Interventional Pain Management Reports
ISSN 2575-9841 Volume 3, Number 4, pp 105-109

Case Report

A Case Report on Sternocleidomastoid Syndrome - A True Pain in the Neck

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ABSTRACT

The Sternocleidomastoid (SCM) muscle may develop myofascial trigger points and the physical manifestation of pain and signs and symptoms due to these trigger points is commonly referred to as SCM syndrome. The diagnosis of SCM syndrome can be onerous as non-specific signs and symptoms associated with SCM syndrome may vary in presentation. The subject in this case report had post-acceleration-deceleration induced injury to SCM with hypertrophy of sternocleidomastoid muscle on the left side. The patient reported pain over the left SCM muscle, forehead, around the ipsilateral eye, over the cheek, the tip of the chin, sternoclavicular joint and deep in the throat upon. The patient was ascribed various diagnosis before a definitive diagnosis was made. The patient underwent three trigger point injections under ultrasound guidance, last one with BOTOX, and reported long-lasting pain relief. Overall with intensive physical therapy and trigger point injections the acute symptoms resolved.

Key words: Sternocleidomastoid syndrome, myofascial pain syndromes, trigger points, ultrasound, Botox

The sternocleidomastoid (SCM) muscle is composed of clavicular (short head) and sternal (long head) divisions (1). Both heads of the SCM muscle attach to the head of the mastoid process with the sternal head attaching to the sternum and the deeper clavicular head attaching posteriorly and laterally onto the clavicle (2). This unique insertion of 2 heads confers the SCM with a complex multidirectional pattern of movement (1,2). The SCM muscle may develop myofascial trigger points that are palpable, taut, ropy or knot-like bands and sensitive to pressure when compressed. The physical manifestation of pain and signs and symptoms due to these trigger points are commonly referred to as SCM syndrome, which is classified as a myofascial pain syndrome (3). This manuscript discusses a case report of SCM syndrome as a result of acceleration-deceleration induced injury to SCM and will also analyze the unique pathomechanics, diagnostic challenges and the conservative methods employed by the authors.

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CLINICAL VIGNETTE

We present a case history of a 62-year-old man who consented for this case report to be published. He presented to the pain clinic with a diagnosis of tension headaches and chronic neck pain. He complained of intermittent pain in the left neck for the last 6 months. The patient reported that he was in a road traffic accident in which he was a restrained driver and was rear-ended by a car. He had an acute pain in the neck for 2 weeks and the computed tomography (CT) scan did not report any acute pathology. He insidiously developed a chronic dull ache in the left neck which was associated with intermittent spasms, swelling, and pain on movement of the neck in all quadrants, with an increased severity of pain associated with sudden movement of the neck on the right side. He

reported that, along with a significant decrease in range of motion (ROM) of his neck due to pain, his neck "locked" intermittently at times. He described his neck pain as cramping and throbbing with an intensity rating of 6 to 8 out of 10 on the Numeric Rating Scale (NRS-11), associated with 4 to 5 episodes of neck spasms daily and radiating to the left shoulder along with a sharp, stabbing pain in the throat with an intensity of 8 to 10 out of 10 during mastication and swallowing. He also reported experiencing intermittent frontal headaches and facial pain for the last 2 months along with lacrimation and coryza. The pain was alleviated by rest, hot compresses, and 4 to 6 tablets of ibuprofen every day. He was also prescribed 10 mg of cyclobenzaprine 3 times a day (started post accident) and 300 mg of gabapentin 3 times a day (taken over the past 10 years for diabetic neuropathic pain); he noted a poor response to cyclobenzaprine. He also underwent a neurological evaluation and was diagnosed as having spasmodic torticollis along with facial and glossopharyngeal nerve neuralgia. On a simultaneous visit to an otolaryngologist's office for evaluation of facial, throat, and temporomandibular joint (TMJ) pain, chronic sinusitis was suspected and a Water's view x-ray was ordered, which was negative for any pathology.

The patient's comorbidities were limited to well-controlled hypertension (10 mg of lisinopril once a day and 25 mg of Metoprolol XL once a day) and type 2 diabetes mellitus, which was well controlled on oral hypoglycemic agents (500 mg of metformin twice a day). His surgical history was positive for C3-4 cervical fusion and right inguinal hernia repair 10 years prior. The patient was within healthy weight parameters and denied alcohol and tobacco use, but reported that he used edible marijuana occasionally to help with the pain. He disclosed that he worked as an electrician and that the pain interfered with his job. An impending divorce was also a major stressor in his life at that point. A series of recent blood and urine tests were reviewed and found to be unremarkable.

