

Thalamic Pain Syndrome (Central Post-Stroke Pain) in a patient presenting with right upper limb pain: a case report

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In the elderly, pain of a widespread nature can often be debilitating. It is not uncommon to attribute this widespread pain to osteoarthritis within the spinal column structures and peripheral joints or to other musculoskeletal etiology. However, chiropractors should remain wary regarding pain experienced by the elderly, especially if pain is widespread and exhibits neuropathic features. Common features of neuropathic pain involve the presence of allodynia, hyperpathia and hyperalgesia. This characteristic widespread pain can sometimes be the sequelae of a central nervous system lesion such as a "Thalamic Pain Syndrome", or "Central Post-Stroke Pain", which are terms commonly used to describe pain that originates in the central nervous system.

Following is the case of a 90-year-old patient presenting with widespread pain attributed to Thalamic Pain Syndrome or Central Post-Stroke Pain. Discussion of the characteristics of neuropathic pain and bedside testing techniques are presented to help the chiropractor identify a patient who may be presenting with Central Post-Stroke Pain.

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KEY WORDS: thalamic diseases, cerebrovascular disorders/complications, chiropractic, osteoarthritis, hyperalgesia, neuralgia.

Les douleurs irradiantes, chez les personnes âgées, peuvent souvent devenir incapacitantes. Il est fréquent d'attribuer ce type de douleur à l'arthrose, qui touche les structures de la colonne vertébrale et les articulations périphériques, ou encore à une autre étiologie musculo-squelettique. Cependant, les chiropraticiens devraient se montrer circonspects devant les cas de douleur chez les personnes âgées, surtout si celle-ci couvre une grande région et présente des caractéristiques neurologiques. Les cas de douleur neurologique sont habituellement associés à la présence d'allodynie, d'hyperpathie et d'hyperalgésie. Ce type de douleur irradiante peut parfois être une séquelle d'une lésion du système nerveux central, comme le syndrome de douleur thalamique ou la douleur post-accident vasculaire central. Ces termes sont couramment utilisés pour désigner une douleur qui trouve son origine dans le système nerveux central.

Voici le cas d'un patient de 90 ans qui souffre de douleur irradiante, attribuée au syndrome de douleur thalamique ou à une douleur post-accident vasculaire central. Suit une discussion sur les caractéristiques de la douleur neurologique et une présentation des techniques d'évaluation au chevet des patients, dont l'objectif est d'aider le chiropraticien à repérer les personnes pouvant présenter une douleur post-accident vasculaire central.

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MOTS CLÉS : maladies thalamiques, complications ou troubles cérébro-vasculaires, chiropratique, arthrose, hyperalgésie, névralgie.

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Introduction

In the elderly, it is not uncommon to attribute widespread pain to osteoarthritis within the spinal column structures and peripheral joints. In other cases, malignant processes or other less common pathologies are the causes of widespread pain. Chiropractors should remain wary of widespread pain experienced by a geriatric patient. Central Post-Stroke Pain is one possible cause of widespread pain after a cerebrovascular accident. Clinically this syndrome can be accompanied by musculoskeletal allodynia, hyperpathia and hyperalgesia.

Case report

A 90-year-old lady presented to a chronic pain clinic with severe pain of the left neck and left upper limb. She was complaining that her arm felt “numb” and “dead” in the morning. She had been referred by a rheumatologist.

Past history

In 1972 she had fractured her left humerus and recovered without complication. In 1988, while in Florida, she had developed congestive heart failure and had been admitted to hospital. During that episode she developed widespread pain affecting the left side of her body (a diagnosis of this pain was not made at that time). Subsequently she had a series of admissions to hospital for related conditions which included congestive failure, arrhythmia and NSAID-induced gastrointestinal bleeding and anemia. There had also been an episode of stroke involving her left face and arm which was treated clinically (which was not investigated with imaging). In 1997 she had been referred to the rheumatologist with lateral epicondylitis. She was prescribed splint therapy and cortisone injections and ultimately responded well to these therapies. Despite this improvement, she had continued to complain of the left-sided widespread pain, which prompted referral to the pain clinic.

Recent treatment

Recent treatment for the neck and upper limb pain included a surgical collar which was worn during the day, with a towel worn at night producing equivocal benefit. She attended physiotherapy for laser, ultrasound and traction of the neck with no resolution of symptoms. Soft tissue therapy utilizing a vibrating modality over the neck and upper back musculature, and heat application at night yielded no abatement in pain sensation.

Chief complaints

Pain radiated from the left side of the neck to the fingers of the left hand. The left arm felt “numb” or “dead” when she awoke in the morning, and the left upper back and arm were oversensitive to stimulation. She experienced occasional headaches that did not occur daily (the headaches were not otherwise characterized).

