Major neurovascular complications of clavicle fracture surgery

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Abstract

Clavicle fracture fixation is becoming an increasingly common operation, with good clinical outcomes and a low rate of significant complications. However, there are several reports of rare but potentially life or limb threatening, neurovascular complications. Arterial injuries are usually pseudoaneurysms associated with prominent screws. These may be clinically silent for several years before presenting as subcritical upper limb ischaemia. Venous injuries are a result of tearing of the vessel wall by fracture manipulation, drills or implants. This produces intra-operative haemorrhage and potentially air embolism, which can be fatal if not rapidly recognized and managed. Brachial plexopathy is the result of traction on adherent plexus or impingement by fracture fragments or callus. It presents as severe arm pain and paralysis immediately postoperatively. Neurovascular injuries can be avoided by a combination of pre-operative planning, communication with anaesthetic staff and strategic surgical technique. The plane of the surgical exposure, release of the soft tissues, drill direction and depth and screw length are all important factors.

Keywords

Air embolism, brachial plexopathy, complication, clavicle, internal fixation, pseudoaneurysm

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Introduction

Plate fixation of midshaft clavicle fractures has become more common since the recognition of the importance of symptomatic malunion as a complication of non operative management.^{1,2} Patients are typically young, with manual occupations and active lifestyles.^{3,4}

Major neurovascular injury has been well documented in patients with clavicle fractures that were managed non-operatively. Direct injury to the vessels and brachial plexus can occur at the time of the fracture,^{5,6} or present in a delayed manner as pseudoaneurysms,^{7,8} deep vein thrombosis⁹ or thoracic outlet syndrome (TOS) caused by abundant fracture callus.¹⁰

Iatrogenic neurovascular complications are rare. The incidence of brachial plexus injury is reported to be 0% to 1.5%.^{11,12} A meta-analysis of 582 cases of clavicle fixation¹¹ did not report a single vascular complication. It is therefore extremely unlikely that a large enough cohort of patients could be produced to estimate the true incidence of vascular complications after clavicle fixation.

Given their rarity, we could find no high level studies that looked specifically at neurovascular injuries that were directly attributable to clavicle fracture surgery, rather than the original injury mechanism. However, there are several case reports and small series of limb threatening and even fatal neurovascular complications following clavicle surgery.^{13–26} This represents a potentially devastating outcome from an operation that is treating a problem that is neither life nor limb threatening.

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Figure 1. Right anterior neck dissection with clavicle present (A) and clavicle removed (B). (A) Neurovascular structures adjacent to the medial two thirds of the clavicle. (B) Brachial plexus and subclavian artery posterior to scalenus anterior (white arrow). Subclavian vein crossing first rib anterior to scalenus anterior and directly posterior to the medial clavicle and sternoclavicular joint (excised). At the medial end of the clavicle, the subclavian artery and brachial plexus are relatively protected by the scalenus anterior. White arrow = scalenus anterior muscle. Colouring of the neurovascular structures has been enhanced to demonstrate them clearly. Subclavian/axillary artery: red; subclavian/axillary vein: blue; brachial plexus, yellow; sternoclavicular joint (excised): black.

Anatomy

The key neurovascular structures around the clavicle are highlighted in Figure 1. All measurements presented have been rounded down to the nearest 1 mm.

Scalenus anterior

The scalenus anterior muscle is an important landmark for understanding the position of the neurovascular structures, relative to the clavicle (Figure 1B). It originates from the transverse processes of C2 to C7, passes posterior the clavicle and attaches to the first and second ribs.

The subclavian vein is anterior to the muscle and directly adjacent to the medial clavicle. By contrast, the artery is posterior to the muscle and a mean of 26 mm from the clavicle.²⁷ The artery and the brachial plexus are relatively protected as a result of this distance and interposed muscle.

Lateral to the muscle, this protection is lost and subclavian artery and vein converge. At the lateral border of the first rib, the vessels change name from subclavian to axillary. In the middle third of the clavicle the vessels are postero-inferior to the bone. They lie a mean distance of 17 mm and 13 mm, respectively, from the clavicle but can be as close as 5 mm.²⁷ In the lateral third, the vessels are directly inferior to the clavicle and are between 46 mm²⁸ and >60 mm²⁷ from the bone.

