Case Report

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Entrapment of the Suprascapular Nerve Due a Complex Scapular Fracture: A Case Report

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Abstract

Introduction: Suprascapular nerve (SSN) entrapment is rare. Direct contusion or traction of the SSN may occur due to fracture fragments within the narrow scapular notch. Decompression of contused or entrapped SSN may release weakness of supraspinatus/infraspinatus muscles when recognized in time.

Case report: A young male patient with a complex fracture of the right scapula suffered from an additional lack of muscle activity (M0/5 = complete paralysis of the supraspinatus/infraspinatus muscles) innervated by the SSN. The fracture itself was no indication for surgery but involved the scapular notch, resulting in entrapment of the SSN. Based on the clinical and CT findings, surgical open exploration of the SSN was performed. By careful dissection, the fragments of the fracture were identified pushing over each other like scissors and squeezing the SSN. Continuity was preserved. Release of the SSN was achieved by widening the scapular notch. 6 weeks postoperatively, the SSN has recovered with a muscular activity of M3 for external rotation and M4 for abduction.

Discussion: SSN entrapment in the differential diagnosis of complex scapular fractures involving the scapular notch has to be considered. Emergency imaging (X-ray, CT) will only demonstrate the bone structure - a scapular fracture, threatening the SSN in the scapular notch by sharp fragments, must be assumed in case of pain related weakness or paralysis.

Conclusion: The judgement of a fracture alone is not sufficient to indicate surgery. In case of weakness of the corresponding muscles, clinical suspicion or uncertainty, exploration of the fracture site including decompression of the involved nerve is recommended. With prompt decompression relieve of symptoms may occur. Without exploration, compression of the SSN may have been missed and the nerve may have been further damaged by the fracture.

Keywords: Suprascapular nerve; Neuropathy; Posttraumatic entrapment; Decompression; Incisura scapulae.

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Introduction

Anatomy: The suprascapular Nerve (SSN) arises from the brachial plexus (origin C4 to C5 cervical spine roots) [1], as a mixed motor and sensory nerve. Motorically, it supplies supraspinatus and infraspinatus muscles and carries sensory pain fibers for the glenohumeral and acromioclavicular joints. SSN has no sensory fibers for the skin. After exiting the upper trunk of the brachial plexus, it runs along the lateral edge of the scapula on the superior border of the scapula to the scapular notch. The scapular notch is a small depression at the upper edge of the scapula and forms a tunnel which is bounded towards the top by the superior transverse scapular ligament (Figure 1). The SSN and suprascapular vein run under the superior transverse ligament whereas the suprascapular artery runs above the ligament [2]. Due to this natural narrowing, the scapular notch is the most common site where the SSN is compressed, thus causing nerve injury and entrapment [2,3].

Nerve injuries: There are three categories of nerve injuries: neurapraxia (least severe, involves focal damage of the myelin fibers around the axon, without axonal damage), axonotmesis (more severe, involves injury to the axon itself), and neurotmesis (complete disruption of the axon, little likelihood of normal regrowth) [4]. The different mechanisms of nerve injury include direct pressure, repetitive microtrauma, and stretch or compression. The degree of injury is related to the severity (neurapraxia, axonotmesis, neurotmesis) and very important to the extent (time) of compression [4,5].

Etiology/clinic: Abnormalities, that may indicate a SSN injury are vague pain at the posterolateral shoulder joint [6], abnormal muscle strength by external rotation and lateral abduction [4] with atrophy of the supraspinatus and infraspinatus muscles [2]. Entrapments of the SSN are often due to direct compression in the scapular notch or compression of the nerve distal to the scapular notch by a dorsally located ganglion from the glenohumeral joint. Mostly, there is a history of repetitive microtrauma during overhead sports [7] or massive cuff tears with significant tendon retraction [1]. There are different risk factors (basketball, volleyball) [8]. Patients then suffer from deep diffuse pain in the posterior and lateral region of the shoulder with partial radiation into the arm. In the course of the disease, the muscles may atrophy and result in weakness of external rotation and abduction with pronounced atrophy when not compensated by the deltoid muscle [3]. The occurrence of entrapment in association with trauma is even rarer. Direct trauma to the SSN is very unlikely due to its coverage by the trapezius muscle and the protection of the intact scapular notch. Much more likely are traction injuries caused by, for example, complex movements of the scapula in the presence of fractures. This is also be expected in our case report [3]. Still rare, yet the diagnosis of suprascapular neuropathy is becoming more common [9]. The literature predominantly describes damage of the SSN due to chronic exposure during sports injuries and repetitive movements. There is only a low association of SSN neuropathy with massive rotator cuff tears (RCT) and no evidence to support a routine SSN release when RCT repair is performed [10]. Less frequently, however, direct trauma causes SSN injury [11].