Mental status examination

Mental status examination was conducted by a psychiatrist, (without psychometric testing), which revealed a euthymic individual. Memory recall was intact throughout the course of the interview. She was able to organize and relate her symptoms without any signs of disorientation.

Physical examination

On postural examination, she presented with marked anterior translation of her head with sloped shoulders. Active neck rotation to the right and left was limited to about half of the expected range due to pain. Lateral flexion of the neck was markedly limited due to pain. She was very protective of her left shoulder due to pain.

On musculoskeletal examination, the patient related that the pain radiated into the third and fourth fingers of the left hand. Upper limb deep tendon reflexes were normal and symmetrical, and motor strength was normal. Assessing with light touch induced pronounced amounts of pain within the region being tested (allodynia) in the area of the left side of the neck, left supraclavicular area and all areas of the left upper limb. Light prick induced an exaggerated amount of pain (hyperalgesia) on the left side, compared to a normal response on the right. Areas of hyperalgesia included the left side of the face, the left supraclavicular area, left arm, the front of the chest and the back down to the beltline all on the left side. Hyperalgesia was not present distal to the beltline. Scratching with a pin along the upper arm produced a persistent “pricking” sensation in the hand (hyperpathia). Scratching on the left face produced radiating sensations of sharpness (hyperpathia) surrounding the area being scratched, in contrast to a normal response on the right (pain did not spread). Once the pain was “stirred-up” in the left upper body and extremity, it persisted for over a minute. Hyperpathia was also present by pin-pricking and scratching the back, the shoulder and the front of the chest. These noxious stimuli aggravated the pain and also referred pain to the left arm. It was also

aggravated by a firm hand grip over the left arm or forearm.

Diagnostic imaging and laboratory work-up

Recent radiographic examination of the left shoulder and cervical spine revealed moderate osteoarthritis and degenerative disc disease with no definitive bony encroachment of the intervertebral foramina. A mild anterior compression fracture of the C6 vertebral body of long standing was noted. A bone scan investigation did not reveal the presence of metastases. However, increased uptake was noted throughout the cervical, thoracic and lumbar spinal columns, shoulder joints, distal right ulna, hands, knee joints, ankle joints and feet. The increased uptake was consistent with osteoarthritis. Blood work revealed a high erythrocyte sedimentation rate of 71 mm/hr.

Clinical impression

The features of this pain were consistent with a neuropathic type pain most likely arising from a stroke incident. The widespread nature of the pain on the left side of the body suggested "thalamic syndrome".

Differential diagnoses

Other conditions were considered, and indeed had been considered by the referring rheumatologist. The patient had an elevated ESR of 71 mm/hr but did not meet diagnostic criteria for polymyalgia rheumatica; proximal muscular pain and morning stiffness/weakness. Additionally, the pain was unilateral despite onset in 1988 (and as it turned out later, responded to a low dose of anticonvulsant). ESR is a screening test with false positives and negatives, and increases with age, female sex, various inflammatory conditions, and in anemia, and by itself is not confirmation of systemic disease. Blood work did not support the probable diagnoses of bony metastatic disease or liver, kidney, or hematological disease.

Whole-body bone scan had demonstrated multiple uptake in cervical, thoracic and lumbar spinal columns, shoulders, bases of metacarpals, distal right ulna, knees, ankles, and both feet. The bone scan findings were interpreted as due to osteoarthritis which was consistent with the clinical findings, and inconsistent with rheumatoid arthritis or bony metastatic diseases. However, osteoarthritis could not explain the specific localization of left upper body pain and sensitivity, nor the neuropathic quality of

pain. Reflex sympathetic dystrophy was not a possibility, given the complete lack of neurovascular changes, dystrophy, sweating, coolness, or visible changes in the distal limb. Postherpetic neuralgia of such a distribution would require a history of typical lesions, and would be accompanied by depigmented scars, which were not present.

Discussion

Chiropractors should be vigilant regarding pain experienced by the elderly, especially if the pain is of a widespread and diffuse nature and is accompanied by neuropathic features. In this case, the plain film radiography and bone scan results can inappropriately lead one to conclude that the pain is related solely to osteoarthritis. As a result, a diagnosis of "osteoarthritis only" could then be erroneously made.

There are three points that this case report illustrates. First, the above case illustrates the fact that a patient initially suffering from musculoskeletal pain may have some underlying pathology other than or in combination with musculoskeletal pain at a later presentation. Second, pain of neuropathic origin may be evident on a musculoskeletal examination and masquerade as musculoskeletal pain (since all tissue subserved by the neuropathic afflicted area will hurt). Third, one should complement a musculoskeletal examination by conducting a neurological screening examination that will help determine whether or not there are characteristics indicative of neuropathic origin pain.

