Iatrogenic postoperative brachial plexus compression secondary to hypertrophic non-union of a clavicle fracture

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ABSTRACT

The brachial plexus is related intimately to the clavicle such that injury can occur primarily and most commonly at the time of trauma through traction or it can occur secondarily, mainly owing to hypertrophic non-union with exuberant callus formation, causing compression of the plexus. The movement-dependent rearrangement of the subclavicular space is restricted with rigid internal fixation, thereby placing inappropriate pressure on the plexus from the deep hypertrophic tissue. This case highlights another cause of brachial plexopathy of which to be aware.

KEYWORDS

Clavicle – Fracture – Non-union – Hypertrophic – Brachial plexus – Injury – Delayed – Iatrogenic

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Clavicle fractures account for up 4% of all fractures.⁸ Of these, the middle third position is the most frequent point of fracture, representing 69–82% of cases.⁵ Conservative management is fairly predictable in these fractures with a non-union rate of 14.5%.^{10,15} The brachial plexus is related intimately to the clavicle (Fig 1) such that injury can occur primarily and most commonly at the time of trauma through traction or it can occur secondarily, mainly owing to hypertrophic non-union with exuberant callus formation, causing compression of the plexus. There is a 1.8% incidence of primary brachial plexus injury following a clavicle fracture.¹⁹ There are only 30 described cases of secondary plexus injury due to hypertrophic callus in the literature.^{1,2,5,4,6,7,9,11,12,15,14,15,17,18} We add a further case but describe the first case of iatrogenic brachial plexus compression from a hypertrophic non-union of clavicle with no preceding neurology.

Case history

A 43-year-old, non-smoking housewife presented to the emergency department with a right midshaft minimally displaced clavicle fracture (Fig 2) with no neurovascular impairment. The mode of injury was a simple mechanical fall on the kerb of a pavement. The mechanism was direct impact on to the proximal humerus, causing a transmitted force on to the clavicle shaft. This was managed conservatively in a broad arm sling.

The patient persisted to have pain around the fracture site and on movement of the arm over the coming

months. Radiography taken four months later revealed exuberant callus formation and a hypertrophic non-union (Fig 3). Computed tomography (CT) was not carried out as the history, examination and plain radiography gave enough clinical suspicion that this was a non-union. There was no resolution to her pain so open reduction internal fixation

Figure 1 Illustration of the intimate relationship of the brachial plexus and the clavicle

Figure 2 Initial radiography displaying middle third fracture of clavicle

Figure 3 Radiography at four months displaying evidence of hypertrophic non-union

Figure 4 Radiography following open reduction internal fixation with autograft from hypertrophic non-union

Figure 5 Post-exploration radiography with debridement of callus, removal of reconstruction plate and excision of middle third clavicle

with autograft from the hypertrophic callus site was carried out eight months following the fracture by the senior author (Fig 4).

Surgery was through a standard supraclavicular incision. The fracture site was identified with exuberant surrounding callus. This was enucleated from the clavicle but scar and deep hypertrophic tissue, particularly posterior and inferior, was left in situ owing to likely adhesions to the plexus. Fracture ends were mobilised and anatomical reduction achieved fixation with a reconstruction plate, using superior to inferior cortical screws. To protect the underlying plexus during plate fixation, a McDonald dissector was placed flush with the inferior surface of the clavicle. This protected the plexus from the drill, depth gauge and screws. Postoperatively, British Medical Research Council (MRC) grade 0/5 power from C5–T1 coupled with absent two-point discrimination in this distribution was observed. There was, however, MRC grade 3/5 power to wrist extension and two-point discrimination in the superficial radial nerve distribution, suggesting an incomplete plexus injury with sparing of the posterior cord.

Given that this paresis was acute and identified immediately postoperatively, our impression was towards an iatrogenic traction neurapraxia, caused when mobilising the fracture site for reduction. However, causes of immediate injury other than traction would also include compression due to haematoma, direct injury due to drill bit, long screws and dissection during mobilisation of fracture ends.

