Diagnosis and Treatment of an Axillary nerve injury

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Abstract

The diagnosis of axillary nerve injury can be challenging as a result of asymptomatic or subclinical presentation. While a complete and thorough physical examination should detect the identifying patterns of nerve injury, deltoid dysfunction initially may be masked in young patients with excellent compensatory mechanisms or those with difficulty complying with the examination. Objective: To determine the efficacy of nerve transfer procedures in the setting of axillary nerve palsy and factors affecting clinical outcomes. The purpose of this article is to diagnosis and treatment of an axillary nerve injury.

KeyWords: Diagnosis, Nerve Injury, Axillary Nerve, musculocutaneous nerve.

DOINumber: 10.14704/NQ.2022.20.12.NQ77351 **NeuroQuantology2022;20(12):3417-3421**

Introduction:

The axillary nerve is one of the terminal branches of the posterior cord of the brachial plexus and its fibers are usually derived from the ventral rami of the fifth and sixth cervical nerves (1). It lies behind the axillary artery and vein, superior to the radial nerve, and lateral to the median and ulnar nerves. The relation of the axillary nerve with other branches of the brachial plexus is relatively constant but with the musculocutaneous nerve, may vary. While **Tubbs et al.**(2)revealed the axillary nerve as the most superior of structures, **Apaydin et al.**(3) demonstrated the musculocutaneous nerve as the most superior in 20% of cases.

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Relevant conflicts of interest/financial disclosures:
The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Pathophysiology of Nerve Injury:

The severity of peripheral nerve injuries is graded primarily on the basis of the amount of axonal discontinuity observed. The 2 most commonly referenced systems that are used to describe nerve injury are the Seddon classification system and the Sunderland classification system **(Table 1)** (4).

Table (1): Seddon and Sunderland Classification Systems for Peripheral Nerve Injuries (4).

Seddon	Sunderland	Injury Type	Neurological Deficits
Neurapraxia	I	Intrafascicular edema with conduction block, possible segmental demyelination	Neuritis, paresthesias
Axonotmesis	II	Severed axon, intact endoneurium	Paresthesia, episodic dysesthesias
	III	Severed axon, disruption of endoneurium	Paresthesia, dysesthesia
	IV	Disruption of endoneurium and perineurium	Hypoesthesia, dysesthesia, neuroma
Neurotmesis	V	Complete nerve discontinuity	Anesthesia, intractable pain, neuroma
	VI	Combination of above	Mixed presentation

Patient Evaluation

The diagnosis of axillary nerve injury can be challenging as a result of asymptomatic or subclinical presentation. While a complete and thorough physical examination should detect the identifying patterns of nerve injury, deltoid dysfunction initially may be masked in young patients with excellent compensatory mechanisms or those with difficulty complying with the examination (5).

Injuries resulting in incomplete paralysis may not be obvious when only the anterior or posterior portion of the deltoid muscle is affected. Additionally, in some cases, the patient may retain nearly full abduction of the shoulder secondary to supraspinatus compensation. Clinical Examination Evaluation begins with a thorough history. A recent history of blunt trauma or shoulder dislocation makes an axillary neurapraxia more likely, whereas penetrating trauma or recent surgery may point toward laceration of the nerve. It is important to note that a patient age of 50 years and a lack of concentric glenohumeral reduction for 12 hours are known risk factors for axillary nerve injury following shoulder dislocation (6).

While the testing of deltoid muscle strength can be used to determine axillary nerve function, patients who have had a recent operation or traumatic episode may have difficulty performing such testing, which may present a challenge when pain prevents a comprehensive physical examination (7).

Special physical examination techniques have been described to aid in the identification of isolated deltoid muscle dysfunction. The swallow-tail test **(Fig. 1)** is performed by having the patient bend forward at the waist and maximally extend the shoulders, first with elbows extended and then with elbows flexed. A difference in shoulder extension between the normal and affected sides of .20° indicates a positive test (8).

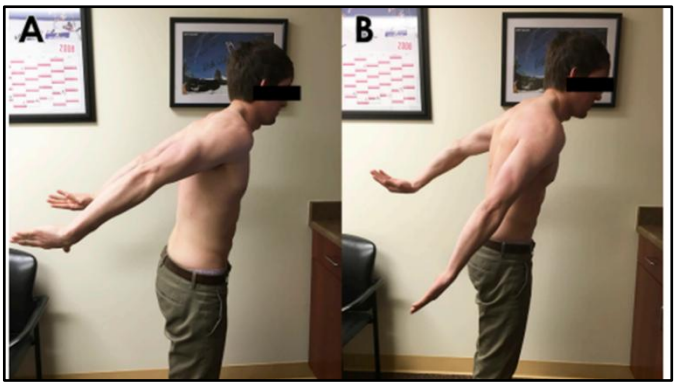

Figure (1): Photographs illustrating the swallow-tail test. In a normal test **(Fig. 5-A),** both shoulders are fully extended to the same level. However, in an abnormal test **(Fig. 5-B),** the posterior fibers of the deltoid muscle fail to fire, thus limiting full extension of the affected shoulder (8)

The deltoid extension lag test involves the examiner maximally extending the shoulder and elbows of the patient and then asking the patient to hold that position. A lag in shoulder extension on the affected side as compared with the contralateral side is considered a positive test (9).

