Neurophysiologic Characterization and Long-Term Analgesic Strategies in Painful Sensory Neuronopathy: A Case Report

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Introduction

Sensory neuronopathies (SN) are a rare subgroup of peripheral nervous disorders that are characterized by damage to the dorsal root ganglion [1]. This condition can typically occur in a subacute to chronic time course [2]. Sensory neuronopathies tend to result from various causes, including autoimmune, infectious, drug-induced, paraneoplastic and hereditary factors [3]. Rarely, SN can be idiopathic in nature [4]. Sensory neuronopathies are typically defined by a non-length dependent pattern of degeneration associated with decreased or absent sensory nerve action potentials (SNAPs) in neurophysiologic studies [1][5].

Case

This patient initially developed numbness in his bilateral feet in 2019. This evolved into diffuse non-length dependent sensory deficits and dysesthesias that slowly spread throughout his entire body over a span of a few years. He described his pain as feeling like “electric shocks”. He also felt like his body was “pressing against glass shards”. At its usual peak, his pain severity was an 8/10 but would intermittently decrease to a 4/10. EMG/NCV studies demonstrated absent sensory responses in the left sural nerve, left superficial peroneal nerve and left ulnar nerve (Figure 2). These studies also showed a reduction in amplitude in sensory response in the left radial nerve and left median nerve (Figure 2). The patient also had an autonomic reflex screen completed which showed no abnormalities. Despite extensive diagnostic testing, we were unable to determine the etiology of his newly diagnosed sensory neuronopathy. For neuropathic pain control, the patient was initially treated with pregabalin and amitriptyline. These medications were self-discontinued by the patient due to socioeconomic barriers and lack of efficacy. He also tried over the counter analgesics, including ibuprofen and acetaminophen, without meaningful pain control. The patient reported that recreational daily cannabis usage (inhaled and edible formulations) provided the most effective symptomatic pain control. The patient was then evaluated in a pain clinic and offered neuromodulation with a spinal cord stimulator but he decided not to pursue this intervention.

Results

MNC

Nerve / Sites	Muscle	Latency ms	Amplitude mV	Distance mm	Velocity m/s
L Peroneal - EDB					
Ankle	EDB	4.3	1.3	70	
FibH	EDB	15.2	1.2	400	36.7
Knee	EDB	17.9	1.2	100	37.6
L Tibial - AH					
Ankle	AH	4.4	4.5	90	
Knee	AH	16.4	2.1	500	41.9
L Peroneal - Tib Ant					
FibH	Tib Ant	3.1	5.2	100	
Knee	Tib Ant	4.6	5.1	90	59.6
L Median - APB					
Wrist	APB	3.3	6.0	70	
Elbow	APB	8.9	5.7	280	50.2
L Ulnar - ADM					
Wrist	ADM	2.8	8.2	70	
B.EL	ADM	8.1	7.0	250	47.5
A.EL	ADM	10.6	6.7	130	52.0

Figure 1. Motor Nerve Conduction Studies

F Wave

Nerve	F Lat ms
L Peroneal - EDB	67.8
L Tibial - AH	65.4
L Median - APB	35.5
L Ulnar - ADM	35.5

SNC

Nerve / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	Amp µV	Segments	Distance mm	Vel. m/s
L Sural - Ankle (Calf)							
1	Ankle	NR	NR	NR	1 - Ankle	120	NR
L Superficial peroneal - Ankle							
Calf	Ankle	NR	NR	NR	Calf - Ankle	100	NR
L Median - Digit II (Antidromic)							
Wrist	Dig II	2.9	3.6	2	Wrist - Dig II	130	45
L Ulnar - Digit V (Antidromic)							
Wrist	Dig V	NR	NR	NR	Wrist - Dig V	120	NR
L Radial - Anatomical snuff box (Forearm)							
Forearm	Wrist	1.7	2.4	4	Forearm - Wrist	100	58

Figure 2. Sensory Nerve Conduction Studies and F wave

EMG

EMG Summary Table									
	Spontaneous			MUAP					Comment
Muscle	IA	Fib	Fasc	Dur	Amp	% Polys	Recruit	Activation	.
L. Tibialis anterior	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. Gastrocnemius (Medial head)	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. Extensor hallucis longus	Normal	None	None	+1	+1	+1	Sl Decr	Normal	.
L. Vastus medialis	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. Rectus femoris	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. Tensor fasciae latae	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. First dorsal interosseous	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.
L. Triceps brachii	Normal	None	None	Nl	Nl	Normal	Normal	Normal	.

Figure 3. EMG Summary Table

Discussion

- Early recognition of pathology and a multimodal pain management strategy is critical for adequate treatment of neuropathic pain in this disease process. A combination of centrally acting pain medications, topical analgesic ointments, non-pharmacological interventions and proper treatment of any psychiatric co-morbidities are all likely to play a role in proper pain management.
- While neuromodulation has not been systematically studied in patients with idiopathic SN, this case suggests that neuromodulation with a spinal cord stimulator may be an option for symptomatic pain relief in carefully selected refractory cases.
- This patient reported that he dealt with years of undertreatment of his pain symptoms due to a lack of understanding about idiopathic sensory neuronopathy among healthcare providers. He also had a history of bipolar disorder, major depressive disorder and anxiety which were likely aggravating factors for his pain and served as barriers to adequate pain treatment.
- Future studies will be necessary to appropriately characterize pain symptomatology in patients with idiopathic sensory neuronopathy to avoid undertreatment of pain. Additional research will also be needed to evaluate the efficacy and safety of neuromodulatory interventions such as spinal cord stimulation and dorsal root ganglion stimulation in patients with SN.

References

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