Traumatic injuries: Traumatic injuries (scapular fractures, clavicular fractures, proximal humeral fractures, and dislocation of the acromioclavicular or shoulder joint) are potential risk factors for compression or traction injury of the SSN in the notch [2,12,13]. Traumatic conditions include scapula fractures (scapular notch fractures) [14], shoulder dislocation, massive cuff tear, distractive trauma and penetrating trauma [15]. A CT scan may be useful to detect fractures that might compress the nerve [15,16].

Scapula fracture: Fractures of the scapula in young patients are predominantly caused by high energy trauma with significant associated injuries to the thorax, spine, head, or abdomen [17]. The indications for scapular fracture surgery are changing with the increasing understanding of symptomatic malunion, improved technologies and increase in patients. Indications for surgery are based on angular deformity and displacement [18]. Three-dimensional diagnosis by CT scan has improved the possibility for understanding the fractures and allows accurate measurement of displacement and angulation [9,18]. In some cases, surgical indications are based on concomitant upper limb lesions or, as in our case, isolated SSN compression. Surgical treatment should be considered for injuries that affect the function of the glenohumeral or scapulothoracic joints. Surgical interventions have at least as good results as non-surgical treatments. Indications include lateral column displacement of at least 25 mm, angular deformity of at least 45°, intra-articular steps of 3-5 mm or double disruptions of the superior suspensory complex [17].

Treatment: For entrapments, patients may often be treated with non-invasive measures; for injuries that do not respond to conservative treatment, surgery may be recommended after 3-6 months. Surgical intervention is also considered for patients with nerve compression by an external source [19]. Decompression of the SSN may be accomplished through an open approach, although arthroscopic surgical approaches have become more common in the past several years. This is especially true if the patient has an identifiable cause for the nerve compression, as in our patient example [20]. The arthroscopic approach is also used under ultrasound guidance. Ultrasound ensures reliable non-invasive localisation of the SSNs. The arthroscope may be used to visualise and release the nerves in a three-dimensional controlled manner. However, you need at least 2 portals to decompress the nerve. The arthroscopic instruments are very effective, but the procedure also carry the risk of neural injury especially in scarred areas or if fracture fragments are present [21].

Synopsis: SSN entrapment is rare and, like scapular fracture, occurs mainly in patients under 40 years of age. The medical history is very important. In the absence of trauma, the ligament is more likely to affect the nerve. In chronic problems, the location of the muscle atrophy may help to localize the cause of the entrapment. Ganglia often cause isolated atrophy of the infraspinatus muscle. If the ligament is causative, then combined atrophy of the supraspinatus and infraspinatus muscles usually occurs [3].

In the literature, scapular fracture is described as a rare but well-known cause of SSN injury [11]. If there is a history of trauma, SSN injury should be considered in the differential diagnosis, with persisting shoulder pain [11].

Case report

A 29-year-old right-handed patient was referred by the ambulance service after a brawl. Diagnosis reveals a complex right shoulder fracture and right dorsolateral rib series fracture (6th-8th rib). The clinical examination shows failure of the SSN with a lack of muscle activity (M0/5 = no muscular activity, complete paralysis) of the muscles innervated by SSN (supraspinatus/infraspinatus muscles). There was no paresthesia or loss of sensation in the affected right limb. CT examination revealed a high degree of suspicion of traumatic lesion of the SSN in a scapular corpus fracture through the scapular notch. With regards to the type of fracture, no surgery was indicated. However, the fracture involves the scapular notch (Figure 2), resulting in entrapment of the SSN. Nerve exploration and decompression was performed based on the clinical and CT findings.

Due to a COVID-infection, surgery was delayed. On the 9th day after the trauma, the SSN was explored, seen in continuity and decompressed. Surgery was performed by an inter disciplinary team of peripheral nerve/hand surgeons and orthopedic surgeons.

