CLINICAL CORRESPONDENCE

Cervicogenic headache caused by myofascial trigger points in the sternocleidomastoid: a case report

JK Roth¹, RS Roth², JR Weintraub¹ & DG Simons³
¹Michigan Head Pain and Neurological Institute, ²University of Michigan Health System, Department of Physical Medicine & Rehabilitation, Ann Arbor, MI, and ³Emory University, Department of Rehabilitation Medicine, Atlanta, GA, USA

Julie K. Roth, M.P.T., Michigan Head Pain & Neurological Institute, 310 Professional Dr., Ann Arbor, MI 48104, USA. Tel. +1 734 677 6000, fax +1 734 677 2422, e-mail jroth@mhni.com Received 12 October 2006, accepted 7 December 2006

Disturbances in the integrity of the upper cervical spine are now well known to be associated with head pain and can give rise to chronic daily headaches (1–3). Head pain that derives from cervical dysfunctions or disease is now contained under the category of cervicogenic headaches (4), which describes a heterogeneous group of disorders that commonly refer pain from structures in the cervical spine region (e.g. joints, muscle, nerve) to various regions in the head. The mechanisms that underlie head pain referred from the neck are not entirely understood. There is general consensus that the trigeminocervical complex, which is located in the lower brainstem and upper three cervical spinal segments and receives multiple afferent inputs from both the trigeminal nerve and cervical peripheral afferents, serves as the centre of convergence for the projection of perceived pain in the head that originates from cervical pain generators (1). However, this model may not explain all forms of cervicogenic pain, in particular myogenic referred head pain that derives from myofascial trigger points (MTrPs) of the cervical and upper back musculature (5, 6). An accurate diagnosis of the aetiology of cervicogenic headache can be challenging for the clinician due, in part, to the overlap of pain and associated symptoms between cervicogenic headache and primary headache (3, 7, 8) and the multiple abnormalities of the cervical spine and musculature that can be a primary source of head pain (9–12).

Several authors have emphasized the role of anaesthetic blockade as a diagnostic tool has paralleled the expansion of the use of interventional procedures for the treatment of chronic cervicogenic headache (14–16). Interventional diagnostic measures tend to follow from abnormal radiographic findings, such as spondylosis, disk disease or facet arthropathy (1,9), and rely on patient report of diminished pain to anaesthetic challenge as a guide to diagnosis. Increasing reliance on nerve blockade as a diagnostic tool may encourage de-emphasis on the clinical examination of the musculature. Reliance on an interventional approach to the diagnosis of cervicogenic headache that targets joint arthropathies or possible nerve involvement tend to ignore the potential contribution of MTrPs or intersegmental motion restrictions as primary causes of cervicogenic headache (10, 17–19). Identification of the musculoskeletal dysfunctions requires a careful and informed clinical examination. Lack of familiarity with the characteristic myofascial referred pain patterns frequently leads to misdiagnosis and ineffectual headache interventions (10).

We report a case of a male with a 25-year history of chronic headaches despite an extensive course of medical evaluations and treatment. After a recent hospitalization and further interventional procedures to identify a cause of his pain, a careful physical examination of the cervical musculature identified MTrPs in the sternocleidomastoid (SCM) muscle as a primary source of his head pain. Physical therapy that focused on reducing the myofascial dysfunction and addressing perpetuating factors felt to be maintaining his MTrP activity brought about dramatic reduction in his pain within 6 weeks, which was maintained at 6 months.
Case report

A 43-year old White male was referred to a head pain specialty clinic for evaluation of refractory headache. He described the onset of right-sided head pain at age 18, primarily in the right supraorbital region, with no history of significant cervical injury. Pain severity gradually increased and became constant by age 28. At age 34 years, right-sided cervical and occipital pain began. Over the years, the patient saw many neurosurgeons, neurologists, pain specialists and a chiropractor and underwent five trials of physical therapy. The head pain was not accompanied by auras or prodrome. Aggravating factors included sleep deficit, weather changes, exercise and exertion, orgasm, neck movements, chewing, driving, Valsalva manoeuvres and coughing. Associated symptoms that fluctuated with head pain severity included decreased appetite, right-sided nasal stuffiness and drainage with conjunctival injection, tearing and right eye redness, cognitive impairment, inability to sleep more than 1–2 h continuously, fatigue and hearing a rushing, pulsating sound in the right ear. He rated his daily pain as moderate to severe and had a medical history of overuse of pain medication. Current medications included valdecoxib, gabapentin, hydrocodone and paracetamol.

