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HIV Lumbosacral Radiculoplexus Neuropathy Mimicking Lymphoma: Diffuse Infiltrative Lymphocytosis Syndrome (DILS) Restricted to Nerve?

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Abstract

Diffuse infiltrative lymphocytosis syndrome (DILS) is a hyperimmune reaction against HIV. It leads to MHC-restricted clonal expansion of CD8 T-cells characterized by circulating CD8 hyperlymphocytosis and CD8 T-cell infiltration in organs. Our patient presented with painful lumbosacral radiculoplexus neuropathy and tested positive for HIV. Nerve biopsy showed large collections of CD-8 lymphocytes suspicious for lymphoma. Symptoms, signs and repeat biopsy improved with antiretroviral treatment. The presentation and treatment response suggest this case is localized DILS.

Keywords

diffuse infiltrative lymphocytosis syndrome (DILS); peripheral neuropathy; HIV; lumbosacral plexopathy; lumbosacral radiculoplexus neuropathy

A number of different types of peripheral neuropathy occur in HIV patients,¹ including distal symmetric polyneuropathy, multiple mononeuropathy, acute or chronic inflammatory demyelinating polyradiculoneuropathy, cauda equina syndrome, autonomic neuropathy, herpes zoster radiculitis, sensory ganglioneuritis, painful sensory neuropathy due to toxicity of antiviral drugs and a neuropathy associated with the diffuse infiltrative lymphocytosis syndrome (DILS).

DILS occurs exclusively in HIV patients.^{2–7} It is characterized by MHC-restricted monoclonal expansion of CD8 T-cells resulting in circulating CD8 lymphocytosis and infiltration of the CD8 T-cells in multiple organs, including the peripheral nerve. The most

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common manifestations of DILS are bilateral parotid gland enlargement, sicca syndrome, lymphocytic interstitial pneumonitis and organomegaly.

The neurological manifestations of DILS include unilateral or bilateral facial nerve palsy, aseptic meningitis, myositis⁸ and peripheral neuropathy.⁹ Peripheral neuropathy can be the presenting symptom of DILS, and as such it occasionally has led to the diagnosis of HIV. It has been described as an acute or subacute painful, symmetric, length dependent, axonal neuropathy mainly involving the distal lower extremities. Cerebrospinal fluid (CSF) analysis usually shows lymphocytosis with elevated protein. Nerve biopsy reveals dense perivascular epineurial and endoneurial mononuclear infiltration. Sheets of T cells may be seen in the epineurium, remote from the angiocentric sites. Patients with DILS (who all have HIV) tend to have higher CD4 counts, and therefore they are less susceptible to opportunistic infections and have a longer survival time. DILS responds well to prednisone and/or antiretroviral treatment with complete or partial improvement of the neuropathy.

We present a case of a patient with painful lumbosacral radiculoplexus neuropathy (with both proximal and distal involvement) and HIV disease who was initially suspected of having peripheral nerve lymphoma. However, with triple antiretroviral therapy the patient's symptoms, and signs and inflammatory infiltrates on the nerve biopsy improved. This pattern confirmed the cause of his neuropathy as likely DILS.

Case Report

A 57-year-old man (EJF) presented with an 8 month history of gradual, symmetric, progressive pain and weakness in his lower limbs. He initially noticed soreness and stabbing pain in his feet, imbalance, and pricking and tingling sensation with contact allodynia (bed sheets rubbing against his feet caused terrible pain). This was followed a few weeks later by weakness in the feet and charley horse type cramping pain in his calves. He had difficulty getting up from a low seated position and needed a cane to prevent himself from falling. There was no weight loss, night sweats, fever, cranial nerve involvement, sexual dysfunction, bowel or urine incontinence, dryness of the eyes or dryness of the mouth.

He had mild hyperlipidemia, was a homosexual, and drank two shots of whiskey per day. He denied intravenous drug use. There was no family history of similar symptoms or neuromuscular disorder. He took no medication.

