DOI: 10.4274/jarem.galenos.2025.88155
J Acad Res Med 2025;15(3):146-51

Frequency of Hypermobility in Patients with Ulnar Entrapment Neuropathy

Ömer Faruk Bucak[1], Pınar Öztop Çiftkaya[1], Emel Ece Özcan-Ekşi[2], Demet Ofluoğlu[3]

[1]University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Department of Physical Medicine and Rehabilitation, İstanbul, Türkiye
[2]Acıbadem Bağdat Street Medical Center, Clinic of Physical Medicine and Rehabilitation, İstanbul, Türkiye
[3]Bahçeşehir University Faculty of Medicine, Department of Physical Medicine and Rehabilitation, İstanbul, Türkiye

Cite this article as: Bucak ÖF, Öztop Çiftkaya P, Özcan-Ekşi EE, Ofluoğlu D. Frequency of hypermobility in patients with ulnar entrapment neuropathy. J Acad Res Med. 2025;15(3):146-51

ABSTRACT

Objective: To evaluate the association between generalized joint hypermobility (GJH) and electrodiagnostically confirmed ulnar neuropathy at the elbow (UNE), and to examine relationships between hypermobility measures and electrophysiological severity.

Methods: In a cross-sectional study at a tertiary center, 96 adults were enrolled: 48 UNE patients (confirmed by standardized nerve conduction studies) and 48 age/sex-matched controls. Hypermobility was assessed with age-specific Beighton thresholds, following the 2017 framework. GJH status incorporated the five-part questionnaire when borderline. Primary electrophysiological outcomes were distal motor latency (DML) and across-elbow/below-elbow motor conduction velocity (AE-BE MCV). Group comparisons used t-test/χ^2; associations used Spearman correlation (two-tailed α=0.05).

Results: Hypermobility indices were higher in UNE versus controls: Beighton score 3.4±2.1 vs. 2.0±1.5 (p=0.021) and GJH prevalence 68.8% vs 16.7% (p<0.001). Among UNE patients, age correlated with worse electrophysiology (DML: r=0.33, p=0.027; AE-BE MCV: r=-0.30, p=0.034). Higher Beighton scores are related to longer DML (r=0.28, p=0.041) and lower AE-BE MCV (r=-0.27, p=0.041). Longer symptom duration showed similar patterns (DML: r=0.34, p=0.023; AE-BE MCV: r=-0.32, p=0.028). Body mass index was not associated with the measured outcome (p>0.05). The presence of GJH correlated with higher DML (r=0.22, p=0.040) and lower AE-BE MCV (r=-0.24, p=0.036).

Conclusion: GJH is more prevalent in UNE and is linked to electrophysiological evidence of segmental conduction impairment at the elbow. Recognizing hypermobility may help stratify risk and expedite early evaluation and tailored prevention.

Keywords: Electromyography, ulnar nerve entrapment, joint instability, joint hypermobility, peripheral nerve injuries, numbness

INTRODUCTION

Ulnar neuropathy at the elbow (UNE) is the second most common entrapment neuropathy after carpal tunnel syndrome and represents a significant cause of upper extremity disability (1). Clinically, UNE presents with numbness, paresthesia, muscle weakness, and functional impairment in the forearm and hand, leading to a substantial reduction in quality of life and work productivity (2,3). Epidemiological data suggest that the prevalence of UNE can be as high as 5.9% in the general population, and increases further among those exposed to repetitive elbow movements in occupational settings (4). The resulting work disability and increased healthcare expenditures highlight UNE not only as a clinical problem but also as a significant socioeconomic burden one study reported that half of UNE patients received wage replacement for more than six months, with average direct and indirect costs totaling around USD 35,000 per case (5).

The pathophysiology of UNE involves several mechanisms, including compression within the cubital tunnel, traction during repetitive flexion-extension, and dynamic instability of the ulnar nerve. Known risk factors include prolonged elbow flexion, external compression, and elbow trauma (6,7). However, UNE does not develop in all individuals exposed to these factors, suggesting the contribution of intrinsic host-related susceptibility in addition to mechanical stress (8).