Prior diagnostic work-up had included a cervical X-ray indicating ‘very small’ spurs posteriorly at the C4-5 and C5-6 levels, magnetic resonance imaging (MRI) revealing a mild left C5-6 disc protrusion without evidence of canal stenosis or nerve root involvement, and a computed tomographic (CT) scan of the brain was negative. Prior treatment had included multiple facet blocks at C3, C4 and C5 with temporary reduction of neck pain, and no resolution of head pain, radiofrequency ablation at C3, C4 and C5 that increased occipital pain, selective nerve block at C2 level that severely increased his pain, and multiple occipital nerve blocks that aggravated his pain. Prior medication trials had included zonisamide, gabapentin, divalproex and rofecoxib. Despite repeated adverse responses to occipital nerve blockade, the referring neurologist concurred with the prevailing diagnosis of occipital neuralgia and referred the patient for specialty head pain management.

At the head pain clinic, neurological examination revealed a healthy-appearing man reporting constant pain at the right lower occipital region, with radiating sharp pain in a right supraorbital distribution. The remainder of the neurological examination was entirely within normal limits. Further radiographic work-up included a brain MRI that showed several small foci of increased signal intensity in the white matter bilateral hemispheres that was felt to be a non-specific finding. Maxillofacial CT indicated moderate left ethmoid sinus mucosal thickening. The diagnosis was hemicrania continua or a possible secondary cervicogenic headache with medication overuse headache, with a recommendation for multidisciplinary treatment and admission to the facility’s in-patient head pain unit.

The 16-day hospital course included an i.v. medication protocol (i.v. droperidol, diphenhydramine and magnesium); behavioural intervention; and multiple interventional procedures including a right suboccipital injection which worsened his pain, and a C2-C3 facet block which provided 24 h of head pain relief. The discharge medication regimen included: for head pain prevention, topiramate, quetiapine, verapamil; and to abort head pain, indomethacin, Midrin® and quetiapine. After discharge, the patient returned home where he underwent selective nerve blockade at C0-C1 and C1-C2 that provided moderate but temporary relief. He returned to the head pain clinic for radiofrequency ablation of the C2-C3 facet joint. This resulted in no appreciable change in his pain symptoms. At this point, the patient was then referred to physical therapy (J.K.R.) for examination for a possible musculoskeletal contribution to the head pain.

The physical therapy assessment noted ongoing constant level 2/5 head pain (0 no pain at all, 5 severest pain) that increased to a severe level 5/5 two episodes per week. Activities that increased head pain included repetitive cervical motion, lifting his head while in left side-lying position and use of his right upper extremity. Previous courses of physical therapy and soft tissue massage had produced only temporary reduction in pain. Physical examination of posture and alignment revealed a slumped sitting posture. Range of motion (ROM) testing revealed restriction in right cervical rotation and cervical extension. Muscle tightness was noted in the right pectoralis muscle. His headache was reproduced with active motions including cervical extension and right upper extremity flexion. On palmar palpation of the right SCM, the most significant finding was immediate and dramatic reproduction of the patient’s primary occipital and supraorbital pain. MTrPs were noted in the midmuscle belly superficial (sternal) head of the right SCM. The patient was assessed to have a primary myofascial pain involving the SCM, with contributing muscle imbalances and postural deviations. He volunteered that during previous evaluations by
Discussion

Forty years ago Travell (19) described the influence of biomechanical factors in the aetiology and maintenance of chronic headache, with particular reference to faulty posture and its effect on muscle strain and overload of the SCM, associated with MTrPs that caused head pain. Much has been written about myogenic headaches that derive from MTrPs (10, 20–22) and the various referred head pain patterns that result from MTrPs of the cervical musculature are now well described (10). Unfortunately, there has been longstanding debate on the contribution of MTrPs in the aetiology of acute and chronic musculoskeletal pain. Recently, Simons (10, 23, 24) has proposed the integrated hypothesis to explain the basic mechanisms of referred pain that results from MTrPs. As a result, a consensus on the pathophysiology of chronic myofascial pain and its associated referred pain is now developing, and recent experimental investigations have provided preliminary support for this model (6, 25–27). Despite these advances, there continues to be a prevailing view among some pain practitioners that musculoskeletal dysfunction is rarely a cause of intractable pain (28), that has led to an undervaluation of myofascial pain as a source of persistent pain. The case described here illustrates that this holds equally true for myogenic head pain that is repeatedly dismissed as cervicogenic headache.

