

Management of Atraumatic Posterior Interosseous Nerve Palsy

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The posterior interosseous nerve (PIN) is susceptible to a number of traumatic and atraumatic pathologies. In this article, we aim to review our current understanding of the etiology, pathology, diagnosis, treatment options, and published outcomes of atraumatic PIN palsy. In general, the etiology of atraumatic PIN palsy can be divided into mechanical, which is caused by an extrinsic compressive force on the nerve, and nonmechanical, which is caused by an intrinsic inflammatory reaction within the nerve. As per this discussion, there are 3 causes for atraumatic PIN palsy. These are entrapment neuropathy, Parsonage-Turner syndrome, and spontaneous “hourglass” constriction. The typical presentation of atraumatic PIN palsy is a patient with spontaneous onset of weakness of fingers/thumb metacarpophalangeal joints extension. However, the wrist extension is preserved with radial deviation due to preservation of extensor carpi radialis longus/brevis function. Magnetic resonance imaging is the imaging of choice and neurophysiology is indicated in all patients. If there is an obvious structural cause of the nerve palsy, prompt decompression and removal of the causative lesion are recommended to avoid irreversible damage to the nerve/muscles. Otherwise, in general, we would recommend consideration for exploration should there be no sign of recovery after 6 weeks of observation. (*J Hand Surg Am.* 2017;42(10):826–830. Copyright © 2017 by the American Society for Surgery of the Hand. All rights reserved.)

Key words Atraumatic, interosseous, palsy, posterior, nerve.

THE POSTERIOR INTEROSSEOUS NERVE (PIN) is susceptible to a number of traumatic and atraumatic pathologies. Atraumatic causes of PIN dysfunction include compression neuropathy, neuralgic amyotrophy, and spontaneous “hourglass” constriction (SHGC)¹ of the nerve. Posterior interosseous nerve palsy may be confused or regarded interchangeably with radial tunnel syndrome; however, this warrants clarification. Radial tunnel syndrome essentially is a lateral forearm pain syndrome

without any neurological deficit and electrophysiologic tests are typically normal. In contrast, atraumatic PIN palsy presents with spontaneous-onset weakness or paralysis of the muscles innervated by the PIN and pain is not necessarily a constant feature.

In this article, we aim to review our current understanding of the etiology, pathology, diagnosis, treatment options, and published outcomes of atraumatic PIN palsy.

ANATOMY

The radial nerve is a direct continuation of the posterior cord. It courses around the spiral groove of the humerus as it innervates the triceps and anconeus. It then pierces the lateral intermuscular septum to enter the anterior compartment of the upper arm approximately 10 cm proximal to the lateral epicondyle. It then innervates the brachioradialis (BR), extensor carpi radialis longus (ECRL), and extensor carpi radialis brevis (ECRB) proximal to the epicondylar

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line. It crosses the elbow anterior to the lateral epicondyle between the BR and the brachialis, then divides into the superficial radial nerve and the PIN 8.0 ± 1.9 cm distal to the lateral intermuscular septum and 3.6 ± 0.7 cm proximal to the leading edge of the supinator.² The PIN courses under the dorsal surface of the radial neck, with which it has contact (opposite to the radial tuberosity) in 25% of cases. After that, it passes under the arcade of Frohse and enters the forearm between the 2 heads of supinator. The arcade of Frohse is a fibrous arch formed by the most proximal part of the superficial head of the supinator 3 to 5 cm distal to the lateral epicondyle. The PIN exits the supinator muscle 3.8 ± 0.9 cm distal to the proximal margin and divides again into the medial and lateral branches.² The medial branch innervates the extensor carpi ulnaris (ECU), extensor digitorum communis (EDC), extensor digitorum quinti (EDQ), and the lateral branch innervates the abductor pollicis longus (APL), extensor pollicis longus (EPL), extensor pollicis brevis (EPB), and extensor indicis proprius (EIP) in that order.³

EPIDEMIOLOGY

In 2006, Latinovic et al⁴ reported an incidence for radial neuropathy of 1.4 in women and 3.0 in men per 100,000 people in the United Kingdom. This included all forms of abnormality affecting the radial nerve. In 2012, Quignon et al⁵ found 264 cases of PIN palsy reported in the literature over a 50-year period. However, the reported incidence is likely to be an underestimation owing to cases that did not present to the medical professionals, underreporting, and misdiagnosis.

