Brachial plexus paralysis in a patient with clavicular fracture, medico-legal implications

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Abstract. Clavicular fractures make up 2.6-4% of all fractures in adults. The most frequent mechanism of injury is a fall with direct trauma to the shoulder during sports or road accidents. These fractures can have acute complications such as vascular lesions, nerve injuries, pneumothorax, and musculoskeletal injury. Primary brachial plexus injuries are rare events, both in the adult and paediatric population, have an incidence of less than 1% and are usually caused by direct compression of the fragments. We describe a case of midshaft clavicular fracture treated conservatively with a figure-eight bandage, associated with acute brachial plexus injury, and possible medico-legal repercussions thereof. It is important to recognize the progression of neurological deficits early on, in order for appropriate treatment to be undertaken promptly. Patients must be monitored and re-evaluated within few days after the injury to check the correct positioning of the brace, its degree of tolerability, and the possible onset of neurological deficits, because some clavicular fractures can be associated with compression of the brachial plexus. (www.actabiomedica.it)

Key words: brachial plexus injury, paralysis, clavicular fracture, neurological deficits, medico-legal implications.

Introduction

Clavicular fractures make up 2.6-4% of all fractures in adults (1). The most frequent mechanism of injury is a fall with direct trauma to the shoulder during sports or road accidents. The portion of the clavicle most frequently involved is the middle third (69-82%), followed by the lateral third (12-26%) and the medial third (2-6%) (2,3).

These fractures can be treated surgically or conservatively. This decision is based on the type of fracture, site, patient age, functional requirements, and degree of displacement (2-4).

These fractures can have immediately (vascular-nerve injuries, pneumothorax) and over time (pseudarthrosis) complications. Acute complications, such as brachial plexus injury, vascular injury, pneumothorax, or musculoskeletal injury, occur in 1-3% of patients with clavicle fractures (5-6).

Both in the adult and paediatric population, primary brachial plexus injuries are rare events, with an incidence of less than 1%, and are typically caused by direct compression of the fragments (8). Iatrogenic neurovascular complications are rare, with an incidence between 0-1,5% and generally caused by traction of the brachial plexus, which adheres to bone fragments following inflammatory-healing reactions during reduction manoeuvres (9).

Given the scarcity of cases reported in scientific literature, we describe a case of midshaft clavicular...
fracture treated conservatively with a figure-eight bandage, associated with acute brachial plexus injury, describing the possible medico-legal repercussions.

Case Report

On 29.05.2019 a healthy 49-year-old right-handed woman reported an accidental fall with direct trauma to left shoulder.

Evaluated in another hospital, she underwent clinical and instrumental investigations with the diagnosis of isolated displaced multi-fragmentary mid-shaft fracture of the left clavicle, in the absence of peripheral neurological deficits. A conservative treatment was chosen with an eight-figured brace.

Two days later, the patient was admitted to the emergency room of the IRCCS Orthopedic Institute Galeazzi (Milan, Italy), complaining of poor tolerance to the brace and reporting the appearance of paraesthesia on the dorsal side of the forearm and left hand, with associated reduction in grip strength and difficulty in stretching out her fingers.

From the orthopaedic and neurological examination, performed at the entrance, an asthenia in the extension of the left-hand fingers was highlighted, equal to M2 according to the classification of the Medical Research Council (10), in the absence of wrist extension deficit. The osteotendinous reflexes were normal and symmetrical respect to the contralateral limb. There were no tactile sensitivity deficits and the axillary Tinel test was negative. New radiographic examinations were performed to assess any secondary decomposition of the fragments (Figure. 1).

The standard radiograph showed a decalage of the lateral fragment of the clavicle, often observable due to the muscular anatomical conformation. However, given the motor deficit thus detected, diagnostic investigations by magnetic resonance imaging (MRI) were performed in order to evaluate the coexistence of any lesions of the brachial plexus.

The iconographic findings pointed to thickening and oedema of the plexus bundles just above the first rib, even in the absence of interposition of bone fragments that could exert a local compressive action. Furthermore, no plexus ruptures or medullary alterations were noted (Figure. 2).