Brachial plexus

At its closest point the plexus is a mean of 12 mm from the clavicle, occurring at a point three-fifths along the length of the bone (measured from its medial end).²⁹

Thoracic outlet

The strict anatomical definition of the thoracic outlet is a subject of debate.³⁰ However, there is agreement that the region of the upper limb commonly referred to as the thoracic outlet consists of three zones. Medially is the scalene triangle, bounded by the medial and lateral borders of the scalene muscles (anterior, medius and posterior). The costoclavicular space is central and bounded by the clavicle superiorly and the first rib inferiorly. The most lateral region is the sub-coracoid (retropectoralis minor) space.^{31,32} The brachial plexus and axillary vessels pass through the thoracic outlet. The subclavian artery passes through the scalene triangle portion but the vein does not.

Clavicle dimensions

Galley et al. reported that the maximum thickness of the clavicle was 17 mm.²⁸ The minimum distance from the superior clavicle to the artery has been reported as 17 mm³³ to 22 mm.²⁸

Presentation and pathology

Reports of vascular injuries associated with clavicle fixation are summarised in Table 1.

Arterial injury

All reports of arterial injury^{14–17,21} have consistent features. There are no reported fatalities, there is no mention of significant bleeding at the time of the initial surgery and the injury was always associated with a prominent screw in the medial two thirds of the clavicle.

Table 1. Iatrogenic vascular injuries associated with clavicle surgery.

Author	Diagnosis	Injury to Surgery Interval	Complication	Cause	Presentation	Treatment	Outcome
Neer (1960) ²²	Mid-clavicle Non-union	4 years	Subclavian vein tear	Mobilization of fragments	Intra-operative bleeding	Not stated	Good
Bain et al. (2013) ¹³	Mid-clavicle Delayed union	6 weeks	Subclavian vein laceration and air embolus	Drilling medial clavicle	Intra-operative bleeding and shock	Resuscitation and ligation of subclavian vein	Fatal
Ding et al. (2012) ¹⁴	Mid-clavicle Acute fracture	Not stated	Subclavian arteriovenous fistula	Medial screw perforated artery	Pulsatile supraclavicular mass and arm weakness at 3 months	Vascular graft	Full recovery
Bain et al. (2010) ¹⁵	Mid-clavicle Non-union	Not stated	Pseudoaneurysm	Medial screw perforated artery	Arm pain, claudication and pallor at 6 years	Vascular stent	Full recovery
Shackford (2003) ¹⁶	Mid-clavicle Non-union	2 years	Pseudoaneurysm	Screw immediately lateral to the fracture site perforated artery	Pain and paraesthesia medial arm at 8 years. Acute presentation of critical arm ischaemia at 10 years	Vascular graft	Full recovery
Casselman et al. (1997) ¹⁷	Mid-clavicle Delayed union	Not stated	Pseudoaneurysm	Screw medial to fracture site perforates artery	Claudication at 8 years	Vascular graft	Full recovery
Johnson & Thursby (1996) ²¹	Mid-clavicle Acute fracture	Not stated	Pseudoaneurysm	Second most medial screw perforated artery	Claudication at 22 months	Vascular graft	Returned to full activity. Moderately reduced forearm arterial pressures after exercise

Figure 2. (A) Plain radiograph of left clavicle (AC, acromion; CL, clavicle, R, first rib). The arrow depicts a prominent medial screw lateral to the 1st rib. (B) Arteriogram (AA, axillary artery; SA, subclavian artery). The arrow depicts penetration of the axillary artery by the prominent medial screw. A pseudoaneurysm has formed. The axillary artery is filled with contrast distal to the pseudoaneurysm. (©International Journal of Shoulder Surgery. From Bain GI, Galley IJ, Keogh ARE and Durrant AV. Axillary artery pseudoaneurysm after plate osteosynthesis for a clavicle nonunion: a case report and literature review. *Int J Shoulder Surg* 2010; 3: 7982.).

