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Acute brachial plexus deficit due to clavicle fractures

Leïlani A. Delaune, MD *, Laurent Wehrli, MD, Yael Maeder, MD, Frédéric Vauclair, MD, Kevin Moerenhout, MD

Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland

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Clavicle fractures are frequently observed in emergency departments, representing 2.6%-4% of all fractures seen in hospital emergency admissions.[13] The mean age of patients in whom they occur is 35 years-old.[30] However, a bimodal age distribution is observed in male patients (<30 and >70 years), whereas a unimodal distribution is described in older female patients.[32] Fractures of the midshaft are the most common (69%-82%).[13,21] Clavicle fractures may lead to several immediate or delayed complications. Acute complications, such as neurovascular injuries (brachial plexus injury, subclavian vein and/or artery injury, stenosis, or thrombosis), pneumothorax or lung injuries, or associated musculoskeletal injuries, occur in 1%-3% of patients with clavicle fractures.[23,29]

Brachial plexus injury is a rare complication of adult clavicle fractures, and only a few cases have been reported in the literature. Most of these reports describe compression of the brachial plexus secondary to hypertrophic callus formation or due to secondary displacement of the fracture.[2,7,9,26] Iatrogenic brachial plexus injuries are rare after clavicle osteosynthesis (0%-1.5%), usually incompletely affecting the upper trunk or the suprascapular nerve.[6]

Given the paucity of reported cases, we describe 2 cases of midshaft clavicle fractures associated with acute brachial plexus injury and present a review of the literature.

Case presentation

Case 1

A right-handed, healthy, 28-year-old patient involved in a road accident was admitted to a level 2 trauma center. Physical and radiographic examinations showed an isolated, multifragmentary displaced midshaft right clavicle fracture, without any neurologic deficits (Fig. 1). The patient was immobilized with a sling after being assessed by an orthopedic surgeon.

The patient was admitted to our emergency department 2 days later owing to the onset of paresthesia at the medial aspect of the forearm and an extension deficit of the thumb. The clinical examination showed hypoesthesia of the medial border of the forearm and volar aspect of the ring and little fingers. Weakness of thumb extension as well as finger abduction was graded M3 according to the classification of the Medical Research Council. Tendon reflexes were normal. A standard radiograph showed a long intermediate bone fragment facing downward. Computed tomography (CT) and magnetic resonance imaging (MRI) scans were performed to precisely assess the brachial plexus. The findings confirmed compression of the plexus caused by the bone splinter, protruding 32 mm under the lower border of the clavicle, pushing back the surrounding soft tissues. There was an important soft-tissue infiltration surrounding the brachial plexus. However, the clavicle bone splinter did not penetrate the brachial plexus and therefore did not cause a nerve laceration (Figs. 2 and 3).

Because of the acute onset of the neurologic symptoms emerging 24 hours after the accident, as well as the probable lower-trunk brachial plexus compression, we performed open reduction, repositioning of the intermediate fragment, and plating of the right clavicle (Fig. 4). Given the delayed onset of neurologic deficits and the MRI scan showing continuity of the trunks, we did not explore the brachial plexus. The operation was performed without complication.

At the 3-month follow-up, the patient mentioned an absence of pain and had recovered full range of motion. Regarding the motor deficit, a clinical progression was observed; the extension of the thumb and abduction of the fingers recovered to grade M4. The

* Corresponding author: Leïlani A. Delaune, MD, Centre Hospitalier Universitaire Vaudois, Rue du Bugnon 46, 1011 Lausanne, Switzerland.
E-mail address: leilani.delaune@chuv.ch (L.A. Delaune).

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sensibility on the medial aspect of the forearm started to recover as well. One month later, the patient showed a complete clinical recovery and returned to his full-time job as a carpenter.

Case 2

A 67-year-old, right-handed female patient was admitted to our emergency department after a fall down an escalator onto her right shoulder. Her main medical background was a treated hypothyroidism, as well as clinically stable secondary progressive multiple sclerosis. Physical and radiographic examinations showed a displaced midshaft clavicle fracture (Fig. 5) and a nondisplaced intra-articular right distal radius fracture. Clinically, as the patient was assessed by an orthopedic surgeon, intermittent sensory and motor deficits were observed, but no muscle was graded less than M3. Conservative treatment by sling immobilization was initially recommended.