ETIOLOGY

In general, the etiology of atraumatic PIN palsy can be divided into mechanical, which is caused by an extrinsic compressive force on the nerve, and nonmechanical, which is caused by an intrinsic inflammatory reaction within the nerve. The net effect of both is nerve ischemia reducing the capacity of the axons to transmit action potentials. The severity depends upon the magnitude and duration of the underlying insult. Chronicity of the insult can result in focal demyelination, followed by axonal damage that in turn leads to neural scarring and, therefore, less chance of functional recovery.^{1,6-8} As per this discussion, there are 3 causes for atraumatic PIN palsy: entrapment neuropathy, Parsonage-Turner syndrome (PTS), and SHGC.

Entrapment neuropathy

Mechanical compression can be due to normal anatomical structures such as the proximal edge of the supinator,⁹ the distal edge of the supinator,¹⁰ the ECRB,³ the recurrent leash of Henry,³ and the arcade of Frohse¹¹; or pathological structures such as lipomas,¹² ganglions,¹³ fibrous adhesions,¹⁴ bulging synovium or cysts in rheumatoid patients.¹⁵ It can also occur owing to a combination of both normal and abnormal structures causing a compressive effect, for example, a space-occupying lesion within the substance of the supinator compressing the nerve against the radial neck.¹⁶ After emerging from the supinator, the nerve can be compressed before or after it bifurcates into the medial and lateral branches, although compression most commonly occurs at the proximal edge of the supinator before it bifurcates.¹⁷

Parsonage-Turner syndrome/neuralgic amyotrophy

The PTS, also referred to as idiopathic brachial¹⁸ plexopathy or neuralgic amyotrophy, is a condition of unknown etiology that could be associated with recent viral illnesses, immunization, and generalized diseases like systemic lupus erythematosus. Patients present with abrupt-onset unilateral upper extremity pain followed by progressive neurological deficits, including weakness, atrophy, and occasionally sensory abnormalities. The exact cause and pathophysiology of PTS are incompletely understood, although autoimmune, genetic, infectious, and mechanical processes have all been suggested. Underlying all these suggested causes is an inflammatory process that has been presumed as the common pathology.^{19,20}

Spontaneous "hourglass" constriction of PIN

The SHGC of PIN is a condition associated with torsion of fascicles during forearm movements and is also weakly associated with vasculitic disorders such as polyarteritis or allergic angioneuropathy. There is hypoperfusion of the nerve causing intrafascicular edema, which then heals with segmental fibrosis and constriction.¹⁷ Further histopathological examination of the constricted nerve segment has revealed abundant lymphocytes and neutrophils in the walls of the small perineural feeding arteries. This leads to swelling of the arterial wall and narrowing of the lumen. The net result is adjacent axonal fibrosis, which supports an inflammatory etiology.¹⁸

CLINICAL PRESENTATION

The typical presentation of atraumatic PIN palsy is a patient with spontaneous onset of weakness of fingers/

thumb metacarpophalangeal joints extension. However, wrist extension is preserved with radial deviation owing to preservation of ECRL/B function. Within a matter of weeks, atrophy of the forearm extensor muscles becomes apparent. Lateral forearm pain may or may not be a constant feature.

The pattern of weakness could suggest the point at which the nerve is affected. Weakness of the ECU, EDQ, and EDC suggests that the medial branch is affected. Weakness of the APL, EPB, EPL, and EIP suggests that the lateral branch is affected. Weakness of all of these muscles would suggest the PIN has been affected proximal to its bifurcation.

There are clinical features that may suggest the different etiologies of PIN palsy. Severe pain usually precedes paralysis in neuralgic amyotrophy. There might be a recent history of a common cold, influenza, minor trauma, vaccination, major surgery, acute hepatitis, or toxic exposure, even though this is subject to recall bias. The degree of paralysis in neuralgic amyotrophy can be variable and is sometimes reversible. Examination may reveal additional paralysis and possible sensory disturbance around the shoulder girdle and upper arm.

Sensory deficit is not encountered in entrapment syndrome or SHGC. In entrapment neuropathy, pain is usually absent or mild. Paralysis due to entrapment tends to be partial initially and then may become progressive and complete with time. In contrast, pain can be a feature in SHGC.