Due to the acute onset of neurological symptoms within 48 hours of the trauma, as well as the probable compression of the brachial plexus, the eight-figured bandage was immediately removed and replaced with a Desault-type brace.

The neurologist who was consulted during a collegiate visit recommended therapy with neurotrophic drugs, Acetyl levocarnitine hydrochloride 1500mg/ day given in 3 divided doses for 30 days, and intramuscular therapy with cortisone, such as Dexamethasone 0.4 mg/kg/day in 2 divided doses for 9 days, with dosage to be reduced by 50% every 3 days. Twelve days after the trauma we proceeded with the open reduction surgery of the fracture, with careful realignment of the fracture fragments, avoiding...
traction manoeuvres and verifying that there were no interposed nerve structures. Final fixation was obtained with plate and screws (Figure 3). The operation was performed without complications. Given the outcome of the MRI, certifying the continuity of the main trunks of the brachial plexus, excluding the interposition of nervous structures between the fracture fragments and the absence of fibro-cicatricial adhesions, it was decided to postpone exploration of the brachial plexus for 3 months, in order to evaluate possible improvements with conservative therapy.

At the follow-up, set 1 month after surgery, the patient did not present any painful symptoms, and reported a progressive improvement in the extension of the thumb and fingers. Clinical evaluation showed excellent stability of the fracture and complete range of motion recovery.

Informed consent to participate in the study was granted by the patient, pursuant to an agreement to publish all the necessary information.

Discussion

Given its topographical relationships with the anatomical structures of the neck and shoulder girdle, the brachial plexus is frequently exposed to traumatic injuries.

Brachial plexus injuries are often associated with fractures of the cervical spine, ribs, proximal humerus (11), scapula and clavicle (12).

The most frequent cause of brachial plexus paralysis is road trauma, particularly in motorcycling, where the plexus suffers traction, contusion, or compression injuries. In fact, the main mechanism is the violent stretching of the nerve roots which simultaneously determine a downward traction of the shoulder and submaximal hyperextension-inclination of the head towards the opposite side (13,14). In such circumstances, the nerve roots are pulled beyond the elastic resistance limit, leading to their tearing.

The direct contusive genesis of brachial plexus paralysis can instead be linked to mechanical trauma with high kinetic energy, where the neurogenic lesion is associated with other significant anatomical-pathological manifestations of the district, such as fractures of the proximal humerus, of the scapula, of the first ribs, the clavicle and axillary artery laceration.

Moreover, post-traumatic neurological aftermath suffering can be determined by the compressive effect exerted “ab estrinseco” on the nerve roots by the contiguous anatomical structures. In this context, a significant role is attributed to diffuse bleeding in the deep cervical muscles.

The causes that most often result in brachial plexus damage after a clavicular fracture are sudden traumatic strains, with a distraction-elongation mechanism, often due to high-energy trauma. Conversely, compression injuries are rarely linked to such fractures. Neurologic symptoms may manifest immediately after trauma or have a slower onset. Compression of the brachial plexus is usually due to bone fragments from the fracture, especially if dislocated, or to oedema of the nearby soft tissues; this most commonly occurs when the fracture is in the middle third of the clavicle (15,16).

Due to the anatomy and muscle insertion points, the lateral fragment of the clavicle can reduce the subclavicular space, leading to compression of the surrounding neurovascular structures.

In our case there was no direct injury to the brachial plexus, but compressive suffering, caused by fracture-related oedema and partly secondary to the use of the eight-figured brace.

In the presence of acute neurological symptoms secondary to the compression of the brachial plexus, appropriate instrumental examinations will be necessary to identify the injury type, and then proceed with early surgical stabilization of the fracture and anti-

Figure 3. X-ray of clavicle after open reduction and fixation with plate and screws.
inflammatory neurotrophic therapy with Cortisone, and anti-oedema medication in order to reduce soft tissue oedema and facilitate neurological recovery.

Surgical exploration or decompression of the brachial plexus, often associated with executive difficulties with possible production of iatrogenic lesions, do not seem to improve the outcomes in patients with brachial plexus lesions as opposed to patients only treated with open reduction of the fracture and fixation with plate and screws (15,16). Such interventions could be reserved for cases where no improvement in symptoms is observed 3 months after osteosynthesis surgery.

