Management of Atraumatic Posterior Interosseous Nerve Palsy

Kohila Vani Sigamoney, MBChB, MRCSEd,* Abbas Rashid, MBBS, BSc,*
Chye Yew Ng, MBChB (Hons)*

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.
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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.

*From the *Trauma and Orthopaedics Department, Wrightington, Wigan & Leigh NHS Foundation Trust, Wigan, UK.*

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Corresponding author: Kohila Vani Sigamoney, MBChB, MRCSEd, Wrightington Hospital, Hall Lane, Appley Bridge, Wrightington, WN6 9EP, UK; e-mail: kohilavani_sigamoney@yahoo.com.

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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

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 al[4] 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 al[5] 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 Frohse[11]; 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 brachial[18] 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/