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LEPROSY NEUROPATHY MIMICKING CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY: A DIAGNOSTIC DILEMMA

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ABSTRACT

Background: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated peripheral neuropathy in which the clinical phenotype should be supported by objective evidence of demyelination. Motor-predominant presentations and severe axonal loss may create substantial diagnostic difficulty. **Case presentation**: A 33-year-old man had seven months of worsening neurological symptoms. He developed motor problems especially he could not lift the foot upward (dorsiflex ankle) while pushing the foot downward (plantar flexion) remained normal. Sensory tests done at the bedside were said to be normal. Sharp pain triggered by a gentle touch indicated allodynia. Cranial nerves were not affected. Ultrasound of nerves showed uneven thickening of both ulnar nerves both common peroneal nerves and the left greater auricular nerve. Higher blood flow was seen in the right ulnar and peroneal nerves. A skin biopsy taken from a lesion on the upper back revealed chronic inflammation, around nerves and skin appendages; it also suggested borderline leprosy as a possibility. Nerve-conduction studies of all four limbs found no CMAPs in the common peroneal and right ulnar nerves. The right median nerve had a CMAP amplitude. Sensory responses that were tested stayed normal. F-waves could not be recorded. The formal conclusion was motor axonal neuropathy affecting all four limbs. HBsAg and HBeAg tests were reactive. Hbv DNA was 1.93 × 10^8 IU/mL (log 8.29). **Conclusion**: The available evidence supports a chronic painful motor-predominant polyneuropathy with severe motor axonal abnormalities and peripheral nerve enlargement. CIDP is an important clinical consideration but is not established by the available electrodiagnostic data. Possible borderline leprosy, vasculitic neuropathy and HBV-associated neuropathy remain relevant alternatives.

KEYWORDS: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP); Motor-predominant neuropathy; Axonal neuropathy; Leprosy; Hansen disease; Alodynia; Hepatitis B; Peripheral neuropathy.

INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuropathy is a chronic immune-mediated disorder of the peripheral nervous system, classically presenting with progressive or relapsing weakness, sensory dysfunction, and reduced or absent deep tendon reflexes.[2] Motor-predominant phenotypes are recognized; however, diagnosis requires appropriate electrodiagnostic evidence of demyelination together with clinical correlation and exclusion of alternative causes.

Several different diseases can mimic CIDP.[2,8,9] These include motor neuropathy, nerve problems caused by infections nerve damage from blood vessel inflammation, inherited nerve disorders and other immune-driven nerve diseases. Severe axonal loss may produce marked weakness and absent motor responses, potentially complicating interpretation of electrodiagnostic studies.

This case is unusual because several possible disease processes are present at the time. The patient has a

painful motor-predominant neuropathy enlarged peripheral nerves, skin biopsy showing possible borderline leprosy, active hepatitis B with a high viral load and a history of long-term steroid use, exogenous Cushing syndrome and relapsed Hansen disease.[1] These factors create a diagnostic dilemma between CIDP and other similar conditions

CASE PRESENTATION

A 33-year-old man was evaluated for a neurological illness of approximately 7 months' duration.

The dominant functional deficit was inability or marked impairment of ankle dorsiflexion. Plantar flexion remained intact. Despite the predominantly motor phenotype, bedside sensory examination was reported as intact.

The patient experienced severe pain with minimal touch, clinically consistent with allodynia. Cranial nerves were reported to be uninvolved.

The clinical records also documented chronic hepatitis B, chronic steroid exposure/exogenous Cushing syndrome, a history described as relapsed Hansen disease, peripheral neuropathy, iron deficiency, reactive thrombocytosis in the clinical documentation, right-eye cataract, and consideration of small-vessel/vasculitic neuropathy.

The combination of chronic progressive motor dysfunction and there was a chance of inflammation in the nerves a motor-predominant immune neuropathy such, as CIDP became an important consideration.

CLINICAL EXAMINATION

The clinical examination demonstrated a predominantly motor neurological deficit. Ankle dorsiflexion was unable or markedly impaired, whereas plantar flexion remained intact. Bedside sensory examination was reported as intact despite the patient's significant pain with minimal touch. The pain was clinically consistent with allodynia. No cranial-nerve involvement was documented. Referral information additionally described lower-limb pain, numbness involving the feet and numbness involving the upper limbs.

