FEATURE ARTICLE

The Lymphatic Theory of Notalgia Paresthetica

CE 1.5 Contact Hours

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ABSTRACT: Notalgia paresthetica is a dermatologic condition with prominent primarily left unilateral pruritis and raised erythematous rash with hyperpigmentation at the medial or inferior scapula. The etiology is unknown. A comprehensive review of the literature was performed with a structured analysis of previous theories. There is no consistent imaging or functional test to support any of the previously proposed mechanisms. A new theory is presented with a unifying theme of all previous treatments and is supported with results of the first electrical impedance myography testing in this condition.

Key words: Notalgia Paresthetica, Pruritis, Electrical Impedance Myography, Lymphatic Theory

Notalgia paresthetica (NP) is a dermatologic condition with prominent primarily left unilateral pruritis and raised erythematous rash with hyperpigmentation at the medial or inferior scapula. There are associated less severe symptoms of pain, paresthesia, numbness, or hyperesthesia. Despite an initial description in 1934, the etiology is still unknown and there is no standard treatment (Astawzaturow, 1934; Ellis, 2013). The general consensus at this time is that NP is a sensory neuropathy with competing camps of thoracic or cervical origin and each of these with possible central or peripheral neural compression. A fifth possible etiology is presented here for the first time as primarily a lymphatic source.

CURRENT THEORIES

Most of the evidence that has been presented advocates for a thoracic etiology, but this does not account for all of the clinical features. The distribution is adjacent to the T2–T6 vertebrae, and degenerative changes of thoracic spondylosis have been found in 60% and 75% of patients, which led to the conjecture that spinal nerve impingement may contribute to the pathogenesis of NP (Raison-Peyron, Meunier, Acevedo, & Meynadier, 1999; Savk & Savk, 2005). This is based on the reasoning that there is nerve compression from the spondylosis as the nerve roots exit the vertebral column at each level through the neural foramen. However, this seems unreasonable because not all patients have thoracic spondylosis; in those who do, the spondylosis is not limited to only the T2–T6 locations, and most importantly, the location of NP is only medial, and not a dermatomal pattern, as would be expected from a neural foraminal impingement. Importantly, electrodiagnostic studies have not confirmed this. However, there was a single report in the 1980s that identified electromyographic paraspinal denervation at T2–T6 (Massey & Pleet, 1984). This was never repeated until 2016 where electrodiagnostic findings did not substantiate this theory and instead revealed that some patients had denervation of paraspinal muscles with positive waves and other patients had normal findings (Terzi & Bekdik-Sirinocak, 2016). There is no consistent imaging or electrodiagnostic evidence for a thoracic etiology of nerve impingement. In addition, a thoracic etiology of nerve impingement can be anatomically excluded based on the isolated medial distribution of NP, which is not dermatomal.

A similar theory suggests a peripheral thoracic compression etiology involving only the dorsal rami of the T2–T6 locations. This theory attempts to explain the lack of a full dermatomal pattern with the limited unilateral medial location with nerve compression in adjacent musculature. It has been suggested in the 1970s that there is compression of the posterior divisions of only these five dorsal roots based on a unique anatomic variant where only these levels of the posterior rami traverse a 90° course through the multifidus muscle with paraspinal musculature neural compression (Pleet & Massey, 1978). However, this anatomical variant of the course of the thoracic dorsal rami limited to only the T2–T6 levels was not referenced in this article of six clinical

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cases, and it has not been reported by any other independent source. More importantly, it contradicts three descriptions of Gray's anatomy of the corresponding distributions of the thoracic dorsal rami (Gray, 1918, 2016b; Mancall & Brock, 2011). The multifidus spinae muscle consists of a number of fleshy and tendinous fasciculi, which fill up the groove on either side of the spinous processes of the vertebrae, from the sacrum to the second cervical vertebrae, and there is nothing known to be unique to the T2–T6 levels. The dorsal rami divide into medial, intermediate, and lateral branches, and these distributions of the T2–T6 levels have far more lateral locations, which are not consistent with the distribution observed in NP. The medial branch includes the skin overlying the tip of the spinous process and is too medial for NP. The lateral branch innervates the longissimus thoracis and iliocostalis muscles and then proceeds as the lateral cutaneous branch for sensory innervation lateral to these muscles, which is too lateral for NP (Ishizuka, Sakai, Tsuzuke, & Nagashima, 1976). These facts anatomically exclude myofascial compression of the thoracic T2–T6 dorsal rami as the etiology of NP.

