Scholar: Pilot and Validation Studies ISSN: 2689-7644
Volume 2 Issue 2 Pages: 11 – 18 DOI:10.32778/SPVS.71366.2021.21

An osteopathic approach to Notalgia Paresthetica: case report, review of the literature, and proposal of an autonomic-neurologic-lymphatic model of notalgia paresthetica

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Keywords: Notalgia paresthetica; osteopathic manipulative medicine, osteopathic manipulative treatment; neuropathy, sensory neuropathy, somatic dysfunction, autonomic, somato-somatic reflex, neuropathic pruritis.

Article History: Date received: 11/20/2020 Date revised: 09/10/2021 Date accepted: 10/15/2021
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Introduction:

Notalgia paresthetica (NP) is an underdiagnosed condition involving chronic itch or paresthesia in a localized area along the area of skin medial to a unilateral scapula or immediately below the scapula. The condition was first described, and named, by the Russian neurologist Astwazaturow in 1934.[1]

The etiology of NP is commonly thought to be a sensory neuropathy, although there is no clear consensus on the specific etiology, with many authors suggesting that the condition is multifactorial. There is evidence for thoracic and cervical spine contributions to NP, as well as consideration for muscular compressive neuropathy and, perhaps, a lymphatic component.[1-4] There is no single treatment shown to be significantly effective for NP, which is why multiple treatments are often tried, including topical lidocaine and capsaicin, oral gabapentin and pregabalin, transcutaneous electrical nerve stimulation, physical therapy, and acupuncture.[3]
There have been two case studies published demonstrating the benefit of using osteopathic manipulative treatment in the care of patients with NP.[5,6]

This case demonstrates the benefit of utilizing an osteopathic manipulative medicine approach in the care of a 71-year-old woman and suggests several different mechanisms that may have been involved in her beneficial response.

Cases Report and Review Results:

A 71-year-old woman presented to the office upon the referral of her dermatologist for itching below the left scapula for two years, which the dermatologist had recently diagnosed as notalgia paresthetica. The itch had been consistent for the past two years. There was no associated burning, numbness, or tingling. The itch would flare with scratching, which she was careful not to do intentionally (sometimes, she would wake up scratching).

Her past medical history included mild depression, anxiety, and osteopenia. Her depression and anxiety were not problematic at the time of presentation. She had right shoulder surgery six years earlier, to repair a fracture.

Her social history included a remote history of smoking with occasional alcohol use. She was retired and walked six days a week and participated actively with her church. Her current medication consisted of Zoloft. She had no known drug allergies.

Review of systems was unremarkable except for the following: infrascapular itching that often awakened her at nights and was worsened by hot showers, occasional mild numbness in the bilateral hands over the past four years, some neck stiffness, and occasional stiffness and soreness at the bases of both thumbs. She has intermittent back pain that had not been a problem recently.

She had tried over-the-counter medications with no benefit. She was sent for OMM evaluation

and treatment upon diagnosis of NP by her dermatologist.

Her exam revealed normal overall appearance and normal eyes, ears, nose, throat, neck, heart, lung, and skin findings; specifically, there was no rash on her dorsal thoracic area. Neurologic exam showed normal findings, except for a positive Phalen test, bilateral hands, at 15 seconds. Her upper extremity motor and deep tendon reflex findings were normal, as were the Tinel tests over the carpal tunnels. Spurling test was negative. She did have markedly limited motion of the head and neck, especially with rotation, left more restricted than the right. She had a mildly increased cervical lordosis and thoracic kyphosis with significant anterior head carriage. She also had Heberden's nodes on most fingers. There was no edema in the extremities.

