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Case Report

Acute brachial plexopathy secondary to surgical management of an atrophic nonunion of a clavicle fracture

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ABSTRACT

We report the case of a 28-year-old patient who developed acute transient brachial plexopathy secondary to surgical management of an atrophic clavicle nonunion. The treatment was conservative, with symptom resolution at 4 months after surgery. This is the first reported case with electromyographic and neuroconduction follow-up, demonstrating complete and spontaneous resolution of axonal damage. The limited number of cases reported in the scientific literature allows for exploring some underlying causes of the acute plexopathy depending on the nature of the non-union (hypertrophic or atrophic).

Introduction

The incidence of nonunion following conservative management of a midshaft clavicle fracture is 15% [1]. The percentage of symptomatic nonunion ranges from 30 % to 80% [1], with clinical manifestations usually being pain [5] or occasionally insidious neurological symptoms associated with compressive phenomena due to hypertrophic nonunion [2].

Surgical treatment of a symptomatic nonunion through open reduction and grafting is a procedure with satisfactory outcomes; however, the frequency of associated complications is close to 17 % [9], the vast majority related to cutaneous irritation from the hardware, superficial infections, and healing failure. Few case reports have been published on acute postsurgical brachial plexopathies following the treatment of a clavicle nonunion [3,7].

We report the case of a 28-year-old patient who developed acute transient brachial plexopathy secondary to surgical management of an atrophic clavicle nonunion. The treatment was conservative, with symptom resolution at 4 months after surgery. This is the first reported case with electromyographic and neuroconduction follow-up, demonstrating complete and spontaneous resolution of axonal damage. Patient Consent was obtained.

Report of the case

A 28-year-old male patient with no significant medical history presented two weeks after a sports-related trauma while playing soccer with a fracture of the midshaft of the left clavicle (Fig. 1). The fracture was managed conservatively. After five months of follow-up, no radiographic evidence of bone healing was observed, and the patient developed an atrophic nonunion (Fig. 2).

The patient presented with mechanical pain radiating to the anterior shoulder, worsening with abduction and internal rotation,

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without clinical signs of nerve compression. The patient underwent surgery to correct the nonunion through open reduction, osteosynthesis, and local bone graft application. The immediate postoperative period in the recovery room was uneventful.

However, 72 h after surgery, the patient returned with neuropathic pain and weakness along the radial and axillary nerve distribution (posterior cord) and the musculocutaneous nerve, with reduced superficial touch sensation in the C5 to T1 dermatomes. Additionally, there were vasomotor changes in the limb without expanding hematomas, and the radial and ulnar arterial pulses were well-preserved.

A magnetic resonance imaging (MRI) of the brachial plexus was performed to rule out space-occupying lesions as possible etiological causes of the nerve impairment (Fig. 3). However, no findings suggesting compression were observed. Therefore, a conservative therapeutic approach involving neuromodulator medications, regular clinical observation, and physical therapy was chosen.

After one week of follow-up, the vasomotor manifestations in the extremity had resolved. From the 4th week onward, the patient began to show partial improvement in neurological symptoms, which continued until the symptoms almost entirely resolved at four months (Fig. 4).

At two months post-surgery, electromyography with nerve conduction studies was performed to assess the location of the injury due to persistent neurological symptoms. The neurophysiological findings revealed an infraclavicular lesion of the brachial plexus involving structures of the posterior cord and the left musculocutaneous nerve, with axonal characteristics and subacute evolution. After 12 months of follow-up, a neuroconduction re assessment showed normal nerve conduction of the brachial plexus.

One year after the initial surgery, strength and sensitivity were fully restored to normal levels. X-ray images demonstrated appropriate fixation of the osteosynthesis material with complete fracture healing (Fig. 5).

Discussion

The incidence of brachial plexus involvement after surgical treatment of a non-union in a midshaft clavicle fracture is 2.8% [9]. The limited number of cases reported in the scientific literature allows for exploring some underlying causes, depending on the nature of the non-union (hypertrophic or atrophic).

In the case of hypertrophic non-union, the non-removal of the callus located on the posterior and inferior edge of the medial fragment of the clavicle, following reduction and osteosynthesis, can lead to compressive phenomena of the lateral and/or medial cord by obliterating the interval between the clavicle and the first rib [8]. Thavarajah et al [8] reported a case of a 49-year-old patient who developed a hypertrophic non-union of a simple midshaft clavicle fracture and underwent surgery (due to pain) for resection of the hypertrophic callus and osteosynthesis (they left the callus located posterior-inferiorly in situ due to its proximity to the plexus). In the immediate postoperative period, the patient developed neurological symptoms, including loss of sensation and strength in areas corresponding to the medial and lateral cord of the plexus. Subsequently, she underwent exploration of the plexus, revealing that the remaining callus was compressing the nerve cords. The patient underwent excision of the midshaft clavicle and neurolysis, leading to a satisfactory postoperative course with resolution of the neurological symptoms. The authors conclude that subclavicular space becomes restricted after osteosynthesis, exerting inappropriate pressure on the plexus due to deep hypertrophic tissue.