From a medico-legal standpoint, since the preponderant share of clavicular fractures associated with suffering of the brachial plexus derives from road accidents and/or injuries (including sports, domestic and professional accidents), it is essential that the quantification of the damage is consistent with the extent of the nerve injury and its functional repercussions.

Usually, the degree of permanent disability related to uncomplicated clavicular fractures is low, ranging between 2% and 8% in the context of private accident insurance (17), or between 2% and 5% in the context of civil liability (18). However, in case of major repercussions on the function of the upper limb brought about by the neurological lesion, the evaluation must necessarily take into account the degree of pathological involvement (19).

Furthermore, it is appropriate that the medico-legal evaluation takes place once the definitive stabilization of the neuropathological conditions has occurred, on average over 12 months (especially in young subjects), when spontaneous improvements can no longer be expected, not unlike other neurological lesions affecting different body areas (20, 21).

The extent of nervous damage will in any case be directly proportional to the severity of the lesion, also in light of the nosographic forms of greater clinical observation.

The most frequent form is *Erb's or superior palsy*, in which the fibres of the 5th and 6th cervical roots are damaged. The deltoid, external rotors of the shoulder, pectorals, brachioradialis, biceps brachialis and supinator muscles are therefore involved in various combinations. The limb is inert and placed in a suspended position, abducted, and internally rotated. It is impossible to raise and rotate the arm or flex and lock the forearm. Sensitivity disorders generally consist of mild hypoesthesia in the shoulder and along the outer surface of the arm. Radial and bicipital reflexes are weak or absent. Involvement of the rhomboid and serratus muscles may indicate a very proximal plexus injury, as the dorsal scapula nerves and the long thoracic nerve originate very high in the plexus. According to the medico-legal barèmes previously cited (16,17), this neuropathological condition is attributed a degree of permanent disability between 44% and 63.5% in terms insurance against private accidents, ranging between 40% and 45% in civil liability.

In Klumpke's or inferior palsy, the C7, C8, and T1 fibers are damaged. This results in a paresis of the small muscles of the hand and the flexors of the fingers, while the extensors of the hand and fingers are generally spared. Sensory disturbances mainly concern the ulnar side of the hand and forearm; the triceps reflex is often absent. This picture matches a permanent disability value similar to that expected in the case of superior paralysis.

There is also a middle root syndrome, Remak type, in which the clinical condition is attributable to a lesion of the seventh cervical nerve, with similar but more limited characteristics than the lower paralysis; such a condition is usually ascribed a permanent disability level ranging between 22% and 35% in the accident sector and between 20% and 25% in civil liability.

Total plexus paralysis is relatively rare and presents clinically as a combination of superior and inferior paralysis. Immediately after a trauma, a complete paralysis of the plexus can be observed, which however tends over time to take the form of an upper or lower lesion. In the case of total stabilized paralysis, the forensic assessment ranges from 60% to 85% in private accidents and from 55% to 60% in civil liability.

In patients with clavicular fracture, a preliminary and accurate clinical-objective evaluation is essential to rule out the presence of any neurological deficits.
A complete description of the semeiotic findings is recommended to best document the state of affairs and provide evidence as to the appropriateness of the diagnostic-therapeutic approach undertaken, in the event of litigation.

Patients must be monitored and re-evaluated within few days after the injury to check the correct positioning of the brace, its level of tolerability, and the possible onset of neurological deficits, given how some clavicular fractures can be associated with compression of the brachial plexus.

It is important to recognize the progression of neurological deficits early, so that appropriate treatment can be undertaken promptly.

In relation to the very high disabling impact of brachial plexus injuries brought about by clavicular fractures, adverse events should be promptly identified and adequately treated, in order to avoid litigation with medical negligence charges, which can severely impact doctors and even the healthcare system as a whole.

