CASE REPORT

Brachial neuritis following a corticosteroid injection

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SUMMARY

This report presents a case of brachial neuritis following a subacromial corticosteroid injection. The patient developed an anterior interosseous neuropathy shortly after the injection, with no other trigger being identified. This neuropathy has unfortunately not shown any sign of recovery at 2 years. The authors propose that corticosteroid injection be added to the list of possible triggering events of brachial neuritis and highlight the frequent use of oral corticosteroids in its treatment. (1) The injection of local anaesthetic and corticosteroid should be considered as a potential trigger for brachial neuritis. (2) Brachial neuritis should be considered in the differential diagnosis for patients presenting with severe arm pain and weakness. (3) The nerves originating from the upper trunk of the brachial plexus are most commonly affected. (4) The anterior interosseous nerve is involved in one-third of cases.

BACKGROUND

Brachial neuritis is a relatively rare condition which frequently presents with severe shoulder pain, followed by variable patterns of peripheral neuropathy. Because of this variable presentation, the diagnosis is often delayed. Numerous triggers have been identified and commonly involve either a physical or immunological insult to the body. A significant proportion of cases present with no identifiable trigger. This report describes a case of brachial neuritis and proposes a preceding corticosteroid injection as a triggering factor. This has not previously been reported in the literature and is not a documented adverse effect of corticosteroid injections.

CASE PRESENTATION

A 37-year-old right hand dominant male mechanic presented to a sports medicine clinic with a 6-month history of worsening pain in his dominant right shoulder. Ten years previously he had suffered a traumatic dislocation of this shoulder, followed by a surgical stabilisation and labral repair. He had made a full recovery and had largely been asymptomatic. The patient had received a subacromial injection of corticosteroid and local anaesthetic into this shoulder from his general practitioner 4 weeks previously, with minimal symptom relief. Two days following this injection he had suddenly developed severe pain, felt diffusely throughout his uninjured (contralateral) left shoulder and along the medial side of both his upper and lower arm. This had gradually subsided over 4–5 days but at this point he began noticing difficulty using his left thumb and index finger. This was especially evident with dexterous tasks at work. The patient was

systemically well, had not been ill in the preceding few weeks and had no signs or symptoms to suggest an underlying malignancy. He did occasionally suffer migraines but did not take any regular medication. His father and paternal aunt have Parkinson's disease.

Further examination of his left arm revealed normal neurology apart from isolated weakness of the distal interphalangeal joints of his thumb, index finger and, to a lesser extent, his middle finger ([figure 1](#)). There was no sensory loss. His cervical spine examination was unremarkable. This pattern suggested an isolated lesion of the anterior interosseous nerve (AIN). The preceding severe diffuse shoulder pain was consistent with brachial neuritis, proposed to be secondary to the subacromial corticosteroid injection.

DIFFERENTIAL DIAGNOSIS

After a period of observation the patient was referred to a neurologist. At this point his symptoms had been present for 3 months and had shown no sign of improving. It was agreed that the isolated AIN lesion was likely to be secondary to brachial neuritis. Nerve conduction studies and electromyography, performed a year after the onset of symptoms, confirmed this isolated nerve lesion,

Figure 1 An inability to flex the interphalangeal joint of the left thumb.

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with normal sensory potentials throughout the left arm but persistent defibrillation potentials and no motor unit activity in flexor pollicis longus. More proximally supplied muscles showed evidence of chronic denervation with re-innervation and no active fibrillation potentials. The possibility of a structural lesion compromising the AIN was investigated with MRI of the forearm. This revealed no oedema or mass in the region of the nerve and no fibrous tissue thickening. Fatty infiltration was observed in the atrophied flexor pollicis longus and in the radial half of flexor digitorum profundus.

It is the authors' opinion that this case of brachial neuritis may have been triggered by a corticosteroid injection. Other documented triggers of brachial neuritis include infection, immunisation, trauma, an immune-mediated inflammation, tumour infiltration and radiotherapy. There was no evidence of any of these precipitants in this case. Brachial neuritis can also occur without an identified trigger and this must also be considered in this example.

TREATMENT

This patient was managed with observation and was encouraged to continue with his normal activities as much as possible. No pain relief was required, as his pain had resolved at the time of consultation. Regular follow-up appointments were made, in order to assess his progress.

OUTCOME AND FOLLOW-UP

At the time of writing, the patient had not experienced any recovery of the strength of his thumb, index and middle fingers. He continues to find dexterous tasks difficult and has some problems at work. MRI was performed 2 years after the initial onset of his symptoms and its findings of persistent denervation changes in the affected muscles, as well as a lack of inflammatory changes in the vicinity of the AIN, indicate a quiescent nerve with little hope of recovery.

