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A CASE REPORT ON DISTAL SYMMETRICAL SENSORY AUTOIMMUNE NEUROPATHY

Periya Karuppan A. R.[1], Visali K.[2], Yazhini S.[3], Keerthiga J.[4], Danush M.[5], Nalinishri R. S.[6], Dr. M. Dheenadayalan*[7]

[1]Pharm.D – Doctor of Pharmacy-Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India. arpr.periyakaruppan@gmail.com
[2]Pharm.D – Doctor of Pharmacy-Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India visalikannan0402@gmail.com
[3]Pharm.D – Doctor of Pharmacy-Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India. yazhiniarulsamy2915@gmail.com
[4]Pharm.D – Doctor of Pharmacy-Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India. keerthigajaisankar@gmail.com
[5]Pharm.D – Doctor of Pharmacy, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India. danushmerick@gmail.com
[6]Pharm.D – Doctor of Pharmacy, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies (VISTAS), Chennai, India. rshrinalini8@gmail.com
[7]Pharm.D., (Ph.D)., Assistant Professor, Department of Pharmacy Practice, School of Pharmaceutical Sciences, VISTAS, Pallavaram, Chennai – 600117. mdheenadayalan.sps@vistas.ac.in,

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***Corresponding author:**

Dr. M. Dheenadayalan

Pharm.D., (Ph.D)., Assistant Professor, Department of Pharmacy Practice, School of Pharmaceutical Sciences, VISTAS, Pallavaram, Chennai – 600117.

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ABSTRACT

Background: The Distal Symmetrical Sensory Autoimmune Neuropathy (DSSAN) is a rare autoimmune disease that affects the peripheral nervous system resulting in distal sensory impairment. This condition is known to cause symptoms like numbness, tingling, pain, sensory ataxia, and instability when walking. Given that DSSAN can be confused with diabetes, nutrition-related, infection-induced, or hereditary neuropathy, diagnosis is difficult. **Case Presentation:** This is the case of a 52-year-old man had a history of gradual numbness, tingling sensations, burning pain, and difficulty balancing while walking for six months. Neurological examination showed symmetrical distal sensory loss with abnormal vibration and proprioception sensation but normal motor sensation. Blood tests ruled out metabolic and infectious causes of neuropathy. Autoimmune profile was found to have a positive anti-nuclear antibody (ANA) and anti-Ro/SSA antibody with raised inflammatory markers. Nerve conduction study was done that showed predominance of sensory neuropathy. The CSF test showed increased protein levels with no pleocytosis. The patient was treated with intravenous methylprednisolone, oral prednisolone, azathioprine, pregabalin, and physiotherapy. **Conclusion:** The patient demonstrated marked improvement clinically after being treated with immunomodulatory and rehabilitative therapy. The current case report emphasizes that autoimmune neuropathy should be considered in patients with progressing symmetrical sensory deficits. Early diagnosis and prompt treatment play vital roles in averting any neurological impairment among such patients.

KEYWORDS: Distal Symmetrical Sensory Autoimmune Neuropathy, Autoimmune Neuropathy, Peripheral Neuropathy, Sensory Ataxia, Nerve Conduction Study, Immunotherapy, Case Report.

INTRODUCTION

Distal Symmetrical Sensory Autoimmune Neuropathy (DSSAN) is an uncommon autoimmune disease whereby the body's immune system malfunctions and begins to attack the sensory nerves within the peripheral nervous system. These nerves play a vital role of conducting various senses like touch, pain, warmth, vibratory, and kinesthetic sensations to the brain. When this happens, the patient starts to experience symptoms like numbness, pins-and-needles sensation, painful burning, and loss of sensation. In most cases, DSSAN affects the distal nerve ends like those located in the hands and feet. This disease develops slowly over time.[1,2]