Technique: By palpation of the scapular spine and the AC joint, a longitudinal incision over the shoulder (coup de sabre) and the scapular notch was performed. After the dissection down to the trapezius muscle, the trapezius is split in the fiber course and detached from the scapular spine medially to the acromion. Under the trapezius muscle, the supraspinatus muscle was exposed. The trapezius muscle was held to the side medio-cranially. The dissection was continued above the supraspinatus muscle up to the superior border of the scapula. The supraspinatus muscle was retracted. The scapular notch could now be palpated. The transverse superior scapular ligament was severed. The scapular notch was completely displaced by two overlapping bone fragments. The fracture fragments push over each other like scissors and squeeze the SSN. The medial fragment of the scapula was reduced under traction. After this maneuver, the SSN was identified at the scapular notch. After careful dissection, there was compression of the SSN by the fracture fragments. The continuity of the SSN was preserved (Figure 3). The nerve was dissected free over 6 cm. The nerve only shows discrete signs of compression at the scapular notch. The epineurium and the fascicular structures were intact without interruption. However, the set reduction of the fracture fragments could not be maintained spontaneously. The scapular notch was therefore extended medially along the upper edge to enlarge the scapular notch. Release of the SSN was achieved by widening the scapular notch by using a bone punch. The insertion of the omohyoid muscle was partially disinserted. After the incision has been extended medially by about 1 cm, the branch to the supraspinatus muscle is free of tension. Dynamic examination with mobilization the arm showed no longer compression of the SSN. There has still been a lateral part of the SSN demonstrating slight tension when running over the bone. The lateral bone edges were rounded off with a rasp. Now the SSN was enveloped by Tisseel[®] (Baxter). After rinsing the wound hemostasis was performed. The wound was closed in layers with readaptation of the trapezius muscle using multiple Z-sutures (Vicryl[®] 1-0). Superficial wound irrigation and control of hemostasis were performed. The skin was closed with subcutaneous (Vicryl® 3-0) and intracutaneous sutures with resorbable suture material (Monocryl[®] 3-0). Postoperative restriction of active shoulder abduction (only passive abduction/flexion up to 80°) was established for six weeks. Six weeks postoperatively, ENMG diagnostics and clinical followup were performed.

Clinical, radiological and ENMG examination 6 weeks postoperatively

Six weeks postoperatively, the patient reported occasional pain in the right shoulder and a slight restriction of shoulder mobility after relative immobilisation. He had consistently adhered to the adapted gilchrist orthosis for 6 weeks. Regular passive physiotherapy were performed.

Findings, right shoulder: Inconspicuous scar (Figure 4), no perifocal pain on pressure. Clinically, there were no muscle atrophy. Passive glenohumeral mobility was almost free. Active arm abduction could be performed spontaneously (M4) up to 110°. Arm adduction and internal rotation was possible with M5. External rotation was attenuated with M3 to 60°. Elbow flexion/ extension, finger flexion/extension, finger abduction/adduction, thumb opposition were estimated M5. There was a normal muscle tone without fasciculations, symmetrical sensitivity to the opposite side, and symmetrically resolvable BSR, TSR and BRR. Global shoulder function included flexion/extension of 140-0-30°, abduction of 110° and external rotation of 60°. Active external rotation was slightly weakened compared to the opposite side, the infraspinatus muscle could be well activated. Axillary nerve and deltoid muscle function were intact.

X-ray: unchanged fracture anatomy, increasing consolidation. Glenohumeral joint centered.

ENMG: The ENMG showed borderline pathological spontaneous activity in the needle myography of the supraspinatus muscle. There was abundant pathological spontaneous activity in the infraspinatus muscle. Electrophysiologically, there was evidence of an axonal lesion of the right SSN with particular involvement of the infraspinatus muscle and abundant evidence of denervation.

Procedure: Adequate course of healing. The gilchrist orthosis could be completely removed and the mobility of the right shoulder could be fully trained with physiotherapy, maximum additional weight bearing of 2-3 kg for a further 6 weeks, followed by successive strength building. 100% incapacity for work was certified until next consultation. Clinical and radiological follow-up 4 months postoperatively was foreseen.



