Case Report

Simultaneous central and peripheral nervous system involvement in the same patient: An interesting case report

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ABSTRACT

Involvement of the central and peripheral nervous systems (PNS) in the same patient is a rare phenomenon. The causes can be clubbed as infective, demyelinating diseases like multiple sclerosis, or may be post-vaccination. Neurotoxins are also associated with demyelination. Either of the two may present earlier, followed by the other one. Due to the involvement of both central and peripheral, the patient usually becomes moribund much earlier. If not diagnosed earlier, the patient may have difficulty surviving. Here, we present an interesting case in which both the central and PNSs were involved in the same patient. The cause we found out was infectious, and the patient improved with therapy and has been under follow-up for the last 3 months on treatment. The case in itself is very essential for primary care physicians, as non-specific multiple neurological symptoms, if diagnosed early, may benefit the patient.

Key words: Highly active antiretroviral therapy, Human immunodeficiency virus, Peripheral neuropathy, Progressive multifocal leukoencephalopathy

Demyelinating disorders, typically categorized into central and peripheral nervous system (PNS) pathologies, rarely coexist within a single patient, making such presentations diagnostically and clinically challenging. Central demyelination, as seen in conditions like multiple sclerosis, primarily affects the brain and spinal cord, whereas peripheral demyelination, such as chronic inflammatory demyelinating polyneuropathy, involves peripheral nerves. Each entity has distinct pathophysiological mechanisms, clinical manifestations, and therapeutic approaches. However, in rare instances, both forms may manifest simultaneously or sequentially, termed combined central and peripheral demyelination. This overlapping syndrome can present with varied neurological signs, requiring a high index of suspicion and comprehensive evaluation for accurate diagnosis.

Here, we present an intriguing case of a patient exhibiting both central and peripheral demyelinating features, shedding light on the clinical course, diagnostic complexities, and management strategies. This case is noteworthy because it demonstrates the rare coexistence of central and PNS involvement in a patient with previously undiagnosed advanced human immunodeficiency virus (HIV) infection [1]. The simultaneous occurrence of magnetic resonance imaging (MRI) findings suggestive of progressive multifocal leukoencephalopathy (PML) and electrophysiologically proven sensorimotor polyneuropathy highlights the broad neurotropic potential of HIV and the diagnostic challenges posed by multifocal neurological presentations [2]. Reporting this case increases clinician awareness of HIV as a unifying diagnosis in patients presenting with concurrent central nervous system (CNS) and PNS manifestations, facilitating earlier diagnosis and treatment.

CASE REPORT

A 63-year-old female patient from Hamirpur, Himachal Pradesh, India, presented with bilateral lower limb weakness and urinary incontinence for the last 4 days. The lower limb weakness was insidious in onset and progressive in nature and associated with pain in the bilateral lower limbs. No history of fever was present. A history of forgetfulness and decreased talking with family members was also present. She had a history of hypothyroidism for which she was taking levothyroxine 50 mg.

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On examination, the patient was conscious and oriented. Her pulse was 82/min. Her Blood pressure was 116/70 mmHg and Respiratory rate was 16/min. On general physical examination, there was no pallor, icterus, clubbing, pedal edema, cyanosis, or lymphadenopathy. On abdominal examination, it was soft and non-tender, non-distended, with no organomegaly. Chest was clear with bilateral air entry present. Higher mental function and the cranial nerves examination were unremarkable. No neck rigidity, Kernig sign, or Brudzinski signs were present. Abnormal findings were confined to the neurological examination as detailed in Table 1.

The patient was investigated radiologically for back pain. An MRI of the lumbar spine was suggestive of a mild disk bulge at the L4-L5 level, causing indentation of thecal sac with mild spinal narrowing.

She developed a fever a few days after hospital admission. A summary of the investigations done is given in Table 1. The result of electrophoresis was polyclonal, which was consistent with chronic inflammation. On a low-dose computerized tomography scan, she was found to have mild bilateral pleural effusion, which was attributed to hypoalbuminemia.