Peripheral Neuropathy is one of the most frequently encountered neurological diseases all around the world, which can be attributed to a variety of reasons, such as diabetes, vitamin deficiency, infection, toxicity, medication use, genetic disorders, and even autoimmune diseases. Out of these several possible factors, autoimmune neuropathies account for only a small number of cases, yet they are very significant, owing to the fact that they are curable in some instances. The cause of autoimmune neuropathies lies in the attack of the immune system on the peripheral nerves, thereby causing inflammation and damage to the nerves. Sensory-predominant neuropathies target sensory nerves and spares the motor nerves.[3,4]

Distal symmetrical sensory autoimmune neuropathy patients usually complain of progressive numbness, paresthesia, burning pain, electrical shocks, and gait disturbances caused by problems maintaining balance. The symptoms usually develop starting from the toes and feet before moving up the body in a "glove-and-stocking" distribution that usually involves the hands later. The symptoms usually become worse, causing difficulties when walking, particularly in the dark, since the condition affects the person's ability to maintain balance. While muscle strength is not affected in the early stages, the condition will eventually cause disability if left untreated.[5,6]

The diagnosis of distal symmetrical sensory autoimmune neuropathy involves a complete clinical evaluation as well as exclusion of the common causes of neuropathy. The laboratory tests conducted to check for blood sugar, vitamin B12 deficiency, thyroid hormones, autoimmune reactions, and infections are essential. Nerve conduction studies are vital in determining the presence of sensory nerves and the level of damage caused. In addition, the cerebrospinal fluid test helps identify increased levels of proteins associated with inflammation and immune system reactions. Moreover, autoimmune antibodies like antinuclear antibody (ANA) and anti-Ro/SSA antibodies assist in making the correct diagnosis.[7,8]

The timely identification ocoupled with early use of immunomodulatory drugs will be vital to enhancing outcomes in patients with autoimmune sensory neuropathies. Immunosuppression by corticosteroids and other drugs will help prevent any progression of the neuropathy. However, there should be more awareness about this disease since it may not only lead to permanent damage but also cause disability in patients when not diagnosed and treated in time. In this case report, a patient with Distal Symmetrical Sensory Autoimmune Neuropathy was diagnosed using various tests and experienced notable improvement following treatment.[9,10]

CASE PRESENTATION

Patient Demographics

A 52-year-old Indian male presented to the Neurology Outpatient Department of a tertiary care teaching hospital with progressively worsening sensory disturbances involving both lower and upper extremities. The patient weighed 72 kg, had a height of 170 cm, and a body mass index (BMI) of 24.9 kg/m². He was married, living with his family, and was independent in activities of daily living prior to symptom onset. The patient was cooperative, conscious, and oriented to time, place, and person during the clinical evaluation.

Chief Complaints

The patient presented with the following complaints:

- Gradually progressive numbness in both feet for 6 months.
- Tingling sensation ("pins and needles") in both lower limbs for 5 months.
- Burning sensation over the soles of both feet for 4 months.
- Numbness involving fingertips of both hands for 3 months.
- Difficulty maintaining balance while walking, especially in darkness, for 2 months.
- Occasional unsteadiness and sensory gait disturbances for 2 months.

History of Present Illness

The patient was apparently healthy six months before presentation when he developed mild numbness over the toes of both feet. The symptoms progressed gradually in a symmetrical fashion to involve the entire foot and lower legs. Subsequently, tingling sensations and burning pain developed, particularly during nighttime. Three months later, similar sensory symptoms appeared in both hands, primarily affecting the fingertips.

The patient reported increasing difficulty in perceiving uneven surfaces while walking and experienced instability during ambulation in poorly illuminated environments. There was no history of muscle weakness, muscle wasting, diplopia, dysphagia, bowel or bladder dysfunction, seizures, loss of consciousness, or cognitive impairment.

Past Medical History

- Hypertension diagnosed 5 years earlier.
- No history of diabetes mellitus.

