

CASE REPORT **GENERAL RECONSTRUCTION**

Acute thoracic outlet syndrome due to clavicular fracture callus: a case report

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Introduction

Thoracic outlet syndrome (TOS) results from compression of neurovascular structures in the cervicoaxillary canal. In the case of neurogenic TOS, the brachial plexus or sympathetic nerves are impinged.¹ Potential mechanisms include direct impingement in the setting of a clavicle displaced fracture, malunion or hypertrophic bone callus.² We report an unusual case of acute neurogenic TOS secondary to excessive bone callus following a conservatively-managed mid-clavicular fracture.

Case

A 54-year-old man presented following a pushbike accident with multiple injuries including thoracic transverse process fractures, multiple right-sided rib fractures with associated haemopneumothorax, right clavicle (**Figure 1A**) and scapular fractures, right hemipelvis fracture and left sacral alar fracture. The patient had no significant past medical or smoking history and was right-hand-dominant. Following admission to the intensive care unit, he was managed with right-sided intercostal catheter insertion, fixation of right ribs three to seven, and right acetabular open reduction and internal fixation. A neurovascular assessment was not documented.

The closed clavicle shaft fracture was assessed with erect X-ray nine days after the injury (**Figure 1B**). This showed 1 cm superior displacement of the medial clavicle with two inferiorly displaced butterfly fracture fragments. There were no neurologic concerns and it was deemed appropriate for nonoperative management with reassessment and repeat imaging in four weeks.

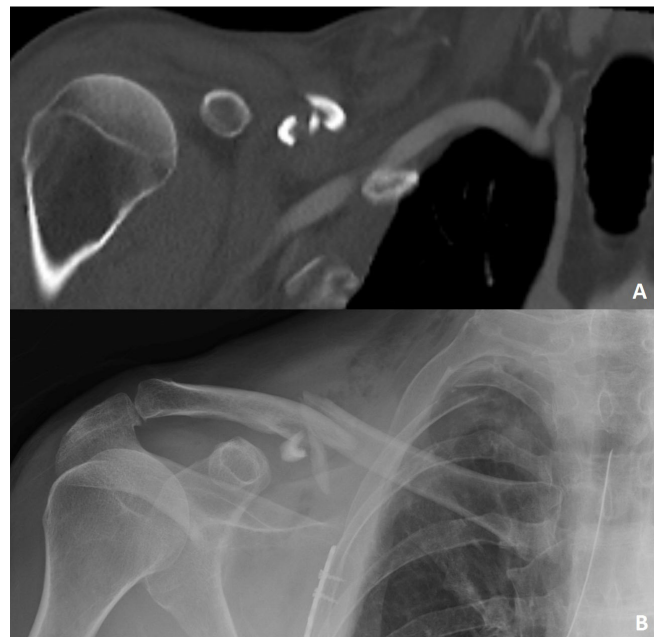


Fig 1. (A) CT demonstrating fracture fragments around the brachial plexus and subclavian artery; (B) erect X-ray demonstrating moderately displaced midshaft clavicle fracture with two butterfly fragments displaced inferiorly

Three weeks following the initial injury, the patient noted new right-hand weakness and presented for assessment. Examination demonstrated global weakness of right shoulder movement, with Medical Research Council (MRC) scale for muscle strength M4 for all movements. Examination was complicated by pain associated with the right-sided clavicle and rib fractures. Right elbow extension was M2 and elbow flexion M4. Right wrist extension and flexion were both M4. Extension of the thumb and digits as well as intrinsic muscles were M0. Finger flexion was M4. These findings were new, escalating and significant neurologic deficits.

A CT angiogram of the brain and carotid showed no intracranial pathology. A CT of the cervical and thoracic spine was unremarkable apart from a large callus associated with the clavicular shaft fracture and two inferiorly displaced fracture fragments. The arterial phase showed no flow disturbance in the subclavian artery (**Figure 2A**) at rest. On initial presentation, the costoclavicular distance was 18.4 mm. This was reduced to 10.4 mm with the formation of bone callus. An MRI of the brachial plexus demonstrated large callus of the midshaft clavicular fracture resulting in narrowing of the right thoracic outlet (**Figure 2B**). The distal trunks and proximal divisions showed inferior displacement with mildly increased T2 signal in this region.