The injury may be a direct puncture or a result of vessel abrasion against screw during postoperative arm movement.²¹

Pseudoaneurysm. A pseudoaneurysm (Figure 2) is an extravascular haematoma that freely communicates with the intravascular space through a defect in the vessel wall.³⁴ The wall is formed by the compressed tissues surrounding the haematoma, as distinct from the three layers of arterial wall that surround a true aneurysm.^{8,35}

Pseudoaneurysms are most frequently seen following femoral artery puncture (0.05% to 4%).³⁶ They enlarge slowly and may be clinically silent for many years until they cause local compressive symptoms or present with embolic symptoms.^{17,36,37} All four pseudoaneurysm patients (Table 1) presented with late upper limb claudication between 2 years and 10 years following surgery. Three had an acute deterioration to limb threatening ischaemia as a result of emboli to the upper limb.^{15,16,21}

In each case, the pseudoaneurysm was treated with a vascular stent or graft procedure and the outcomes were reported as excellent.

Arteriovenous fistula. Acquired arteriovenous (AV) fistulae are the result of a penetrating injury to the walls of both the artery and vein.¹⁴ With time, the arterial wall is weakened and the fistula dilates. The pressure in the venous side of the fistula increases, causing swelling and venous engorgement. Left untreated, persistent venous hypertension can result in congestive heart failure or limb threatening ischaemia.¹⁴

The reported case of AV fistula¹⁴ presented as a pulsatile supraclavicular mass and ipsilateral arm weakness 3 months post clavicle fixation.

Venous injury

By contrast to arterial injuries, the two reported venous injuries were identified intra-operatively. One case was a result of tearing during mobilization of the bone

fragments²²; minimal details were provided for this case, although the patient's reported outcome was good. In the second case, the patient developed a fatal air embolism as a result of the vessel injury.¹³ Profuse bleeding was noticed on removal of the drill from the medial plate hole. A Bristow elevator had been placed under the clavicle as drill stop, which may have deflected the drill. The patient went into shock despite aggressive fluid resuscitation and control of the bleeding. Twenty millilitres to 30 mL of air was aspirated from the right atrium. The coroner's report identified a 25-mm tear of the subclavian vein.

Venous air embolism. An air embolism occurs when air or gas is admitted into the vascular system.³⁸ It most commonly occurs with central venous catheterisation but is also well recognized in posterior cranial fossa surgery,³⁹ total hip arthroplasty and prone spinal surgery.^{40,41}

As a result of the negative intrathoracic pressure, the lumen of the subclavian vein also has a negative pressure. Any breach of the vessel wall creates a pressure gradient, causing any surrounding air to be sucked through the defect and into the lumen.⁴²

The right ventricle struggles to pump the compressible air, creating an 'air lock'.⁴³ This reduces cardiac output, which can lead to cardiac and cerebral hypoperfusion and even complete cardiovascular collapse.^{38,40,41,44}

Similar to an embolism from any other cause, air in the pulmonary circulation leads to pulmonary vasoconstriction, release of inflammatory mediators, bronchoconstriction and ventilation/perfusion mismatch.⁴⁰ If there is a patent foramen ovalae, the embolus has the potential to enter the cerebral circulation.⁴⁰

The first signs of an air embolus in an anesthetized patient will be a decrease in end-tidal carbon dioxide, decreased arterial oxygen saturation and hypercapnoea. The effects of an air embolism depend on both the rate and the volume of air entering the circulation.^{38,40} The lethal volume of air is estimated to be 200 mL to 300 mL (3 mL/kg to 5 mL/kg). The closer the vein to the right heart, the smaller the lethal volume becomes.⁴⁰

Deep vein thrombosis (DVT). There are multiple reports of DVT associated with non-operative management of clavicle fractures.^{9,45–47} However, we could only find one report of DVT associated with clavicle fracture surgery,⁴⁸ and that report expressed some doubt as to whether the DVT was a result of the surgery or an underlying form of TOS characterised by venous thrombosis (Paget–Schroetter syndrome).

Given the lack of evidence, we cannot comment further on the pathoanatomical factors involved in DVT following clavicle fracture surgery.