The diagnosis of cervicogenic headache can be a significant clinical challenge. In this case, there was significant symptom overlap between head pain secondary to SCM MTrPs (see Fig. 1) and the clinical features of hemicrania continua, including continuous and persistent one-sided head pain, nasal congestion and conjunctival injection. Of particular note was the presence of autonomic features that are well known to be associated with hemicrania continua (4) but are less well appreciated as common symptomatic findings for MTrPs in the sternal division of the SCM (10). In addition, diverse anatomical pathways can cause cervicogenic headache. This patient had undergone multiple medical examinations by several medical specialties as well as receiving five previous courses of physical therapy. It is noteworthy that despite the many previous practitioners, all had failed to examine the SCM as a possible source of pain. This is probably because individuals with head pain from the SCM do not report pain along the distribution of the muscle, but rather across the occipital, supraorbital and temporal regions of the head (10). The failure to consider referred pain in locating musculoskeletal pain complaints is common in clinical practice and can lead to diagnostic confusion and misdirected interventions (8, 10). This is not the case with visceral sources of referred pain such as cardiac and gallbladder disease. With knowledge of the various referred pain patterns associated with cervical MTrPs, the pain clinician is enabled to work within a framework that relates specific patterns of pain with their associated TrPs, leading to their identification in the troubled muscle or muscle groups.

Moreover, as this case illustrates, when cervicogenic headache secondary to chronic myofascial pain is effectively treated by a multimodal approach,
there can be surprisingly rapid and prolonged diminution in clinical morbidity, even in longstanding cases (19, 20). The treatment of chronic myofascial pain requires a comprehensive approach directed to the underlying MTrP source, while simultaneously addressing the central importance of perpetuating factors that maintain TrP activation (10, 29). As noted, the physical therapy regimen prescribed was multifaceted, with attention directed to manual techniques to reduce TrP activity, including stretching to restore muscle length and elasticity, relaxation to decrease TrP activation and instruction to correct faulty postures. This report indicates that individual and isolated physical medicine modalities often fail to manage cervicogenic headache effectively, but that manual therapies combined with therapeutic exercise and posture re-education can produce significantly positive outcomes.

The SCM can be a particularly enigmatic and common source of myofascial cervicogenic headache (30). As the SCM does not overlie the posterior cervical spine, it is easily ignored during a physical examination for cervical dysfunctions that may underlie a headache and patients typically do not complain of neck pain with the presence of SCM MTrPs. SCM MTrPs and muscle hypertonicity are common sequela of whiplash injuries from motor vehicle accidents (31–33) and whiplash injuries are a known and frequent initiating event for cervicogenic headaches (34). Forward-head posture can contribute to the development of SCM MTrPs. The SCM produces cervical flexion as its primary function. Compromised endurance of deep flexor muscle of the cervical spine is related to abnormal forward-head posture (35). There is also evidence that relative excessive EMG activity of the SCM, which could give rise to TrPs if recurrent, is related to weak deep cervical flexors (33). Several clinical studies have documented an association between deep cervical flexor weakness and cervicogenic headache (33, 36, 37).

Anaesthetic nerve blockade to confirm the diagnosis of cervicogenic headache has become increasingly popular (14, 16). Anaesthetic cervical blocks can identify, at times, the level of spinal root mediation of pain, but not necessarily pinpoint the abnormal cervical abnormality generating the pain signal. Indeed, the clinical utility of nerve blocks has been challenged, based on their lack of diagnostic specificity and the absence of empirical evidence to confirm long-term therapeutic benefit (38–40). In this case, the patient had variable responses to multiple (often repeated) cervical blocks and no appreciable benefit from neurotomy of the C2-C3. In contrast, manual techniques, that include assessment of intervertebral segmental motion, muscle length and joint restrictions, have been found to be reliable and equal to facet blockade in confirming
the level of facet dysfunction that mediates a cervicogenic headache (41). Moreover, careful clinical examination that emphasizes myogenic dysfunction may be more comprehensive and effective in identifying the range of cervical abnormalities that can produce head pain. This case report highlights the relative advantage of a hands-on physical examination, with careful palpation of the cervical musculature, when evaluating a patient with suspected cervicogenic headache.

Acknowledgement

The authors thank Todd Rozen MD for his very helpful comments on an earlier draft of this manuscript.

References