Three days later, the patient again presented for consultation because of worsening of the neurologic symptoms, in addition to severe shoulder pain. The clinical examination showed superficial

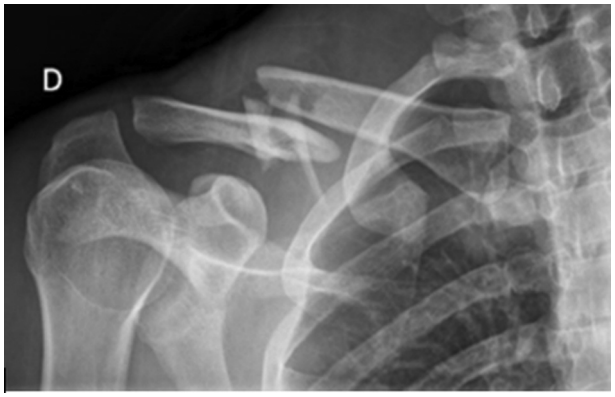
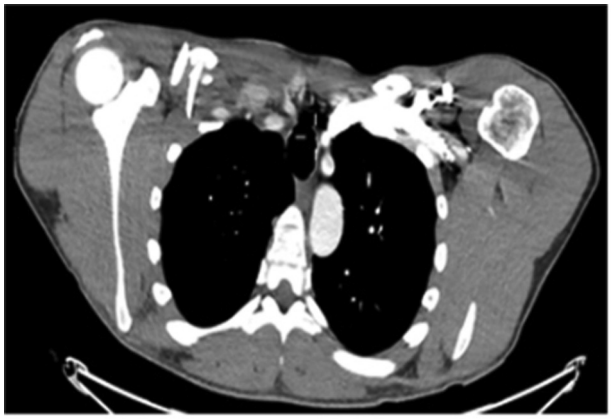


Figure 1 Radiograph of multifragmentary displaced midshaft right clavicle fracture in a 28-year-old male patient (case 1). D is for droit, which means right in french.



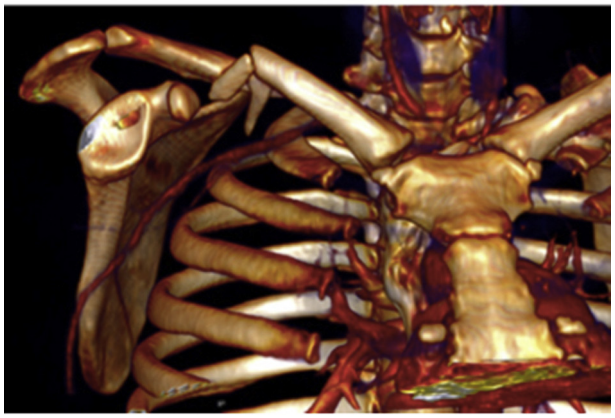
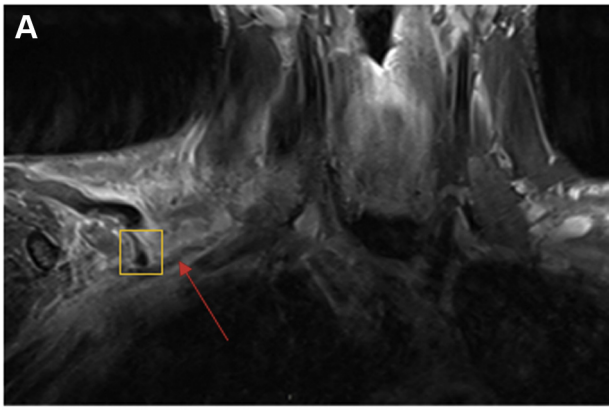


Figure 2 Computed tomography scan of the scapular waist in case 1, showing a multifragmentary displaced clavicle fracture, with a bone splinter pushing the soft tissues surrounding the brachial plexus.



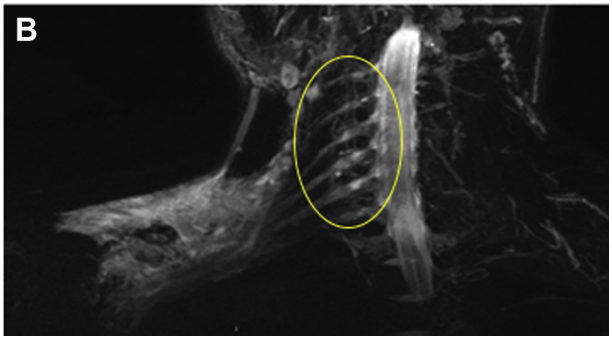


Figure 3 Brachio-cervical magnetic resonance imaging scans in case 1. (**A**) T1 coronal view, showing that the bone splinter ( ) is causing compression on the brachial plexus ( ). (**B**) T2 STIR (short tau inversion recovery) coronal view, showing intact brachial plexus roots ( ).