The combination of severe motor dysfunction and preserved bedside sensory examination, together with prominent pain, was clinically atypical for a simple motor-predominant CIDP phenotype and prompted further investigation.

INVESTIGATIONS

Peripheral Nerve Ultrasound

Peripheral nerve ultrasonography demonstrated asymmetric thickening of multiple peripheral nerves. The right and left ulnar nerves, right and left common peroneal nerves, and left greater auricular nerve were reported to be enlarged. Thickening of individual fascicles was described.

Mildly increased vascularity was reported in the right ulnar and peroneal nerves. The overall ultrasound impression was suggestive of polyneuritis.[4,5]

The measurements were: 0.37 cm^2 over 7 cm for the ulnar nerve 0.18 cm^2 over 4 cm for the left ulnar nerve 0.33 cm^2 over 6 cm for the right common peroneal nerve 0.19 cm^2 over 3.2 cm for the left common peroneal nerve 0.10 cm^2 for the right greater auricular nerve and 0.40 cm^2, for the left greater auricular nerve.

The presence of multiple enlarged peripheral nerves supported a pathological peripheral nerve process but was not specific for CIDP.

Skin Histopathology

the punch biopsy taken from the right upper-back skin lesion revealed thickening of the outer skin layer and a slight flattening of the skin ridges. In the underlying skin there were inflammatory cells around nerves and skin appendages with clusters of mononuclear cells such, as histiocytes and lymphocytes. No well-formed granulomas were observed. The pathologist noted that borderline leprosy might be present and recommended correlation and additional tests.

The histopathology is therefore suggestive rather than diagnostic of leprosy and should be interpreted with clinical and microbiological correlation.

Thus, the biopsy was suggestive rather than diagnostic of leprosy and needed to be interpreted together with the clinical history, nerve examination and appropriate microbiological investigations.

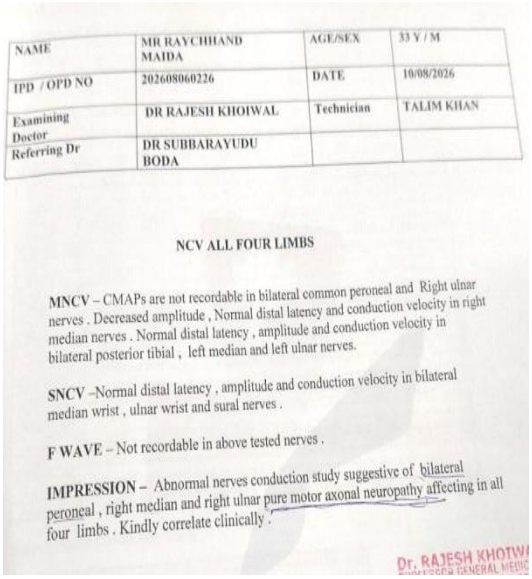
Nerve-Conduction Study

Four-limb nerve conduction studies demonstrated severe motor abnormalities. Compound muscle action potentials were non-recordable in the bilateral common peroneal nerves and the right ulnar nerve. The right median nerve showed reduced CMAP amplitude.

The bilateral posterior tibial nerves were reported to have normal distal latency, amplitude and conduction velocity. The left median and left ulnar nerves were reported as normal. Tested sensory responses were reported as normal. F-waves were not recordable in the tested nerves.

The formal electrophysiological impression was bilateral peroneal, right median and right ulnar pure motor axonal neuropathy affecting all four limbs.[2,8]

This was the important finding, in the diagnosis. The study showed motor axonal abnormalities but the study did not show a generalized demyelinating pattern. Because of this the study alone was not enough to diagnose CIDP.



NAME	MR RAYCHHAND MAIDA	AGE/SEX	33 Y / M
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Examining Doctor	DR RAJESH KHOIWAL	Technician	TALIM KHAN
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NCV ALL FOUR LIMBS

MNCV – CMAPs are not recordable in bilateral common peroneal and Right ulnar nerves . Decreased amplitude , Normal distal latency and conduction velocity in right median nerves . Normal distal latency , amplitude and conduction velocity in bilateral posterior tibial , left median and left ulnar nerves.

SNCV –Normal distal latency , amplitude and conduction velocity in bilateral median wrist , ulnar wrist and sural nerves .

