Upper Extremity Injection Nerve Injury: Black, White, and Shades of Gray

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Abstract

Upper extremity nerve injury can be due to multiple causes and has a constellation of effects depending on the site, method, and, if applicable, injected substance. While many injuries result from mechanical etiologies such as needles for vaccines and nerve blocks, secondary chemical injury may occur, usually presenting with a different set of symptoms and timeline. Management of such injuries is tiered as well, ranging from conservative measures—physiotherapy and analgesics—to surgical neurolysis. Injury avoidance is paramount; preventative measures are possible for many of the more common etiologies of upper extremity nerve injuries. The purpose of this paper is to assemble etiologies, signs and symptoms, diagnosis, and management of the most common nerve injuries of the upper extremity.

- nerve injection injury
- nerve palsy
- intramuscular injections
- injection injury

Introduction

Injection-induced peripheral nerve injuries in the upper extremity vary in accordance with the mechanism of injury. Some injuries are mechanical, such as intravenous (IV) central line placement, joint aspiration, nerve blocks, or intramuscular (IM) injections, while others may occur indirectly as a result of the toxicity from the injected substance. The brachial plexus (BP), axillary, median, and radial nerves are among the most commonly injured nerves in the upper extremity, usually due to mechanical injury. Upper extremity nerve injuries frequently present as neuromotor or neurosensory dysfunction, with the specific symptoms depending on the proximity of the nerve to the spinal cord.[1-3] It is prudent to obtain a thorough medical history including past reactions to injected substances. The treatment modality is dependent on the nerve and mechanism of injury but usually begins with conservative management with analgesics and progresses toward neurolysis. In this paper, we review the literature on injection injuries of nerves of the upper extremity, the common mechanisms of injury, pathology involved, options in management of these cases, and potential precautions to be taken.

Brachial Plexus Injection Injury

BP blocks were first performed by Halsted in 1885[4] and have generally been associated with a low complication rate. Kulenkampff and Persky reported a series of 1,000 cases of BP blocks but only one had nerve irritation.[5] The anatomical approach tends to affect the incidence of brachial plexus injection injury (BPII), but most methods have the advantage of complete anesthesia with one injection. General methods of injury include direct needle trauma, neurotoxic chemical injury, and axillary artery laceration with subsequent injury from hematoma formation.

Mechanisms of Injury

The supraclavicular injection for BP block is the most commonly used method, but can be associated with pneumothorax, hematoma formation, Horner's syndrome, and phrenic or recurrent nerve paralysis.[4] Interscalene BP blocks through

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the neck are associated with a higher risk of BPII as compared with other approaches to the BP.[6,7] Infraclavicular blocks have been reported to be associated with up to 21% incidence of BPII.[8,9] Tsao and Wibourn[1] have suggested that BPII caused by axillary arteriography is similar to that caused by an axillary nerve (AN) block, another cause of BPII. Axillary arteriography is frequently used when the femoral route is contraindicated or not possible due to diseases such as aorto-iliac disease, abdominal aortic aneurysm, and aortic coarctation among others.[10] Laceration of the axillary artery can produce compartment syndrome due to physical limitations of the axillary sheath,[10] with 24 hours of compression causing Wallerian degeneration. While performing an axillary block, one can assume the axillary sheath being breached by the following indicators: paresthesias, arterial puncture, venous puncture, and sheath tethering. Expansile hematoma or pseudoaneurysm formation in the axillary sheath can cause medial brachial fascial compartment syndrome where microcirculation compression causes nerve injury presenting with delayed neurologic symptoms. It is important to note that the distal pulses may be intact while the nerve injury is occurring. Axillary artery arteriography has not directly been a cause of BPII, with numerous studies reporting no permanent damage,[11-13] and it is speculated that surgical exploration due to hematoma formation may be the cause of this nerve injury. BPII presents in accordance with the mechanism of injury. Mechanical injury can be varied and result in a varied distribution of symptoms but a predilection toward musculocutaneous nerve (100%) and AN (85%) has been reported. When BPII is found after repeated attempts at subclavian vein catheterization, the lower trunk is the most vulnerable to injury with the upper trunk being at risk with deep insertion of the needle.[14-19]