The patient was referred to the peripheral nerve injury unit at the Royal National Orthopaedic Hospital in Stan-

more. There the brachial plexus was explored and extensive callus compressing the plexus was identified. This was removed along with the reconstruction plate used for fixation and the middle third of the clavicle was also excised (Fig 5). A subsequent infraclavicular incision was made so that neurolysis of the plexus could be carried out. Intraoperative neurophysiology was performed after decompression, which found the lateral, posterior and medial cords to be working. Postoperatively, the patient has made an encouraging recovery with MRC grade 4/5 power from C5–T1 and a corresponding return of two-point discrimination.

Discussion

All cases described in the literature to date describe preoperative neurology as a result of plexus compression from hypertrophic callus. Our case is unique in that there was no neurology preoperatively so no emphasis was placed on removing the entire callus. The result was that at anatomical reduction of the fracture site, the scar and deep hypertrophic callus tissue engaged with the plexus, causing a motor and sensory paresis.

Although clavicle non-union and pseudarthrosis is relatively rare, it remains one of the most common reasons for the development of late or delayed brachial plexus paresis after a traumatic event.³ Secondary injuries to the brachial plexus may also be associated with manipulation of a fracture (causing traction injury of plexus), overshoot of the drill, long screws, expanding false aneurysm of the subclavian artery caused by fracture spike and haematoma collec-

tion.⁷ Infrequent causes for delayed brachial plexopathy are axillary tumour progression in breast cancer patients and radiation sequelae.⁵

To ascertain the cause of the plexus injury, imaging that could help with diagnosis would include radiography to assess for hypertrophic non-union and, if doubt still remained, then CT could be requested. These investigations would also help postoperatively to assess for screw length. Ultrasonography could also be used to assess for haematoma or subclavian aneurysm causing plexus compression. Furthermore, magnetic resonance imaging may have a role in defining the anatomy of the plexus preoperatively. Finally, if doubt remains as to whether this is indeed a plexus injury, nerve conduction studies could be carried out to assess for a more distal lesion. However, this would only serve to complement a thorough neurological clinical examination.

With movement of the shoulder joint, the subclavicular space undergoes considerable changes in its dimensions and geometry. This potential space can compensate efficiently for a certain amount of additional tissue (callus), which also takes part in the process of movement dependent rearrangement, the reason patients may remain asymptomatic for a certain period of time after clavicular non-union is diagnosed.⁷

The typical deformity of a midclavicular fracture is downward with posterior displacement of the lateral fragment (due to the weight of the shoulder), bringing it into direct contact with the neurovascular bundle. The medial fragment is elevated owing to the upward and backward pull of the sternocleidomastoid muscle. When malunion occurs in this position, the excess callus places pressure on the medial cord of the brachial plexus as this is where it consistently crosses the first rib.⁵ Treatment options for decompression of the brachial plexus secondary to hypertrophic callus formation include osteotomy, bone graft (autograft from hypertrophic site or vascularised fibula) and plate internal fixation. Partial clavicle excision, as in this case, or excision of hypertrophic callus alone have been reported.⁵

The functional role of the clavicle has long been debated. There are several facts that would support the concept of minimal function. Children with cleidocranial dysostosis (congenital absence of the clavicle) often function well. In fact, older vascular surgical references recommended excisional clavicular osteotomy as an approach to the subclavian vessels.²⁰ Nevertheless, there are some important biomechanical functions of the clavicle such as its function as a strut. Having this strut allows the shoulder to reach into cross-body and internal rotation positions without medial collapse. The strut function of the clavicle allows the thoracohumeral muscles to maintain their optimal working distance. The clavicle therefore increases the strength of shoulder girdle movements. Consequently, middle clavicle excision would result in reduced strength of shoulder girdle movements.²¹

The prognosis of brachial plexus decompression depends on several factors including the degree of plexus damage and time of management (latency of plexus damage), age and motivation of the patient. Early exploration in

a young and motivated patient shows excellent prognosis, even to the extent of complete recovery of all plexus elements.^{7,16}

Conclusions

Clavicle non-union surgery remains fairly successful and predictable. However, this case raises the question of whether deep hypertrophic tissue should be left in situ or removed. The movement dependant rearrangement of the subclavicular space is restricted with rigid internal fixation, thereby placing inappropriate pressure on the plexus from the deep hypertrophic tissue. This case highlights another cause of brachial plexopathy of which to be aware.

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