F WAVE – Not recordable in above tested nerves .

IMPRESSION – Abnormal nerves conduction study suggestive of bilateral peroneal , right median and right ulnar pure motor axonal neuropathy affecting in all four limbs . Kindly correlate clinically .

Dr. RAJESH KHOIWA

Figer 1: Nerve conduction velocity (NCV) study.

NCV study demonstrating bilateral peroneal and right median/right ulnar pure motor axonal neuropathy, with non-recordable CMAPs in bilateral common peroneal and right ulnar nerves, reduced CMAP amplitude in the right median nerve, and non-recordable F-waves.

Source: Original investigation of the patient.

HEMATOLOGICAL AND BIOCHEMICAL EVALUATION

The hematological profile showed hemoglobin of 12.02 g/dL, hematocrit of 36.5%, MCV of 61.5 fL, MCH of 20.3 pg, MCHC of 32.9 g/dL, RDW of 20.4%, total leukocyte counts of 11,500/µL, neutrophils of 82%, platelet count of 384 ×10³/µL and CRP of 14.41 mg/L.

The hematology morphology report described a microcytic hypochromic blood picture with neutrophilic leukocytosis and thrombocytosis. Parasites were not seen.

The marked microcytosis and low MCH were compatible with the documented history of iron deficiency.

INFECTIOUS AND IMMUNOLOGICAL WORK-UP

The test results showed that the person had a HBsAg and a reactive HBeAg. The HBeAg index was 10.700. The HBV DNA level was very high at 1.93×10^8 IU/mL. This level is equivalent to a value of 8.29.

The HCV antibody test was not reactive. The HAV IgM test was not reactive. The HEV IgM test was also not reactive. The ANA IgG test was not. The index was 0.096.

A urine culture did not find any bacteria. The urine protein-to-creatinine ratio was 0.14. The procalcitonin levels recorded were 0.056 ng/mL and 0.225 ng/mL on dates.

The fact that the ANA was not positive does not rule out all types of neuropathy. The combination of pain a neuropathy and an active HBV infection still made an immune-related or vasculitic cause a possible explanation.[7]

Hepatitis B and Liver Evaluation

The patient had a HBsAg test and a positive HBeAg test. The HBV DNA level was 1.93×10^8 IU/mL whichs equivalent to a log value of 8.29.

An abdominal ultrasound showed that the liver had grade I fat buildup. There was no sign of any fluid-filled lump, in the liver.

Liver elasticity testing showed that the liver stiffness was 9.69 kPa. This was described as fibrosis, which matches the Metavir F2 stage.

The HBV results were important because HBV can cause immune-related and blood vessel-related nerve problems. These issues can lead to nerve conditions. However just having a HBV infection does not mean that HBV caused the patients nerve problem.

Cushing. Chronic Steroid Exposure

The medical history shows that the patient has had long-term use of steroids /exogenous Cushing syndrome.
This finding is important in the interpretation of the case. Prolonged corticosteroid exposure can modify manifestations and is particularly relevant in a patient in whom an infectious neuropathy, including Hansen disease is being considered.

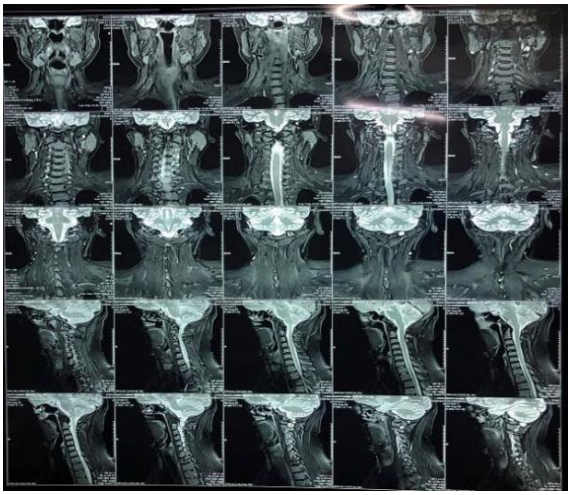
The history also becomes important because the case records describe relapsed Hansen disease. Chronic exposure may potentially influence infectious disease activity and its clinical expression.