The individual's osteopathic structural findings included: suboccipital strain, C3 extended, rotated right, sidebent right, C5 extended, rotated right, sidebent right, hypertonic bilateral middle scalenes, restricted bilateral first ribs, elevated right first rib, T6 flexed, rotated left, sidebent left, T7 flexed, rotated left, sidebent left, the left sixth and seventh ribs were posteriorly translated, with a left posterior 6th rib tenderpoint, and there were fascial strains across the thoracic inlet, left wrist, and right elbow. She had restricted middle scalenes, levator scapulae, sternocleidomastoid, pectoral, and upper trapezii muscles.

A recent thoracic spine x-ray report, which she brought with her from her dermatologist, reported multilevel thoracic spondylosis with endplate osteophyte formation.

The initial diagnoses were notalgia paresthetica, osteoarthritis of the cervicothoracic spine and hands, and mild bilateral carpal tunnel syndrome with somatic dysfunction of the cranial, cervical, thoracic, rib, and upper extremity areas. It was also noted that she had postural imbalance consistent with upper crossed syndrome.

She was treated with osteopathic manipulative treatment (OMT) that included myofascial release (MFR), balanced ligamentous tension (BLT), Fulford percussor, Still Technique, counterstrain, ligamentous articular strain (LAS), and muscle energy technique. Specifically, treatment was started with the patient seated, treating the T6 and T7 vertebral restrictions with Fulford percussor technique, then treating the posterior left sixth and seventh ribs with Still technique. The right first rib was also treated with Still technique. The patient was then treated supine with direct-then-indirect MFR of the suboccipital area. The C3 vertebral dysfunction was treated with BLT followed by Still technique. The C5 dysfunction was treated with BLT and the middle scalenes were treated with LAS. The thoracic outlet was treated with indirect MFR. The posterior left sixth and seventh ribs were treated with BLT and the left posterior 6th rib tenderpoint was treated with counterstrain. LAS and MFR were used to treat her wrist and elbow dysfunctions. She responded well to OMT, with increased motion of her areas of somatic dysfunction and she had no discomfort after treatment.

As part of the first treatment, the patient was taught scalene, levator scapulae, and pectoral stretches to address her postural imbalance. She was also taught wrist stretches for her carpal tunnel syndrome. She tolerated the stretches well and was given instructions sheets for each of them. She was also directed to try a trial of glucosamine 1500mg, chondroitin 1200mg daily for her osteoarthritis of hands and spine.

Upon return two weeks later, she reported a marked (more than 50%) improvement with her periscapular itching. The itching had not awakened her since her last visit. It was still noticeable with hot showers. The tinging in her hands remained mild and intermittent. Her neck was still stiff, although she did have more movement. Her physical exam was very similar to her first visit, including a positive Phalen sign at 15 seconds. She had a similar set of osteopathic structural findings; however, she did have more motion present in the cervical, thoracic, and rib areas of dysfunction. She was doing her home exercises daily. She was treated again with OMT, similar to her first visit, with a good response. The stretches given last visit were reviewed with her. She had started taking glucosamine and chondroitin.

The patient returned four weeks later. She noted marked improvement for the two weeks following the second visit. She did note some mild recurrence of the itch at the third week. The overall amount of itching was greatly improved since her presentation.

She was seen again at two weeks, then twice more at one-month intervals. She was treated with a

similar regimen of OMT each visit. She continued to do her exercises and take her supplements. Her itching greatly improved (almost resolved) with her noting mild episodes at night about once a month. She no longer had any daytime itching. She also noted improved motion and less stiffness in her neck and upper back. She was instructed to follow up should she have any recurrence of her itching.

