Shoulder pain after stroke: case report and review

A 65 year old man with multiple vascular risk factors was admitted after a stroke. Five days later, he complained of left shoulder pain. This was a dull ache, worse on movement and on touching the shoulder. He also voluntarily described a second different pain, as a “clawing at the flesh” that was constant. Abnormalities on examination were a global decrease in the range of left shoulder movement, dysarthria, left pyramidal weakness, left hemisensory diminished pinprick sensation and decreased sensation to touch and temperature in the left upper limb. “Referred” allodynic pain was experienced in the shoulder joint, when the skin over the left scapula was touched. A left shoulder radiograph was normal. Central post stroke pain (CPSP) was diagnosed and amitriptyline 25 mg nightly was started. Adhesive capsulitis could not be excluded and paracetamol as required was added. He was rehabilitated and discharged while taking amitriptyline at day 17. His shoulder symptoms had improved but not resolved. Subsequently, despite worsening pain, the patient did not seek help until three weeks after discharge. There was a global decrease in all shoulder movements and passive abduction only possible to 60°. The previous sensory abnormalities were still present. A diagnosis of frozen shoulder in conjunction with CPSP was made. Magnetic resonance imaging showed no local shoulder abnormality but multiple discrete infarcts in the white matter of the brain, both thalamus, left parietal lobe and the pons (fig 1). The amitriptyline was increased to 50 mg every day and the shoulder injected with corticosteroid. The pain and sensory changes improved and had resolved by four months with full range of shoulder movement.

This case illustrates some interesting clinical features of shoulder pain after stroke. We also describe a phenomenon of “referred” allodynia, which to our knowledge has not been previously described.

Our patient exhibited classic symptoms and signs of central pain, including abnormalities of temperature sensation, which are a universal feature and allodynia, which is pathognomonic. Allodynia is the elicitation of pain from a normally non-painful stimulus. We could find no previously reported cases of referred allodynia within a joint as a result of somatic stimulation and believe this to be the first described. CPSP however, is well documented. A recent study showed that 8% of stroke patients suffer limb pain attributable to CPSP. The current only evidence-based treatment is amitriptyline, to which our patient responded. Magnetic resonance imaging demonstrated a number of infarcts, including in the brain stem, the left and right thalamus. Infarcts in all these areas have been associated with CPSP.

In addition, he developed symptoms suggestive of adhesive capsulitis. This is more common after stroke but the pathophysiology is not understood. He improved after increasing the dose of amitriptyline and the corticosteroid injection. Thus, although this patient seemed to have two distinct types of pain, it is certainly possible that the peripheral pathology to some extent “drove” the post stroke central pain.

The true incidence of post stroke shoulder pain is unknown but was documented in a “stroke complications” audit as 4%. Our ongoing work suggests this is an underestimate. The causes are heterogeneous but can be classified into regional (for example, peripheral soft tissue, exacerbation of arthritis, trauma), central (CPSP and complex regional pain syndrome) or mixed (peripheral and central). Our work suggests the latter is under recognised. The roles of shoulder subluxation, poor handling techniques, abnormal tone and capsular contracture have all been implicated but remain unvalidated.

To conclude, physicians need to be aware of the potential different causes of shoulder pain after stroke and to provide prompt and appropriate treatment. Further research is needed into epidemiology and causation to maximise established and future treatments.

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