One such intrinsic factor is generalized joint hypermobility (GJH), characterized by increased connective tissue laxity and excessive joint range of motion (9). The prevalence of GJH varies depending on age, sex, and ethnicity, but rates as high as 10% have been reported in young adults (10,11). Women and younger

ORCID IDs of the authors: Ö.F.B. 0000-0003-3995-8120; P.Ö.Ç. 0000-0002-1718-8762; E.E.Ö.E. 0000-0001-5704-7019; D.O. 0000-0003-4273-5518

Corresponding Author: Ömer Faruk Bucak, MD;
E-mail: omerbucak46@gmail.com
Received Date: 02.09.2025 **Accepted Date:** 23.10.2025
Epub: 10.11.2025
Publication Date: 02.12.2025

J Acad Res Med 2025;15(3):146-51

individuals are disproportionately affected; in one recent adult cohort, the prevalence of GJH was 48.2% in females versus 20.4% in males (12).

Beyond musculoskeletal symptoms, GJH is linked to joint instability—including recurrent subluxations, ligamentous alterations, and soft tissue injuries—that can impose increased mechanical stress on peripheral nerves (13). In the elbow joint, laxity of the supporting connective tissues may predispose the ulnar nerve to subluxation or luxation during flexion, thereby amplifying friction and traction forces that facilitate UNE development (14). Recent studies lend support to this hypothesis. Dynamic ultrasonography has demonstrated a higher frequency of ulnar nerve instability during elbow flexion in hypermobile individuals (15). Similarly, surgical series have reported greater intraoperative mobility of the ulnar nerve in patients with joint hypermobility (16).

Nevertheless, the available literature remains limited. Most studies are small in scale, use heterogeneous definitions of hypermobility, and frequently lack electrodiagnostic (EDX) confirmation (17,18). Given that EDX studies are considered the gold standard for UNE diagnosis, this represents an important methodological gap (19). Current guidelines recommend standardized conduction protocols—including short-segment "inching" stimulation across the elbow—as well as defined thresholds for conduction velocity and amplitude changes, which provide high sensitivity for early diagnosis and accurate severity grading (15,18).

Against this background, the relationship between GJH and UNE warrants systematic investigation using contemporary diagnostic standards. Therefore, the present study aimed to evaluate the association between GJH and by EDX confirmed UNE in adults. We hypothesized that GJH may represent an independent host susceptibility factor, associated with UNE beyond the effects of age and sex.

METHODS

Study Design and Ethics

This cross-sectional observational study was conducted at the department of physical medicine and rehabilitation, a tertiary care university hospital, between April 2017 and November 2017. The study protocol was approved by the Başkent University Institutional Ethics Committee (decision no: 16/05, project no: KA15/382, date: 12.01.2016). All procedures adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Participants

Patients referred to the electroneuromyography laboratory with a preliminary clinical diagnosis of UNE were screened consecutively. Inclusion criteria were: (1) age between 18 to 65 years, (2) the presence of typical clinical symptoms (paresthesia in ulnar digits, nocturnal worsening of symptoms, weakness in intrinsic hand muscles), and (3) confirmation of UNE by both clinical and electrophysiological criteria. Exclusion criteria were (1) occupational risk factors with high repetitive elbow strain, (2) systemic diseases associated with neuropathy (e.g., diabetes mellitus, hypothyroidism, rheumatoid arthritis, crystal arthropathy), (3) history of elbow fracture, trauma, or surgery, (4) coexisting cervical radiculopathy, brachial plexopathy, or generalized polyneuropathy, (5) inability to complete standardized evaluations. Eligible patients were assigned to group 1 (UNE group) while age- and sex-matched healthy volunteers without neurological or rheumatological disease comprised group 2 (control group). Demographic and clinical data recorded included age, sex, body mass index (BMI), hand dominance, occupation, medical history, symptom duration, and symptom characteristics. Neurological examination included manual muscle testing of intrinsic hand muscles, sensory examination of the upper limb, and evaluation of Tinel's sign at the elbow (Figure 1).