Discussion:

NP typically presents with itching along the medial border of the scapula or the infrascapular area, sometimes accompanied by paresthesias or pain. The symptoms are localized and continuous or frequent, often lasting for two to three years, seen more often in women, especially in mid-to-late adulthood, often on the left side.[1,4] There is frequently, but not always, a hyperpigmented macule or lichenified area in this region. This has been found to be due to chronic scratching or rubbing; the histopathology is consistent with scratching.[1,4,7,8]

Associated Cervical and Thoracic Degenerative Spinal Processes:

Some studies, using radiological imaging, including MRI, have found an association of thoracic and/or cervical degenerative disc and spinal changes associated with NP.[2] Various authors presume the problem to arise from the posterior rami of the upper thoracic spinal nerves, especially T2-6 nerve roots, while others believe the symptoms of NP to arise primarily from lower cervical degenerative disease, especially C5-6.[1,3,8] Some of these authors speculate that there may be other factors that are not easily visualized on radiological studies that may contribute to NP, such as cervical spine strain, osteoarthritis, muscle spasms, cervical fibrous bands or other cervical spine pathology.[2] They propose that NP spinal nerve compression may trigger functional, rather than recognizable structural changes in cutaneous nerves, causing NP.[2,3] Other studies have found no correlation of NP with findings on spinal MRI imaging.[7] EMG studies have been inconclusive.[4]

Possible Lymphatic Component:

An alternative etiology of NP has been proposed, by one author, to be lymphatic. The author points out that the localized symptoms of intense itching with NP do not fit well with dermatomal or dorsal rami models of spine pathology, nor does it fit with the model of the dorsal scapular nerve involvement, which has also been suggested.[4] This author proposes that lymphatic congestion, via thoracic duct dysfunction, may play a role, especially since the most posterior portion of the thoracic duct is at the level of T2-T6 where it enters the superior mediastinum. Some studies, but certainly not all, indicate a predominance of left-sided findings (the thoracic duct is found on the left 80-95% of the time).[4] Lymph congestion has been shown to be a cause of intense pruritus and the area around the thoracic duct is well innervated with C-fiber and A-delta nerve fibers.[4] This same author published a case in which electrical impedance myography studies were used to measure the soft tissue over the rhomboid major muscles in a patient with left-sided NP and the findings demonstrated increased tissue edema and extracellular fluid on the involved side, consistent with lymphatic congestion.[4]

Sensory Neuropathy:

Despite the lack of clear proof that NP is caused by nerve pathology, there is a large consensus that NP is a sensory neuropathy. This is for good reasons, since the symptoms appear to be neurologically-mediated and often respond, at least partially, to neurological medications. There are suggestions to explore neurological causes at the microneurological level.[7] Understanding the neurophysiology of pruritis will help to better explore the pathophysiology of NP.

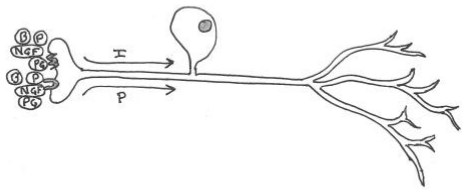
Neuropathic Pruritis:

One of the widely accepted categories of itch is neuropathic pruritis, which is caused by neuronal (or glial) damage or dysfunction.[9] The neurophysiology of itch overlaps considerably with the neurophysiology of pain, often involving the same nerve fibers and similar processes of peripheral and central sensitization.[10] There are selective itch receptors, named prurireceptors, in the skin that are found on small-caliber afferent C-fibers that are reactive to histamine. These histamine-sensitive prurireceptors are also weakly activated by capsaicin, which is why they have been termed "itch-selective" rather than "itch-specific", because they are selective, but not specific for itch.[11] These same fibers also carry nociceptors.[9] There are also histamine-

ISSN: 2689-7644
Volume 2 Issue 2 Pages: 11 – 18
DOI:10.32778/SPVS.71366.2021.21

independent prurireceptors likely involved in chronic itch.[10] There may be other itch receptors on A-delta fibers (See Figure 1).

Figure 1: C-fiber with itch-selective receptors (prurireceptors) and nociceptors.



I=itch, P=pain/nociception. B=Bradykinin, NGF=Nerve Growth Factor, P=Substance P, PG=Prostaglandin.