The abduction in internal rotation test **(Fig. 2)** is a preferred method for evaluating deltoid muscle function as it has been shown to be superior to both the deltoid muscle extension lag test and swallow-tail test (1).

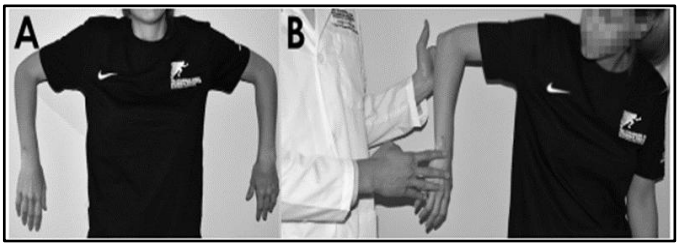

Figure (2): Photographs illustrating the abduction in internal rotation test. **Fig. 6-A** The patient is instructed to abduct and internally rotate the shoulder actively with both elbows flexed. **Fig. 6-B** If any discrepancies are noted, the examiner repeats the maneuver (passively) and the patient is instructed to maintain the position. If the patient is not able to do so, the test is positive(10).

It is performed by placing the patient's shoulder in full internal rotation with the elbow flexed and asking the patient to abduct the shoulder. If the patient is unable to fully abduct the shoulder in this position, the examiner assists with abduction and asks the patient to maintain this position. Any abduction lag indicates a positive test. The internal rotation of the shoulder in this maneuver displaces the supraspinatus tendon insertion anteriorly, decreasing its ability to aid in shoulder abduction and increasing the sensitivity of the examination. Further clinical examination following the identification of axillary nerve injury often allows for determination of the location of the lesion as it relates to the quadrilateral space. If the axillary lesion spares the posterior portion of the deltoid and teres minor muscles, the lesion is distal to the quadrilateral space (9).

Etiologies:

Non-Surgical Injuries:

Understanding the etiology or mechanism of injury is of paramount importance as the treatment of an axillary nerve injury is based on the severity and type of injury that has been sustained. A detailed clinical history is vital for treatment decision-making as each mechanism has a characteristic injury pattern. Operative treatment typically is not indicated for low-energy, non-penetrating traumatic injuries. Such injuries are usually traction, torsion, or compression neurapraxias in which the axon is not disrupted. Rather, neurological symptoms occur following trauma as a result of a conduction block due to transient demyelination or intrafascicular edema in the nerve (10).

Shoulder dislocation is one of the leading causes of traction injury of the axillary nerve, with the reported rates of nerve injury following dislocation ranging between 5% and 54% (11).

Injuries to the nerve occur in association with anteroinferior shoulder subluxation, frank dislocation, and Bankart lesions. When nerve damage occurs in association with acute shoulder dislocations, complete spontaneous recovery usually occurs, with reported rates in the literature ranging from 87.5% to 100% of cases (12)

In the study by **Visser et al.** (13) of 32 patients in whom a severe axillary nerve injury was confirmed on the basis of both clinical examination and EMG findings failed to recover full function. However, only minor residual symptomatology remained. Interestingly, those authors did not find an increasing severity of nerve injury, but they did find that the risk of nerve injury in association with dislocation increased with age (p , 0.007). Results have been similarly favorable in patients with low energy torsional and compression-type injuries.

Guerra and Schroeder (14) reported on 5 patients with torsional nerve injury, all of whom had good or excellent restoration of motor function after a mean duration of follow-up of 33 months.

Surgical Injuries:

Iatrogenic injury is a frequent and preventable cause of axillary nerve palsy. Open injuries are more likely to result in axonotmesis or neurotmesis of the nerve, which require operative repair. Mechanisms of intraoperative nerve injury include limb malpositioning, inadvertent suturing, heat penetration, aggressive retractor placement, and excessive inferior dissection of the shoulder capsule (15).