Hypermobility Assessment

GJH was operationalized in line with the 2017 International Classification framework for hypermobility spectrum disorders and hypermobile Ehlers-Danlos syndrome (hEDS) (20). We did not attempt to diagnose hEDS; the exposure of interest was GJH as defined by age-specific Beighton thresholds. The Beighton examination (0-9) was performed bilaterally following a standardized script and without warm-up or stretching; borderline elbow/knee hyperextension was verified with a goniometer. Cut-offs were ≥5 for adults aged 18-50 years and ≥4 for those >50 years. In accordance with the 2017 framework, participants scoring one point below the relevant cut-off (i.e., Beighton= 4 for ages 18-50; Beighton= 3 for >50) completed the five-item historical hypermobility questionnaire (5PQ); therefore, a 5PQ score ≥2 was considered evidence of historical hypermobility and such individuals were classified as GJH-positive (20-22). All joint laxity assessments were performed independently by blinded physiatrists (masked to case/control status and EDX results).

Electrophysiological Examination

Electrophysiological examinations were done bilaterally, including the following techniques: (1) sensory orthodromic nerve conduction studies of the median and ulnar nerve were registered at the wrist stimulating the third and fifth digits, respectively; (2) median motor nerve conduction study was registered at the abductor pollicis brevis muscle stimulating the wrist and antecubital fossa; (3) ulnar motor nerve conduction study was registered at the abductor digiti minimi muscle stimulating the wrist, below elbow, and above elbow; (4) short segment technique at the elbow for ulnar nerve (stimulating 6 points separated by 2 cm segments from 4 cm distal to 6 cm proximal to the medial epicondyle). During electrophysiological examinations, subjects were lying in the supine position, and their elbows were flexed at 90° for an ulnar nerve conduction study. American Association of Neuromuscular and Electrodiagnostic Medicine criteria were used for the diagnosis of cubital tunnel syndrome (23,24).

J Acad Res Med 2025;15(3):146-51

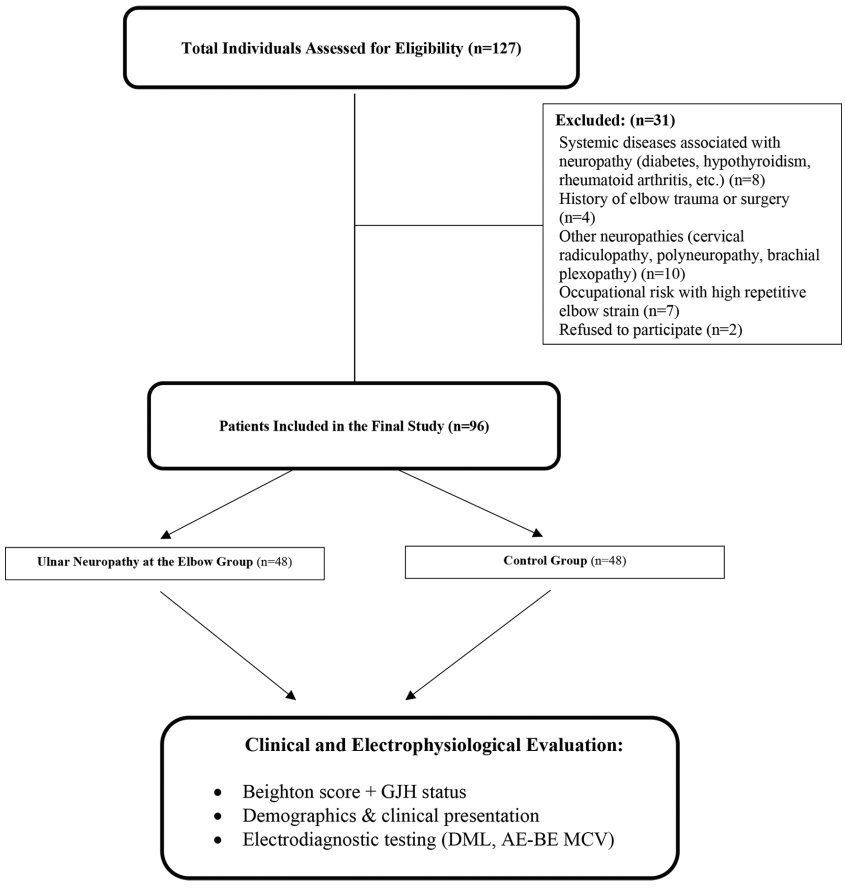

Figure 1. Flow chart of the study
GJH: Generalized joint hypermobility, DML: Distal motor latency, AE-BE MCV: Across-elbow motor conduction velocity