Thalamic Pain Syndrome or Central Post-Stroke Pain is a term that is in common currency to describe widespread pain of central nervous system origin. However, thalamic pain is not always attributed to a lesion within the thalamus and its nuclei. Sites of involvement can also include lesions within the dorsal horn, ascending pathways of the spinal cord, brainstem, subcortical white matter and cerebral cortex.¹ As a result, Central Post-Stroke Pain (CPSP) is also a commonly used term to describe pain of central origin arising from lesions of the central nervous system as a result of cerebrovascular accidents.

Strokes afflicting the central nervous system are one of the most common causes of central pain, followed by multiple sclerosis, spinal cord injuries, traumatic brain injuries, syringomyelia, and other neurological disorders.²⁻¹¹ About 85% of all central post-stroke pain (CPSP) can be attributed to infarcts.² In a prospective study, the incidence of central pain occurred in as many as 8% of all stroke

patients during the first year after experiencing a stroke. Within that group of stroke patients, 63% experienced pain within one month after onset of the stroke.¹⁰ The preceding investigators were not aware of any subjects that might have resolved spontaneously. Additionally, the study did not address treatment regimens.

In Canada the stroke incidence is estimated to be 138 per 100 000 people.¹² In a Statistics Canada survey of residents in health care institutions, the prevalence of stroke was estimated to be 4% for those aged 65 or older living in households and 22% for those living in institutions.¹³

Within the stroke population, there are many possible disabilities that can involve cognitive impairments, language difficulties, motor deficits, emotional changes and other related disturbances. It can easily be conceived that pain is often forgotten and overlooked by the relatives and physicians of these patients.¹⁴ Since strokes contribute to disability within industrialized countries, it is important to determine whether patients are experiencing neurological difficulties associated with post-stroke pain.

Patients experiencing cerebrovascular lesions from strokes can experience pain immediately after the event, or experience a few years delay between stroke and onset of pain.^{11,15} The pain experienced can develop initially as a sensory disturbance and muscle weakness or impairment and can then improve.¹⁶ However, the pain may often remain permanently, with varying intensities.¹⁷ In a patient with central post-stroke pain, there are many external and internal events that can evoke pain for extended periods of time. These events include cutaneous stimuli (wearing clothes), body movements (changes in body position, walking, extremity movements), visceral stimuli (full rectum or urinary bladder), loud noise, bright light, emotions and changes of mood.^{2,15}

Additionally, the area of pain in CPSP patients is often diffuse in location.² Many stroke patients can exhibit extensive pain afflicting the whole left or right side of the body or a body quadrant such as the body on one side except the face, or the arm and/or the leg on one side, or the face on one side and the extremities on the other side, or exclusively the face.² Pain can also be restricted to smaller areas of the body such as the hand.¹⁵

There may also be several differing qualities of pain exhibited simultaneously. A wide variation in quality of pain is reported from patient to patient, even with those patients sharing the same lesions and diagnosis.¹⁵ Quali-

ties such as burning, aching, lancinating, pricking, lacerating and pressing are commonly reported.¹⁸

There are many hypotheses that are invoked to help explain CPSP. A commonly held theory is that central pain arises due to lesions within the medial lemniscal pathway. This results in the prevention of this pathway from providing tonic inhibition upon the spinothalamic projection zones responsible for pain and temperature.²

However, many patients with central pain exhibit normal tactile and vibration sensibility. As a result, it is thought that lesions within the spinothalamic pathways are usually crucial for the development of central pain.^{2,19-21} There are many studies that support the hypothesis that the spinothalamic deficit is a necessary prerequisite for central pain.^{10,21} However, this explanation is not entirely complete. A deficit in this tract (indicated by a disturbance or loss of thermal sensation), can be found in greater than half of stroke patients, however these same patients do not necessarily experience pain.¹⁰ As a result, it is difficult to predict the development of central pain syndrome based upon sensory deficit.¹⁴

The hypothesis remaining is that central pain is not due to a particular characteristic mechanism but a multifaceted phenomenon. There may be several mechanisms at work that may contribute to central pain. There may be plastic changes to the spared sensory fibres, the secondary pathways, and spinothalamic pathways. Alternatively, there may also be a loss of specific mechanisms which are part of the inhibitory process for the central nociceptive systems. These changes could lead to an alteration of central sensory nuclei that results in abnormal firing patterns (central sensitization) which are interpreted as pain.^{2,21}