Table 1: Investigations during hospitalization

Investigation category	Parameter	Result
Hematological	Hemoglobin	5.9 g/dL
	Total leukocyte count	$2.57\times10^3/\mu L$
	Platelet count	$198\times10^3/\mu L$
	Erythrocyte sedimentation rate	>120 mm/h
	Reticulocyte count	0.8%
Inflammatory markers	C-reactive protein	57.6 mg/L
	Procalcitonin	0.53 ng/mL
Renal function	Creatinine	1.2 mg/dL
Liver function	Total bilirubin	0.3 mg/dL
	Aspartate aminotransferase	125 U/L
	Alanine transaminase	71 U/L
	Alkaline phosphatase	582 U/L
	Albumin	1.4 g/dL
	Albumin/globulin ratio	0.2
Iron profile	Serum iron	19 µg/dL
	Total iron-binding capacity.	98 µg/dL
	Ferritin	465.2 ng/mL
	Transferrin saturation	19%
Immunoglobulin profile	IgA	4.13 g/L
	IgG	50.00 g/L
	IgM	4.52 g/L
	Kappa free light chain	468.07 mg/L
	Lambda free light chain	470.40 mg/L
	Serum protein electrophoresis	Polyclonal pattern suggestive of chronic inflammation
Infectious/autoimmune workup	HIV rapid antigen test (initial)	Negative
	HIV ELISA (repeat)	Positive
	VDRL, IGRA, Cryoglobulin, Cryofibrinogen	Negative
	Viral hepatitis markers	Negative
	Carcinoembryonic antigen, alpha-fetoprotein	Negative
Radiological investigations	Magnetic resonance imaging lumbar spine	Mild L4-L5 disk bulge with indentation of thecal sac and mild spinal canal narrowing
	Low-dose CT thorax	Mild bilateral pleural effusion
Cerebrospinal fluid analysis	Cell count	3 lymphomononuclear cells/µL
	Protein	222 mg/dL
	Glucose	62 mg/dL
	Adenosine deaminase	11.4 U/L
	Gram stain, India ink, AFB stain	Negative
	Cryptococcal antigen	Not detected
	HSV DNA	Not detected

HIV: Human immunodeficiency virus, ELISA: Enzyme-linked immunosorbent assay, IgA: Immunoglobulin A, IgG: Immunoglobulin G, IgM: Immunoglobulin M, CT: Computed tomography, VDRL: Venereal disease research laboratory, IGRA: Interferon-gamma release assay

She was started on broad-spectrum antibiotics and symptomatic treatment, but her fever failed to subside, and she also developed difficulty in eating due to oral ulcers, for which she was examined and diagnosed with oral candidiasis. She was started on fluconazole alongside broad-spectrum antibiotics. In due course of time in hospital, the power in both her lower limbs progressed to 0/5 with some sensory involvement in both upper limbs and lower limbs as detailed in Table 1.

The patient was again investigated for HIV by enzyme-linked immunosorbent assay and was found to be HIV positive this time, a few days after the hospital admission. Her CD4 count and HIV viral load were sent. Cerebrospinal fluid (CSF) analysis was done, which showed 3 lymphomononuclear cells per μL, 222 mg/dL protein, 62 mg/dL glucose, 11.4 U/L Adenosine deaminase, and negative on gram, India ink, and acid-fast bacilli staining. Cryptococcal antigen as well as HSV DNA was not detected in CSF.

Motor and sensory nerve conduction studies (NCS) were performed for all limbs. Motor NCSs were carried out in the median, ulnar, peroneal, and posterior tibial nerves (Fig. 1a). Sensory NCSs were performed in the sural, median, and ulnar nerves (Fig. 1b). NCSs showed bilateral median sensory nerve action potential (SNAP) with reduced bilateral ulnar sensory conduction velocity. Right median and bilateral post-tibial compound muscle action potential (CMAP) and distal latency were prolonged. The right ulnar, left tibial, and bilateral peroneal CMAP amplitude was reduced. Furthermore, the conduction velocity of the right ulnar, bilateral median, tibial, and peroneal nerves was reduced. Hence, NCS was suggestive of sensorimotor axonal polyneuropathy. She was then started on intravenous (IV) immunoglobulin (2 g/kg/day) and pneumocystis, toxoplasma, and bacterial pneumonia prophylaxis. Screening for other blood-borne viruses and opportunistic infections was all negative. Suspecting polyradiculopathy due to cytomegalovirus (CMV), oral Ganciclovir was also added. Her CD4 count report came out to be 11 cells/μL, and HIV viral load was 10,10,000 IU/mL. Highly active Anti-retroviral therapy (HAART) comprising Tenofovir/Lamivudine/Dolutegravir (300/300/50) was started.