On examination, his vital signs were normal, and there was no lymphadenopathy or parotid gland enlargement. His cranial nerve and upper extremity examination revealed normal strength, sensation and reflexes. He had mild weakness of his gluteus maximus and hamstring (25% weak) and moderate weakness of his iliopsoas and quadriceps (50% weak) muscles. He had severe weakness in dorsiflexors and plantar flexor muscles of the feet and toes (more than 75% weak). There was profound sensory loss (to touch, pin, vibration and proprioception) in a stocking distribution up to his ankles with absent knee and ankle reflexes. He could only walk with assistance and had a steppage gait. His Neuropathy Impairment Score (NIS) (a composite score of weakness, reflex change and sensory loss with a maximum score of 244) showed a moderate to severe neuropathy (Table 1).¹⁰

Extensive laboratory tests including complete blood count (CBC), erythrocyte sedimentation rate (ESR), vitamin B12 and folate levels, thyroid stimulating hormone (TSH), antinuclear antibody (ANA), extractable nuclear antigen (ENA), rheumatoid factor (RF), antinuclear cytoplasmic antibody (ANCA), hepatitis B and C serology, cryoglobulin, Lyme disease screen, heavy metal screen, fasting blood sugar and HbA1c were normal. Protein monoclonal study showed hypergammaglobulinemia. His HIV-1 screen was reactive, and the western blot was positive. The quantitative HIV-1 RNA level was 42,066 copies/ml. The

CD8 count was elevated at 1100/ul (152-838), and the CD4 count was low at 298/ul (401-1532). Tests for toxoplasmosis and syphilis were negative. Serum CMV-IgG, IgM and polymerase chain reaction (PCR) were negative.

CSF examination showed a markedly elevated protein, normal glucose and a pleocytosis that was almost all lymphocytes (Table 1). Cytology and immunophenotyping and gram stain and cultures for various infectious agents were negative. CMV IgG, CMV IgM and PCR were negative. CSF Lyme disease, venereal disease research laboratory (VDRL) and Cryptococcus antigen were negative.

Nerve conduction studies and electromyography (EMG) showed a predominantly axonal neuropathic disorder with reduced or absent compound muscle action potentials (CMAP) and sensory nerve action potentials (SNAP) and fibrillation potentials and large motor unit potentials in multiple muscles. These abnormalities affected lumbosacral greater than cervical or thoracic levels and were interpreted as a polyradiculoneuropathy or as a radiculoplexus neuropathy (Table 2). Quantitative sensory testing using CASE IV showed pan-modality (vibration, cooling and heat-pain) raised thresholds at the left foot.

Enhanced chest, abdomen and pelvis computer tomography (CT), magnetic resonance imaging (MRI) of lumbosacral spine without Gadolinium, MRI of lumbosacral plexus, metastatic bone survey, bone marrow aspirate and biopsy were normal. Enhanced MRI of the head showed subtle diffuse dural enhancement (Fig. 1, A – arrows) with bilateral deep white matter changes.

A right sural nerve biopsy, showed large collections (sheets) of mononuclear inflammatory cells located perivascularly in epineurial and perineurial sites (Fig. 2, A-D). These cells reacted strongly for leukocyte (CD-45) and weakly for macrophage (CD-68) preparations, indicating that they were predominantly lymphocytes. Further immunostaining revealed that the lymphocytic infiltrates were predominantly CD-8 positive with a few cells reacting for CD-4 and fewer for CD-20 (a B-cell marker) (Fig. 2). T-cell gene rearrangement was negative. The biopsy was reviewed by a hematopathologist (PJK), who was concerned about lymphoplasmacytic lymphoma, plasma cell tumor, myeloma or lymphocytosis secondary to his HIV infection.

The patient was treated with a combination antiretroviral regimen consisting of zidovudine, abacavir, and lamivudine from the time of his evaluation, and there was marked improvement of his neuropathy on clinical examination. He became much stronger and had less difficulty walking or getting up from a low chair. His NIS substantially improved (by greater than 50%) at one and four month visits after beginning antiretroviral treatment (Table 1). A repeat CSF analysis one and four months after antiretroviral treatment showed reduction in the number of the total nucleated cells and protein levels (Table 1). At four months, nerve conduction studies and EMG were unchanged, whereas quantitative sensory testing showed improvement in vibration, cooling and heat-pain thresholds.