The patient underwent right brachial plexus exploration, demonstrating extensive circumferential firm callus several centimetres

around the clavicular fracture (**Figure 3A** and **3B**). A formal, complete, inferior anterior scalenotomy was undertaken. The brachial plexus and subclavian artery were found to be impinged within the costoclavicular interval with significant contusions of the lower and middle trunks, posterior cord elements and subclavian artery. The callus was resected, widening the costoclavicular interval. All involved and compressed elements of the brachial plexus as well as the subclavian vessels were dissected and found to be in continuity. Consequently, no nerve repairs were required. The first thoracic rib was not resected. The fracture was reduced and handed over to the orthopaedic team, and the clavicle was plated using a Synthes 2.7 mm clavicle shaft plate CS2 (Synthes, Pennsylvania, United States) with acceptable reduction on intraoperative image intensifier (**Figure 3C**).

Immediately following the operation, the patient noted improvement in thumb extension (M3). Early outpatient follow-up three weeks postoperatively demonstrated improved extension at the wrist and proximal interphalangeal joints (M4). The patient reported cessation of all pain medications at six months after injury. At his final 7.5-month follow-up he was extremely happy with his restored limb function. He had no sensory deficit of concern, normal elbow flexion and extension, normal wrist flexion and extension, and normal digital flexion and extension, with the exception of a 20 degree lag in middle finger metacarpophalangeal extension, which was of no concern to the patient. Pain was absent and function was so well restored that no further follow-up was required or requested by the patient.

Discussion

Impingement of the brachial plexus during its passage through the cervicoaxillary canal is termed neurogenic TOS, which is the most common form of TOS.¹ It typically occurs in the setting of a predisposing anatomical abnormality and a precipitating factor, such as trauma or functional changes.³ Potential areas of compression include the interscalene triangle, the costoclavicular interval and the retropectoralis minor or subcoracoid space. In the absence of defined anatomical abnormalities (such as lateral insertion of costoclavicular ligament), the costoclavicular distance has been independently associated with TOS.⁴ Clavicle fractures are common, with an incidence of 29–64 per 100,000, and most commonly affect the shaft.⁵ Managed

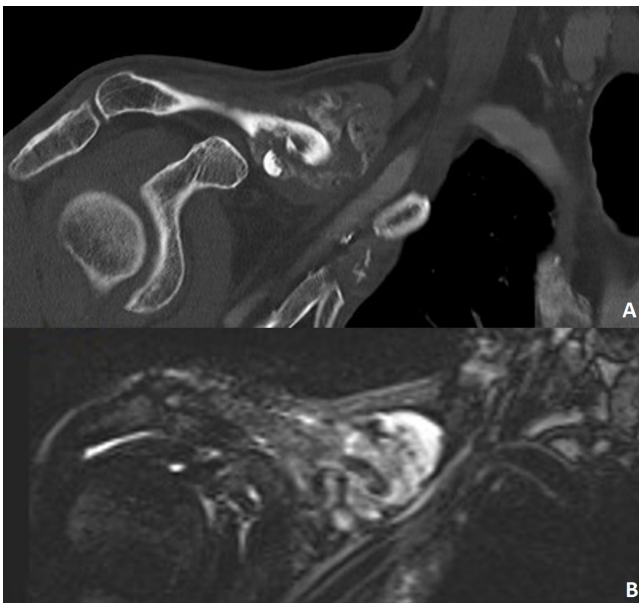


Fig 2. (A) CT following presentation with upper limb weakness demonstrating extensive callus; (B) MRI demonstrating brachial plexus compression with mildly increased T2 signal