- No history of thyroid disorders.
- No history of chronic kidney disease.
- No history of liver disease.
- No history of tuberculosis.
- No history of connective tissue disorders.
- No previous neurological illnesses.
- No prior hospital admissions for similar complaints.

Medication History

At presentation, the patient was receiving:

Medication	Dose	Frequency	Indication
Amlodipine	5 mg	Once daily	Hypertension

Allergy History

- No known drug allergies.
- No known food allergies.

Social History

The patient belonged to a middle socioeconomic class family.

- Non-smoker.
- No history of tobacco chewing.
- No alcohol consumption.
- No recreational drug use.
- Mixed vegetarian and non-vegetarian diet.
- Adequate sleep pattern prior to symptom onset.
- Moderate physical activity level.

Occupational History

The patient was employed as an accountant in a private organization for more than 25 years.

- Sedentary desk-based occupation.
- No exposure to heavy metals.
- No exposure to industrial toxins.
- No exposure to pesticides or organic solvents.
- No occupational radiation exposure.

Family History

- No family history of peripheral neuropathy.
- No family history of hereditary neurological disorders.
- No family history of autoimmune diseases.
- No family history of diabetes mellitus.
- No family history of connective tissue disorders.

General Physical Examination

On examination, the patient was conscious, cooperative, and well-oriented.

General Findings

- Moderately built and nourished.
- No pallor.
- No icterus.
- No cyanosis.
- No clubbing.
- No generalized lymphadenopathy.
- No pedal edema.

Vital Signs

Parameter	Observation
Temperature	98.4°F
Pulse Rate	78 beats/min
Respiratory Rate	18 breaths/min
Blood Pressure	128/82 mmHg
Oxygen Saturation (SpO_2)	98% on room air
Height	170 cm
Weight	72 kg
BMI	24.9 kg/m²

Neurological Examination

Mental Status Examination

- Conscious and alert.
- Fully oriented to time, place, and person.
- Normal memory functions.
- Intact language and speech.
- Normal attention and concentration.
- No cognitive impairment.

Cranial Nerve Examination

All cranial nerves (I–XII) were intact.

- Pupils equal and reactive to light.
- Extraocular movements normal.
- Facial sensation preserved.
- Facial muscle strength normal.
- Hearing intact.
- Palatal movements normal.
- Tongue movements normal.

Motor System Examination

Parameter	Finding
Muscle Bulk	Normal
Muscle Tone	Normal
Muscle Strength	5/5 in all limbs
Fasciculations	Absent
Muscle Wasting	Absent

No evidence of motor neuropathy was observed.

Sensory System Examination

Superficial Sensations

Sensory Modality	Findings
Pain	Reduced distally in both feet and hands
Temperature	Reduced distally
Light Touch	Mildly impaired distally

Deep Sensations

Sensory Modality	Findings
Vibration Sense	Markedly impaired at toes and ankles
Joint Position Sense	Impaired in toes
Proprioception	Reduced bilaterally

A symmetrical glove-and-stocking pattern of sensory loss was noted.

Reflex Examination

Reflex	Right	Left
Biceps	+2	+2
Triceps	+2	+2
Knee Jerk	+2	+2
Ankle Jerk	+1	+1
Plantar Response	Flexor	Flexor

Coordination Examination

- Finger-nose test: Normal.
- Heel-knee-shin test: Mild impairment due to sensory deficits.
- Romberg test: Positive.
- Tandem walking: Impaired.

Gait Examination

- Broad-based sensory ataxic gait.
- Increased instability with eyes closed.
- Difficulty walking on uneven surfaces.

Systemic Examination

Cardiovascular System

- S1 and S2 heard normally.
- No murmurs.
- No added heart sounds.

Respiratory System

- Bilateral air entry equal.
- Normal vesicular breath sounds.
- No adventitious sounds.

Gastrointestinal System

- Abdomen soft and non-tender.
- No organomegaly.
- Bowel sounds normal.

Genitourinary System

- No abnormalities detected.
- Normal bladder and bowel control.