Pseudoaneurysm or hematoma formation results in a gradual worsening of function over time, secondary to compression, usually with median nerve (MN) deficits (93%), followed by ulnar nerve lesions (53%).[10] No other nerve has been found to be damaged in isolation other than the median or ulnar nerves.[1] Unlike mechanical injury that affects larger fibers and causes motor disturbances, chemical injury tends to affect smaller-diameter fibers causing loss of pinprick, hyperalgesia, hypesthesia, paresthesias, and/or disturbed sympathetic conduction.[4] Various neurotoxic agents have been reported including thiopental, chlorpromazine, oil-based preparations (more so than water-based preparations), efocaine, and xylocaine (possibly due to copper ion contamination).[20] With neurotoxic injury, smaller-diameter fibers are more commonly involved through loss of pinprick sensation, hyperalgesia, paresthesias, and disturbed sympathetic innervation.[4]

Presentation and Diagnosis

BPII can present in multiple ways. Neuromotor or neurosensory dysfunction, especially at the time of nerve blockade, may indicate ongoing traumatic injury—although this sign is far from definitive, specific root or nerve pain, or extreme pain, indicating primarily neurosensory dysfunction. Patients have been known to complain of attacks of pain in the night and arm lethargy during the daytime.[4] Risk factors for injury include general anesthetic use and adult age.[21-23] Postinjury sequelae usually consist of neurapraxia, especially with AN blocks, but according to one study almost all cases had resolved by 4 weeks.[8]

BPII is best diagnosed using clinical presentation and history, and an electromyogram (EMG) can be helpful when done 3 to 4 weeks after the patient presents with paresthesias; the nerve should be stimulated proximal to the injury to perceive decreased amplitude with possible fibrillation accompanied with positive sharp waves. Magnetic resonance imaging and sonography can be helpful in delineating mechanical causes of nerve compression such as hematomas.[24-26]

Management and Preventive Measures

Treatment of BPII depends on the degree of damage, types of roots involved, lag between injury and repair, and patient's age and gender. Stratification of treatment is generally done depending on the severity of injury and ranges from conservative management to nerve grafts. Preserving the biceps muscle function should be a priority. Generally, if nerve fascicles are intact, conservative management is indicated and if degenerative lesions exist, patients may benefit more from graft repairs along with conservative measures.[27,28] The main conservative options, which should be started immediately, are nonopioid analgesics, carbamazepine, amitriptyline, and physiotherapy.[29,30] Upon the failure of conservative therapy, or if degenerative nerve damage exists, surgical options should be strongly considered. Severe early loss of motor function after axillary arteriography may be an indication for surgical exploration, especially if immediate postarteriography sensory changes exist, which may be a warning sign of impending or developing BPII. Motor dysfunction is an especially ominous early sign and warrants quick surgical intervention. The presence of a hematoma warrants rapid surgical evacuation to reduce symptoms.[31] In the case of an expansile hematoma in the axillary sheath causing MBFC, prompt surgical decompression is prudent; patients undergoing evacuation within 4 hours of injury do better than post-4 hours of injury.[32] EMG proven degenerative lesions are best treated with nerve grafting, frequently from intercostal nerves.

Avoidance of BPII, when placing a subclavian line, is best done by proper technique, avoiding multiple attempts, having experienced providers, and expecting possible anatomical abnormalities. The subclavian IV line should not be attempted too deeply or laterally lest injury to the artery or BP is risked.[14,24] For patients with an acute need for dialysis, the internal jugular line is preferable over the subclavian line as there is a higher chance for stricture formation with subclavian lines.[33] Avoiding chemical injury is best done by using time-tested agents and cautiously using newer potentially neurotoxic agents.[4]

Axillary Nerve

The terminal branch of the posterior cord of the BP becomes the AN, whose fibers derive from the ventral rami of C5 and C6.[34,35] It descends inferolaterally on the anterior surface of

the subscapularis, and upon passing through the quadrilateral space at the inferior border of the subscapularis, the anterior and the posterior branches form, which innervate the teres minor and the three heads of the deltoid, respectively. The posterior branch travels more superficial than the anterior branch, gives sensation to the skin over the lateral shoulder and the glenohumeral joint, and ascends with the posterior circumflex humeral artery around the surgical neck of the humerus. Using surface landmarks, the AN runs 1.5 cm below the acromion; the average level of the AN is 5.29 cm from the anterolateral corner of the acromion and 5 cm from the posterolateral corner.[36] The cutaneous branch, arising from the posterior branch, gives sensation to the skin over the lateral shoulder and the glenohumeral joint.[35,37] The AN is of particular importance due to IM deltoid injections used to inject small amounts of nonirritating medications (up to 1 mL).