If any of the following findings were found in the study, the results were accepted as ulnar nerve entrapment at the elbow: an absolute nerve conduction velocity above elbow-to-below elbow of <50 m/sec, an above elbow-to-below elbow conduction velocity >10 m/sec slower or a 20% slowing compared with the below elbow-to-wrist segment, a decrease in compound muscle action potential peak amplitude from below elbow-to-above elbow of >20%, a significant change in compound muscle action potential configuration between the above and below elbow sites.

In addition to the above-mentioned techniques, ulnar nerve entrapment at the elbow was considered if any latency exceeded 0.7 msec in the short segment study technique. All electrophysiological examinations were done by an experienced physiatrist, using a Nihon Kohden® electrophysiological device.

Statistical Analysis

All statistical analyses were performed using SPSS statistics version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Normality of continuous data was assessed using the Kolmogorov-Smirnov test and inspection of histograms. Between-group comparisons of continuous variables (e.g., Beighton score, age, BMI) were performed with the independent-samples Student's t-test for normally distributed data. Categorical variables (e.g., sex distribution, prevalence of GJH) were compared using the chi-square test (χ^2) or Fisher's exact test when appropriate. Electrophysiological parameters, including distal motor latency (DML) and across-elbow/below-elbow motor conduction velocity (AE-BE MCV), were analyzed as continuous outcomes. Associations between clinical variables (age, Beighton score, symptom duration, BMI, and GJH status) and electrophysiological parameters were examined using the Spearman rank correlation test, as data were not normally distributed. Two-tailed p-values <0.05 were considered statistically significant.

RESULTS

A total of 96 adults were enrolled (48 with by EDX confirmed UNE and 48 controls); in total, 192 ulnar nerves underwent conduction studies. Baseline characteristics were comparable between groups: age 44.0±14.4 vs 45.7±9.7 years (p=0.502), female sex 32/48 (66.7%) vs 33/48 (68.8%) (p=0.830), and right-hand dominance 46/48 (95.8%) vs 47/48 (97.9%) (p=0.564). Within the UNE cohort, involvement was 54.2% left (26/48), 16.7% right (8/48), and 29.2% bilateral (14/48); the mean symptom duration was 157.6±18.8 months. The most frequent presenting symptom

J Acad Res Med 2025;15(3):146-51

was numbness in digits IV and V (29/48), followed by hand pain (9/48), nocturnal numbness (5/48), weakness (3/48), and multiple symptoms (2/48) (Table 1).

Hypermobility indices were higher in UNE: Beighton score 3.4±2.1 vs 2.0±1.5 (p=0.021), and GJH prevalence (2017 framework) 68.8% (33/48) vs 16.7% (8/48) (p<0.001) (Table 2).

Age was positively correlated with DML (r=0.33, p=0.027) and negatively correlated with AE-BE MCV (r=-0.30, p=0.034). Beighton score showed a positive correlation with DML (r=0.28, p=0.041) and a negative correlation with AE-BE MCV (r=-0.27, p=0.041). Symptom duration was positively correlated with DML (r=0.34, p=0.023) and negatively with AE-BE MCV (r=-0.32, p=0.028). No significant correlation was found between BMI and electrophysiological parameters (DML: r=0.08, p=0.492; AE-BE MCV: r=-0.12, p=0.287). The presence of GJH (GJH, 2017 framework) was positively correlated with DML (r=0.22, p=0.040) and negatively correlated with AE-BE MCV (r=-0.24, p=0.036) (Table 3).

