

# Progressive Brachial Plexus Palsy after Osteosynthesis of an Inveterate Clavicular Fracture

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## What to Learn from this Article?

*Unique scenario of progressive brachial plexus palsy following osteosynthesis of clavicle.*

### Abstract

**Introduction:** The thoracic outlet syndrome (TOS) is a rare complication of clavicular fracture, occurring in 0.5-9% of cases. In the literature from 1965–2010, 425 cases of TOS complicating a clavicular fracture were described. However, only 5 were observed after a surgical procedure of reduction and fixation. The causes of this complication were due to the presence of an exuberant callus, to technical surgery errors or to vascular lesions. In this paper we describe a case of brachial plexus palsy after osteosynthesis of clavicle fracture

**Case Report:** A 48 year old female, presented to us with inveterate middle third clavicle fracture of 2 months duration. She was an alcoholic, smoker with an history of opiate abuse and was HCV positive. At two month the fracture was displaced with no signs of union and open rigid fixation with plate was done. The immediate postoperative patient had signs of neurologic injury. Five days after surgery showed paralysis of the ulnar nerve, at 10 days paralysis of the median nerve, radial and ulnar paresthesias in the territory of the C5-C6-C7-C8 roots. She was treated with rest, steroids and neurotrophic drugs. One month after surgery the patient had signs of complete denervation around the brachial plexus. Implant removal was done and in a month ulnar and median nerve functions recovered. At three months post implant removal the neurological picture returned to normal.

**Conclusion:** We can say that TOS can be seen as arising secondary to an "iatrogenic compartment syndrome" justified by the particular anatomy of the space cost joint. The appropriateness of the intervention for removal of fixation devices is demonstrated by the fact that the patient has returned to her daily activities in the absence of symptoms and good functional recovery in about three months, despite fracture nonunion.

**Keywords:** brachial plexus palsy, clavicle fractures, outlet thoracic syndrome.

### Introduction

The thoracic outlet syndrome (TOS) is a rare complication occurring in less than 1% of surgically treated clavicle fractures [1]. The most commonly recognized etiology is compression, supported by the

exuberant callus in the presence of delayed union or non-union. In a smaller percentage of cases, a vascular genesis [2] is recognized. On the basis of this, we have considered relevant to describe a case of TOS with progressive paralysis of the brachial plexus having an

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#### Author's Photo Gallery



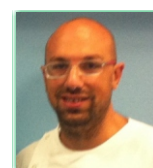
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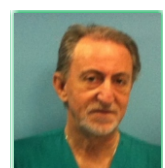
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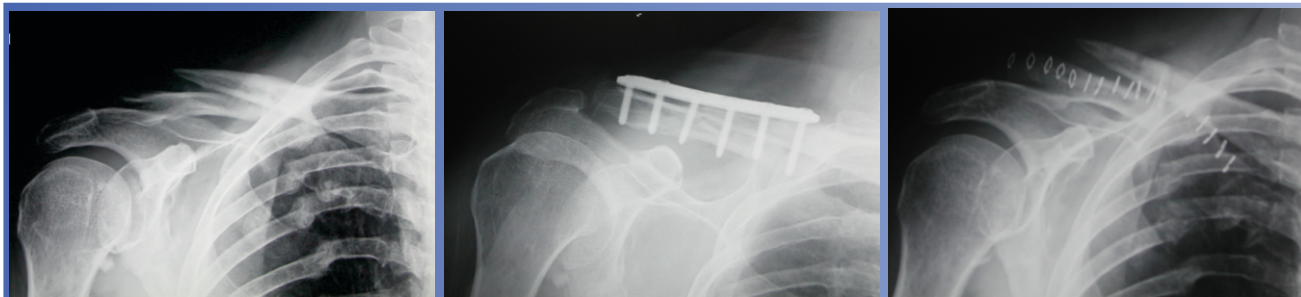


Fig 1: a) inveterate right clavicle fracture. b) clavicular fracture fixed with plate. c) after plate removal

unusual genesis and arising after an osteosynthesis operation of inveterate clavicular fracture.

### Case Report

In June 2009, C.M., (female, 48 years old) after a motorcycle accident, reported the middle third right clavicular fracture with associated multiple rib fractures and ipsilateral hemithorax (the first and second rib were free). The patient was conservatively treated with bandage "shape of eight". After about four months from the traumatic event, we observed the displacement of the fragments and no radiographic signs of consolidation (Fig. 1a). The patient had a complex history of opiates and alcohol abuse, heavy smoking, psychopharmacological treatment for depressive syndrome, and was tested positive for HCV. Her physical examination was negative for vascular or nerve deficit of the right upper limb and no emerging central or peripheral neurological disorders, such as canalicular syndromes or cervico-brachialgia, were noted. The patient and her relatives were informed about the non-surgical option, but she preferred to undergo the intervention. The patient was then surgically treated with an open reduction internal fixation and the fracture was stabilized with a plate (Fig. 1b). The post-operative course was without complications and the patient was discharged two days later.

One week after the surgery, the patient reported onset of numbness and tingling in the fingers of her right hand near the ulnar nerve. The vascular Adson test was good, and the peripheral pulses were palpable and symmetrical. After two weeks, numbness and tingling in the median nerve area occurred. Moreover, the flexor carpi radialis, the opponens pollicis muscle and the interossei muscles strength were reduced to 4/5. After about three weeks after surgery, the radial nerve deficit also appeared, with weakness of the carpi ulnaris extensor (CUE) and worsening of the deficit of the interossei muscles strength (3/5). The bone-tendon reflexes were sluggish to the upper right limb, while normal bright to the upper left and lower limbs.