Peripheral Sensitization:

Peripheral sensitization for itch is similar to that for nociceptive processes, including increased sensitivity of C-fibers to inflammatory mediators, such as bradykinin, serotonin, and prostaglandins, as well increased nerve growth factor, which in turn leads to further upregulation of neuropeptides, especially substance P.[10] Substance P is a pruritogen as well as a probable neuronal sensitizing agent.[10]

Central Sensitization:

Central sensitization for itch has also been shown to be similar to that for nociceptive processes, involving the spinothalamic tracts as well as areas of the thalamus, subcortical and various cortical regions.[9] With central sensitization for itch, normal tactile and painful stimuli are perceived as itching, which is termed alloknesis (knesis is an ancient Greek word for itching). This likely involves hypersensitivity to small caliber C-fiber input, similar to that seen with chronic pain syndromes.[10] There are close parallels between pain and itch patterns of sensitization with allodynia correlating with alloknesis and hyperknesis, which explains why medications, such as gabapentin, which suppress C-fiber activity, are used for both neuropathic pain and chronic itch.[10] Such sensitization, once established, would be consistent with persistent itch out of proportion to tactile stimuli, such as is seen with NP. However, the question remains, which nerve structures are being facilitated?

Autonomic Components:

An area that has not been explored in NP is the role of autonomic pathways, particularly sympathetically-mediated pathways and reflexes. The sympathetic nerves system may play a key role in NP.

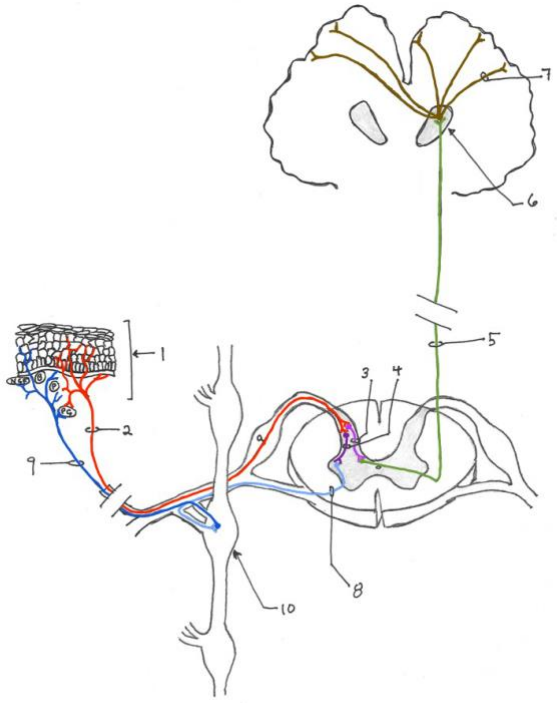
The autonomic nervous system is both an efferent and afferent system; all nerves containing autonomic efferent axons also carry primary afferent (sensory) fibers; the majority of these sympathetic and parasympathetic primary afferent fibers are small-caliber C-fibers.[10] These autonomic primary afferent fibers target T1-L2, S2-S4, and the vagal complex.[12] These fibers respond to nociceptive stimuli and other stimuli. Some are capable of eliciting a neurogenic inflammatory response in the surrounding tissue as well as affecting local vasculature.[12] Input from these fibers can influence both sympathetic and somatic reflexes at multiple levels, including the spinal cord, brainstem, and forebrain levels.[12]

Sympathetically-Maintained Itch:

Neuropathic pruritis, in some ways, can be seen as analogous to neuropathic pain, with the difference being itch rather than pain. Neuropathic pain can be generated and maintained either by sensory nerves alone or by inappropriate action of the sympathetic nervous system, the latter condition is known as sympathetically-maintained pain.[13] Sympathetically-maintained pain can be caused by neurogenic inflammation, which is triggered by inflammatory and stimulatory mediators released by the terminal sympathetic fibers.[13] This can lead to sympathetic-sensory coupling in which sympathetic nerve terminals contribute to activation and sensitization of C-fiber nociceptors via several mechanisms, including the release of substance P, bradykinin, prostaglandins, and increased nerve growth factor.[13] As mentioned above, itch-selective receptors are found on the same C-fibers as nociceptors and they also respond to substance P, bradykinin, prostaglandins, and nerve growth factor. It is quite probable that sympathetically-maintained itch follows much the same pathway as sympathetically-maintained pain with the thoracic

spinal sympathetic nerves contributing to the pathophysiology of NP (See Figure 2).