Brachial plexus injury

The reports of brachial plexus injury following clavicle fracture surgery are summarized in Table 2.

All reported cases of plexus injury had the same presentation of severe radicular pain in the immediate postoperative period, most commonly in the arm and shoulder.^{18–20} Sensory loss and paralysis was usually profound but incomplete.

The brachial plexus is susceptible to injury through traction, compression and entrapment.⁴⁹ The inflammatory reaction associated with a fracture can cause any adjacent tissues, including the plexus, to become adherent to the fracture fragments and callus.¹⁸ Reduction of bone fragments will generate traction force on any adherent plexus elements.

Jeyaseelan et al. reported a series of 21 cases of brachial plexus injury following delayed fixation of a displaced clavicle fracture (mean time to surgery was 19 days post initial injury).¹⁸ The brachial plexus was tethered by scar to the posterior surface of the fracture fragments in all but one case, where it was tethered between two prominent screws. It was proposed that the traction injury occurred during fracture reduction.

Jeyaseelan et al. also noted that the clavicle was fixed in a shortened position (>0.5 cm) in 24% of cases.¹⁸ They reported that this provided an additional compressive mechanism to the plexus injury.¹⁸ Other compressive injuries have been attributed to hypertrophic callus²⁰ and to a malunited bone fragment.¹⁹ This fragment was finally demonstrated by three-dimensional computed tomography (3D CT), after plain radiographs and magnetic resonance imaging had failed to identify it.¹⁹

Transient brachial plexus traction injury has been reported after intramedullary fixation of acute clavicle fractures.²⁶ The proposed mechanism was traction that occurred during reaming of the fragments.

For both the plexus compression case reports^{19,20} and the intramedullary fixation series,²⁶ it was noted that only a minimal amount of subperiosteal dissection was performed at the time of initial surgery to preserve the blood supply.

Jeyaseelan et al. recommended that, in cases of delayed fixation, all adherent soft tissues must be thoroughly released from the inferior clavicle before the fragments are mobilized.¹⁸ This is in direct contrast to other studies emphasizing the need for minimal soft tissue stripping to preserve the blood supply.⁵⁰

Recovery from postoperative brachial plexus injury occurs over many months and the final outcome is variable. All three cases reported by Ring and Holovacs²⁶ were managed non-operatively and had complete resolution of symptoms within 6 months. In the series reported by Jeyaseelan et al., all patients had a neurolysis performed.¹⁸ Four patients had nerve ruptures that were treated with nerve transfers or grafts. By 12 months, 71% of the 21 patients had recovered their Medical Research Council power grade by 4, and a further 14% by 3.¹⁸

TOS

TOS represents a group of disorders characterized by either nerve (brachial plexus) or vascular (subclavian/axillary vein or, less commonly, subclavian/axillary artery) compression at the thoracic outlet.³¹ Although the scalene triangle is the most common location of compression across all forms of TOS, in cases involving clavicle fracture, the costoclavicular space is most common region of compression.

TOS has been well described following non-operative management of clavicle fractures. The factors involved are abundant callus and infero-lateral displacement of the lateral fragment, which compromises the costoclavicular space.^{10,18,31} TOS as a result of fracture callus has also been reported following clavicle fixation^{23–25} (Table 3). Over reduction of the fracture in the presence of abundant callus and/or comminuted fragments may introduce an additional compressive factor in these cases.

In two of the cases,^{23,24} the patients had symptoms within 24 hours of their fixation surgery. In one of these two cases,²⁴ the callus was not visible on radiographs, although it was presumed to be present but not yet calcified. The third case²⁵ presented 5 months following fixation of an acute fracture sustained in a motor vehicle accident. It could be argued that this case is probably not a true iatrogenic TOS: a delay of this length is more typical of TOS following non-operative management,¹⁰ suggesting the compression was the result of a callus that formed after the fixation procedure, rather than mobilization of a pre-existing callus during fracture reduction.

In all cases, the patients had angiographic evidence of normal flow in the subclavian/axillary vein following decompression of the costoclavicular space.^{23–25}

Table 2. Iatrogenic brachial plexus injuries associated with clavicle fracture surgery.