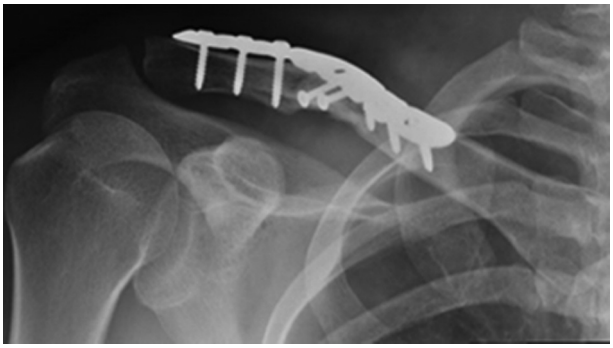


Figure 4 Radiograph after open reduction and internal fixation with a plate in the right clavicle in case 1.

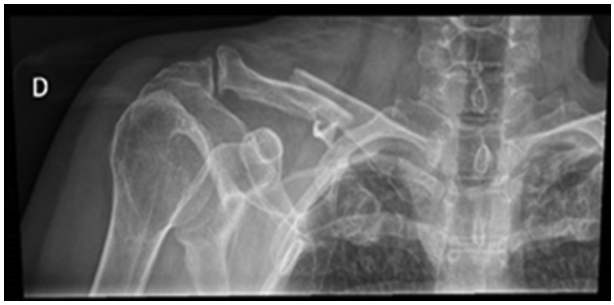


Figure 5 Radiograph of displaced midshaft right clavicle fracture in a 67-year-old female patient (case 2).

hypoesthesia in the following territories: axillary, radial, median, and ulnar nerves. Motor deficiencies were documented and graded according to the Medical Research Council classification; the muscular groups of the shoulder were evaluated as M0. Flexion and extension of the elbow were impossible, as were wrist and finger extension. Movements of flexion of the wrist and fingers, as well as abduction of the fingers, were classified as M3. Pronation and supination were maintained. Tendon reflexes (triceps, biceps, and brachioradialis) were absent. A CT scan showed multiple right costal fractures and a reduced space between the intermediate fragment of the clavicle and the first rib measuring 7 mm. An MRI scan revealed important soft-tissue infiltration around the brachial plexus resulting in compression of the plexus (Fig. 6). Electroneuromyography confirmed axonal lesions by showing fibrillations in the supraspinatus, infraspinatus, deltoid, biceps, brachioradialis, and extensor digitorum communis muscles.

Because of the worsening neurologic symptoms and clinical signs of upper-, middle-, and lower-trunk brachial plexus compression, we performed an open reduction with repositioning of the intermediate fragment and plating of the clavicle (Fig. 7), without any surgical exploration of the nerves. The severe pain in the shoulder completely disappeared after surgery.

At the 6-week follow-up, there was a clinical progression of the motor deficiencies (M4) of the shoulder, except for the supraspinatus and infraspinatus (M0) and deltoid (M1). Dysesthesia was still present on the lateral aspect of the shoulder and arm and on the posterolateral surface of the forearm. No sensory deficiency of the hand was observed. Electroneuromyography was performed 3 months after surgery, demonstrating good electrical recovery of the infraspinatus and biceps muscle (normal effort potential and disappearance of abnormal rest potentials), as well as partial recovery of the deltoid muscle. At the 5-month follow-up, the patient was satisfied with the neurologic recovery observed. The supraspinatus and infraspinatus were graded M3; the triceps and deltoid, M4; and the other muscular groups, M5. Follow-up at 8 months showed excellent motor recovery to M5 for all muscle groups, except the infraspinatus muscle, being at grade M4. We noted a mild hypoesthesia only on the anterior aspect of the arm.