Laboratory Investigations

Hematological Parameters

Parameter	Result	Reference Range
Hemoglobin	13.8 g/dL	13–17 g/dL
Total WBC Count	7,800 cells/mm³	4,000–11,000
Platelet Count	2.6 lakh/mm³	1.5–4.5 lakh
ESR	42 mm/hr	<20 mm/hr

Biochemical Parameters

Parameter	Result
Fasting Blood Sugar	92 mg/dL
HbA1c	5.4%
Serum Creatinine	0.9 mg/dL
Blood Urea	26 mg/dL
AST	24 U/L
ALT	22 U/L
CRP	12 mg/L

Nutritional Assessment

Parameter	Result
Vitamin B12	520 pg/mL
Folate	Normal

Autoimmune Profile

Investigation	Result
ANA	Positive (1:320 Speckled)
Anti-Ro/SSA Antibody	Positive
Anti-dsDNA	Negative
Rheumatoid Factor	Negative
ANCA	Negative

Infectious Disease Screening

Test	Result
HIV I & II	Negative
HBsAg	Negative
Anti-HCV	Negative
VDRL	Non-reactive

Nerve Conduction Study (NCS)

Sensory Nerve Conduction Findings

Nerve	SNAP Amplitude	Conduction Velocity
Sural Nerve	Reduced	Mildly Reduced
Superficial Peroneal Nerve	Reduced	Reduced
Median Sensory Nerve	Reduced	Mildly Reduced
Ulnar Sensory Nerve	Reduced	Mildly Reduced

Motor Nerve Conduction Findings

Nerve	Distal Latency	CMAP Amplitude	Conduction Velocity
Median Motor	Normal	Normal	Normal
Ulnar Motor	Normal	Normal	Normal
Tibial Motor	Normal	Normal	Normal
Peroneal Motor	Normal	Normal	Normal

Electrophysiological Interpretation

The study demonstrated:

- Predominantly sensory axonal neuropathy.
- Symmetrical distal sensory nerve involvement.
- Preservation of motor nerve function.

- Findings consistent with distal symmetrical sensory autoimmune neuropathy.

Cerebrospinal Fluid (CSF) Analysis

Lumbar puncture was performed under aseptic precautions.

Parameter	Result
Appearance	Clear and Colorless
Opening Pressure	Normal
Total Cell Count	2 cells/mm³
Differential Count	Lymphocyte Predominance
Protein	68 mg/dL
Glucose	68 mg/dL
Gram Stain	Negative
Bacterial Culture	No Growth

CSF Interpretation

The cerebrospinal fluid showed:

- Mildly elevated protein concentration.
- Normal glucose level.
- Absence of pleocytosis.

These findings indicate albuminocytologic dissociation, supporting an immune-mediated peripheral neuropathic process.

DIAGNOSIS

The patient was evaluated for progressive, symmetrical sensory symptoms, including numbness, tingling, burning sensations, impaired vibration sense, reduced proprioception, and sensory ataxia involving both upper and lower extremities. Neurological examination revealed a characteristic glove-and-stocking pattern of sensory loss with preserved motor strength and no evidence of muscle weakness. Nerve conduction studies demonstrated reduced sensory nerve action potentials and abnormal sensory conduction with normal motor nerve parameters, confirming a sensory-predominant peripheral neuropathy. Autoimmune investigations showed positive antinuclear antibody (ANA) and anti-Ro/SSA antibody positivity, along with elevated ESR and CRP, suggesting an underlying immune-mediated inflammatory process. Cerebrospinal fluid analysis revealed elevated protein levels with a normal cell count, supporting an inflammatory neuropathy. Common causes such as diabetes mellitus, vitamin B12 deficiency, thyroid dysfunction, infections, toxic exposures, hereditary neuropathies, and central nervous system disorders were excluded through appropriate investigations. Based on the clinical presentation, electrophysiological findings, autoimmune markers, and exclusion of alternative etiologies, the final diagnosis was Distal Symmetrical Sensory Autoimmune Neuropathy (DSSAN).