Alternative explanations to these two thoracic etiologies for NP are that it is primarily a cervical neurologic source, also either central or peripheral. Evidence to support the central cervical theory includes two individual case reports with associated cervical disc disease (Alai et al., 2010). There is also a report of benefit with cervical traction in NP (Low, Swanson, & Swanson, 2017). This theory is based on the correlation with the cutaneous sensory location of the dorsal scapular nerve. This nerve arises from C5 in 70%, C4 in 22%, and C6 in 8% of individuals, where it innervates only the levator scapulae muscle in 48% of individuals and innervates the levator scapulae, rhomboid major, and rhomboid minor muscles in 52% of individuals (Nguyen, Liu, Rosales, & Reeves, 2016). However, in the case report of these two individuals, there were no other locations of pain consistent with neuroforaminal stenosis of any of the C4, C5, or C6 nerve roots. For this reason, a central cervical etiology can also be anatomically excluded.

A final theory to account for the location of pain as only medial scapula without arm involvement is that the dorsal scapular nerve experiences extraforaminal peripheral impingement. This condition has been described as dorsal scapular neuropathy, and a possible direct link of this condition with NP has been suggested. The cited weaknesses of understanding and inconsistencies with testing and treatment outcomes have been reported to "require further study" (Muir, 2017). Clinically, the dorsal scapular nerve is an unlikely etiology based on the prominence of pain in dorsal scapular neuropathy in contrast to mainly pruritis in NP (Sultan & Younis El-Tantawi, 2013). In addition, there is no electrodiagnostic confirmation in any reported case.

Both cervical and thoracic central neural compression theories fail based on the lack of a complete dermatomal pattern and are inconsistent with the isolated medial location of NP. The incidence of underlying imaging abnormalities in asymptomatic individuals suggests this is an association and not a source (Shumway, Cole, & Fernandez, 2016). In addition, anatomic correlation with neural foraminal stenosis and/or spondylosis is not universally present in all cases. The peripheral compression theories for both cervical and thoracic sources also fail based on the above reasoning and also because of the inability to account for the prominence of pruritis compared with pain. Meralgia paresthetica with compression of the lateral femoral cutaneous nerve is a typical example of a peripheral neural compression. In these conditions, tingling, numbness, and burning pain are the prominent symptoms, and pruritis is not associated.

The most striking feature of these four lines of reasoning of central or peripheral, cervical, or thoracic etiologies is that none of them adequately explains the prominence of pruritis as the overwhelming clinical feature. Pain is a minor symptom that is observed in less than 30% of patients with NP (Lal, Kazlouskaya, Elston, & Goldstein, 2014). Although neurogenic sources of pruritis are known, in these conditions, pain is the most severe symptom and pruritis is a minor and secondary clinical concern.

The low numbers of all of these reports of NP preclude meaningful conclusions, and taken together, there is no consistent imaging or functional test for any of these proposed mechanisms. All of them fail based on either the clinical symptoms or the location of NP. All of these theories are based on the premise that the main symptom of NP is neuropathic itch, where neural damage can be caused by compression or degeneration of the nerve fibers in the extracutaneous peripheral nerves or in the central nervous system (Stumpf & Stander, 2013). However, this is an elusive condition because the cellular and molecular mechanisms of neuropathic itch are still unknown. All neurological disease categories have been implicated, and the same neurological illnesses that cause neuropathic pain can also or instead cause itch (Oaklander, 2011).

It seems likely that none of these theories of cervical or thoracic spinal with central or peripheral neural compression is correct. It would be reasonable to consider another source and develop a hypothesis for the etiology of NP around the prominent symptom of pruritis with anatomic awareness of symptom distribution.

MECHANISM OF PRURITIS

The basic mechanisms mediating itch have been extensively studied and involve both Aδ and C fibers. The complexity of stimuli and central processing have been summarized to include "a myriad of mediators capable of stimulating these afferent nerves leading to itch, including biogenic amines, proteases, cytokines, and peptides. Some of these mediators can also evoke sensations of pain and the sensory processing underlying both sensations overlaps in complex ways" (Potenzieri & Undem, 2012). The neurologic anatomy and exacerbating factors have been described as the itch impulse being transmitted from the peripheral nerves to

the dorsal horn of the spinal cord, across the cord via the anterior commissure, and ascending along the spinothalamic tract to the laminar nuclei of the contralateral thalamus. Thalamocortical tracts of tertiary neurons are believed to relay the impulse through the integrating reticular activating system of the thalamus to several areas of the cerebral cortex. Factors that are believed to enhance the sensation of pruritus include dryness of the epidermis and dermis, anoxia of tissues, dilation of the capillaries, irritating stimuli, and psychological responses (Yonova, 2007).