However the supplied records do not provide verified endocrine data to independently establish the biochemical severity or current activity of Cushing syndrome. Therefore the diagnosis is presented here as a documented history of exogenous Cushing syndrome/ steroid exposure without adding unsupported endocrine findings.

Cervical Spine MRI

MRI of the spine demonstrated straightening of the cervical spine likely related to spasm or positioning along with small marginal osteophytes.

There was disc degeneration and desiccation at C3–C4, C4–C5 and C5–C6. Small posterior osteophyte disc complexes are pressing on the anterior subarachnoid space causing a narrowing of the central canal. The cervical cord looks normal in thickness. Signal with no intramedullary signal. These findings in the spine do not explain the diffuse four limb motor.



Figer 2: MRI of the cervical spine.

MRI cervical spine demonstrating multilevel degenerative disc changes with disc degeneration/desiccation at C3–C4, C4–C5 and C5–C6, without documented cervical cord signal abnormality. Source: Original investigation of the patient.

CARDIAC AND ABDOMINAL EVALUATION

2D echocardiography showed a ventricular ejection fraction of 60 percent. Left ventricular systolic and diastolic function were normal. There was no wall motion abnormality. Trivial mitral and tricuspid regurgitation were found. Heart chambers were normal. No clot, vegetation or pulmonary embolism was present. The inferior vena cava was normal and collapsible.

Abdominal ultrasonography showed grade I fatty infiltration of the liver. Liver elastography demonstrated fibrosis corresponding to Metavir F2.

These investigations did not identify a cardiac or structural explanation for the neurological presentation.

DIAGNOSIS REASONING

The chronic progressive motor phenotype, plus peripheral nerve enlargement initially suggested CIDP as a diagnosis.

However several findings argued against labeling the patient with CIDP. First the nerve conduction study was read as a motor neuropathy, not a demyelinating neuropathy. Second the electrophysiological report did not show accepted criteria for demyelination. Third sensory responses were preserved in the nerves that were tested. Fourth severe allodynia was a feature.

In addition peripheral nerve ultrasound showed enlargement of nerves. A skin biopsy revealed perineural and periadnexal inflammation and suggested borderline leprosy. The patient also had hepatitis B with a markedly elevated viral load together with a history of chronic steroid exposure/exogenous Cushing syndrome and relapsed Hansen disease.

Therefore the overall clinical picture could not be explained by labeling the patient with CIDP.

The defensible formulation based on the available data is. chronic painful motor predominant polyneuropathy with severe motor axonal abnormalities and peripheral nerve enlargement with histopathological findings that raise the possibility of borderline leprosy. CIDP remains a diagnosis. Is not confirmed by the current electrodiagnostic data.

DIFFERENTIAL DIAGNOSIS

Motor-Predominant CIDP

The chronic progressive course, marked motor dysfunction and evidence of nerve enlargement support considering CIDP. Motor predominant variants of CIDP can present with findings. However the nerve conduction study was read as a motor neuropathy. Full accepted electrodiagnostic criteria for demyelination were not shown in the report. Therefore CIDP cannot be regarded as established based on the evidence.

Multifocal Motor Neuropathy

Multifocal motor neuropathy is a differential because it causes motor weakness while sensory responses are relatively preserved. The patient's motor abnormalities and preserved sensory responses fit this pattern. However severe pain and allodynia are less typical for motor. Also definite conduction block data and anti GM1 antibody results were missing from the records.[8]

Leprosy-Associated Neuropathy

Leprosy associated neuropathy is especially important in this case because many peripheral nerves are enlarged, such as the ulnar, peroneal and greater auricular nerves. A skin biopsy showed perineural and periadnexal inflammation. Suggested borderline leprosy. The clinical history also shows relapsed Hansen disease. However the biopsy was not definitive. The records do not give microbiological evidence to confirm that leprosy caused the neuropathy.[4,6]

Vasculitic Neuropathy

Vasculitic neuropathy can cause pain and mainly axonal abnormalities on nerve conduction studies. The patient's pain motor neuropathy and systemic inflammatory findings make this a relevant possibility. However systemic vasculitis was not confirmed by the investigations.[9]

HBV-Associated Neuropathy or Vasculitis

The patient had hepatitis B with HBsAg and HBeAg and a very high HBV DNA level. HBV-associated immune mediated or vasculitic neuropathy is therefore a differential in a patient with painful axonal neuropathy.[7] However HBV positivity alone does not prove causation.