Figure 2: Notalgia Paresthetica Segmental Sympathetic and Spinothalamic Innervation.



1. Skin and underlying structures. 2. C-fiber afferent neuron (red). 3. Spinal cord interneuron (dark purple). 4. Spinal cord interneuron (light purple). 5. Spinothalamic tract neuron (green). 6. Thalamus. 7. Thalamocortical neurons (brown). 8. Preganglionic sympathetic neuron (light blue). 9. Postganglionic sympathetic neuron (dark blue). 10. Sympathetic paraspinal chain. B=Bradykinin, NGF=Nerve Growth Factor, P=Substance P, PG=Prostaglandin.

Cervical and Thoracic Sympathetically-Mediated Components:

Another probable sympathetically-mediated component of NP is from cervical spine processes, such as degenerative disc disease, degenerative joint disease (including facet arthropathy), and cervical somatic dysfunction.

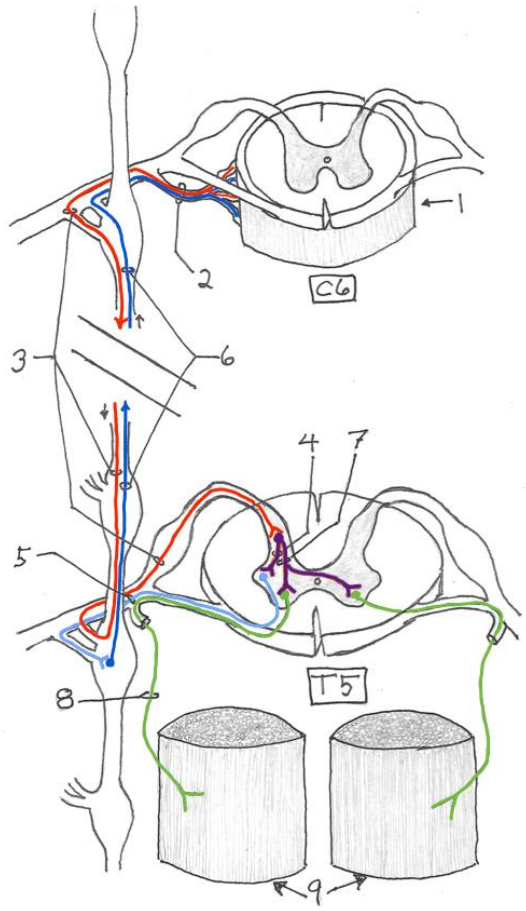
An example of a cervical-upper thoracic sympathetically-mediated reflex (which includes these same thoracic C-fibers) is the reflex changes found in the area of T1-T5 in patients with cervical nociceptive input, such as seen with cervical degenerative disc disease and nerve root impingement.[3,9]

The sympathetic efferent supply to the neck arises from the lateral horns of the T1-5 spinal cord segments and travels out the ventral horns to the paraspinal chain ganglia.[14,15] From there, they ascend to the cervical spine sympathetic ganglia, where they contribute efferent sympathetic fibers to the cervical spinal nerves, some of which innervate the cervical spine structures. These same pathways also carry sensory afferent information, from small-fiber primary afferent fibers.[9] The recurrent meningeal nerve (a.k.a. sinuvertebral nerve) is a nerve of particular interest because it is a sensory-sympathetic nerve that carries both sensory and sympathetic nerve fibers that innervate the dura, disc, ligaments, and periosteum of the spinal canal.[9]

