CASE REPORT

Concurrent Peripheral Pathologies and Complex Regional Pain Syndrome Type 1 as Contributors to Acute Post-Stroke Shoulder Pain: A Case Report

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Abstract- Post-stroke shoulder pain is associated with either a peripheral or central pathology. However, most of the time, it is challenging to establish a cause-and-effect relationship between the suggested pathology and shoulder pain reported. We report a 66-year-old man who developed a right hemiplegic shoulder pain two months post stroke with initial investigations suggestive of peripheral pathologies. Pharmacological and non-pharmacological treatment did not improve his shoulder pain. Later he developed complex regional pain syndrome (CRPS) of the right hand and the initial shoulder pain subsequently relieved following resolution of the CRPS.

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Keywords: Stroke; Shoulder pain; Complex regional pain syndrome

Introduction

Shoulder pain involving the hemiplegic arm is a common complication post stroke and the etiology remains poorly understood. Its prevalence ranges between 5% and 84%, (1) with 17% of patients experienced shoulder pain within the first week (2), 55% within two weeks (3), 87% within two months (3), and 75% within the first year after stroke (4). Both central neuropathic and peripheral nociceptive mechanisms have been suggested in the pathophysiology of post stroke shoulder pain, with central neuropathic mechanisms thought to play a larger role in the chronic stage than previously assumed (5).

Shoulder pain is more frequent in subjects with complex regional pain syndrome (CRPS) (6), limited shoulder external rotation range of motion (7), subluxation (3,6), spasticity (3), impingement (7), biceps tendonitis (7), and adhesive capsulitis (7). However, it is always difficult to establish a cause-and-effect relationship between the suggested factors and shoulder pain reported. Treatment of the associated factors may not necessarily reduce the post stroke shoulder pain. Nevertheless, treatment is still warranted if it has an association with motor impairment, activity limitation, quality of life or prolongation of the stroke rehabilitation process.

We report a case of acute post stroke shoulder pain which was initially thought to cause by shoulder pathologies, but later had an association with CRPS of the hand. Resolution of the CRPS has caused reduction of the initial shoulder pain and improvement of the upper limb function.

Case Report

A 66-year-old man was recently diagnosed to have stroke when he presented clinically with progressive slurring speech and right body side weakness. CT scan of the brain within 48 hours showed hypodensities in the left thalamus, right cerebral peduncle and both sides of pons.

On examination, the patient's vital signs were stable and there was dysarthria with upper motor neuron palsy of right 7th cranial nerve. He had right hemiplegia with an increased tone and hyperreflexia of the right upper and lower limbs, together with an up going plantar response. The muscle power of both upper and lower limb was generally 3) according to Medical Research Council (MRC) grade, except for wrist flexors and extensors, finger flexors and ankle plantar and dorsiflexors with MRC grade 1). Sensation to pin prick was normal.

After the first month, there was a considerable increased of muscle strength (4 proximally and 3

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distally) of the right upper and lower extremities. Mild spasticity was noted on his right shoulder and elbow (Modified Ashworth Scale grade 1). He also showed gradual improvement in his activities of daily living (ADL) and mobility.

The patient started to complain of the right shoulder pain on the second month after stroke. It has an insidious onset and there was no history of trauma or fall. The pain was described as dull, gnawing or cramping and worse in the evening. It most commonly occurred when the shoulder or arm was under physical exertion or load. Lying on the stroke-affected side of the body would also exacerbate the pain. Pain score according to visual analog scale (VAS) was 10.

Shoulder examination did not reveal any signs of acute inflammation, shoulder subluxation or nerve injury. The Speed and Yergason tests were positive. An ultrasound study of the right shoulder revealed synovitis of the long head of biceps with very small linear tear of the anterior slip of supraspinatus tendon, but there was no impingement shown. Non-steroidal anti-inflammatory drugs (NSAIDs) were prescribed to treat the pain and relative immobilization of the shoulder joint was advised. Despite the treatment, his pain score was still the same.