After treatment, the patient did not improve and significantly deteriorated with altered sensorium and cognitive decline. Suspecting immune reconstitution syndrome, IV Dexamethasone (1 g/day) was added to HAART, and Contrast-enhanced MRI (CEMRI) brain was ordered. CEMRI was suggestive of asymmetric areas of confluent T2/fluid-attenuated inversion recovery (FLAIR) hyperintensities seen involving centrum semiovale and subcortical white matter of bilateral fronto-parietal lobes (left>right). No evidence of diffusion restriction or blooming on diffusion-weighted images. No post-contrast enhancement was seen. Multiple T2/FLAIR hyperintensities were seen involving the right thalamus, left external capsule, and subcortical white matter of bilateral fronto-parietal lobes. A thin rim of diffusion restriction was seen along the ependymal lining of the bilateral lateral ventricles and the fourth ventricle. Tiny foci of diffusion restriction are seen involving the left cerebral peduncle, the left hemi-pons, at the level of the cervico-medullary junction, the corona radiata of the left frontal lobe, and the anterior limb of the right internal capsule. Minimal IVH is noted involving the dependent portion of the occipital horns of bilateral lateral ventricles. Small foci of blooming on SWI are seen in the left putamen and right cerebellum with microhemorrhages. A small linear area of blooming is seen in the right frontal region. Diffuse cerebral atrophy with prominent extra-axial spaces and ventricular system was seen. The possibility of PML was kept (Fig. 2). The bad prognostic nature of the illness was explained to the patient's relatives. Due to prolonged hospitalization and the progressive nature of the disease, attendants wanted to take their patient home.

The patient was given discharge on request with a Ryles tube (RT) and a Foley's *in situ*. The attendants were explained regarding the basic care of RT, Foleys and the patient. After 3 weeks, the patient was brought by attendants for follow-up, and there was no significant change in the patient's neurological condition. Symptomatic treatment, along with HAART, was continued. After that, the patient was lost to follow-up.

DISCUSSION

Neurological disorders are frequently encountered in individuals with HIV infection and may involve any part of the neuraxis, including the brain, spinal cord, nerve roots, peripheral nerves, and muscles. The clinical presentation is often heterogeneous, particularly in advanced disease,

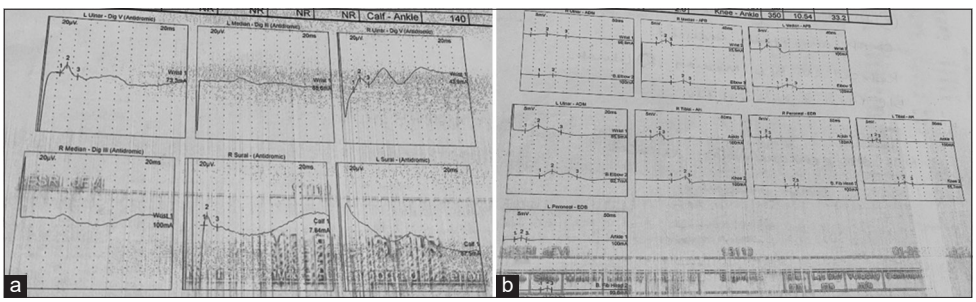

Figure 1: Motor and sensory nerve conduction studies (NCS) were performed for all limbs. (a) Motor NCSs were carried out in the median, ulnar, peroneal, and posterior tibial nerves; (b) Sensory NCSs were performed in the sural, median, and ulnar nerves