Author	Diagnosis	Complication	Cause	Presentation	Treatment	Outcome
Ring & Holovacs (2005) ²⁶	Acute mid-clavicle fracture (1 week to 3 weeks)	One complete Two upper trunk	Traction on plexus during reaming of intramedullary nail	Postoperative weakness and sensory disturbance	Observation only	Full recovery by 6 months post injury
Namdari et al. (2012) ¹⁹	Mid-clavicle non-union 9 months	Incomplete C5-T1 injury	Malunited fragment compressing plexus after plating and bone grafting	Postoperative neck and arm pain with weakness	Excision of bone spike	Immediate pain relief. Partial motor recovery at 4 months
Thavarajah et al. (2013) ²⁰	Mid-clavicle hypertrophic non-union	Incomplete global injury	Hypertrophic callus compressing plexus following plating and bone grafting	Postoperative pain and paralysis	Neurolysis	Power recovered to MRC grade 4/5. Duration of follow-up not reported
Jeyaseelan et al. (2013) ¹⁸	Mid-clavicle, medial and lateral fractures. 4 days to 31 days from injury	Plexopathy following plate fixation (21 cases)	Traction injury to plexus. A result of reduction of displaced bone fragments without release of adherent tissues	Postoperative pain and paralysis. 90% involved upper plexus distribution	Neurolysis. four cases of nerve rupture treated with nerve transfer or grafts	At 12 months: pain resolved 100% Significant sensation improvement – 86%. ≥ MRC grade 4 power – 76%

MRC, Medical Research Council.

Table 3. Iatrogenic thoracic outlet syndrome associated with clavicle fracture surgery.

Author	Diagnosis	Injury to surgery interval	Complication	Cause	Presentation	Treatment	Outcome
Skedros et al. (2010) ²⁴	Mid-clavicle delayed union, multiple failed fixation procedures	52 days from initial fracture, 26 days post revision procedure	TOS with total SCV occlusion	Mobilised callus compressed vessel	Altered sensation immediately post-operatively Progressive swelling and intermittent cyanosis on day 1 to day 14	Revision fixation with curved plate and intercalary graft	Returned to full activity, subtle upper extremity fatigue when hammering
Papagelopoulos et al. (2005) ²³	Mid-clavicle non-union	18 months	TOS with vascular and neurological involvement	Not identified	Altered sensation, venous congestion and diminished radial pulse when standing. Day 1	Revision fixation with curved plate and intercalary graft	Excellent at 2 years
Sheikh & Bashir (2005) ²⁵	Mid-clavicle acute fracture	Not stated	TOS with SCV compression	Callus formation	pain, swelling and discolouration of upper limb, beginning 4 months post fixation	Scalenectomy, first rib resection and staged SCV angioplasty	Not stated

TOS, thoracic outlet syndrome; SCV, subclavian vein.

Management of neurovascular injuries in clavicle surgery

Vascular injury

Arterial injury. Iatrogenic arterial injury does not present peri-operatively but rather as subcritical ischaemia between 4 months and 10 years following surgery. Often, there was a history of claudication, which progressed to critical ischemia. A patient with a previous history of clavicle fixation, who presents with subcritical ischaemic symptoms in the upper limb, should have an expedient angiogram and referral to a vascular surgeon.

Venous injury. The subclavian vein is at significant risk during clavicle surgery because it is adjacent to the clavicle, is thin-walled and has a substantial blood flow. Profuse bleeding may be apparent but can also be concealed in the chest cavity, which would manifest as uncontrollable hypotension.

Once the injury is diagnosed, the two main goals are to gain rapid control of the bleeding and prevent air entering the circulation. An urgent vascular review should be sought because the surgical repair of a subclavian vein represents a demanding procedure that may require grafting.