The cervical spine sensory afferent fibers and the efferent sympathetic fibers travel along the paraspinal sympathetic ganglia to and from the upper thoracic segmental levels of the spine, in which the sympathetic fibers originate.[12,14] The recurrent meningeal nerve afferent fibers terminate in the dorsal horn of these thoracic spinal levels, where they connect through interneurons with ventral horn fibers. Connected motor neurons in the ventral horn can cause changes in resting tone of the associated paraspinal musculature, which in turn may cause somatic dysfunction of these vertebra and associated ribs (because the posterior ribs articulate with the respective transverse processes of these vertebrae).[14] The proximity of the sympathetic ganglia to the medial aspect of the posterior ribs may also play a role in the reflex changes causing somatic dysfunction at the costovertebral junctions.

This neurologic connection explains why thoracic somatic dysfunction of the associated vertebrae, costotransverse joints, and connected tissues is found with cervical nerve root impingement and other nociceptive processes of the cervical spine. A similar process may well be involved in patients with NP who have cervical degenerative changes or other nociceptive conditions that can contribute to the already overactive sympathetic activity at the thoracic level, especially if there is somatic dysfunction associated with this process (See Figure 3).

Figure 3: Segmental Sympathetic and Spinothalamic Innervation



1. C6 vertebra. 2. Recurrent meningeal (sinuvertebral) nerve. 3. Small-fiber primary afferent sensory neuron (red). 4. Spinal cord interneuron connecting sensory afferent and sympathetic efferent neurons (purple). 5. Preganglionic sympathetic neuron (light blue). 6. Postganglionic sympathetic neuron (dark blue). 7. Spinal cord interneuron connecting sensory afferent and ventral motor neurons (purple). 8. Ventral motor neurons to paraspinal muscles (green).

This cervical-thoracic connection has also been documented with the finding of somatic dysfunction of the cervical spine being associated with findings of upper thoracic somatic dysfunction, which is likely mediated through the same sympathetic pathway, involving the recurrent meningeal nerve.[16] This anatomic correlation is why cervical spine dysfunction can refer pain to the upper thoracic and periscapular region.[14]

Thoracic Sympathetically-Mediated Viscerosomatic Reflexes:

The T2-6 medial scapular area is also the site of commonly found viscerosomatic reflexes involving the heart and lungs, which are mediated by visceral primary afferent C-fibers, via the sympathetic nervous system.[17] It is possible that there could be vagally-mediated autonomic contributions as well.[12] Viscerosomatic reflexes have long been documented in the osteopathic literature and are commonly used in practice.[15,18] These reflexes are mediated by autonomic reflexes, such as the sympathetically-mediated viscerosomatic reflexes between the lungs and thoracic paraspinal and rib areas, which involve small-fiber C-fiber visceral sensory afferents as well as sympathetic efferents.[15] Neurogenic inflammation may play a role in these reflexes.[12] Treatment of the somatic dysfunction component with osteopathic manipulative treatment can have a beneficial aspect on the involved visceral structure, such as the lungs.[18] The reflex changes of somatic dysfunction of the thoracic vertebra and associated costovertebral joints has been found to be associated with other sympathetically-mediated reflex sources as well, including ENT, cardiac, and upper extremity nerve problems such as carpal tunnel syndrome.[15,19] All these structures also derive sympathetic supply from the upper thoracic spine.

In summary, the location of the symptoms of NP coincide with sympathetically active areas known to be associated with sympathetic-mediated reflexes from multiple structures.

Somato-Somatic Autonomically-Mediated Sensitization:

It is probable that the medial scapula and infrascapular areas of itching in patients with NP involve components of thoracic, costal, and cervical somatic dysfunction that facilitate a somato-somatic autonomically-mediated sensitization process, possibly involving neurogenic inflammation. This process likely maintains these facilitated itch pathways, which include the small-caliber prurireceptor C-fibers involved in the neuropathic itch of NP. This sympathetically-mediated facilitation, related to underlying somatic dysfunction, may not be the only factor; however, it may well play an important role in maintaining this condition. The neurologic component of this process

may be further aggravated by lymphatic congestion associated with the underlying somatic dysfunction of the structures related to the area of itch along the scapula.

