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Edited by Ruby Singh

E-mail: bioinformationruby@gmail.com

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Development of long flexor tightness in low ulnar nerve palsy: A review

Pankaj Gupta*, Deepak Raghav & Gopal Yadav

Department of Orthopaedics (Physiotherapy), Santosh Deemed to be University, Ghaziabad, India; *Corresponding author

Affiliation URL:

<https://www.santosh.ac.in/>

Author contact:

Pankaj Gupta - E-mail: therapist_pankaj2002@yahoo.co.in

Deepak Raghav - E-mail: deepak.raghav@santosh.ac.in

Gopal Yadav - E-mail: hod.orthopaedics@santosh.ac.in

Abstract:

Proximal interphalangeal joint stiffness is a common complication of ulnar nerve palsy, particularly in leprosy. Therefore, it is of interest to examine the pathophysiological mechanisms underlying stiffness associated with low ulnar nerve palsy. The condition compromises hand function, restricts daily activities and may delay corrective surgery for hand deformities. Unlike traumatic stiffness, nerve-related stiffness involves skin contracture, extrinsic muscle tightness, long flexor tightness and capsular changes. Understanding these mechanisms can improve clinical assessment and guide timely management of hand dysfunction.

Keywords: Proximal interphalangeal (PIP) joint stiffness, long flexor tightness, skin contracture, capsular contracture, leprosy, ulnar nerve palsy

Background:

Leprosy remains a public health concern in India, with more than 100,000 new cases reported annually despite substantial progress in disease control [1]. The disease primarily affects the skin and peripheral nerves, particularly the ulnar, median, common peroneal, facial and trigeminal nerves, resulting in deformities such as lagophthalmos, claw hand, wrist drop, ape thumb deformity and foot drop when left untreated [2]. The widespread implementation of multidrug therapy (MDT) has markedly reduced the global burden of leprosy; however, early diagnosis and prompt initiation of WHO-recommended MDT remain essential to prevent nerve damage, disability and permanent deformities [3]. In addition to physical impairments, individuals affected by leprosy frequently experience stigma, discrimination, psychological distress and socioeconomic hardship, which contribute to delayed healthcare seeking and advanced disease at presentation [4]. Delayed diagnosis often leads to irreversible nerve damage, secondary complications such as joint contractures and poorer functional outcomes following reconstructive surgery [5]. Therefore, it is of interest to report the pathokinesiology underlying proximal interphalangeal (PIP) joint stiffness in the context of ulnar and median nerve involvement in leprosy.

PIP joint stiffness due to low ulnar nerve palsy:

The ulnar nerve is the peripheral nerve most frequently affected in leprosy and its involvement results in paralysis of the interossei, medial two lumbricals, adductor pollicis and the ulnar half of the flexor digitorum profundus, leading to significant functional impairment of the hand [6]. Consequently, patients develop loss of protective sensation, clawing of the fingers, impaired finger abduction and adduction, collapse of the metacarpal arch and reduced grip and pinch strength, all of which compromise hand function [6, 7]. Clawing results from an imbalance between the intrinsic and extrinsic muscles, producing asynchronous finger flexion and extension and reversal of the normal sequence of finger movement. This abnormal motor pattern is further exaggerated when both the ulnar and median nerves are involved, resulting in inefficient hand use and maladaptive cortical reorganization that reinforces dysfunctional movement patterns during daily activities [7, 8]. Repeated use of the hand with altered biomechanics, together with loss of protective sensation, predisposes the fingers to recurrent microtrauma involving the collateral ligaments, dorsal extensor apparatus and periarticular soft tissues. These injuries initiate a cascade of edema, inflammation, reduced joint mobility

and disorganized collagen deposition, ultimately leading to contracture and stiffness of the proximal interphalangeal (PIP) joints [8, 9]. Secondary structural changes include tightening of the long flexor tendons, shortening of the collateral and oblique retinacular ligaments, attenuation of the extensor mechanism and habitual wrist flexion, all of which further restrict joint motion and perpetuate deformity [9]. Unlike trauma-related PIP stiffness, where immobilization and inflammation are the primary contributors, stiffness associated with leprosy is predominantly a consequence of chronic neuromuscular dysfunction, abnormal movement patterns and repetitive mechanical stress. These pathomechanical changes progressively transform a reversible motor deficit into fixed joint contractures, thereby limiting hand function and reducing the effectiveness of reconstructive surgical interventions [10].

This cascade can be summarized as follows:

Nerve impairment → Muscle imbalance → Altered movement patterns → Limited range of motion → Tissue adaptation (contractures) → Cortical maladaptation. Importantly, in leprosy, it is not muscle imbalance alone that initiates stiffness, but rather the maladaptive and asynchronous use of the hand. These dysfunctional patterns lead to cortical changes and abnormal feedback loops, further reinforcing the development of PIP joint contractures. While the brain is affected secondarily, cortical adaptation is not the root cause of stiffness-it is the result of continued dysfunctional motor use [11].