McGillvray et al. [6] reported a similar case, involving a 35-year-old patient who developed plexopathy in the immediate postoperative period following the management of a painful clavicle non-union with open reduction and internal fixation and bone grafting. They describe performing material extraction within the first 16 h, resulting in partial improvement of symptoms. Subsequently, an MRI of the plexus was conducted, documenting an osseous callus along the posterior and medial segment of the clavicle, compressing the nerve cords. In a second procedure, they explored the plexus and completely resected the callus. After 10 months of follow-up, the patient had completely resolved the neurological symptoms.

In the case of an atrophic or oligotrophic non-union, plexopathy is usually secondary to a traction phenomenon caused by elongation of the plexus while correcting the anatomical length of the clavicle. Johnson et al. [3] described the case of a 54-year-old patient

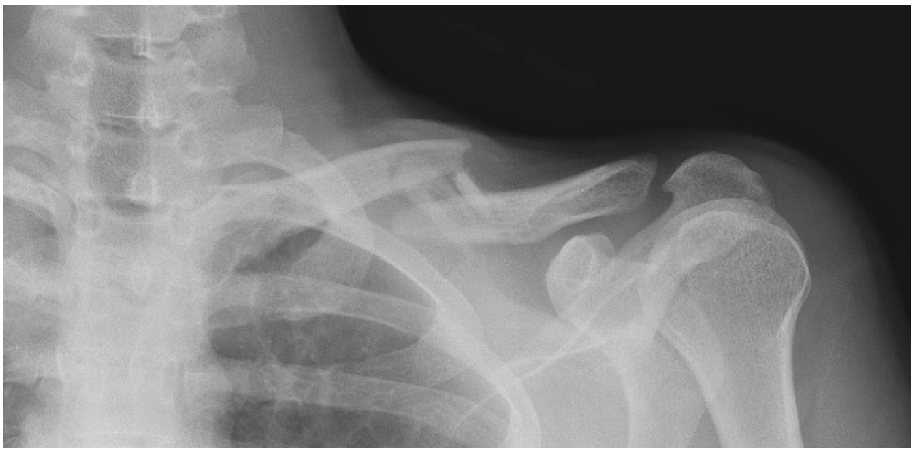

Fig. 1. Initial X-ray showing a midshaft fracture of the middle third of the clavicle.

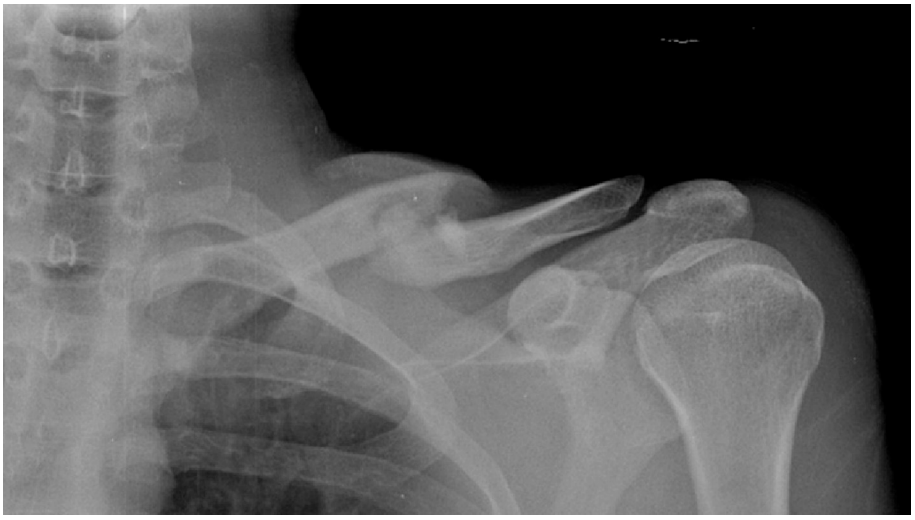

Fig. 2. X-ray at 5 months, showing an atrophic non-union of the fracture.