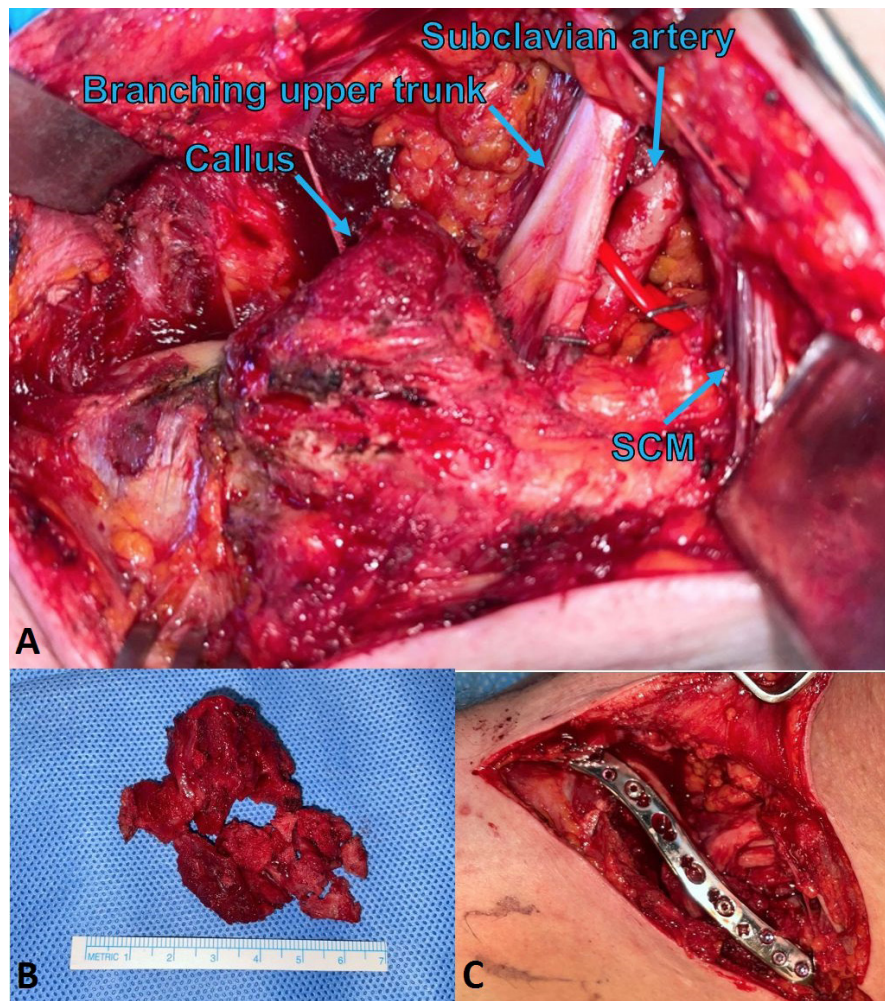


Fig 3. Operative photography demonstrating (A) excessive callus overlying the branching upper trunk of the brachial plexus and subclavian artery (the sternocleidomastoid muscle is also seen); (B) removed callus (same orientation); (C) reduced clavicle fracture with internal fixation

nonoperatively, they can result in neurogenic TOS through direct impingement of displaced fragments, hypertrophic callus formation or malunion.² This occurs through narrowing of the costoclavicular space causing compression of the brachial plexus. With alleviation of compression, recovery is expected within approximately three months.⁶ Prompt diagnosis in this setting can be difficult because the patient may present with shoulder pain, sensory or motor changes, or vascular symptoms with additional injuries or comorbidities. Timing of onset following injury can also vary, with the literature demonstrating acute injury with a displaced bone fragment or later onset due to malunion or hypertrophic callus formation.^{7,8} Optimal management is surgical decompression of the costoclavicular space, although this can be challenging. Described methods include excision of excessive bone callus with or without open reduction and internal fixation, first rib resection, scalenectomy, cleidectomy, corrective osteotomy with internal

fixation, and nonoperative management.⁹ In terms of prevention, conservatively-managed fractures that are significantly displaced warrant more frequent review and neurological examination in the months following injury to monitor for compressive callous formation.

The first notable aspect of this case is that the patient presented three weeks after the initial injury. This is unique in that most cases are either associated with direct injury from displaced fragments or from hypertrophic callus or malunion months or years from initial injury. A large amount of callus had formed in a short period of time in this case. Other cases with brachial plexus compression in the weeks following clavicle fracture were found,^{8,10} however, only one of these cases was associated with hypertrophic callus. The more common aetiology was direct impingement by fracture fragments or the clavicle itself.

The second notable aspect is that this patient's costoclavicular distance prior to the formation of

hypertrophic callus was 18.4 mm, which is larger than the mean of 12.3 mm found in the anatomical study by Duarte and colleagues.⁴ Dissection of the brachial plexus and excision of the hypertrophic callus with subsequent improvement of the patient's neurology demonstrates that restoring an adequate costoclavicular space was therapeutic for this patient. In light of this, surgical exploration should be considered for acute TOS following clavicular fracture in the setting of any motor dysfunction. Additional interventions on other components of the thoracic outlet are not always required. All thoracic outlets are different, requiring the sites of impingement to be determined. Interventions must then strive to mechanically restore sufficient space around the neurovascular structures to successfully resolve the thoracic outlet compression.

Conclusion

Our case is a reminder of the importance of thorough neurovascular assessment and reassessment in the setting of clavicular fractures. This serves to identify acute or delayed disruption or compression of the brachial plexus. Additionally, our case demonstrates that timely and targeted surgery can successfully decompress the brachial plexus and lead to prompt reversal of the neurologic impairment of acute TOS.

Patient consent

Patients/guardians have given informed consent to the publication of images and/or data.

Conflict of interest

The authors have no conflicts of interest to disclose.

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