On physical examination, the patient had an obvious red, serpentine-like mass on the left side of his neck in the region of the left SCM, which appeared bigger than the right SCM, conferring a slight degree of unevenness to the neck as a whole. Postural evaluation revealed decreased cervical lordosis consistent with his history of cervical fusion as well as forward positioning of the head with the shoulder girdle protracted, along with a slight thoracic kyphosis. Cervical ROM was limited to a reduced flexion, severe reduction in rotation on the right side, with right rotation decreased by 60% and associated with intense pain; as compared to the left rotation, which was limited to 20% and accompanied by unexplained lacrimation. Palpation of the cervical spine revealed no step-off deformity or percussion pain. There was significant myofascial pain in the paravertebral, occipital, and trapezius muscles. A superficial palpation of the left SCM revealed a hypertrophied muscle with tense knots. Provocative testing with deep palpation of the trigger points in the sternal head was associated with sharp pain at the point of palpation along with a referred pain in the throat, which was similar in nature (dull and achy) and intensity to the pain experienced by the patient during mastication and swallowing. A constant pressure on the clavicular head of the SCM was associated with pain in the left shoulder, and the exam was interrupted as the patient reported feelings of dizziness and nausea. Furthermore, a gentle tap at the trigger points elicited a "jump sign" due to involuntary contraction of the SCM muscle. Further examination did not reveal any sensory or motor deficits with normal and bilaterally equal tendon reflexes. The patient's sensory response to pinprick and gentle touch tests was unremarkable; muscle strength tests were 5 out of 5 and symmetric throughout.

The patient was diagnosed as having dystonia and myofascial pain of the neck muscles along with left-sided neck pain due to contracture of the SCM muscle. He was prescribed diclofenac gel, 75-mg enteric-coated tablet of diclofenac twice a day, and 500-mg tablet of methocarbamol three times a day as a substitute for ibuprofen and cyclobenzaprine. Furthermore, he was also scheduled for left neck trigger point injections under ultrasound guidance (USG) and given a referral for physical therapy (PT). In lieu of previous neck surgery and hardware in situ, an urgent CT scan of the neck was ordered along with an ultrasound exam of the neck, bilaterally, with a focus on SCM muscles to rule out any benign or malignant mass.

His CT scan reported intact C3-5 hardware and the USG of the SCM didn't report any anomalies.

The patient underwent USG trigger point injections (4 in the sternal head, 3 in the clavicular head, and 4 in the body of the left SCM) under sedation with 2 mg of midazolam intravenously (IV) and 100 mcg of fentanyl IV. The block solution used was 10 mL of preservative-free 0.5% bupivacaine mixed with 40 mg of methylprednisolone (Depo-Medrol; Pfizer, New York, NY). Using a 25-gauge 5/8th-inch needle, each trigger point was injected with one mL of block solution, in a fan-like motion, ensuring not to extend beyond the muscle.

At 12 weeks after initial presentation, the patient reported a good response to trigger point injections for 6 to 8 weeks with a decrease in his left neck spasms to 1 to 2 episodes per day, a resolution of his headaches and facial pain, and an overall decrease in pain intensity to 4 to 5 out of 10 on the NRS-11. Furthermore, he stated that during provocative maneuvers such as movement of the head on the right side and upon swallowing, the pain had decreased to 5 to 6 out of 10 and was tolerable. He reported that the ROM of his neck was still significantly limited and that he was unable to sleep at night after taking methocarbamol, which was substituted by tizanidine (2 mg three times a day as needed). At this point, after retrospectively examining the symptoms and the clinical response to the trigger point injections, he was diagnosed as having SCM syndrome. The patient was also given a referral to an otolaryngologist for consideration of bipolar SCM muscle release, but he refused surgical intervention in favor of conservative management.

The patient underwent a second trigger point injection 3 months after initial presentation and reported a similar degree of relief to what he reported after his first trigger point injection. He was then scheduled for a botulinum toxin (BOTOX) injection of the SCM muscle under USG along with a second referral for goal-directed PT. He underwent a BOTOX injection under USG and under IV sedation with 4 mg of midazolam and 100 mcg of fentanyl. The block solution used this time was preservative-free 0.9% chloride and 100 units of BOTOX. Using a 25-gauge 1.5-inch needle, the block solution was injected at the same location and using the same technique as the trigger point injections. At 12 weeks after the BOTOX injection, and at 5 months after initial presentation, the patient reported a significant decrease in his pain to 2 to 3 out of 10, along with resolution of his neck spasms, headaches, and facial pain. In the interim, the patient underwent two 3-week sessions of PT and 4 sessions of psychological supportive therapy. He reported that he used the muscle relaxants and diclofenac tablets sparingly. He stated that he has regained some of his ROM at the neck but still has some pain and discomfort on lateral rotation, for which he continues to perform the neck exercises at home. Upon examination of his left neck, the SCM swelling had reduced considerably and the physician was unable to elicit the jump sign. The patient further stated that he felt a sense of accomplishment that he was able to take care of his pain without any opioid prescriptions.