Osteopathic Approach - Neurologic, Fluid, and Biomechanical:

A well-rounded osteopathic manipulative medicine (OMM) approach addresses patients and their problems from three perspectives - neurologic, fluid, and biomechanical.[20] While these aspects often overlap, evaluating and treating patients from each of the three perspectives is worthwhile.

A neurologic aspect of an OMM approach to patients with NP would be to identify and treat somatic dysfunction of the cervical, thoracic, and costal areas, to decrease and balance overactive autonomic activity at both the thoracic and costal levels, as well as to maximize function at the spinal nerve level. A fluid aspect of this approach would be remove any restrictions and correct any somatic dysfunction of the first ribs and thoracic outlet, to facilitate lymphatic drainage of the upper thoracic area and perhaps address any dysfunctions involving structures related to the diaphragm, to maximize the lymphatic pumping mechanics of breathing. Normalizing sympathetic tone may also improve lymphatic flow, via dilation of large lymphatic vessels. The biomechanical approach overlaps with the neurologic and fluid, by making sure there is optimum motion in the cervical and thoracic vertebrae and rib cage as well as addressing any myofascial restrictions that could affect neurologic, fluid, and mechanical function.

Likely Role of Somatic Dysfunction, and Its Treatment, in This Patient with NP:

This patient had a classic presentation of NP. While the initial cause of her two-year course of NP is not known, it is likely that her cervical and thoracic osteoarthritis as well as her cervical, thoracic, costal, and upper extremity somatic dysfunction contributed to a process of facilitation and sensitization of her neuropathic itch, leading to chronic NP. Her somatic dysfunction likely impacted her at a neurologic level, involving spinal nerves and sympathetically-mediated reflexes (thoracic and cervical), as well as at a lymphatic level. Her postural imbalance may also have contributed to this process as well.

This patient responded well to treatment of her cervical, thoracic, costal, and upper extremity somatic dysfunction with OMT, with greater than 50% improvement following the first evaluation and treatment. The home stretches likely helped long-term with her postural imbalance and eventually the glucosamine and chondroitin supplements may have helped her osteoarthritis of the spine and hands. Her immediate and sustained response to OMT suggests that OMT was the primary reason for her improvement.

Specifically, treating her thoracic and costal somatic dysfunction with OMT likely improved biomechanics as well as improving and balancing sympathetic and spinal nerve function. Treating her cervical somatic dysfunction may have helped decrease sympathetically-mediated reflex contributions to her middle thoracic and costovertebral process. Treating her suboccipital region may have helped normalize any vagally-mediated contributions to her NP. Treating her thoracic outlet, first rib dysfunction, as well as her lower rib and thoracic vertebral dysfunctions, likely improved her lymphatic drainage, maximizing lymphatic return from the thoracic duct, which may have contributed to her left-sided NP. Addressing her long-standing pattern of postural dysfunction with home stretches likely improved her biomechanical function and may have also helped with her neurologic and lymphatic function.

Conclusion:

Patients with NP may have components of somatic dysfunction contributing to their ongoing problem, including autonomically-mediated components related to somatic dysfunction of the cervical, thoracic, and costal areas, as well as lymphatic components. It is worthwhile to look for, and treat, underlying components of somatic dysfunction and postural imbalance in patients with NP. Utilizing an OMM approach, paying attention to neurologic, fluid, and biomechanical aspects of the patient, treating with OMT and home exercises, is a worthwhile approach to consider in patients with NP.

Author Contributions:

David B. Fuller, DO is the sole author. He is also the creator of the figures in this article.

Funding Sources: None.

Potential Conflicts of Interest Disclosures:
The author discloses that there were no conflicts of interest or financial support in the development of this project. All data is authentic and accurate.

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