Figure 1: Anatomy of the scapular notch. SSN runs under the superior transverse ligament (green arrow), suprascapular artery runs above the ligament.





Figure 2: 3D reconstruction of the CT examination (view from A: dorsocranial, B: ventromedical, C: Cranial, D: DorsalE: Lateral). Incisura scapulae (green arrow). Ac Dislocation with elevation of the lateral clavicle end (by Approximatly 9 mm), consistent with a tossy lesion II- III. Minimally dislocated fracture of the lateral clavicle end. Multi-fragmentry fracture of the acromion and scapula (IIB). Centered position of the humeral head in relocation to the glenoid fossa. Dislocation of the fracture over a distance from the green to the red star (Normally the green and the red star fit over each other).



Figure 3: Intraoperative images of the decompression of the suprascapular nerve. Visualization of the fracture fragments. Picture A shows the SSN pulling through the incisura scapulae. Picture B shows the sharp fragments of the fracture over which the SSN is stretched. Picture C shows the suprascapular artert (green arrow) and the SSN (green arrow) after decompression by extension and widening of the scapular notch.



Figure 4: Scar conditions after 1 month postooperatively. Access. Coup de sabre.

Conclusion: There was clinical and electrophysiological evidence of an axonal lesion of the right SSN with affection of the infraspinatus muscle 6 weeks postoperative. The SSN demonstrated recovery with M3 muscle activity for external rotation and M4 activity for abduction compared to no muscle activity (M0/5 = no muscle activity) preoperatively.

Discussion

To our knowledge, there are only a few case reports of SSN entrapment due a scapula fracture involving the scapular notch [22,23]. This represents the only recent case report of post-traumatic entrapment of the SSN due to a scapular fracture. Although SSN entrapment is an unusual presentation after scapula fracture, proper diagnosis and treatment are critical to prevent chronic and irreversible supraspinatus and infraspinatus muscle weakness and atrophy.

SSN injury due to trauma has also been reported (shoulder dislocation, humeral neck fracture, scapular fracture, clavicular fracture) [24]. Scapula fractures through the superior lateral angle of the scapula potentially involve the scapular notch. These are type 111 fractures, according to the classification of DeCoulx el al. 1956 [23]. Solheim et al. [22] reported in 1978 a case report of a 32-year-old man with a fracture of the scapular notch associated with a lesion of the SSN. Decompression surgery of the nerve was performed 20 months after the injury. The result demonstrated pain relief.

In our case, there was a division of the SSN with a branch leading to the supraspinatus muscle before the scapular notch contributing to the rapid recovery of the supraspinatus muscle function. This fact shows how important it is, to take anatomical variations into account. Primary wide resection of the scapular notch to avoid nerve compression symptoms has been described as a surgical intervention but not in fractures [22]. An EMG examination is recommended in cases of impaired supraspinatus muscle function in combination with a fracture of the scapular notch to diagnose SSN injury [23].

In addition to the open decompression, there is the option of decompression under combined arthroscopic and ultrasound guidance. This technique is well described and gaining more and more popularity [21]. In our case, this was not an option because the fracture required readiness for a stabilizing osteosynthesis. If the nerve had still been entrapped after enlargement of the incisura scapulae, osteosynthesis would have been necessary to ensure decompression. Furthermore, it had to be possible that microsurgical suture was required in a potential transection of the neural structure. This extension of surgery would have been difficult under combined arthroscopic and ultrasound guidance.

SSN entrapment in the differential diagnosis of complex scapular fractures involving the scapular notch has to be considered. Emergency imaging (X-ray, CT) will only demonstrate the bone structure - a scapular fracture, threatening the SSN in the scapular notch by sharp fragments, must be assumed in case of pain related weakness or paralysis.

Conclusion

SSN entrapment is an uncommon but possible lesion in combination with a scapular fractures. SSN entrapment in the differential diagnosis of complex scapular fractures involving the scapular notch has to be considered and emphasizes the importance of prompt decompression when clinical symptoms indicate a correlation. The judgement of a fracture alone is not sufficient to indicate surgery. In case of weakness of the corresponding muscles, clinical suspicion or uncertainty, exploration of the fracture site including decompression of the involved SSN is recommended. With prompt decompression relieve of symptoms may occur. Without exploration, compression of the SSN may have been missed and the nerve may have been further damaged by fracture fragments.

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