Although many patients with AN injury may present with initial pain at the site of injection, postinjury sequelae may offer clues as to the mechanism of injury. Mechanical trauma through a needle tip usually presents as pain at the time of injection while delayed sensory damage may be due to chemical neuropathy. Immediate sharp, electric-like sensation radiating down the fingers with possible deltoid muscle weakness and neurosensory deficit on the lateral side of the shoulder may be seen.[38,39] Long-standing injury may result in deltoid muscle atrophy and a positive deltoid extension lag sign. Diagnosis of AN injury relies on EMG (done 3–4 weeks after injury), which may show acute denervation of muscle and decreased AN compound motor action potential amplitude. Treatment of AN injury includes conservative measures as well as neurolysis, with more extensive lesions requiring nerve grafting with suture repair. Avoiding AN injury can be best done by injecting 5 cm from the acromion in the middle or anterior head of the deltoid.[40-42]

Median Nerve

The MN derives from the lateral and medial cords of the BP that originate from the C6/C7 and C8/T1 ventral roots, respectively. It enters the arm in the axilla and passes medially between the biceps brachii and the brachialis before crossing the cubital fossa medial to the brachial artery. It then passes between the two heads of pronator teres before entering the hand through the carpal tunnel. The first intervention involving the MN was reported by Kopell in 1958 to manage carpal tunnel syndrome (CTS); the nerve has since been affected for other reasons including IV access in the cubital fossa.[43-49] Local steroid injections continue to be used for the management of CTS, which can ironically injure the MN; the efficacy of the injections is also used as a prognostic factor and can help in the diagnosis of the severity of CTS.[50-53] The degree of damage to the MN, which is usually most affected by interventions concerning the forearm region, depends on the agent and deposition site. Rat studies have found the site of injection to be the most important factor in determining the degree of nerve injury.[54-56] Mechanisms of injury of the MN include direct needle injury, breakdown of the blood-nerve barrier due to steroids, and direct neurotoxicity of steroids.[45-47,55,57,58] The latter is the most relevant due to the increasing prevalence of CTS. All steroids are neurotoxic when injected in the intrafascicular plane of the nerve. Triamcinolone has been found to be the most toxic, affecting small and large fiber groups, followed by methyl-prednisolone with lidocaine, betamethasone, and finally, dexamethasone being minimally toxic. Other commonly used neurotoxic agents include bupivacaine, which can cause axon degeneration in the intrafascicular plane.[50,55,59-61] Less common pressure effects of the steroid and granuloma formation from repeated injections may be implicated in nerve injection injury.[44,62]

Symptoms of MN injection injury (MNII) include shooting pain at the time of injection, with numbness, paresthesias, other sensory defects, and weakness with possible thenar atrophy. Transient exacerbation of CTS symptoms (Phalen's and Tinel's signs becoming positive) may result as well.[45,46,49,50,63-65] Local steroid injections also cause local adverse effects like ischemia, depigmentation, and atrophy of the skin. Transient inflammatory changes associated with steroid injection may cause pain, which must be followed to 48 hours to ascertain MNII.[44-46]

There are several techniques that can be used to diagnose MNII. Sonoelastography, a test that measures the elasticity of a peripheral nerve, is useful for assessing tissue stiffness around the MN; an hypoechoic nerve has been a reported finding in postiatrogenic nerve injury. B-mode ultrasound can also be helpful in localizing the site of nerve injury. Hyperechoic areas on ultrasound and red areas on sonoelastic imaging are associated with hard tissue.[50,63] Nerve stimulation studies can also aid in diagnosis to measure the regenerative capacity of the nerve.[66]

Similar to BPII and ANII, conservative measures such as physiotherapy, nonsteroidal anti-inflammatory drugs, or local steroid injections may be helpful in the case of incomplete MNII. Steroids may offer symptomatic relief, but MN degeneration may be occurring to the point that surgery becomes inevitable.[50,67] Severe neuromotor functional loss, severe debilitating pain, limited recovery 3 months after injury, or if the injected substance has been deemed to be extremely neurotoxic have all been proposed as criteria for surgery. Surgery for this type of injury involves neurolysis with resection of lesions (e.g., a neuroma). Potential problems include limited graft viability, difficulty in obtaining autologous grafts, tissue rejection with homografts, and difficulties with successful cable grafts.[56,57,68] If acute MNII compression is found after steroid injection, immediate carpal tunnel release is indicated.