A repeat MRI of the lumbosacral spine with Gadolinium two months after presentation (Fig. 1) showed diffuse leptomeningeal enhancement. Because there was still concern for a lymphomatous process, a repeat right sural biopsy was performed four months after beginning antiretroviral treatment and revealed marked improvement of the inflammatory infiltrates in the nerve (Fig. 2, E-G). The CD-8 positive cells were now rare and scattered, and the biopsy was no longer concerning for lymphoma.

At five years from onset, the patient continued to do well with only residual mild distal feet weakness and numbness and intermittent tingling of the feet. His pain was gone. His gait and strength were dramatically improved and were almost normal. The lumbosacral

radiculoplexus neuropathy from DILS had essentially resolved and he no longer needed to use a cane when walking.

Discussion

The clinical presentation of our patient is that of a painful, symmetrical lumbosacral radiculoplexus neuropathy. The neuropathy was the initial manifestation of HIV infection in this patient. The differential diagnosis for neuropathy in an HIV patient is largely dependent on the stage of the HIV disease. Our patient's CD4 count was more than 200 cells/ μ l. This indicates that he was less likely to have cytomegalovirus (CMV)-related polyradiculoneuropathy,¹¹ which occurs mainly in HIV patients with CD4 counts of less than 50 cells/ μ l. Patients affected with CMV polyradiculoneuropathy usually have sphincter involvement, an asymmetric presentation, CSF polymorphonuclear pleocytosis and evidence of CMV infection elsewhere in the body (retinitis, hepatitis, pneumonitis and encephalitis). CMV polyradiculoneuropathy is usually fatal if it is not treated. Our patient did not have CMV related polyradiculoneuropathy, as he had CSF lymphocytic pleocytosis, negative serum and CSF CMV PCR and cultures, and his neuropathy improved without specific treatment for CMV infection.

Other conditions also should be considered in the differential diagnosis of peripheral neuropathy in HIV-infected patients. A clinical presentation of bilateral, painful, lumbosacral radiculoplexus neuropathy is very common in diabetic and non-diabetic lumbosacral radiculoplexus neuropathy (DLRPN and LRPN).¹² These conditions begin unilaterally or asymmetrically and have associated weight loss. Our patient was not diabetic, he did not report any weight loss, and his neuropathy was symmetrical. The nerve biopsies in DLRPN and LRPN can show large inflammatory cell collections and evidence of microvasculitis, but the inflammatory infiltrates usually are not as prominent as in our case. Although the CSF protein is elevated in DLRPN and LRPN, it is usually below 200 mg/dL, and our patient's level of 967 would be much too high. There also should not be a pleocytosis present (as in this case). Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) can occur in association with HIV and be essentially indistinguishable from ordinary CIDP, including the response to immune modulating therapy.¹³ CSF findings in HIV patients with CIDP frequently show mild lymphocytic pleocytosis of 10-50 cells/mm³. Thus, the large lymphocytic pleocytosis noted in our patient would be very atypical for HIV-related CIDP. Pain has been reported to occur in 17% of CIDP patients but is unlikely to be as severe as the pain experienced by our patient.¹⁴ CIDP is usually a generalized polyradiculoneuropathy; our patient had exclusively a lower limb syndrome (lumbosacral radiculoplexus neuropathy), and his nerve conduction studies did not meet the electrophysiological criteria for demyelination.