Management of PIP joint stiffness:

The management of PIP joint stiffness in leprosy requires a comprehensive understanding of its underlying pathophysiology and the stage at which the patient presents. Since stiffness develops through progressive muscle imbalance, altered biomechanics and maladaptive motor patterns, early intervention is essential to prevent irreversible contractures [12]. In patients presenting before the onset of secondary soft tissue changes, treatment should focus on preserving coordinated hand movements through structured therapeutic exercises that promote intrinsic muscle function, synchronous finger motion and proper joint alignment, thereby minimizing abnormal movement patterns and preventing cortical maladaptation [12, 13]. For individuals who require additional support or have difficulty adhering to exercise programs, orthotic devices such as knuckle-bender splints or lumbrical slings can help maintain metacarpophalangeal joint flexion, improve finger alignment and reduce mechanical stress on the proximal interphalangeal

(PIP) joints [13]. Preservation of soft tissue extensibility is equally important. Regular passive stretching of the flexor digitorum superficialis, flexor digitorum profundus, collateral ligaments and periarticular tissues-performed manually or with dynamic splints-helps prevent adaptive shortening and maintain joint mobility [14]. Because autonomic nerve dysfunction commonly accompanies sensory loss in leprosy, daily self-care practices, including soaking, scraping and oiling (SSO), are recommended to maintain skin integrity, improve tissue pliability and reduce the risk of secondary injuries [15]. When neuropathy is recognized early and rehabilitation protocols are initiated promptly, the progression to fixed PIP joint stiffness can often be prevented by interrupting the cycle of abnormal movement, repetitive microtrauma and tissue fibrosis [12-15]. Conversely, patients presenting with established contractures require meticulous clinical assessment to identify the predominant soft tissue restraints and determine the most appropriate management strategy, which may include intensive hand therapy, serial splinting, or reconstructive surgical procedures for advanced deformities [16].

Assessment of contributing structures to PIP joint stiffness:

A thorough clinical evaluation of the affected hand is crucial to determine the structures contributing to PIP joint stiffness. Several anatomical and soft tissue components may be involved, each requiring specific diagnostic techniques and therapeutic strategies [17].

Tightness of long flexor muscles:

Prolonged clawing of the fingers, resulting from ulnar and median nerve paralysis, can lead to shortening of the extrinsic long flexor muscles-particularly the Flexor Digitorum Superficialis (FDS) and Flexor Digitorum Profundus (FDP). This tightness can be assessed by sequentially passively extending the wrist, the metacarpophalangeal (MCP) joint, the proximal interphalangeal (PIP) joint and finally the distal interphalangeal (DIP) joint. Pain or discomfort in the forearm during this process is indicative of long flexor tightness [18].

Skin contracture:

In some cases, joint stiffness may be due solely to skin involvement without articular pathology. Skin contractures can be identified by passively extending the PIP joint to its limit. A springy or resistant feel on the volar aspect of the joint during this motion suggests the presence of a skin contracture [19].

Capsular contracture:

A capsular contracture of the PIP joint is typically characterized by a firm, abrupt block to passive extension. This suggests involvement of the joint capsule and intra-articular structures. Early detection is critical, as conservative therapy (e.g., stretching and splinting) is more effective before fixed contractures become established [20].

Ankylosis or fusion of the PIP joint:

In severe cases, the PIP joint may become completely immobile, showing no movement in either flexion or extension. This ankylosis may result from joint degeneration, chronic disuse, or fibrosis. Additionally, secondary changes such as contracture of the Oblique Retinacular Ligament (ORL) or attenuation of the dorsal extensor mechanism may impair PIP joint mobility. The ORL contracture can be suspected when DIP hyperextension occurs during PIP extension-especially with the MCP joint flexed. These findings typically indicate a need for surgical intervention. When complete loss of joint range of motion is observed, radiographic imaging is recommended to differentiate between soft tissue and bony involvement and to guide reconstructive planning [21].

Therapeutic approach based on specific causes of PIP joint stiffness:

Tissue adaptations resulting in PIP joint stiffness may occur at varying rates depending on the extent of hand use and adherence to prescribed exercises or orthotic interventions. Early recognition of the contributing factor is essential to guide effective treatment. Passive extension exercises remain a foundational strategy for preventing and managing contractures. When contractures are already present, these exercises should be supplemented with serial casting or appropriate splinting techniques [22].

The therapeutic approach should be tailored to the underlying cause:

Skin contracture:

When stiffness is attributed to skin contracture, management is generally straightforward. Regular passive stretching combined with the application of cylindrical splints is often sufficient to restore and maintain range of motion [23].

Capsular contracture:

In cases where the joint capsule is involved, a combination of prolonged passive stretching, therapeutic orthosis and daily Soaking, Scraping and Oiling (SSO) of the hand is recommended. These interventions aim to reduce intra-articular stiffness and improve joint pliability [24].