TREATMENT

Following confirmation of Distal Symmetrical Sensory Autoimmune Neuropathy (DSSAN), the patient was initiated on immunomodulatory therapy aimed at reducing autoimmune-mediated inflammation and preventing further nerve damage. Intravenous methylprednisolone was administered at a dose of 1 g daily for five consecutive days as induction therapy. The patient tolerated the treatment well without significant adverse effects. Following completion of pulse therapy, oral prednisolone was initiated at 1 mg/kg/day and gradually tapered over subsequent weeks based on clinical response and inflammatory marker monitoring. Regular follow-up assessments were performed to evaluate symptom progression and treatment efficacy.

To achieve long-term disease control and minimize corticosteroid-related adverse effects, azathioprine was introduced as a steroid-sparing immunosuppressive agent at a dose of 50 mg twice daily. Baseline and periodic monitoring of complete blood count and liver function tests were conducted throughout treatment. The addition of azathioprine was intended to suppress ongoing autoimmune activity, reduce the risk of disease progression, and maintain sustained remission. The patient demonstrated good tolerance to immunosuppressive therapy, with no evidence of hematological or hepatic complications during follow-up.

Symptomatic management was provided to address neuropathic pain and improve the patient's quality of life. Pregabalin 75 mg twice daily was prescribed for the management of burning sensations, tingling, and neuropathic discomfort affecting the distal extremities. The patient reported gradual reduction in pain intensity and improvement in sleep quality following initiation of therapy. Supportive treatment also included vitamin supplementation, adequate nutritional counseling, and patient education regarding the chronic nature of the disease and the importance of medication adherence.

A structured physiotherapy and rehabilitation program was incorporated into the treatment plan to improve balance, coordination, and functional mobility. Balance training exercises, gait rehabilitation, proprioceptive exercises, and lower-limb strengthening activities were performed under supervision. The patient was also advised regarding fall prevention strategies and home safety modifications. During follow-up visits, significant improvement was observed in sensory symptoms, gait

stability, and overall functional status, indicating a favorable response to the combined immunosuppressive, symptomatic, and rehabilitative treatment approach.

S. No.	Drug Name	Dose	Frequency	Route of Administration
1	Methylprednisolone	1 g	Once daily for 5 days	Intravenous (IV) infusion
2	Prednisolone	1 mg/kg/day (approximately 60 mg/day)	Once daily, followed by gradual tapering	Oral
3	Azathioprine	50 mg	Twice daily	Oral
4	Pregabalin	75 mg	Twice daily	Oral
5	Methylcobalamin	1500 mcg	Once daily	Oral
6	Calcium Carbonate + Vitamin D3	500 mg + 250 IU	Twice daily	Oral
7	Pantoprazole	40 mg	Once daily before breakfast	Oral
8	Amlodipine	5 mg	Once daily	Oral

Follow-Up and Outcome

During regular follow-up visits, the patient demonstrated gradual and sustained clinical improvement. At one month, there was a noticeable reduction in burning sensations, tingling, and neuropathic pain, with improved sleep quality and daily functioning. By three months, sensory symptoms had further decreased, and balance during walking improved significantly. Neurological examination revealed partial recovery of vibration and proprioceptive sensations, while motor strength remained preserved. At six months, the patient reported marked improvement in gait stability, sensory function, and overall quality of life. No relapse, disease progression, or significant treatment-related adverse effects were observed, indicating a favorable therapeutic response and successful disease control.