Attention also should be paid to factors that increase the predisposition to iatrogenic injury, including current or previous use of methotrexate, a history of shoulder surgery, a history of radiation treatment, a preoperative passive external rotation range of motion of >10°, and decreased operative time (16).

Several surgical procedures have been implicated in axillary nerve injury as well. Among these, the Latarjet procedure, which is a means of glenohumeral joint stabilization that includes transfer of the coracoid process to the anterior aspect of the glenoid, has become increasingly popular (15).

While the Latarjet procedure is effective, a systematic review of 1,904 such procedures demonstrated a relatively high rate of neurovascular injury (1.8%), including 6 axillary nerve injuries (17).

The cadaveric study by **Freehill et al.** demonstrated consistent postoperative overlap between the musculocutaneous and axillary nerves as well as medial displacement of both nerves relative to their preoperative positions during the Latarjet procedure (18).

Delaney et al.(19) used intraoperative neuromonitoring during the procedure and found the nerve to be most at risk during glenoid exposure and graft insertion. Additionally, 79.4% of the nerve alerts occurred when the arm was in

external rotation, indicating that while the axillary nerve is under the least amount of tension in external rotation, that position also may bring the axillary nerve closest to the surgical field. In order to avoid injury, those authors advised against excessive retraction and a more superior placement of the coracoid graft.

Axillary nerve injury is also a well-documented complication of total shoulder arthroplasty. Neurological injuries occur in association with about 1% of total shoulder arthroplasty procedures, with the axillary nerve being most commonly affected (20).

Associated neural injuries:

Most axillary nerve injuries are low-grade lesions which will recover rapidly and often completely. The detection of associated injuries to the median or ulnar nerve suggests an infraclavicular brachial plexus injury which has a poorer prognosis (21).

The detection of associated nerve deficits should prompt early referral to a specialist centre for further evaluation. Associated nerve injuries need to be identified and documented at the time of initial examination and after any intervention. Failure to do so not only make recognition of injury and duration difficult but also exposes the treating doctor to litigation. Neuropathic pain is associated with intermediate- and high-grade injuries but can also highlight deterioration in low-grade injuries due to pressure from a haematoma or perineural scar formation. Neuropathic pain from the axillary nerve is often reported as a burning or deep aching sensation in the upper lateral cutaneous nerve of arm territory. In a series of brachial plexus palsies associated with arterial injury, nerve-related pain was demonstrated in 20% of all cases either representing neurostenalgia, or less commonly causalgia, as a consequence of nerve traction and ischaemia (22).

Nonoperative Treatment:

With the exception of certain cases of quadrilateral space syndrome, neurapraxic injuries usually are treated nonoperatively. Several case series involving patients who have had complications after shoulder surgery have shown high rates of spontaneous recovery following axillary nerve neurapraxia; however, comparative studies with operative treatment are lacking. Nonoperative treatment should include a rehabilitation program emphasizing passive and active range of motion as well as strengthening of the rotator cuff, deltoid muscle, and periscapular musculature (23).

Operative Treatment:

The results of operative treatment of axillary nerve injuries are optimal when surgery is performed within 3 to 6 months after the injury. Outcomes of surgical treatment for axillary nerve injury are worse beyond this time frame, with only marginal benefits seen when surgery is performed 12 months after the injury (24).

These recommendations are based on case series, and further comparative studies are needed to optimize the timing of surgery. Four standard modalities of operative treatment are generally utilized: neurolysis, neurorrhaphy, nerve-grafting, and neurotization. The choice between treatment modalities is contingent on the findings observed during operative exploration(25).

Predictors of surgical outcome:

Irrespective of the procedure, better results will be achieved with earlier intervention. There is a trend towards better outcomes with intervention at less than 4–6 months following injury (20) with more substantial evidence suggesting significantly poorer results after 9–12 months (26).

There are several factors which will determine the success of grafting or transfer surgery, unfortunately not all under the control of the surgeon:

- Increasing patient age results in worse outcomes. Results of grafting and nerve transfer deteriorate after the age of 30 years, with potential for no recovery after the age of 50 years(26).
- Increasing patient body mass index (BMI) is associated with a worse result.
- The patient with multiple nerve injuries or associated rotator cuff tears will do less well than those with isolated axillary nerve injuries, whatever the intervention (27).
- Perhaps unsurprisingly, those patients who can be managed with neurolysis alone have a better outcome than those who undergo graft or transfer, though this reflects the severity of the injury rather than choice of surgical modality(28).
- Graft length has an unclear bearing on outcome. While some studies have historically suggested there is a worse outcome with respect to abduction with grafts > 6 cm and others have shown trends to support this, others have demonstrated good results with longer grafts, with

results being comparable with both shorter grafts and nerve transfer(29).

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