Lymphomatous neuropathy¹⁵ was considered a likely possibility in our patient, as HIV patients have a higher incidence of non-Hodgkin lymphoma. It can present with severe pain as a lumbosacral radiculoplexus neuropathy. However, this usually tends to be asymmetrical and of the B-cell type (unlike our patient who has symmetrical presentation and was T-cell predominant). The histological findings of DILS are recognized to resemble lymphoma in nerve.¹⁶ The absence of constitutional symptoms and the improvement of the neuropathy with antiretroviral treatment alone would also argue somewhat against lymphomatous neuropathy (although we realize that lymphomas in HIV patients may recede with successful immune reconstitution). However, our hematopathologist (PJK) reviewed both nerve biopsies (from before and after treatment), and he concluded that lymphoma definitely was not the cause of the neuropathy. The types of lymphomas that typically occur in HIV individuals (diffuse large B-cell lymphoma, Burkitt's lymphoma, Hodgkin's lymphoma, and rarely MALT type lymphoma) were excluded based on morphology and phenotype.

Peripheral T-cell lymphoma (very rare in HIV positive individuals and the only other possible entity suggested by the phenotype of the dominant cells in the infiltrate) was excluded based on the results of the immunophenotyping (the T cells had a normal immunophenotype) and molecular genetic studies (no clonal T cell gene rearrangements by a method that has 95% sensitivity for T cell lymphomas).

Another condition worthy of consideration in patients with pathological findings similar to our patient's is the immune reconstitution inflammatory syndrome (IRIS).¹⁷ This is a term that describes a spectrum of clinical syndromes that occur within a few weeks to months following initiation of antiretroviral therapy and present either as worsening symptoms and signs and symptoms of an already diagnosed opportunistic infection or as unmasking of a previously unrecognized subclinical condition. The histopathology is often dominated by CD8+ T-lymphocyte inflammatory infiltrates. These syndromes are a result of antiretroviral therapy-induced restoration of a pathogen-specific immune response. In the central nervous system, IRIS has manifested itself as an atypical presentation of a variety of infections including tuberculosis, progressive multifocal leukoencephalopathy (PML), and cryptococcal infections. Our patient did not have IRIS since he was not receiving antiretroviral therapy when he presented with his neuropathy, and his symptoms did not worsen once antiretroviral therapy was instituted.

We believe that our patient's presentation is that of a localized form of DILS affecting predominantly the peripheral nerve system (the lumbosacral roots, plexus and lower limb nerves). He did not meet all the full criteria for the DILS diagnosis that requires: 1) HIV seropositivity, 2) bilateral salivary gland enlargement or xerostomia and xerophthalmia lasting for more than 6 months, and 3) histologic confirmation of salivary or lacrimal gland CD8 lymphocytic infiltration. However the nerve biopsy findings and the response of his neuropathic symptoms, CSF findings and the resolution of the CD8 lymphocytic infiltration on his nerve biopsy with antiretroviral treatment suggests that his neuropathy likely is a localized form of DILS. Why the peripheral nerve system was more susceptible for the immune reaction in his case is not fully clear. Our patient did not have sicca symptoms, so we did not do a minor salivary gland biopsy or Gallium-67 scintigraphy scan (to detect salivary gland involvement). The markedly elevated CSF protein, the pleocytosis and the meningeal enhancement all suggest that he likely had superimposed asymptomatic meningitis, which is also a recognized feature of DILS. Another explanation for the markedly elevated CSF protein level is a functional CSF block due to the inflammatory response, and its gradual decline may be due to effective reduction of inflammation by the antiviral treatment.

Our case expands the differential diagnosis of painful lumbosacral radiculoplexus neuropathy to include DILS and HIV infection. The histological features of the nerve can be confused with lymphomatous infiltration of the nerve. The absence of characteristic clinical features of DILS such as parotid gland enlargement and pulmonary involvement does not exclude this diagnosis. We believe that our patient has a localized form of DILS affecting predominately the peripheral nerves of the lower limbs that improved with antiviral treatment.