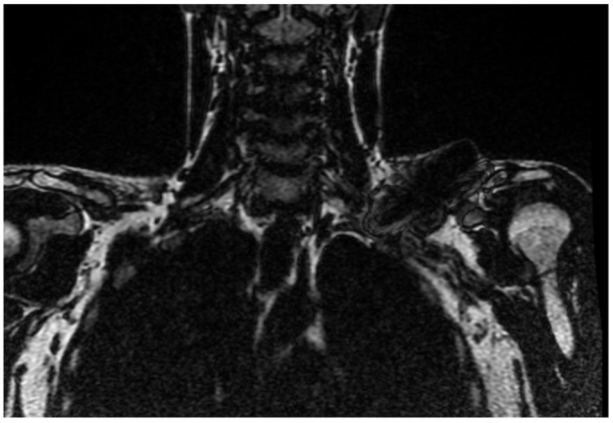

Fig. 3. MRI of the brachial plexus (T2-Weighted IDEAL Fast Recovery Fast Spin Echo). No evidence of space-occupying lesions with compressive effect on the brachial plexus.

with an atrophic non-union of a midshaft clavicle fracture. Within 48 h of the surgery (open reduction, fixation, and grafting), the patient developed paresthesias and progressive neurological symptoms. After ruling out space-occupying lesions near the plexus using tomography, it was decided to continue with clinical observation and physical rehabilitation. The neurological symptoms spontaneously resolved six months after the surgery. The authors concluded that the plexopathy was secondary to iatrogenic traction during clavicle lengthening. Like our case, the plexopathy did not present immediately after surgery (unlike acute plexopathies commonly seen in hypertrophic non-unions during the immediate postoperative period). The resolution time of neurological symptoms was within six months, and the treatment approach was conservative.

Matthews et al. [5] describe the case of a 68-year-old patient with an atrophic non-union, treated with open reduction and fixation, and the use of recombinant human bone morphogenetic protein (rhBMP-2). At 72 h after the surgery, the patient was readmitted due to weakness and paresthesias in the territory of the median, ulnar, and radial nerves. Conservative treatment was administered, and after 3 months, the strength began to improve. At 24 months, the patient was symptom-free. While the authors suggest a possible etiology related to rhBMP-2, based on findings from Margolis et al. [4], who documented axonal drop out in some nerves exposed to rhBMP-2 in an animal study, we do not see a clear causal relationship. From an anatomical perspective, it is unlikely that rhBMP-2 was placed directly over the plexus during the procedure. On the other hand, we believe that the case shares similar findings to those reported by Johnson et al. and ourselves (atrophic non-union, development of symptoms after 72 h, and spontaneous resolution of nerve injury). Therefore, we venture to suggest that the cause was not the recombinant protein but rather iatrogenic traction on the plexus during clavicle length restoration after osteosynthesis.

Indeed, it is also possible for a compressive effect to occur due to a third fragment, which, after reduction, compresses the brachial plexus [7]. This scenario resembles the etiological mechanism of compressive plexopathies seen in the treatment of hypertrophic non-unions and often requires immediate surgical intervention to alleviate the compression and its associated symptoms.

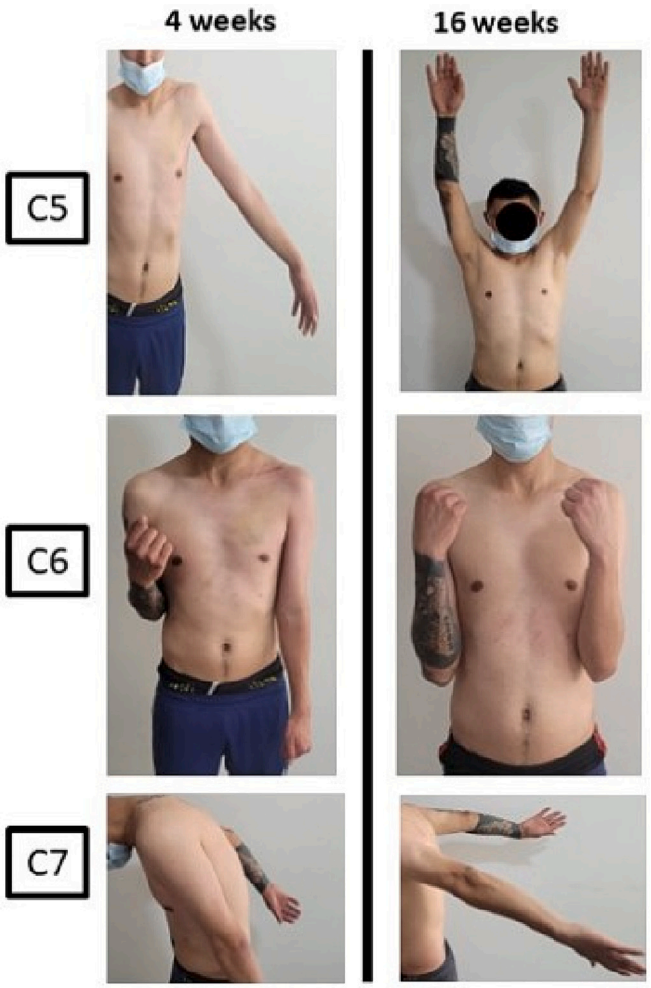

Fig. 4. Clinical evolution demonstrating neurological recovery of the affected fascicles after 4 months of clinical observation and conservative management.