LYMPHATIC THEORY

This mechanism involving pruritis mediated through C-fibers and Aδ nerve fibers is well understood and contrasts with the mechanisms of neuropathic itch, which originates centrally without evidence of neuronal pathology (Tivoli & Rubenstein, 2009). Exploring the C-fiber source further, it is noted that lymphatics is a well-known source of pruritis. This is because the reduction of lymphatic drainage causes a buildup of inflammatory mediators in the skin. The accumulation of immune proteins and cytokines, along with venous and lymphatic edema, results in barrier dysfunction, and improper barrier function eventually leads to dermatitis, which is a cause of an intense pruritic response. This response involves several communication cascades between well-known biochemical mediators and the peripheral nervous system (McCord & Fore, 2007). A lymphatic source would therefore account for the symptoms of NP. The location of NP has been problematic to explain based on the anatomy of the cervical and thoracic spinal nerve roots and the dorsal rami. Essentially, there is no precise correlation with any of these. Further insight into the possible etiology of NP is obtained by combining this theory that there is a lymphatic source for the pruritis with the very localized, specific, and consistent location of the symptoms. NP is a unilateral condition with extreme left-sided prominence. This aspect of a predominantly left-sided location has not been emphasized and may provide clues to the source. It has been repeatedly noted that the most common location of skin manifestations in patients with NP is at the medial border of the left scapula with a left-sided incidence as high as 79% (Chiriac, Podoleanu, Moldovan, & Stolnicu, 2016; Howard, Sahhar, Andrews, Bergman, & Gin, 2017; Pagliarello et al., 2017; Richardson, Say, & Speece, 2009; Robbins & Ferrer-Bruker, 2017).

There is only one structure that is located along the superior left posterior thoracic wall, and that is the thoracic duct. A detailed analysis of the course of the thoracic duct reveals a convincing correlation with the typical location for NP, which combined with the association of lymph as the source of the symptom of intense pruritis, provides a basis for a new theory for the etiology of NP.

The classical description of the course of the thoracic duct is that it "starts from the cisterna chyli at the level of the second or third lumbar vertebra. It then enters the thorax through the aortic hiatus of the diaphragm and ascends in the posterior mediastinum, between the descending thoracic aorta on the left and the azygos vein on the right. When it reaches the level of the fifth thoracic vertebral body, it gradually inclines to the left side and enters the superior mediastinum. It first crosses anteriorly by the aortic arch, and it runs posterior to the left subclavian artery, and forms an arch. Finally, the duct terminates by opening into the junction of the left subclavian and jugular veins" (Kiyonaga et al., 2012).

Computed tomographic scanning of the thoracic duct in sequential cross-sectional planes gives further insight pertinent to this proposed mechanism for NP. It is divided into the abdominal, thoracic, and cervical portions. As it enters the thorax from the abdomen, it is slightly right and gradually traverses left over the vertebral body at T5. It then enters into the superior mediastinum, which is defined inferiorly by the thoracic plane at the inferior border of T4 and superiorly by the thoracic inlet at T1. In the anteroposterior (AP) view with lymphangiography, the thoracic duct is anterior to the vertebra until T5 when is crosses to the left. At this point, it attains its most posterior aspect of the thorax at the level of the aortic arch at T3–T4 and then proceeds anteriorly while traversing the superior mediastinum. At the level of the thoracic inlet and in the cervical portions, it is situated more anteriorly and is located in the middle of the AP dimension at the level of T1 (Adler & Rosenberger, 1981). This left-sided and posterior location matches well with the location of NP.

Further supporting evidence for this etiology is that the percentage of patients with left- versus right-sided NP compares favorably with the known percentages for the left versus right location of the thoracic duct. Although the thoracic duct is most commonly found on the left, it is located on the right in 5%–20% of cases (Kim et al., 2016; Kinnaert, 1973).

The thoracic duct drains all of the body and limbs below the respiratory diaphragm and receives the efferents from the posterior mediastinal lymph nodes and the posterior intercostal lymph nodes of the upper six left levels of the thoracic vertebral column (Gray, 2016a). Lymph flow is a complex process that is dependent on pressure gradients generated by contractile elements in the lymphatics, the intrathoracic pressure, and the venous backpressure in the subclavian vein (Mallick & Bodenham, 2003). Disorders of lymph flow create stagnation, and when these vessels become engorged, lymph can accumulate in the vertebral body and appear on magnetic resonance imaging (MRI) like a hemangioma (Méndez, Hochmuth, Boetefuer, & Schumacher, 2002). This congestion of lymph is a source of intense pruritis, as is observed in NP.