Joint surface involvement (Structural damage or ankylosis):

When stiffness is caused by articular damage or ankylosis, management becomes more complex and often requires a longer course of therapy, typically extending up to six weeks or more. A structured regimen of therapeutic exercises combined with splinting (e.g., gutter splints, cylindrical splints, or dynamic orthoses) may be necessary. If conservative measures fail to improve joint mobility, surgical evaluation should be considered. The selection of splint type and exercise intensity should be continually re-evaluated based on patient progress and compliance, ensuring the approach remains individualized and goal-directed [25]. A thorough review of the literature reveals that managing joint stiffness following hand injuries requires a comprehensive, multidimensional approach. Joint stiffness can result from a range of intrinsic factors, such as

tissue damage and extrinsic influences, including improper rehabilitation or insufficient activity levels. The evidence underscores the critical role of early intervention in preventing permanent joint changes and improving long-term functional outcomes [26]. Key to successful rehabilitation is the restoration of range of motion, particularly at the PIP joint, which is essential for efficient hand function. Both passive and active exercise interventions, along with orthotic use, form the cornerstone of treatment. While the literature consistently supports the efficacy of passive extension exercises, additional measures-such as serial casting or splinting-may be required for patients with severe contractures or joint stiffness [27]. However, treatment strategies must be individualized to account for patient-specific factors, including the underlying cause of stiffness, comorbid conditions and overall rehabilitation objectives. For some patients, long-term management may involve a combination of manual therapy, orthotic interventions and tailored exercises. Throughout this process, a key focus should be the establishment of progressive functional goals, with the aim of enhancing patient independence and quality of life.

Conclusion:

Long flexor tightness is a significant contributor to proximal interphalangeal joint stiffness in low ulnar nerve palsy, particularly in leprosy. Early recognition and targeted rehabilitation may preserve joint mobility and improve hand function. Further research is required to establish the mechanisms and optimize treatment strategies for nerve-related joint stiffness.

References:

- [1] <https://www.who.int/publications/i/item/who-wer9937-501-521>
- [2] Aman M *et al.* *Eur J Med Res.* 2023 **28**:116. [PMID: 36907874]
- [3] Ebenezer GJ & Scollard DM. *Neurotherapeutics.* 2021 **18**:2337. [PMID: 34799845]
- [4] Zhang Z *et al.* *J Clin Invest.* 2021 **131**:e146643. [PMID: 34499625]
- [5] Deps PD *et al.* *Indian J Med Res.* 2025 **161**:120. [PMID: 40257140]
- [6] Khadilkar SV *et al.* *J Neurol Sci.* 2021 **420**:117288. [PMID: 33360424]
- [7] Cruz VA *et al.* *Best Pract Res Clin Rheumatol.* 2025 **39**:102039. [PMID: 39915143]
- [8] Binder-Markey BI *et al.* *J Hand Surg Am.* 2019 **44**:751. [PMID: 31248678]
- [9] Froes Junior LAR *et al.* *Rev Inst Med Trop Sao Paulo.* 2025 **67**:e74. [PMID: 41191719]
- [10] Willis M *et al.* *PLoS One.* 2025 **20**:e0330477. [PMID: 41166248]
- [11] Zink PJ & Philip BA. *Am J Occup Ther.* 2020 **74**:7401205030p1. [PMID: 32078514]
- [12] Rashidi M *et al.* *Cureus.* 2025 **17**:e77799. [PMID: 39991405]
- [13] Calderone A *et al.* *Neurol Int.* 2024 **16**:1492. [PMID: 39585070]
- [14] Chauhan S *et al.* *Rheumatology (Oxford).* 2010 **49**:2237. [PMID: 20724432]
- [15] Han XY *et al.* *Am J Clin Pathol.* 2008 **130**:856. [PMID: 19019760]
- [16] Aride DB *et al.* *Clin Exp Rheumatol.* 2024 **42**:746. [PMID: 38372720]
- [17] Tosti R *et al.* *J Am Acad Orthop Surg.* 2013 **21**:581. [PMID: 24084432]
- [18] Prosser R. *J Hand Ther.* 1996 **9**:378. [PMID: 8994014]
- [19] Cho H *et al.* *J Clin Med.* 2024 **13**:1516. [PMID: 38592344]
- [20] Houshian S *et al.* *J Hand Surg Am.* 2013 **38**:1651. [PMID: 23890503]
- [21] Kawakatsu M *et al.* *J Hand Surg Am.* 2010 **35**:1457. [PMID: 20709466]
- [22] Inkellis E *et al.* *Hand Clin.* 2018 **34**:251. [PMID: 29625644]
- [23] Zan T *et al.* *Zhonghua Shao Shang Za Zhi.* 2016 **32**:463. [PMID: 27562155]
- [24] Sharma M *et al.* *Indian J Orthop.* 2021 **55**:1158. [PMID: 34824716]
- [25] Gashaw M *et al.* *Orthop Res Rev.* 2022 **14**:157. [PMID: 35586200]
- [26] Yates SE *et al.* *J Hand Ther.* 2024 **37**:495. [PMID: 38278697]

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