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Abbreviations

ANA	antinuclear antibody
ANCA	antinuclear cytoplasmic antibody
CBC	complete blood count
CIDP	chronic inflammatory demyelinating polyradiculoneuropathy
CMAP	compound muscle action potential
CMV	cytomegalovirus
CSF	cerebrospinal fluid
CT	computer tomography
DILS	diffuse infiltrative lymphocytosis syndrome
DLRPN	diabetic lumbosacral radiculoplexus neuropathy
EMG	electromyography
ENA	extractable nuclear antigen
ESR	erythrocyte sedimentation rate
LRPN	non-diabetic lumbosacral radiculoplexus neuropathy
MRI	magnetic resonance imaging
NIS	Neuropathy Impairment Score
PCR	polymerase chain reaction
RF	rheumatoid factor
SNAP	sensory nerve action potential
TSH	thyroid stimulating hormone
VDRL	venereal disease research laboratory

Figure 1. Post-contrast MRI of the head (**A**), showing dural enhancement (arrows) (incidentally noted is left arachnoid cyst, asterisk) and of the lumbar spine showing multiple lumbosacral root thickening and post contrast enhancement (**B**) (axial) (arrows) and diffuse leptomeningeal enhancement (sagittal) (arrows) (**C**).

Figure 2.

Left sural nerve biopsy (paraffin sections) taken during the initial evaluation before treatment (**A-D**) from a man with a severe bilateral lumbosacral radiculoplexus neuropathy and newly diagnosed HIV infection. There were large collections (sheets) of epineurial, greater than perineurial or endoneurial inflammatory cells that were frequently perivascular (longitudinal section, H&E, **A**). Serial transverse paraffin sections showed the large collections (H&E, **B**) reacted to CD-8 (**C**) but only minimally to CD-4 (**D**) immunostains. These findings were suspicious for a lymphoma or CD-8 lymphocytosis (DILS). After four months of triple antiretroviral treatment, a repeat left sural nerve biopsy was taken from the proximal stump of the prior biopsy site. Serial transverse paraffin sections (**E-G**) showed a marked improvement of the inflammatory infiltrates with only small, scattered epineurial inflammatory collections remaining. There was now only minimal reactivity to CD-8 lymphocytes (**F**) and little to CD-4 lymphocytes (**G**). These results suggest that the process was DILS and not a lymphoma.

Table 1

Neuropathy Impairment Score (NIS) and spinal fluid evaluation at presentation and in follow-up after antiretroviral treatment in a patient with DILS and lumbosacral radiculoplexus neuropathy

	At presentation	One month	Four months
NIS Score (points)	60	53	28
CSF protein (mg/dL)	967	599	279
CSF cells	184	7	11
CSF lymphocytes	98%	87%	89%

CSF = cerebral spinal fluid, NIS = Neuropathy Impairment Score

Table 2

Initial electrophysiological testing from a patient with DILS showing radiculoplexus neuropathy most severely affecting lumbosacral levels

Nerve Conduction Studies					
Stimulate (Record)	A (mV/ μ V)	CV (m/s)	DL (ms)	F-wave latency (MS)	
R Median sensory (Index)	12 (>15)	49 (>56)	3.3 (<3.6)		
R Median motor (APB)	6.2 (>4)	47 (>48)	3.8 (<4.5)	37.9	
R Ulnar motor (ADM)	6.5 (>6)	41 (>51)	2.8 (<3.6)	33.3	
R Ulnar sensory (Fifth)	13 (>10)	41 (>54)	3.0 (<3.1)		
R Peroneal motor (EDB)	0.3 (>2)	22 (>41)	7.6 (<6.6)		
R Tibial motor (AH)	0 (>4)	--	NR (<6.1)		
R Sural sensory (Ankle)	0 (>6)	--	NR (4.5)		

EMG				
Muscle	Insertional Activity	Fibrillation Potentials	Reduced Recruitment	MUP
				Long Duration High Amplitude
R Deltoid	Normal	0		+
R FDI	Increased	0		++
R Medial gastroc.	Increased	+		+
R Gluteus maximus	Normal	0		++
R rectus femoris	Normal	0	+	+
R TFL	Increased	0		+
R Tibialis anterior	Increased	+++	++	+++
R Vastus medialis	Increased	0	++	+
R Thoracic paraspinals	Increased	+	+	+
R Paraspinal lumbar (upper)	Normal	0	+	+

FDI = first dorsal interosseus, MUP = motor unit potentials, R = right, TFL = tensor fascia latae