This theory of a lymphatic source for NP is dependent on the existence of unmyelinated C-fibers and thinly myelinated Aδ nerve fibers within the mediastinum because these are the nerve fibers that mediate pruritic symptoms. These have been discovered and extensively studied as the cardiopulmonary C-fibers (Wilson & Bonham, 1997). These

cardiopulmonary C-fiber afferents have been determined to be subsets of the vagal nerve (Kollarik, Ru, & Brozmanova, 2010; Lin, Gu, & Lee, 2017). However, these unmyelinated fibers within the mediastinum have also been discovered to be associated with the phrenic nerve (Brown, Perry, Hunt, & Lapper, 1994; Fazan, Rodrigues Filho, Jordão, & Moore, 2009). In addition, these afferent Aδ and C fibers are not isolated to visceral membranes, as phrenic nerve sensory afferents have also been discovered to innervate the parietal pleura of the thoracic cavity (Jammes, Trousse, & Delpierre, 2005). The existence of these nerve fibers within the mediastinum provides anatomical support for a lymphatic source of NP.

PREVIOUS TREATMENTS

The success of previous treatments can now be discussed in terms of this proposed lymphatic source of NP. Many of these have been topical agents or medications with neurologic targets, which can be understood based on the cellular pathology at the site. Biopsies have identified substantially increased epidermal dendritic cells immunoreactive for S-100, which possibly are Langerhans cells, leading to a conclusion that there is an increase in the sensory epidermal innervation in the affected skin areas in NP (Springall, Karanth, Kirkham, Darley, & Polak, 1991). Later, an increase in the number of dermal nerves was identified (Inaloz, Kirtak, Erguven, Karakok, & Inaloz, 2002). It is now known that these changes as well as postinflammatory hyperpigmentation, mild hyperkeratosis, and dermal melanophages are a result of chronic and aggressive scratching (Savk, Dikicioglus, Culhaci, Karaman, & Sendur, 2002). NP has a dermatologic presentation, but all of these changes are a result of scratching from intense pruritis, and there is no primary dermatologic pathology.

Several treatments for the symptoms have been attempted while the search for the source has progressed. Local and topical treatments including topical anesthetics, topical capsaicin, intralesional corticosteroid, and botulinum toxins are preferred local treatment applications. All of these have independently been shown to be effective for pruritis in a variety of conditions (Elmariah & Lerner, 2011; Gazerani, Pedersen, Drewes, & Arendt-Nielsen, 2009; Hong, Buddenkotte, Berger, & Steinhoff, 2011; Lysy et al., 2003). A final local treatment that has been attempted is paravertebral local anesthetic blocks, which are largely ineffective (Pérez-Pérez, 2011). Of these treatments, the most effective has been local or systemic administration of inhibitors of neuronal excitability, especially local anesthetics (Oaklander, 2011). However, the mechanism of action of these treatments does not provide insight into the etiology of NP because they all affect the afferent neurologic pathways and receptors treating the symptom of pruritis, but not the source of the pathology.

Systemic treatments have also been used in NP. Oxcarbazepine was helpful in a few cases, and this would be consistent with the known effects of oxcarbazepine mediated through C-fibers (Brouwer et al., 2015; Savk, Bolukbasi, Akyol, & Karaman, 2001). Gabapentin has also been used with success, and the effect has been determined to be depressed C-fiber-evoked field potentials (Maciel, Cunha, Laraia, & Trevisan, 2014; Tanabe, Murakami, Honda, & Ono, 2006). Amitriptyline has also been shown to suppress the response of human peripheral C-type axons (Freysoldt et al., 2009). These treatments that have effects mediated through C-fibers are expected based on the known neurologic afferents operating with pruritis from multiple sources. Of these, amitriptyline is interesting because it also has effects on lymphatic vessels mediated through histamine (Bernstein, Whitney, & Soltani, 1981; Scallan & Davis, 2014).