Air can be excluded from the field of injury by submerging the wound in saline or applying an occlusive dressing to the wound. The pressure gradient can be reduced by positioning the patient head down and applying positive pressure ventilation.⁴⁰

Established air embolism. Repositioning must still be performed because the greatest risk to the patient is continued entrainment of air.⁴⁰ An attempt to aspirate air from the right atrium can be made if a central venous line is in place. Aggressive fluid loading should be administered. In the event of cardiovascular collapse, the standard measures of cardiopulmonary resuscitation may also help clear the embolism from the right ventricle.^{38,40,41} There is conflicting evidence that placing the patient in a lateral decubitus position (Durant manoeuvre) may help reduce the air lock phenomenon.^{38,40,41}

Brachial plexus injury

All reports of significant brachial plexus injury following clavicle fracture surgery have a characteristic presentation of unremitting radicular pain, profound weakness and sensory loss in the immediate postoperative period. The lesion is usually incomplete, with the upper trunk and suprascapular nerve most commonly involved. A 3D CT scan may help identify any prominent bone compressing the plexus.

Significant postoperative plexopathy has severe symptoms and a variable outcome. We therefore recommend urgent revision surgery, including the support of a brachial plexus surgeon as neurolysis, repair, transfer or grafting may be required.

TOS

Acute iatrogenic thoracic outlet syndrome presents in the immediate postoperative period. In both reported cases, the clavicle fixation was revised with specific emphasis on using intercalary graft and plates with an apex-superior contour to lengthen the clavicle and increase the volume of the costoclavicular space.

Prevention of neurovascular injury

Recommendations

Based on this review of the literature, we present recommendations to prevent these injuries.

Pre-operative planning. We consider the following as higher risk for iatrogenic injury: a medial fracture, comminution, a 2-week to 4-week delay from the time of injury, or non-union, malunion and revision cases. It is now our practice to obtain a pre-operative 3D CT angiogram in complex cases to define the exact location of the vessels relative to the clavicle. Excessive callus that may result in compromise of the costoclavicular space should be identified.

Communication with anaesthetists. Before the operation, the anaesthetic staff must be alerted to the potential for significant vascular injury to occur. This warning should be repeated immediately prior to performing the highest risk steps of the procedure (e.g. drilling and screw insertion).

Positioning. Regardless of the patient position chosen, the table set up must allow the surgeon to rapidly alter the position in the event of an emergency.

Dissection. The chosen plane of dissection should take into account that the subclavian vein may be adherent to the posterior periosteum of the medial clavicle. It is our strong recommendation that all dissection around the medial and inferior clavicle be performed in the subperiosteal plane. Any adherent soft tissue must be released from the fracture fragments to prevent inadvertent traction injury during reduction. The periosteum in this area must not be penetrated.

Fixation. In the medial third of the clavicle, the safest direction for screws is from superior to inferior and in

the middle third it is from anterior to posterior.²⁷ The correct screw length is unlikely to be greater than 18 mm.²⁸ If the drill hole is made eccentrically on the clavicle, this length may be significantly shorter. Sharp drills must be used and great care must be taken to avoid plunging when drilling the far cortex. We caution against the use of blunt retractors below the clavicle.

Conclusions

Although rare, limb or life threatening neurovascular complications from clavicle surgery have potentially devastating implications for young, active patients who typically undergo surgical management of these fractures.

Procedures on the medial half of the clavicle pose the greatest risk of vascular complications. The thin walled vein may be adherent to the posterior periosteum. Venous injuries produce profound bleeding, which may be into the chest and present as hypotension. This requires urgent repair and the prevention of air embolus.

By contrast, arterial injuries usually present years following the surgery as a pseudoaneurysm, with a mass in the supraclavicular fossa and/or embolic symptoms including claudication and critical ischaemia. The most common reported mechanism of iatrogenic brachial plexus injury is a traction injury as a result of mobilization of fragments with adherent plexus elements.

To avoid significant iatrogenic neurovascular injuries, the surgeon must have a detailed understanding of the neurovascular anatomy of the region. A thorough pre-operative planning must be performed and the anaesthetic staff must be warned of the potential risk for significant injury. All dissection around the medial and inferior clavicle should be performed in the subperiosteal plane. Screws should be placed along a safe trajectory and screw length should be accurately measured.

Previous presentation

The present study is not based on a previous communication to a society or meeting

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