Two weeks after the initial treatment, he noticed that his right hand started to swell, became dry and had decreased range of motion. His right shoulder was also getting more difficult to move and the shoulder pain then radiated down to the hand. ADL involving the right upper limb became affected. Examination revealed significant hand edema, dryness, discoloration, reduced temperature and it was allodynic to mechanical and cold stimuli (Figure 1). There was also point tenderness over the right metacarpals and right elbow. CRPS type 1 was main diagnosis. He was started on prednisolone 15 mg daily, gabapentin 900 mg three times a day (*tds*) and acetaminophen 1 g *tds*. Transcutaneous electrical nerve stimulation was also started.

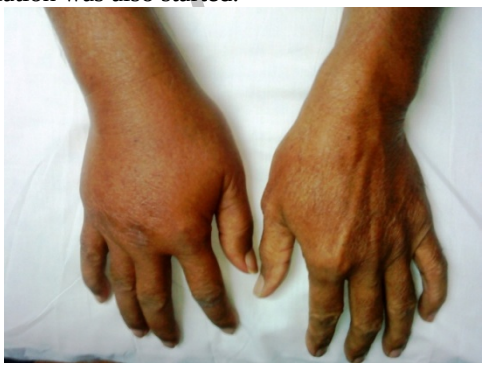

Figure 1. CRPS type 1 with right hand oedema and skin discoloration.

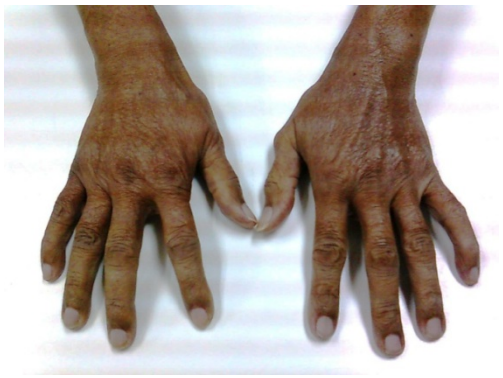

Figure 2. Resolution of right hand oedema and skin discoloration after two months.

Significant improvement was noted after two months with reduction of hand oedema, improvement in hand skin colour and range of motion (Figure 2). There was also improvement in shoulder range of motion and shoulder pain with the VAS pain score reduced from 10 to 3.

Discussion

Shoulder pain is one of the most common disabling complications in stroke survivors. As previously mentioned in this context, mechanism of post-stroke shoulder pain may follow a central pain or nociceptive pathophysiology. It is difficult to determine the exact role of these mechanisms in the contribution of shoulder pain especially in the acute stage. Most studies first evaluate the shoulder pain in later stage, weeks after initiation of stroke rehabilitation. By then, the primary cause may be obscured by common secondary complications such as contracture or trauma (8).

CRPS is an uncommon but significant contributor of post-stroke shoulder pain but a cause-and-effect relationship between shoulder pain and CRPS was difficult to evaluate. This relationship is even more difficult to assess if there are concurrent pathologies affecting the shoulder. In this patient, the clinical and radiological evidences of the long head biceps tendonitis and synovitis with small supraspinatus tendon tear suggested nociceptive pathologies of shoulder pain, but the thalamic involvement together with the occurrence of CRPS suggested a central pathology.

Treatment of CRPS took precedence in this patient with goals of peripheral and central desensitization using a multimodal management when there was no improvement of the initial shoulder pain. Targeting the central element has proven beneficial to reduce the shoulder pain initially caused by biceps synovitis and

tendonitis. This is evidenced by the marked reduction (70%) of shoulder VAS pain score.

In conclusion, we would like to highlight that the cause of post-stroke shoulder pain can be multifactorial. Failure to reduce the pain score with initial treatment may suggest the involvement of other pathologies. In this case, treatment of associated factor such as CRPS causes a reduction of shoulder pain in the presence of other peripheral nociceptive pathologies. Therefore, attempt for an early identification and treatment of associated central pathology such as CRPS in the presence of a peripheral pathology is warranted in post stroke shoulder pain.

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