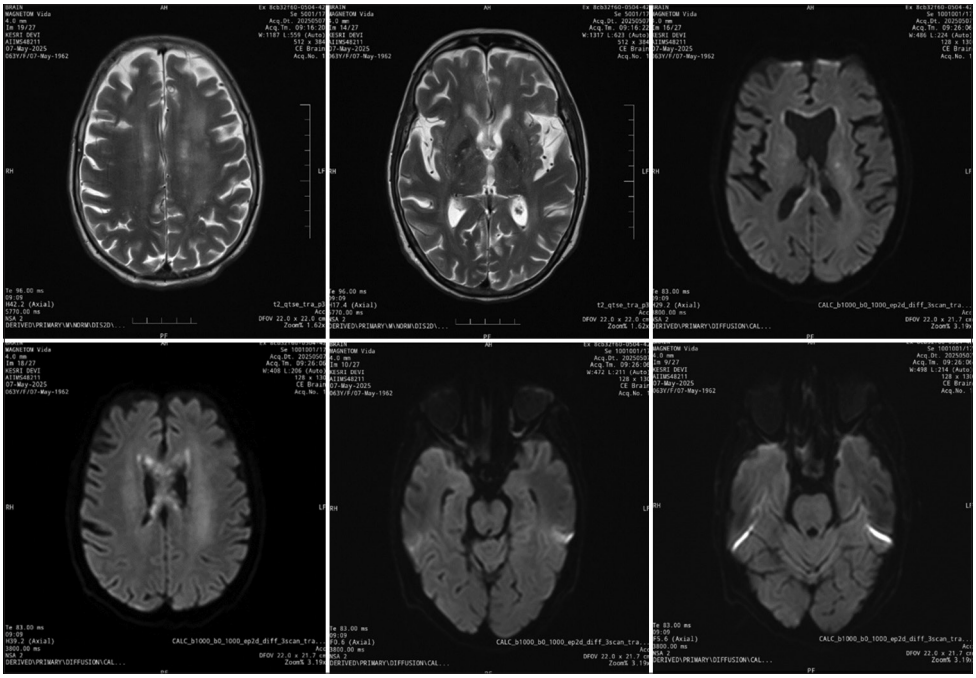

Figure 2: Contrast-enhanced magnetic resonance imaging brain

where multiple neurological processes may coexist and complicate diagnosis. A comprehensive diagnostic approach involving detailed neurological examination, neuroimaging, CSF analysis, and electrophysiological studies remains essential for accurate diagnosis. The severity and nature of neurological manifestations are closely related to the degree of immunosuppression, as reflected by the CD4 cell count and HIV viral load. Advanced HIV infection can simultaneously affect both the CNS and PNS through direct viral neurotoxicity, immune dysregulation, and opportunistic infections [1,2].

The CNS involvement in our patient was suggested by progressive cognitive decline, altered sensorium, and MRI findings demonstrating multifocal asymmetric white matter lesions involving bilateral frontoparietal regions, thalamus, external capsule, brainstem, and periventricular areas. These radiological findings, together with profound immunosuppression (CD4 count 11 cells/μL) and a markedly elevated HIV viral load, were highly suggestive of PML [3,4]. PML is a demyelinating disorder caused by reactivation of the latent JC virus in immunocompromised individuals and remains one of the most important opportunistic neurological complications of advanced acquired immunodeficiency syndrome (AIDS). Although confirmation by CSF JC-virus polymerase chain reaction could not be obtained, the clinical and radiological profile strongly supported the diagnosis [5-7].

Simultaneously, our patient demonstrated significant PNS involvement. NCSs revealed a sensorimotor axonal polyneuropathy characterized by reduced SNAPs, reduced CMAPs, prolonged distal latencies, and reduced conduction velocities. HIV-associated peripheral neuropathy is the most common neurological complication of HIV infection and is particularly prevalent in patients with advanced disease, severe immunosuppression, and high viral loads [8-10]. The underlying pathology is predominantly axonal and is believed to result from direct viral neurotoxicity, activation of infected macrophages, release of inflammatory cytokines, and immune-mediated neuronal injury [11].

Alternative diagnoses were carefully considered in our patient. HIV-associated encephalopathy was considered due to the cognitive decline and altered sensorium; however, the characteristic multifocal asymmetric white matter lesions on MRI were more suggestive of PML than diffuse HIV encephalopathy. CMV polyradiculopathy was also suspected due to rapidly progressive lower limb weakness and profound immunosuppression, prompting initiation of ganciclovir therapy. However, CSF analysis did not demonstrate features typically associated with CMV infection. Primary CNS lymphoma was considered in the differential diagnosis; nevertheless, the absence of contrast-enhancing mass lesions, significant edema, or mass effect on MRI made this diagnosis less likely.

An important aspect of this case is the simultaneous involvement of both the CNS and PNS. Although uncommon, such coexistence has been described in advanced HIV infection. HIV is unique among infectious diseases in its ability to affect multiple levels of the nervous system concurrently [12]. In advanced AIDS, opportunistic CNS infections such as PML may occur alongside HIV-associated peripheral neuropathy or polyradiculopathy, resulting in overlapping neurological manifestations [4]. Therefore, the coexistence of central and peripheral neurological deficits in our patient is best explained by widespread neurological involvement secondary to advanced, untreated HIV infection rather than two unrelated disease processes.

This case highlights the importance of considering HIV infection in patients presenting with unexplained multifocal neurological deficits, especially when both CNS and PNS signs are present. Early HIV testing, prompt recognition of opportunistic neurological

complications, and timely initiation of antiretroviral therapy are crucial to improving outcomes and reducing neurological morbidity.

CONCLUSION

HIV infection can lead to a range of central and PNS complications, with up to 53% of individuals experiencing at least one neurological symptom within the first 12 weeks of diagnosis. These disorders have a wide range of causes, including inflammatory, immunologic, infectious, vasculitic, and neoplastic processes. If HIV is identified early, then early initiation of HAART can prevent the development of PML by improving immunocompromised status. Early identification is crucial, as these neurological diseases can result in substantial morbidity and reduced quality of life. Therefore, HIV testing should be obtained in individuals presenting with multiple new neurological deficits.

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