Avoidance of MNII is best done with appropriate positioning of the needle—an insertion point that is relatively ulnar to the flexor carpi radialis will reduce potential injury to the MN, although it increases the risk of palmar cutaneous branch injury. It is prudent that patients are aware of complaining about pain associated with injection to aid in avoiding MNII. The palmaris longus tendon can be used as an efficient landmark—injecting ulnar to it, in line with the fourth digit, is another way of preventing MNII. If using the flexor digitorum superficialis of the fourth finger to guide

needle insertion, two pops should be felt. Twenty-two gauge needles have been the recommended caliber of needle, both for drug delivery and to prevent nerve penetration.[45,47,50,69] Dexamethasone has been favored over betamethasone with mepivacaine; the latter being associated with a slight increase in CTS symptoms.[47] It has been recommended that steroids be used for temporary relief or if the patient refuses surgery, or in some specific cases like pregnancy (CTS may be in remission after delivery) or myxedema (remission after thyroid hormone replacement). Steroid injections are also useful for diagnostic and therapeutic purposes; injection outside the carpal tunnel may cause skin pigmentation changes and atrophy of the subcutaneous layer.[45-47]

Radial Nerve

The radial nerve (RN) derives from the posterior cord of the BP and fibers from the ventral roots of C5–T1. The nerve passes through the triangular interval and runs along the spiral groove on the posterior humerus. Next, it passes anterior to the lateral epicondyle before dividing into a superficial branch and a deep branch in the forearm. The superficial branch passes through the brachioradialis and continues on to pierce the deep fascia on the back of the wrist while the superficial branch pierces the supinator muscle. The RN is most commonly injured during IM injections into the deltoid and IV catheter insertion into the cephalic vein in the wrist. Upon needle insertion into the cephalic vein, the RN gets fixed and is strained by ulnopalmar flexion of the wrist. Insertion near the radial styloid is especially prone to damage the RN. In the upper arm, the RN runs superficially in the lateral aspect of the middle third of the humerus and risks being injured at that location.[70-73]

Symptoms of RN injection injury (RNII) include nonspecific features like pain, paresthesias, allodynia, pain with radiation to the dorsal surface of the forearm, wrist drop, numbness over the dorsum of the first interosseous space, and Tinel's sign.[70] Specific sensory distribution losses include anesthesia and neurosensory loss over the dorsal surface of the first three fingers.[74] These symptoms can also be caused by a neuroma formation (postinjury sequelae) in the region of the RN. Diagnosis is best done using needle EMG in the extensor digitorum communis, brachioradialis, and triceps (if applicable). In the case of the RN, motor nerve conduction tests are not helpful in distinguishing which patients respond to neurolysis.[75,76]

Factors that affect the recovery of RNII include proximity to the cell body and neuroma formation, which reduces chances of regeneration. Some RNII resolves spontaneously to full or partial recovery. It may be adequate to allow for enough time to be given for spontaneous recovery before deciding on surgical intervention. Medical management includes carbamazepine and melixetine for neuropathic pain, along with centripetal massage, splinting, physiotherapy, and vitamins B1, B6, and B12. Nonconservative options include steroid injections and neuroma management consisting of repeated blocks, steroids, cryoneurolysis, or phenol lytic blocks.[70,77] Surgical management of RNII involves various combinations of tendon transfers (Robert Jones Transfers), which have shown to achieve considerable augmentation of extension of wrist and fingers.[75,78] Given that some RNII resolves spontaneously, 3 months of conservative therapy may be warranted before surgical intervention.[75,79] Neuroma formation and the possibility of tension-free nerve grafting with end-to-end approximation are the two main exceptions for early surgical withdrawal. Bathing the proximal severed end in absolute alcohol and ligating it with sutures is one way of avoiding neuroma formation.[80-82]

In developing countries, injections by untrained professionals are a major cause of RNII, particularly the insertion of an IV needle into the cephalic vein with irregular motions. It has been recommended to avoid the cephalic vein except in cases that no other forearm vein is available, and if attempting cephalic vein catheterization, insert the needle at 5 to 15 degrees to the skin; larger angles can puncture the deep venous wall and injure the superficial RN.[71,75,83,84] Theoretically, the cephalic vein and the superficial branch of the RN intersect at multiple points and no area can be absolutely defined as safe for injection,[85] but the superficial branch does emerge 6 to 11 cm proximal to the styloid process so injections given proximal to the point of emergence may be safe.[85]