DISCUSSION

Brachial neuritis is also referred to as neuralgic amyotrophy (NA) or Parsonage-Turner syndrome. Its idiopathic form (INA) has an incidence of 2–4/100 000, whereas its more severe, recurrent, hereditary form (HNA) is much rarer.¹ For the sake of this case report we will concentrate on INA. The median age of onset is early in the fourth decade, although it can commonly occur between the third and seventh decades.^{1–2} Males are affected more commonly than females, with a ratio of over 2:1 reported in some series.^{3–4}

The condition may present in a wide variety of ways, however manifests in the vast majority as a sudden onset of severe, unremitting neuropathic pain in the shoulder and upper arm. This is often worse at night. The symptoms are generally unilateral, with no difference with respect to hand dominance.^{2–5} A report on 246 patients, diagnosed with both INA and HNA, revealed a mean duration of this initial severe pain of around 4 weeks, although 4.9% and 22.7% experienced resolution within 48 h and 1–7 days, respectively.⁴ These authors also described three stages of pain throughout the course of the condition: the first is a severe neuropathic pain, the second a mechanosensitivity of the affected nerves and the third a musculoskeletal pain secondary to altered joint movement patterns as a result of ongoing nerve palsy.

Within the first week of painful symptoms (within 24 h in 33% of patients), a gradual paresis of individual nerves branching from the brachial plexus begins.⁴ The pattern of paresis is highly variable and may be patchy or confined to a single

peripheral nerve.⁶ Occasionally nerves not originating from the plexus may be involved; however, the upper branches of the plexus are more commonly affected: the long thoracic, suprascapular and axillary nerves.^{4–5} Winging of the scapula is a common examination finding, being observed in up to 66% of cases.⁷ While it was initially thought to only affect motor nerves, over 75% of cases have now been shown to have sensory involvement, of which hypoesthesia is most common.^{4–5}

The anterior interosseous nerve is a branch of the median nerve in the forearm. It provides motor function to flexor pollicis longus, to the index and middle finger portions of flexor digitorum profundus and to pronator quadratus. It also provides sensory input from the distal radioulnar, wrist and intercarpal joints. It is affected in 1/3 of brachial neuritis cases, however an isolated AIN palsy is less common and was first recognised, and eponymously named, by Kiloh and Nevin⁸ in 1952.⁴ Patients present with weakness of flexion of the thumb interphalangeal joint, the distal interphalangeal joints of the index and middle fingers and with weakness of forearm pronation.

The pathophysiology and aetiology of INA is uncertain. It is believed to be a combination of a genetic predisposition to peripheral nerve dysfunction, an immunogenic trigger and possibly a biomechanical insult.⁷ In this case we believe that the injection of corticosteroid served as such a trigger. As opposed to HNA, no genetic mutation has yet been found to be associated with INA. Over 50% of patients report an antecedent immunological or physical insult in the days or weeks prior to the onset of symptoms.^{5–7} The insults described include infection (of which, viral infection is most common) and trauma to any body part.⁵ Documented iatrogenic causes include vaccination, surgery, anaesthesia, radiation therapy, lumbar puncture and radiological contrast dye administration.^{5–9} There is no reference in the literature to suggest either injected local anaesthetic or corticosteroid as a possible trigger for INA. It is, however, interesting to note that intravenous hydrocortisone was coadministered with the radiological dye in the single case report implicating its meglumine component.⁹

This case raises the possibility that a subacromial corticosteroid and local anaesthetic injection may act as a trigger for INA. The mechanism of action may only be speculated on; however, a possible pathway may be via a transient immunosuppression, allowing a subclinical or dormant viral infection to affect the brachial plexus. This is an interesting proposal, as high-dose oral corticosteroids are often used as treatment for acute NA, despite there not being any high-level evidence to support its use.¹ It may be that, following an initial trigger, subsequent administration of corticosteroid serves to control the florid autoimmune response.

As mentioned, corticosteroids are often used to treat the pain associated with brachial neuritis. The evidence supporting this treatment is very limited. A report on 99 patients concluded that neither oral nor intra-articular corticosteroid significantly altered the course of the condition.³ Some patients, however, did report pain relief following treatment. In a larger non-randomised study, oral prednisone was observed to reduce the time until recovery of paresis started and had higher rates of 'full' and 'good' recovery at 1 year when compared with non-prednisone treatment (12% vs 1% and 44% vs 10.7%, respectively).⁴ The most effective treatment for pain control was observed to be a slow-release non-steroidal anti-inflammatory, combined with a slow-release opiate.⁴ Recently, individual reports of rapid recovery from NA following treatment with intravenous immunoglobulins have been published.^{10–11}

An awareness of the possible triggering factors and variable clinical presentation may hasten diagnosis when patients present to medical practitioners, as a median time to diagnosis of 10.5 weeks has been reported. Whereas it was previously thought that the majority of patients recovered within 6–18 months,² mild paresis has been seen to persist beyond 3 years in 69.4% of cases.⁴ Van Alfen⁴ found that 21.7% of patients reported less than 50% functional recovery, 36.8% had been forced to find a different job and 22.3% were unable to work. A recurrence rate of 26.1% was reported in INA patients over an average follow-up period of 6 years, with symptoms affecting either the same or, more commonly, a different peripheral nerve distribution.

Learning points

- ▶ The injection of local anaesthetic and corticosteroid should be considered as a potential trigger for brachial neuritis.
- ▶ Brachial neuritis should be considered in the differential diagnosis for patients presenting with severe arm pain and weakness.
- ▶ The nerves originating from the upper trunk of the brachial plexus are most commonly affected.
- ▶ The anterior interosseous nerve is involved in one-third of cases.

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Competing interests None.

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