At present, there is no pathognomonic quality of pain associated with a specific mechanism causing the central pain. Additionally, central pain may present as either continuous, paroxysmal and spontaneous, or as paroxysms abnormally evoked. The pain can also be described as superficial and/or deep.¹⁸ In some cases, the common primary feature accompanying pain is a partial or complete loss of afferent sensory information with the additional presence of hyperalgesic and hyperpathic phenomena within the painful area. Some patients may exhibit gross sensory deficits while others may exhibit subtle sensory deficits.¹⁴ Sensory loss can involve all the sensibilities, with the spinothalamic functions (cold, warmth, pinprick) being the most common modalities that are impacted.¹⁵

However, a clinical sensory deficit is not a necessary accompanying feature for pain to occur after a stroke.²²

There are several characteristics found in CPSP patients exhibiting pain. Simple bed-side examination techniques can be utilized to elucidate these neuropathic characteristics. A classic feature is the presence of abnormally evoked pain known as allodynia.^{14,18} Allodynia refers to the evocation of pain through applications of non-noxious stimuli. Non-noxious stimuli may include light, mechanical or thermal (cold or heat) stimuli.¹⁴ The presence of allodynia can be determined clinically by lightly stroking with your fingertips or by gently squeezing the patient's region of complaint.^{14,23} If allodynia is present, then the patient will experience abnormal amounts of pain within the region being tested.

A second characteristic is the presence of hyperalgesia. Hyperalgesia is an exaggerated response to normally painful stimuli.¹⁴ Hyperalgesia can be determined clinically by applying pin strokes to the region of complaint. These pin strokes should cause some discomfort of pain. If an exaggerated amount of pain is experienced, then the area of complaint is considered to be hyperalgesic to pin stroking.²³

A third characteristic is the presence of hyperpathia. Hyperpathia arises when explosive cutaneous pain is experienced locally (and possibly also radiated elsewhere in the body) as a result of noxious or non-noxious stimuli.^{23,24} The presence of hyperpathia can be determined by repeatedly lightly tapping a cutaneous region with a pin in one spot. While testing the area of complaint, one should ask the patient "Does the pain travel elsewhere?", "Does the pain worsen over time?", "Does the pain continue after I stop touching you?", "What do the sensations feel like?". If there is pain present and it travels distally or proximally, and/or the pain worsens over time (the "windup effect"), and/or the pain continues after stimulation, then it is suggestive that hyperpathia is present.²³ Hyperpathic pain can continue long after the stimuli have been discontinued. These phenomena are indicative that a neuropathic process is occurring which involves the central nervous system.

Treatment

This patient had been referred to a medical specialist at a pain clinic. Despite repeated hospitalizations and workups over 10 years, the neuropathic factors in the pain had not

been recognized because of the coexisting musculoskeletal and medical problems. A chiropractic resident in the clinic participated in the clinical bedside examination which finally defined this neurological component. By recognizing the neurological component in the diagnosis, it was possible for the attending physician to prescribe carbamazepine, an anticonvulsant which has properties of reducing neuropathic pain. Fortunately in this patient's case, this treatment was very effective against the pain.

Other pharmacological treatments which might have been considered for chronic post-stroke pain include other anticonvulsants, such as amitriptyline, or mexiletine. However, central neuropathic pains of such chronic duration are more often incompletely responsive to pharmacological treatment, and residual muscular aching and reduced function would be common sequelae. In such a case, management of the stiffness and aching and improving spine and limb function, offer good opportunities for the chiropractic clinician to provide further relief and correction of disability. The role of the chiropractor in correct diagnosis, alerting the family physician, continued chiropractic support of the patient in a primary care role, and following up with application of musculoskeletal treatment for residual discomfort, can benefit patients who might otherwise continue to suffer pain and dysfunction.

Conclusion

Central post-stroke pain is present within the stroke population. As members of the health care profession, it is incumbent upon us to recognize the common features of Central Post-Stroke Pain (CPSP). These features are identified through patients' past histories and other clinical features such as allodynia, hyperalgesia or hyperpathia which are suggestive of central origin pain.

This case report has illustrated the fact that a patient initially suffering from musculoskeletal pain may present with another painful disorder other than or in combination with musculoskeletal pain, at a later time. Second, pain of neuropathic origin may be evident on musculoskeletal examination and masquerade as musculoskeletal pain since all tissue subserved by the neuropathic afflicted area will hurt. Finally, a neurological screening examination (implementing the aforementioned bedside techniques) should always complement the musculoskeletal examination for widespread or diffuse pain, to elucidate whether the pain is of neuropathic origin or due to centralized pain.

Once pain of central origin is identified, appropriate care involving other health care professionals can then be initiated which will ultimately benefit the patient.

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