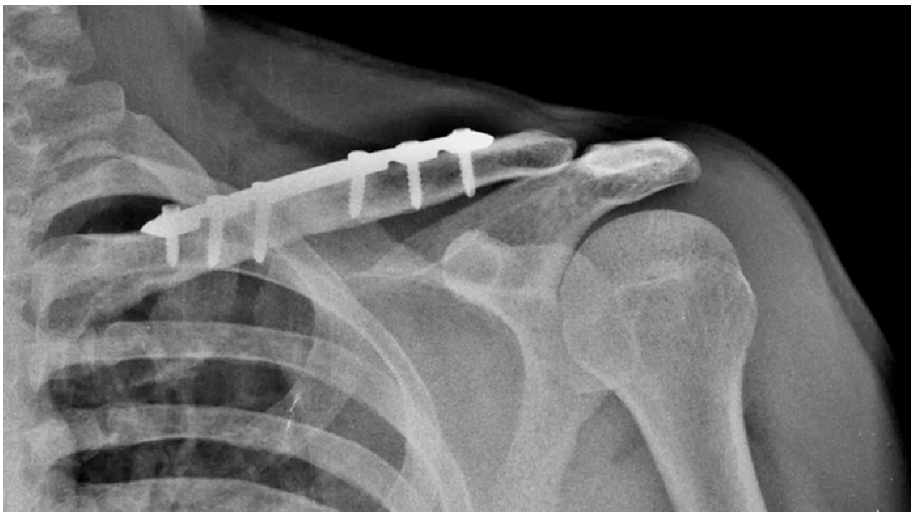

Fig. 5. Radiographic follow-up at 12 months after surgery, showing complete healing of the fracture in the middle third of the clavicle.

The diagnostic approach documented in the published case reports includes the use of imaging studies such as Computed Tomography Angiography and MRI of the plexus [3,5–7] to rule out the presence of potential acute post-surgical compressive etiologies (hematomas, pseudoaneurysms, or issues related to the osteosynthesis material). However, in the cases reviewed, the plexopathy was not secondary to a cause other than those mentioned (compression by hypertrophic callus or traction on the plexus). In our patient's case, an MRI of the plexus was performed, but no compressive findings were documented at this level.

Some studies reported the use of nerve conduction studies to determine the location of the neurological injury and for follow-up [5,7]. Namdari et al. [7] documented involvement of the medial trunk (ulnar and median nerves) with neurological follow-up studies showing axonal reinnervation at 4 months. Matthews et al. [5] reported similar findings with predominantly medial fascicle involvement (ulnar and median nerves). In our patient's case, the radial and axillary nerves (posterior fascicle) and musculocutaneous nerve (lateral fascicle) were predominantly affected, with resolution of axonal damage documented by electromyography and nerve conduction studies at 12 months.

In summary, plexopathy following surgical treatment of a symptomatic non-union of a midshaft clavicle fracture is a rare complication. The etiology of the neurological damage may be related to the type of non-union treated. Hypertrophic non-unions can lead to plexus involvement due to compressive phenomena (from insufficiently resected bony callus), and these cases typically present immediately in the recovery room. The management involves surgical removal of the plate, complete callus resection, and neurolysis.

On the other hand, atrophic non-unions may cause plexopathy due to traction or elongation of the plexus during clavicle length restoration. These cases present in a subacute fashion between the 2nd and 3rd postoperative day, and conservative management through clinical observation and rehabilitation appears to be a reasonable approach.

Functional recovery and nerve damage are reversible in both cases, with minimal residual functional limitations. We present a case of transient plexopathy involving the posterior and lateral fascicles, likely due to elongation after surgical treatment of an atrophic non-union of a clavicle fracture. The patient experienced complete resolution of symptoms after conservative management.

Conclusion

When dealing with acute brachial plexopathy secondary to surgical treatment of a hypertrophic non-union of a clavicle fracture, the management should be surgical, as the mechanical effect of compression can only be resolved through complete callus resection. On the other hand, in cases of acute plexopathy secondary to the treatment of an atrophic non-union (due to plexus traction), a conservative approach could be considered as a viable option.

Patient consent

Patient Consent was obtained.

CRediT authorship contribution statement

Gabriel Larrota: Formal analysis, Investigation, Methodology. **Ricardo Castro Gaona:** Investigation, Supervision. **Néstor Sánchez Dicelis:** Funding acquisition, Resources, Supervision. **Diego A. Sánchez Cruz:** Formal analysis, Investigation, Methodology.

Declaration of competing interest

None.

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