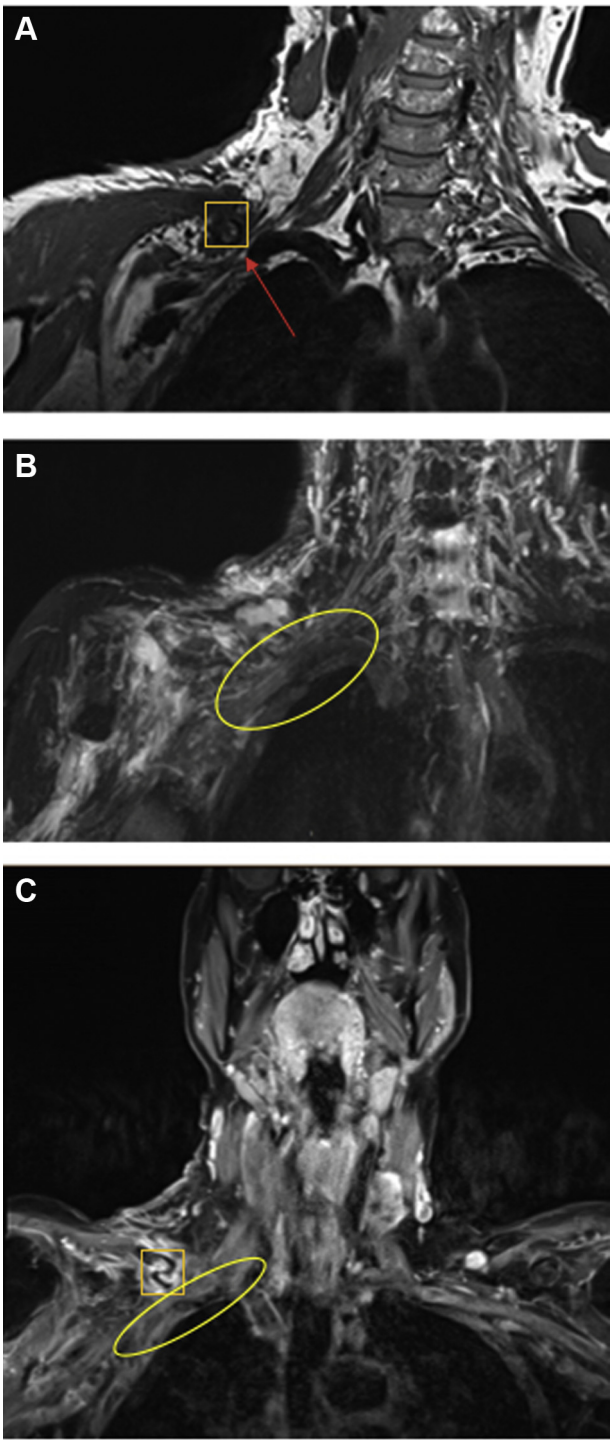


Figure 6 Brachio-cervical magnetic resonance imaging scans in case 2: (**A**) T1 coronal view, showing a bone fragment ( ) causing compression on the brachial plexus ( ). (**B**, **C**) T2 STIR (short tau inversion recovery) coronal view, showing the bone fragment ( ) and intact brachial plexus ( ).

Discussion

Brachial plexus compression is a rare complication of clavicle fractures. Traumatic elongations of the brachial plexus are more common and are frequently due to high-energy traumas. They are often associated with cervical spine, rib, humerus, scapula, and clavicle fractures.[12,18]

In most cases, the appearance of neurologic symptoms associated with conservative management of a midshaft clavicle fracture is delayed. Gradual development of brachial plexus palsy may appear within the month after the trauma, but this is extremely rare.[14,24,25] These delayed neurologic symptoms are often due to compression by hypertrophic callus,[7] nonunion of the clavicle fracture, secondary displacement, or a subclavian pseudoaneurysm.[2] Compression of the brachial plexus generally results in neurapraxia without axonotmesis.[19] This leads to what is known as secondary thoracic outlet syndrome and usually requires surgical

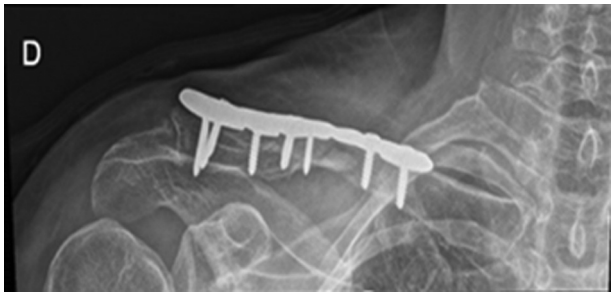


Figure 7 Radiograph after open reduction and internal fixation with a plate in the right clavicle in case 2.

treatment.[27] Symptoms include pain, weakness, rapid fatigability of the shoulder girdle muscles, and numbness or paresthesia in the arm and fingers due to plexus irritation, usually worsening during repetitive movements of abduction and/or flexion of the shoulder.[3] Several surgical procedures can be performed in patients with late neurologic symptomatic clavicle fractures: resection of the first rib, anterior and middle scalenectomy, partial or complete clavicle resection, costoclavicular space decompression with excision of excessive bone callus, and corrective osteotomy of the clavicle associated with internal fixation by a plate or elastic stable intramedullary nails (with or without the use of bone grafting from the iliac crest).[3,5,10,15,16,20,27] Total or partial excision of the clavicle (also called "cleidectomy") has also been performed in the past but resulted in residual pain, impairment in the function of the shoulder, and a poor cosmetic appearance.[1,10] The surgical procedure chosen depends on the etiology of the secondary thoracic outlet syndrome.