DISCUSSION

The SCM may develop trigger points (TPs) that result from chronic irritation induced by stress, overuse of the muscle, poor posture, inadequate work posture and ergonomics, aging, pillow height, neck trauma (whiplash), certain occupations (violinists), weight-lifting, anxiety, stress, hyperventilation syndrome, among other causes (3). The TPs are differentiated as active or latent, with the former causing a dull aching pain that changes character on compression to sharp referral pain away from the SCM muscle, along with motor dysfunction and autonomic phenomena (4,5). Active TPs in the SCM may also cause the head to tilt to that side, because to hold the head upright would induce pain (4). Latent TPs are usually asymptomatic and may cause referred pain in response to compression, stretch, or overload of the affected tissues (5).

The SCM may harbor TPs, with symptoms varying according to the topographical location of the TPs (3). The sternal TPs may refer pain in the area around the sternum, whereas TPs in the middle part of the SCM may refer pain to the ipsilateral cheek, throat, and sinuses; and TPs near the mastoid may refer pain near the occipital ridge, the vertex of the skull, the eye, and the eyebrow (3). Furthermore, sternal TPs are also implicated in the development of autonomic disturbances such as such as excessive lacrimation, conjunctivitis, rhinitis, blurred vision, coryza, and ipsilateral eyelid droop, most likely due to spasm of the orbicularis oculi muscle. In severe cases, blurred or possibly double vision is sometimes reported (3,6).

The clavicular head of the SCM is a short-range cervical rotator, and along with other short-range cervical rotators such as splenius capitis and obliquus capitis inferior, it is extremely important to our sense of equilibrium and is hypothesized to play an important role in disequilibrium symptoms (2). The TPs of the clavicular head may be associated with referred pain to the frontal region, which might cross the midline and involve the entire forehead (3,6). The TPs in the upper aspect of the clavicular region can refer pain in the throat on swallowing and cause popping or ringing sounds in the ipsilateral ear. The symptoms of postural dizziness or disorientation are more commonly reported than vertigo and are usually activated by a sudden turning of the head or may be reproduced from pincer palpation of the SCM; these symptoms last from seconds to hours (3,6). The clavicular TPs may also relate to the autonomic phenomena of localized sweating and vasoconstriction, which appears in the form of blanching and thermographic cooling in the frontal area of the forehead (6). Usually, with the involvement of the clavicular head, there is a triad of concurrent symptoms like dizziness, frontal headache, and dysmetria (6).

The diagnosis of SCM syndrome can be onerous, as the constellation of nonspecific signs and symptoms vary in presentation and in the sequence in which they appear (7). A list of differentials that may be attributed to the myriad signs and symptoms associated with SCM syndrome include atypical cervical neuralgia, Meniere's disease, congenital and spasmodic torticollis, trigeminal neuralgia, facial nerve neuralgia, vestibulocochlear problems, fibromyalgia, cervical sprain and strain, cervical disc disorders, etc. (6,7). Tension headache is the most common diagnosis that is assigned to patients with myofascial pain syndrome of the SCM, as neck pain may not be the first presenting symptom (8). Furthermore, the physician should be cognizant of the fact that certain physical activities can perpetuate and worsen the symptoms associated with SCM syndrome. It is vital to identify established faulty movement patterns in the cervical and thoracic regions, so that appropriate corrective measures can be performed (3,6). The patient's static posture and movement patterns, especially cervical flexion, sit-to-stand, and swallowing, should also be scrutinized closely (6,9). The physical exam should also include other joints that may mimic the symptoms of SCM TPs, such as the sternoclavicular joint and the TMJ; the upper cervical spine should be examined for joint dysfunction; and motion palpation procedures should be applied to the entire spine in order to detect joint dysfunction at lower levels as well (9). PT is the mainstay of treatment and techniques such as ischemic compression, stretching, and post-isometric relaxation are quite effective (9). The proximity of the TPs to the vital structures and blood vessels prompted the authors to use USG precision to inject the block solution into the muscle. Current literature offers evidence that palpation techniques may be suboptimal in identifying TPs (10). USG has been reported to have excellent intrarater reliability for detecting TPs and can supplement the physical exam to objectively identify the TPs; USG may also improve clinical success with various therapeutic measures (11,12). The clinical evidence for the use of BOTOX for TPs is currently equivocal (13). However, recent studies have reported on the safe use of BOTOX for TPs and have commented that BOTOX significantly reduced pain as compared to placebo (14,15). The authors, in order to offer a longer period of relief, decided to use BOTOX. This decision was also based on the fact that the patient was highly motivated to continue with conservative management and experienced significant clinical improvement.

CONCLUSION

The fact that such an array of symptoms as facial pain, lacrimation, dizziness, blurred vision, and eyelid jerking could be generated from an activated trigger point was not a diagnostic possibility entertained by any of the practitioners who previously examined the patient. An effective treatment of SCM syndrome can be very rewarding to both doctor and patient, as when it is severe, it can be very distressing. This case report demonstrates that it is vital to have knowledge of muscle kinesiology and the pathomechanics of myofascial pain syndrome; such knowledge, in conjunction with the application of novel techniques such as USG precision and the use of BOTOX to identify and treat symptomatology associated with TPs, can lead to positive outcomes.

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