Motor Neuron Disease

A motor neurological syndrome could suggest motor neuron disease. However the patient's pain, allodynia,

peripheral nerve enlargement and inflammatory skin biopsy findings make this diagnosis less typical. Also the records do not include a motor neuron exam and electromyography to confirm or rule out motor neuron disease.

DISCUSSION

This case shows a challenge in peripheral neuropathy. A chronic progressive motor picture can look like CIDP even though the nerve tests show motor damage. The nerve conduction study is key. The left and right peroneal nerves and the ulnar nerve did not give any CMAPs and the right median CMAP was low. Sensory tests were normal. The formal reading was a motor neuropathy in all four limbs. Missing CMAPs mean motor damage. Do not prove demyelination. Similarly absent F-waves should not automatically be interpreted as proof of CIDP. Need to be assessed in the context of the complete nerve conduction study.

Peripheral nerve ultrasound provided evidence of pathological nerve involvement. Enlargement of the ulnar, common peroneal and greater auricular nerves with fascicular thickening and increased vascularity in selected nerves suggests an inflammatory or infectious peripheral nerve process. However peripheral nerve enlargement is not specific for CIDP.

The distribution of nerve enlargement is particularly relevant in this case. Involvement of the ulnar and greater auricular nerves with the clinical history of Hansen disease and the biopsy findings makes leprosy-associated neuropathy an important alternative diagnosis.

The skin biopsy demonstrated perineural and periadnexal inflammation and the pathologist specifically raised the possibility of borderline leprosy. Importantly this finding should not be converted into a diagnosis without appropriate clinical and microbiological correlation.

The patients active hepatitis B represents another diagnostic consideration. The tests showed that HBsAg and HBeAg were positive and the HBV DNA level was 1.93×10^8 IU/mL. Hepatitis B can be linked to immune-related diseases and a type of nerve problem called vasculitic neuropathy. In a patient presenting with neuropathic pain and axonal nerve injury this possibility deserves consideration.

Nevertheless the presence of HBV infection does not by itself establish a relationship with the neuropathy. The diagnosis would require clinical, laboratory and where indicated pathological correlation.

The history of steroid exposure/exogenous Cushing syndrome adds another layer to the diagnostic complexity. Term corticosteroid exposure can modify inflammatory manifestations and is particularly relevant when an infectious process such as Hansen disease is being considered. The documented history of relapsed Hansen disease makes the combination of infection and chronic exposure clinically important.

Another unusual feature is the discrepancy between the examination and the patients pain. Although bedside sensation was reported as intact minimal touch caused pain. This significant allodynia suggests dysfunction of sensory pain pathways even in the setting of relatively preserved conventional bedside sensory testing.

The overall picture therefore illustrates why CIDP should not be diagnosed just because a person has long-term weakness and strange nerve test results. It is important to check if the nerve problems are from the covering of the nerves from the nerves themselves or a mix of both.

In this patient the current evidence favors a motor-axonal neuropathy while the enlarged nerves, biopsy findings, HBV infection and Hansen disease history provide several competing explanations.

The case also highlights the risk of anchoring bias. Once CIDP is suspected clinicians may interpret all findings as evidence supporting an immune-mediated demyelinating neuropathy. However the presence of potential mimics should prompt systematic reassessment.

CONCLUSION

This case highlights a diagnostic dilemma in a patient with chronic painful motor-predominant polyneuropathy, severe motor axonal abnormalities, and multiple enlarged peripheral nerves. Although CIDP is an important consideration, the available electrodiagnostic data do not establish a generalized demyelinating neuropathy. The combination of nerve enlargement, suggestive skin histopathology, a history of relapsed Hansen disease, and active HBV infection requires consideration of leprosy-associated neuropathy, vasculitic neuropathy, HBV-associated neuropathy, and other motor neuropathies. Careful correlation of clinical findings, electrodiagnostic studies, nerve imaging, histopathology, and microbiological testing is essential before assigning a definitive diagnosis.

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ETHICAL CONSIDERATIONS AND PATIENT CONSENT

Written informed consent for publication of the clinical information and relevant investigation images was obtained from the patient.

The manuscript has been prepared with appropriate protection of patient identity and confidentiality.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest related to this case report.

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