Neurologic symptoms can also be present shortly after the trauma, as observed in our cases. The compression of the brachial plexus is usually due to displaced clavicle fracture components or bone fragments,[8] and this is more commonly encountered when the fracture is located at the midshaft.[1,14,22,24] Neurologic symptoms are mainly described after trauma with high kinetic energy, as it causes important displacement of the bony fragments, but can also occur due to the high force of impact.[7] Because of the anatomy and muscle attachments, the lateral fragment of the clavicle fracture can reduce the subclavicular space, leading to compression and impingement of the surrounding neurovascular structures.[34] In our cases, there was no direct lesion of the brachial plexus. However, as seen on the MRI scans, fragments of the fracture compressed some of the brachial plexus trunks. Intermittent neurologic symptoms occurred immediately after the initial trauma and worsened during the following days. In 1991, Della Santa et al[7] described a case with pain and paresthesia appearing 5 days after the trauma, followed a few days later by deltoid and radial nerve–innervated muscle palsy. The intermediate bone fragment was found to be compressing and penetrating the posterior cord. Among the 16 patients with late neurologic symptoms described in their series, 50% recalled immediate brachialgia and paresthesia, secondarily affecting the hand.

The time of onset of symptoms in the acute phase has sparsely been described.[4,11,17,19,28,31,33] In 1965, Howard and Shafer[10] reported 4 cases of immediate or progressive acute numbness, paresthesia, and/or weakness due to compression by a middle-third clavicle fracture. Radiographs and surgical findings showed that the downward and posteriorly displaced lateral fragment caused compression on the brachial plexus, but the medical and radiologic means were less advanced at that time. Two cases were treated with open reduction and a Steinmann pin, with complete neurologic recovery at 6 months. One case with clavicle excision at 2 months showed motor regeneration over a period of 12 months. The last case was treated conservatively because of a delay in presentation of 6 weeks, with complete neurologic recovery at 6 months, but this case was complicated by shoulder-hand syndrome.

Brachial plexus injuries due to clavicle fractures may be difficult to diagnose in the acute stage, mainly because of the pain caused by the fractures. A meticulous initial clinical assessment is needed to obtain the patients' baseline neurologic state. Immediate recognition of neurologic deficits—or their worsening—is mandatory so that early surgical treatment can be performed because it increases the chances of a better neurologic recovery.[2,19]

According to the observations made in our 2 cases, early surgical treatment was very effective. It allowed our patients to obtain immediate control of their preoperative pain and an excellent neurologic recovery.

Conclusion

A thorough clinical orthopedic and neurologic assessment and reassessment are mandatory in patients with clavicle fractures to re-evaluate the neurologic status, as some clavicular fractures are associated with compression of the brachial plexus. It is important to recognize the progression of neurologic deficits early so that adequate treatment may be initiated. When a patient presents with acute neurologic symptoms due to compression of the brachial plexus, surgical fixation of the clavicle fracture should be performed to increase the chances of a full neurologic recovery. In our opinion, acute plexus injury associated with a midshaft clavicle fracture is an absolute indication for open reduction and internal fixation of the clavicle.

The take-home messages are as follows:

- Meticulous neurovascular assessment and regular reassessment are mandatory in patients with clavicle fractures.
- In case of clinical neurologic deficits, CT and MRI scans can be useful to analyze the position of bone fragments in relation to the nerves and the costoclavicular space, as well as the integrity of the subclavian artery.
- Brachial plexus injuries associated with clavicle fractures are usually reported in cases of high-energy trauma with a mechanism of elongation but can also occur in cases of low-energy trauma by bony compression.
- Brachial plexus compressions are usually reported in a chronic situation of clavicular hypertrophic callus formation or nonunion, but they may also occur in the acute stage, owing to displacement of bony fragments.
- In case of a secondary appearance of neurologic deficits, surgery is indicated on clinical and radiologic grounds. Open reduction and internal fixation of the clavicle may enable a full neurologic recovery through decompression of the brachial plexus.

Disclaimer

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