Ulnar Nerve

The ulnar nerve is derived from the medial cord of the BP and consists of fibers from C8 and T1. It travels posteromedial to the humerus in the upper arm before entering the anterior forearm between the humeral and ulnar heads of the flexor carpi ulnaris. It then descends along the ulna with the ulnar artery before entering the palm through Guyon's canal. The ulnar nerve is frequently injured as a result of steroid injection for medial epicondylitis (golfer's elbow) as well as CTS. Upper nerve injection injury (UNII) is far less common than other upper extremity nerve injection injuries but sporadic case reports exist. Two of these cases describe UNII, one through improper positioning while injecting into the deltoid, and another through neuroma formation after jet injection for swine flu vaccination. In a retrospective study of 115 patients with upper nerve (UN) injury, only 2 were found to be injection-induced UN lesions.[86-88]

Symptoms of UNII include tenderness over the epicondyle with a positive Tinel's sign and/or palsy leading to clawing of the fourth and fifth digits. Chemical neuritis, such as the one report with swine flu vaccination or methyl-prednisolone injections, is usually transient.[55] Diagnosis is best done using delayed EMG and nerve action potential (NAP). Given positive action potential findings, the recommended treatment is neurolysis with or without transposition and possible nerve transfers. With a negative NAP indicating no regeneration, resection and end-to-end suture or graft repair with or without transposition is recommended.[86,89]

Avoiding UNII is done through proper training, avoiding recurrent traumatic dislocation of the ulnar nerve, especially if predisposed to it by congenital defects, and keeping the elbow in an extended or semiflexed position to protect the nerve when injecting to treat medial epicondylitis.

Prognostic Factors and the Decision for Surgery

There are many previously confirmed prognostic factors for traumatic peripheral nerve injury that can aid in choice of therapy. Two factors that predispose to a good outcome include young patient age and distal injury site.[90] The commonality in each of these situations is that less distance has to be covered by the regenerating nerve. Neurapraxia has a positive prognosis compared with greater degrees of nerve injury.[91] Two major drivers of poor outcome are delayed referral to a qualified peripheral nerve expert and delayed surgical repair.[92] Another factor that increases the likelihood of a negative outcome is concomitant damage to the vascular supply of the nerve.[93] Combined motor and sensory deficits are known to point to a worse prognosis than sensory deficit alone. Patients experiencing palsy on examination have better outcomes than those with dense paralysis.[94] Interestingly, the particular nerve injured seems to play a role in recovery. A prospective study of 393 nerve graft repairs by Roganovic and Pavlicevic[95] showed that sensory recovery was similar between median, ulnar, radial, and musculocutaneous nerves. Contrastingly, recovery of motor function varied between each nerve especially with more proximal injuries. Finally, there are three primary temporal factors to take into account when trying to predict the need for surgery. Repair of segmental demyelination occurs in 8 to 12 weeks, thus a deficit that remains after this time points toward axonal injury.[96] Axonal regeneration occurs at an approximate rate of 2 mm/d in small nerves and 5 mm/d in large nerves.[97] Denervated muscle undergoes irreversible atrophy within 12 to 18 months after injury.[98] These last two factors allow for the use of EMG in surgical planning. Time to target muscle reinnervation can be estimated and the choice for exploration can be made if EMG shows that reinnervation is proceeding at a rate too slow to allow for distal muscle function.[99]

Conclusion

Upper extremity nerve injury is most frequently seen in the BP, axillary, median, radial, and ulnar nerves. They most commonly occur as a result of mechanical injection injury, which can exacerbate a possible chemical injury and may present with differing signs. We find that establishing a timeline of signs and symptoms as well as using EMG and sonoelastography can help to confirm the extent of injury. Mechanical injury often produces a neuromotor cluster of symptoms while chemical injury is more damaging to sensory nerves. EMG and sonoelastography are best used weeks after injury, often leading clinicians to start conservative measures at the onset of symptoms. These conservative measures usually include nonopioid analgesics, carbamazepine, vitamins, and physiotherapy, the latter of which is more cost effective. These measures can be upgraded to neurolysis, tendon transfers, and nerve grafting. It is important to note the preventive measures that may be taken that might significantly decrease nerve injury, which is especially important in developing countries where access to diagnosticians and their methods may be limited. Finally, a high-quality study defining the relationship between predictors and outcomes in injection nerve injuries remains to be done. One would suggest that a multicentric study with a detailed record of patient presentation and specific criteria for treatment protocol be implemented to better elucidate this important topic.

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