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**References**

1. Kihlström C, Möller M, Lönn K, Wolf O. Clavicle fractures: epidemiology, classification, and treatment of 2 422 fractures in the Swedish Fracture Register; an observational study. BMC Musculoskelet Disord 2017; 18: 82.
2. Ramponi DR, Jo Cerepani M. Clavicle Fractures. Adv Emerg Nurs J 2021; 43: 123-127.
3. Donnelly TD, Macfarlane RJ, Nagy MT,Ralte P, Waseem M. Fractures of the clavicle; an overview. Open Orthop J 2013; 7: 329-33.
4. Wiesel B, Nagda S, Mehta S, Churchill R. Management of Midshaft Clavicle Fractures in Adults. J Am Acad Orthop Surg 2018; 26: e468-e476.
5. Mouzopoulos G, Morakis E, Stamatakos M, Tzurbakis M. Complications associated with clavicular fracture. Orthop Nurs 2009; 28: 217-224.
6. Kim MS. Conservative treatment for brachial plexus injury after a displaced clavicle fracture: a case report and literature review. BMC Musculoskeletal Disord 2022; 23: 632.
7. Clitherow HD, Bain GI. Major neurovascular complications of clavicle fracture surgery. Shoulder Elbow 2015; 7: 3-12.
8. Canadian Orthopaedic Trauma Society. Nonoperative treatment compared with plate fixation of displaced midshaft clavicular fractures. A multicenter, randomized clinical trial. J Bone Joint Surg Am 2007; 89: 1-10.
9. Clitherow HD, Bain GI. Association between screw prominence and vascular complications after clavicle fixation. Int J Shoulder Surg 2014; 8: 122-6.
10. Compton A. Aids to the investigation of peripheral nerve injuries. Medical Research Council: Nerve Injuries Research Committee. Brain 2010; 133: 2838-44.
11. Basile G, Avato FM, Passeri A, et al. Atrophic pseudarthrosis of humeral diaphyseal fractures: medico-legal implications and methodological analysis of the evaluation. Acta Biomed 2022; 93: e2022176.
12. Kaiser R, Menci L, Haninec P. Injuries associated with serious brachial plexus involvement in polytrauma among patients requiring surgical repair. Injury 2014; 45: 223-6.
13. Viswamadesh R, Rajendiran S, Pakiri Maheswaran AM, Gomathinayagam K. The Demography of Traumatic Brachial Plexus Avulsion Injuries. Cureus 2022; 3: 14: e25626.
14. Kaiser R, Waldauf P, Ullas G, Krajcová A. Epidemiology, etiology, and types of severe adult brachial plexus injuries requiring surgical repair: systematic review and meta-analysis. Neurosurg Rev 2020; 43: 443-452.
15. Lin CC, Lin J. Brachial plexus palsy caused by secondary fracture displacement in a patient with closed clavicle fracture. Orthopedics. 2009; 32: orthosupersite.com/view. asp?ID=43780.
16. DeLaune LA, Wehrli L, Maeder Y, Vaulclaire F, Moerenhout K. Acute brachial plexus deficit due to clavicle fractures. JSES Int 2020; 5: 46-50.
17. Ronchi E, Mastroboroto L, Genovesi U. Guida alla valutazione medico-legale dell'invalidità permanente"; Milano, Giuffrè Ed.; 2015 p. 316.
18. Buzzi F, Domenici R. Società Italiana di Medicina Legale e delle Assicurazioni-S.I.M.L.A., Linee Guida per la valutazione medico-legale del danno alla persona in ambito civilistico.; Milano, Giuffrè Ed.; 2016. p 337.
19. Amadei F, Bruno MC, Romanò C, Basile G. A case of infection and severe soft tissue loss of the elbow. Planning of surgical treatment in compliance with good clinical-care practices and medical-legal implications. Acta Biomed 2022; 93: e2022106.
20. Basile G, Passeri A, Bove F, Accetta R, Gaudio RM, Calori GM. Pelvic ring and acetabular fracture: Concepts of traumatological forensic interest. Injury 2022; 53: 475-480.
21. Ciatti C, Maniscalco P, Quattrini F, et al. The epidemiology of proximal femur fractures during COVID-19 emergency in Italy: a multicentric study. Acta Biomed 2021; 92: e2021398.

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