Evidence of denervation of the BR or more proximal muscles may be seen in neuralgic amyotrophy but never in entrapment neuropathy or SHGC because only the PIN is affected in the latter conditions.¹⁵

INVESTIGATIONS

Plain radiographs of the elbow can be used to exclude osseous pathology.¹⁶ Magnetic resonance imaging, which is our preferred imaging modality, is employed to exclude space-occupying lesions, to assess neural continuity, and to identify denervation changes in the forearm muscles. Modern high-resolution ultrasonography can also provide clear visualization of the nerve, including the percentage of fascicular constriction. It also has the potential advantage of enabling assessment of dynamic compression of the nerve.^{8,17}

Electrodiagnostic testing is used to confirm the clinical diagnosis and to provide prognostic information about the reinnervation potential of the nerve.¹⁶ Motor nerve conduction studies may demonstrate slowed conduction velocity, prolonged

distal latency, and reduced amplitude compared with the opposite side. Abnormal sensory conduction studies may be seen in neuralgic amyotrophy but not in entrapment syndrome or SHGC. Electromyographic examination will reveal abnormal spontaneous activity in the form of positive sharp waves and fibrillations, discrete recruitment pattern, and decreased recruitment interval in PIN-innervated muscles.^{21,22}

TREATMENT

Treatment is guided by the diagnosis. If there is an obvious structural cause of the nerve palsy, prompt decompression and removal of the causative lesion are recommended to avoid irreversible damage to the nerve/muscles. If imaging has excluded a structural cause, a period of close observation including modification of activity, physiotherapy, and wearing of an orthotic is reasonable. The challenge in clinical practice lies with distinguishing between neuralgic amyotrophy and entrapment neuropathy because the thresholds for exploration may differ. In the former, a longer period of observation is generally accepted owing to the potential for spontaneous recovery. In contrast, in the latter condition, a low threshold for exploration is advised, particularly in those who demonstrate progressive weakness. However, the distinction between the 2 may not always be obvious. In general, we would recommend consideration for exploration should there be no sign of recovery after 6 weeks of observation.¹⁵

At the time of exploration, neurolysis of the PIN is performed. Direct electrical stimulation is also performed to confirm the absence of muscle response. In the rare occurrence of SHGC, the constricted segment should be examined under a microscope. In mild to moderate constrictions, external neurolysis with epineurotomy²³ or interfascicular neurolysis^{8,24} is recommended. If the constriction is severe, the segment is excised and repaired.²² If primary repair is not possible, a nerve graft is then required.¹⁵

After simple decompression, tendon transfer is considered only should there be no recovery or insufficient recovery after a period of rehabilitation.

Surgical approaches

The PIN may be explored via an anterior, a posterior, or a lateral approach.

1. The extended anterior approach²⁵: The anterior approach starts with a lazy S-shaped skin incision 5 cm proximal to the elbow crease along the lateral border of the biceps, which is then curved

transversely in a medial direction for 2 cm at the elbow crease, then extended distally for 5 cm along the medial border of the BR. The lateral antebrachial cutaneous nerve is identified lateral to the biceps tendon and protected. The radial nerve is identified proximally in the interval between the brachialis and the BR. A branch to the BR will be encountered here. The branches to the ECRL and ECRB arise more distally and the origins can be variable. The radial nerve then divides into its 2 main trunks: superficial and deep. The superficial branch is found on the undersurface of the BR. The recurrent tributaries of the radial artery (leash of Henry) and muscular branches must be carefully ligated in order to expose the deep branch (PIN) that courses under the supinator. Beware of the nerve branch to the supinator that comes off the PIN just proximal to the supinator muscle edge. Next, the arcade of Frohse and the superficial head of supinator muscle are released under direct vision (Fig. 1).

2. The posterior approach²⁶: Along an imaginary line between the lateral epicondyle and the distal radioulnar joint, a 5-cm skin incision starting at the radial neck region is made along the groove between the “mobile wad” and the rest of the extensor muscles. Superficial dissection utilizes the interval between the EDC and the ECRB to reveal the supinator. The muscle fibers of the supinator run obliquely to the radial shaft, in contrast to the fibers of the EDC, which are parallel to the forearm. The PIN emerges from under the distal edge of supinator, which can be tendinous. The superficial head of supinator is then carefully released under direct vision. Together with the release achieved via the anterior approach, the PIN is fully decompressed (Fig. 2).
3. The lateral approach¹⁵: A curvilinear incision along the lateral aspect of the elbow is made. After skin flap elevation, the BR is the key muscle for this approach. The proximal part of the PIN is exposed by retracting the BR laterally while the distal part of the PIN is exposed by retracting the BR medially. A BR muscle-splitting approach has also been described.²⁷

Scars may be an aesthetic concern for some patients and this should be addressed before surgery. Utilizing either the anterior or the posterior approach alone may run the risk of inadequate decompression of the nerve. Thus, we favor a combined anterior-posterior approach using 2 separate incisions to ensure the adequacy of exposure.

