ed. New York: Mc Graw Hill Medical; 2015. p. 2674-94.
4. Schütz SG, Robinson-Papp J. HIV-related neuropathy: Current perspectives. HIV AIDS (Auckl) 2013;5:243-51.
5. Kaku M, Simpson DM. HIV neuropathy. Curr Opin HIV AIDS 2014;9:521-6.
6. Sharma PK, Sharma P. Differential diagnosis of neurological and other conditions. In: Kar HK, Kumar B, editors. IAL Textbook of Leprosy. 1st ed. New Delhi: Jaypee Brothers Medical Publishers; 2010. p. 211-26.