The patient underwent an echo color doppler examination for arterial and venous supraclavicular fossa and upper limbs, chest x-ray, right shoulder and cervical spine MRI: all of these tests seemed to exclude the presence of expansive lesions or iatrogenic damage to nerve roots of the brachial plexus or to the vascular structures. Forty days after surgery, an electromyographic and electrical conduction velocity examination was performed: we noted the almost complete denervation on the extensor digitorum muscles, right flexors of the fingers and right first interosseous neurogenic damage with denervation activity on other muscles. From clinical examination and investigations it was clear that patient was suffering from thoracic outlet syndrom most likely secondary to osteosynthesis surgery. Therefore, the patient was again subjected to surgery, 70 days after the first operation, in order to remove the means of synthesis. The not yet consolidated fracture stumps were mobilized to widen the cost-clavicular space diameters. One week after the second operation, the clinical situation seemed to improve: the digitorum communis extensor (DCE) and longus pollicis extensor (LPE) strength was 4/5; communis digitorum flexor (CDF) 3/5; longus pollicis flexor (LPF) 2/5; ulnaris carpi flexor (UCF) force 2/5; cross-finger test could not be performed; hyperhidrosis in the territory of median nerve in the palm. Two months later, the patient recovered almost completely the function of the upper right limb, the cross-finger test was allowed, and the paraesthesias and hyperhidrosis disappeared. In March 2010, the patient underwent a new intervention because the medial stump caused a skin ulcer. We remodeled the proximal clavicle in the beveled way (this was not done the first time) and a new fixation was performed by plate osteosynthesis. The wound healed without further bed sore and without any changes at the neurological pattern.

### Discussion

TOC is rarely reported following clavicle fracture (0.5%). From 1965 to 2010 the literature reported 425 cases of

TOS following a fracture of the clavicle. Among these, only 5 occurred after surgical operation, with main causative factor being exuberant callus and all were associated with neurological as well as vascular symptoms. The case we report is different from the other literature cases on the basis of at least three points:

- 1) the occurred deficits were limited to the neural structures of the brachial plexus, which was involved in a progressive way with the involvement of the anterior root, followed by the lateral one and then by the posterior one;
- 2) the symptoms occurred one week after the surgery, such as "clockwork" and not sharply;
- 3) in the genesis of the TOS no responsibility of the callus has been found.

Within the space, the three trunks of the brachial plexus have a precise position. The lateral root is situated at the rear side and below the subclavian muscle, behind and rear of the vascular bundle, the medial cord is separated from the first rib from serratus anterior and the posterior cord is surrounded by a fat pad. The presence of this fatty shield may explain why the radial nerve has been affected by the last one in the deficit process, while the other two strings closest to the fracture and the less extensible structures were involved earlier. The latency of onset of symptoms can be explained in a "iatrogenic compartment syndrome" rather than in the formation of exuberant callus. During the movement of reposition and abduction of the shoulder (Wright test), the cost-clavicular space is reduced by 50% [3]: in this case, the inveterate clavicular fracture with displacement of the stumps led to a shortening of the clavicle, but also to a simultaneous relaxation of vascular and nerve structures located behind it. At the time of fracture reduction, with the restoration of its physiological length, probably the cost-clavicular space was tensioned laterally. If in acute way this maneuver is innocent in itself, for an inveterate fracture with nerves used to a larger space on a plot of neuropathy for drug, alcohol and tobacco abuse, in HCV positive patients [4] it may be sufficient to damage the nervous structures. What was created as a result of the rigid fixation of the clavicle, was a test of Wright with a reduction of the cost-clavicular space. Our hypothesis is supported by the evidence of the subjective and objective improvement following the removal of plate and a new breakdown of the stumps which allowed the opening up of cost-clavicular space [5].

Unique presentation of this case can help us extrapolate few learning points:

- 1) The space in which the brachial plexus is located, below the clavicle is very small, almost virtual, meaning that the plexus slides within it as "a finger in a glove" with the emergence of a genuine canalicular syndrome by increased internal pressure due to rigid fixation with the plate. It is a modest and ineffective pressure to produce vascular changes, but enough to undermine the weaker nerve fiber.
- 2) In our case, the diagnosis was of exclusion, and not without anxiety, of the severity of the disease together with many inquiries because of its rarity. However, it has been solved by only reducing the inner pressure by removing the plate and then widening sub-clavicular space, allowing the brachial plexus to breathe. Between the two evils, we obviously preferred the lesser one, thus leaving to its fate the fracture of the clavicle and allowing a complete neurological recovery.
- 3) It was not by chance that the ulnar nerve went into crisis first, followed immediately by the median: the lateral cord is in contact with the subclavian muscle, while the posterior cord (and therefore radial) is surrounded by fat, and is protected.
- 4) Even our case confirms that the brachial plexus injuries after clavicular fractures caused by external compression generally have a favorable course, even if the data in literature refer to compression of exuberant callus [6].

### Conclusion

To sum up, we believe that this case study offers several relevant findings which should be taken into consideration for further investigations when dealing with progressive brachial plexus palsy after osteosynthesis of an inveterate clavicular fracture.

### Clinical Message

This is quite an unique scenario where there was progressive compression of brachial plexus after osteosynthesis, primarily because of the plate and also possibly because of stretching of relaxed structures. Although reproduction of such scenario is unlikely, but the case makes us aware of existence of such entity.

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