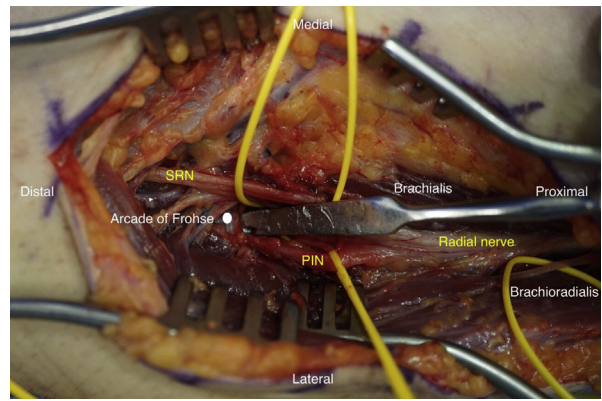


FIGURE 1: Anterior approach. SRN, superficial radial nerve.

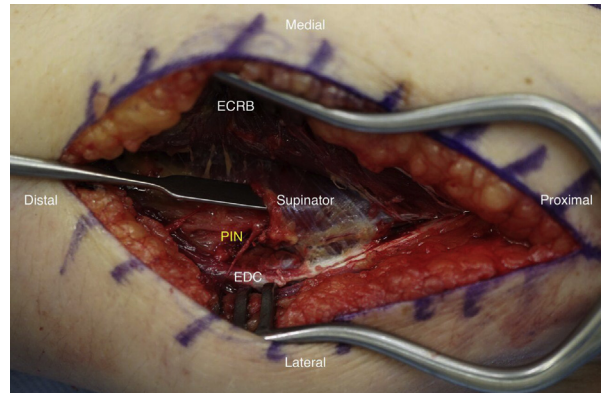


FIGURE 2: Posterior approach.

OUTCOMES

In cases of mechanical compression, good outcomes have been reported with surgical decompression even in the face of poor reinnervation potential on preoperative neurological testing. Complete resolution of symptoms has been reported as long as 2 years after the onset of symptoms²⁴ and partial resolution of symptoms up to 4 years after the onset of symptoms.^{8,21}

Cases due to neuralgic amyotrophy showing clinical signs of recovery within the first month of the onset of symptoms usually go on to do well with nonoperative treatment, with 73% of patients recovering within a year and up to 90% by 3 years. Of those showing no spontaneous clinical improvement within the first month, recovery was much slower with only 21% recovered by the first year and up to 71% recovered by 3 years. Therefore, Ochi et al⁸ recommended surgical treatment to expedite recovery in the latter group in the form of interfascicular neurolysis.

The results of surgery in cases due to SHGC depend on the extent of interfascicular scarring, which can be determined on ultrasound scanning.

Wu et al¹⁵ reported good results (denoted by Medical Research Council grade 4 or 5 power) with interfascicular neurolysis for mild to moderate constrictions (defined as < 75% of fascicle thinning on ultrasonography) at 23 months' follow-up. Hironobu et al²³ suggested that complete recovery of nerve function could be achieved within 5 months in a similar group of 4 patients. Conversely, Ochi et al⁸ reported poor recovery after interfascicular neurolysis in patients with severe constrictions (defined as > 75% of fascicle thinning on ultrasonography). As a consequence, this group of patients should undergo neurolysis or autografting. In their retrospective review of 50 cases, Ochi et al⁸ found that age younger than 50 years at the time of surgery and a preoperative wait of 7 months or less were associated with improved surgical outcome.

DISCUSSION

Atraumatic PIN palsy results in forearm extensor muscle weakness and a variable degree of forearm pain. Clinical assessment may provide clues to the likely underlying etiology. Magnetic resonance imaging is the study of choice and neurophysiology is indicated in all patients. In the absence of a space-occupying lesion, a trial of nonoperative management is advisable. However, should there be no sign of muscle recovery after 6 weeks of observation or if there is progressive weakness, exploration of the nerve is recommended.

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