DISCUSSION

Distal Symmetrical Sensory Autoimmune Neuropathy is an uncommon immune-mediated disorder characterized predominantly by sensory dysfunction with minimal or no motor involvement. The present patient developed progressive numbness, paresthesia, burning sensations, impaired proprioception, and gait instability, which are classical manifestations of sensory autoimmune neuropathy. Similar findings were reported by Sinnreich et al., who described patients presenting with sensory ataxia, impaired balance, and preserved muscle strength due to immune-mediated sensory nerve involvement. Likewise, Oh et al. reported pure sensory chronic inflammatory demyelinating neuropathy characterized by progressive sensory deficits without significant motor weakness. The clinical presentation observed in our patient closely resembles these reports and emphasizes the importance of considering autoimmune etiologies in patients presenting with progressive distal symmetrical sensory symptoms after exclusion of common metabolic and nutritional causes.[11,12]

Neurological examination and nerve conduction studies played a crucial role in confirming the diagnosis in this patient. Electrophysiological testing demonstrated reduced sensory nerve action potentials with preserved motor nerve conduction, indicating sensory-predominant peripheral nerve involvement. Similar findings were described by Shahar Shelly et al who reported sensory dysfunction, gait ataxia, and mild electrophysiological abnormalities in patients with CISP-Plus. Furthermore, Notermans NC et al emphasized that sensory neuropathies frequently demonstrate characteristic sensory conduction abnormalities despite preserved motor function. The electrophysiological findings in our patient correlate strongly with these previous observations and highlight the importance of nerve conduction studies in differentiating autoimmune sensory neuropathies from other neurological disorders.[13,14]

The autoimmune basis of the disease was supported by positive ANA and anti-Ro/SSA antibodies, elevated inflammatory markers, and increased cerebrospinal fluid protein levels. These findings are consistent with previously reported autoimmune sensory neuropathies. Mori et al. demonstrated that patients with autoimmune neuropathies frequently exhibit positive autoimmune serology and inflammatory neurological manifestations. Similarly, Camdessanché et al. reported that sensory neuronopathies often show autoimmune antibody positivity and evidence of immune-mediated neuronal injury. The laboratory abnormalities observed in our patient strongly support an inflammatory autoimmune mechanism affecting peripheral sensory nerves. Identification of autoimmune markers is clinically valuable because it assists in differentiating autoimmune neuropathies from hereditary, toxic, infectious, and metabolic neuropathies, thereby facilitating appropriate immunomodulatory treatment strategies.[15,16]

The patient showed substantial improvement following treatment with corticosteroids, azathioprine, symptomatic therapy, and physiotherapy. Reduction in paresthesia, improvement in gait stability, and recovery of sensory function indicated a favorable response to

immunomodulatory therapy. Similar therapeutic outcomes were reported by Koh JS et al who demonstrated significant neurological recovery following early immunotherapy in chronic immune sensory polyradiculopathy. Likewise, Gorson KC et al observed favorable responses to corticosteroids and immunosuppressive agents in chronic immune-mediated neuropathies. The positive outcome observed in our patient supports previous evidence that early diagnosis and timely initiation of immunotherapy can prevent disease progression, improve neurological recovery, and enhance overall quality of life. Early recognition remains essential to reduce long-term disability associated with autoimmune sensory neuropathies.[17,18]

CONCLUSION

Distal Symmetrical Sensory Autoimmune Neuropathy is a very rare, yet potentially treatable, autoimmune disorder of the nervous system which predominantly affects the peripheral sensory nerves and causes substantial impairment to balance and sensation. In this particular case, Distal Symmetrical Sensory Autoimmune Neuropathy had to be considered following exclusion of the usual differential diagnoses, such as diabetes mellitus, nutritional deficiencies, various infections, and toxic factors, in order to achieve the correct diagnosis. It was essential for proper treatment to carry out a number of different tests for the thorough examination of this rare illness. Immunomodulatory treatment started early on after the diagnosis was made, and the patient had considerable improvement in his state and regained his functions. Therefore, this case study stresses the significance of the need to diagnose and start the appropriate treatment of Distal Symmetrical Sensory Autoimmune Neuropathy in a timely manner, thus avoiding possible progression and complications.

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