Despite the anatomical contradiction of a muscular etiology with peripheral neural compression in NP, there have been a variety of muscular treatments attempted. These include the response of a single patient with osteopathic manipulative treatment with soft tissue techniques and fascial release (Richardson et al., 2009), two cases in which the condition was improved by exercises involving nonpharmacologic active range of motion and strengthening of the scapular muscles as well as stretching of the pectoral muscles (Fleischer & Meade, 2011), five cases of successful treatment with narrow-band UVB (Pérez-Pérez, Allegue, Pabeiro, Caeiro, & Zulaica, 2010), and the use of transcutaneous electrical nerve stimulation to reduce the symptoms of 15 adults with NP (Savk, Savk, & Sendur, 2007). A final muscular source that has been reported is electrical muscle stimulation of the serratus anterior muscle to treat suspected long thoracic nerve injury, leading to serratus anterior dysfunction as the source of NP (Wang, Gowda, Barad, Mackey, & Carroll, 2009). It is possible, if not likely, that the success of these treatments is based on the fundamental treatment of muscular and myofascial sources, unrelated to any associated peripheral compression of dorsal rami nerves. An intriguing explanation for all of these is that they are also all treatments for lymphedema, including the TENS unit (Board & Harlow, 2002; Choi & Lee, 2016). A lymphatic source would also explain the response of a single case of surgical decompression based on an assumption that the dorsal branch of the spinal nerve is compressed by the paraspinous muscles and fascia against the transverse process of these spinal segments (Williams, Rosson, Elsamanoudi, & Dellon, 2010). Surgical manipulation, bleeding, and irrigation would also relieve lymphedema within the muscle.

The most recent comprehensive review concludes that NP remains a condition that is difficult to treat (Howard et al., 2017). This is not unexpected because the source has yet to be identified. Taken together, the response to all of these local, topical, systemic, and myofascial sources would be expected and can be explained if the source of NP is lymphatic congestion. NP was originally described 80 years ago, and the etiology has been elusive. The predominant, left-sided, posterior thoracic location with intense pruritis as the major symptom all makes sense with the

lymphatic theory of NP within the context of understanding the details of the anatomy of the lymphatic system and the mechanisms of sensation of pruritis.

ELECTRICAL IMPEDANCE MYOGRAPHY

Electrical impedance myography is a technique that measures bioimpedance of muscles by creating a circuit with the patient electrically grounded and an electrode placed in selected muscles. The impedance of this system reflects the extracellular ionic environment (Rutkove & Rutkove, 2009). This technique can detect tissue edema with marked alterations on the measured impedance signature, where the edematous condition creates additional extracellular fluid through which the current can flow, which is measured as a decrease in impedance (Jia, Sanchez, & Rutkove, 2014). Bioimpedance has sufficient sensitivity to be useful for the early detection of unilateral lymphedema and for monitoring effects of therapy by using the normal contralateral limb as an individual control (Lukaski, 2013).

CASE REPORT

An 86-year-old man diagnosed with left-sided NP received testing and treatment. An MRI of the cervical spine revealed mild symmetric multilevel spondylosis and mild bilateral C5 neural foraminal stenosis. MRI of the thoracic spine was unremarkable other than findings consistent with a T5 hemangioma. Electrodiagnostic testing was negative for left dorsal scapular mononeuropathy, but there was possible evidence of an old left C5 radiculopathy. He was treated with gabapentin 300 mg 3 times a day and a lidoderm patch every 12 hours with complete relief of symptoms, and the cutaneous appearance in the affected area became normal within 2 weeks.

In an effort to establish initial testing of the lymphatic theory of NP, electrical impedance myography using Neurotherm Electrothermal Spine System Model 20S was obtained. The grounding pad was placed in the midline position in the lower posterior thorax, and an insulated electrode was placed sequentially in the bilateral rhomboid major muscles. The impedance on the affected left side measured 115 ohms, and the normal right side was measured at 136 ohms. These findings are consistent with lymphedema in the affected region of NP where the affected side would be expected to have a lower impedance due to the increased excellular fluid present in lymphedema. The hypothesis is that this patient was clinically improved with symptomatic care of the medications while the underlying source was still present.

SUMMARY

The pathophysiology of NP can be described as beginning with stasis of lymphatic flow in the superior aspect of the thoracic duct. The anatomy of the thoracic duct is such that only the T2–T6 portions are most posterior to the thoracic wall correlating to the location of the symptoms. The lymphatic congestion leads to intense pruritis mediated through Aδ and C fibers within the mediastinum and/or the adjacent posterior thoracic soft tissues. The intense itching leads to the observed skin biopsy results, and these resolve with topical and local treatments as well as systemic medications affecting the neurologic afferents because there is no primary cutaneous source. The various muscular and myofascial treatments have some effectiveness because these modalities are standard treatment with documented effectiveness for lymphedema. Initial electrical impedance myography results are consistent with the theory of a lymphatic source of NP. Future testing to include a larger sample size for electrical impedance myography testing and chest computed tomographic scanning of patients with the infrequent right-sided symptoms to investigate the presence of possible right-sided thoracic ducts with patients with right-sided symptoms is planned and may lead to confirmation of this theory. ■

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