Table 1. Baseline characteristics of participants and clinical presentation in the UNE patients

Parameters	UNE (n=48)	Control (n=48)	p-value
Age (year) (mean ± SD)	44±14.4	45.7±9.7	0.502
Gender (%)			
Female	32 (66.7%)	33 (68.8%)	
Male	16 (33.3%)	15 (31.2%)	0.830
BMI (kg/m²) (mean ± SD)	24.2±3.3	23.5±3.7	0.336
Dominant hand (%)			
Right-handed	46 (95.8%)	47 (97.9%)	
Left-handed	2 (4.1%)	1 (2.1%)	0.564
Involvement side, n (%)			
Left	26 (54.2%)	-	
Right	8 (16.7%)	-	
Bilateral	14 (29.2%)	-	
Symptom duration (months) (mean ± SD)	157.6±18.8	-	
Presenting symptom, n (%)			
Numbness in digits IV-V	29 (60.4%)	-	
Hand pain	9 (18.8%)	-	
Nocturnal numbness	5 (10.4%)	-	
Weakness	3 (6.3%)	-	
Multiple symptoms	2 (4.2%)	-	

Values are **mean ± SD** for continuous variables and **n (%)** for categorical variables
UNE: Ulnar neuropathy at the elbow, BMI: Body mass index, SD: Standard deviation

Table 2. Comparison of hypermobility indices between UNE and control groups

Outcomes	UNE (n=48)	Controls (n=48)	p-value
Beighton score (mean ± SD)	3.4±2.1	2.0±1.5	**0.021***
GJH (2017 framework), n (%)			
Presence	68.75% (n=33)	16.6% (n=8)	**<0.001****
Absence	31.25 % (n=15)	83.4% (n=40)	

Values are **mean ± SD** for continuous variables and **n (%)** for categorical variables
*: Student's t-test, p<0.05 considered statistically significant, **: Chi-square test,
UNE: Ulnar neuropathy at the elbow, GJH: Generalized joint hypermobility, SD: Standard deviation

J Acad Res Med 2025;15(3):146-51

Table 3. Correlation of clinical variables with electrophysiological parameters in UNE patients

Predictor	DML r (rho)	DML (p-value)	AE-BE MCV r (rho)	AE-BE MCV (p-value)
Age (years)	**0.33**	**0.027**	**-0.30**	**0.034**
Beighton score (0-9)	**0.28**	**0.041**	**-0.27**	**0.041**
Symptom duration (months)	**0.34**	**0.023**	**-0.32**	**0.028**
BMI (kg/m²)	0.08	0.492	-0.12	0.287
GJH (2017 framework)	**0.22**	**0.040**	**-0.24**	**0.036**

Spearman rank correlation coefficients (r) and corresponding two-tailed p-values are shown separately. Bold indicates statistically significant correlations ($p<0.05$) Negative r: Indicates inverse association, DML: Distal motor latency, AE-BE MCV: Across-elbow/below-elbow, motor conduction velocity BMI: Body mass index, GJH: Generalized joint hypermobility

DISCUSSION

Our study indicates that GJH is considerably more common in patients with by EDX confirmed UNE than in matched controls. Higher Beighton scores and GJH status on its own align with a less favorable electrophysiological profile, which is reflected by longer DML and slower across-elbow conduction velocity. These patterns mirror the effects of older age and longer symptom duration, whereas BMI shows no meaningful association with nerve conduction. Taken together, the data support a model in which intrinsic connective-tissue properties, rather than general body habitus, influence segmental conduction in UNE, consistent with contemporary views that host factors help shape entrapment neuropathies beyond external mechanical load alone (8,25).

In line with these findings, recent ultrasound studies demonstrate that elbow flexion alters the shape of the cubital tunnel, increases ulnar nerve movement, and can lead to temporary subluxation or dislocation, even in otherwise healthy individuals (15,26,27). This provides a clear structural basis on which connective tissue properties may influence vulnerability to UNE.

From a mechanistic standpoint, ligamentous and retinacular laxity likely amplifies flexion-induced narrowing of the cubital tunnel and raises intra-tunnel pressure, increasing ulnar-nerve excursion, contact stress, and shear, changes that culminate in focal demyelination, resulting in the distal latency/velocity pattern we observed (28).

In parallel, contemporary reviews integrating ultrasound, clinical, and electrophysiological data describe how positional narrowing, intermittent compression, and perineural microvascular instability can converge to impair conduction in cubital tunnel syndrome (25). Importantly, dynamic instability on imaging correlates with greater electrophysiological severity, reinforcing that laxity is not merely an anatomic variant but a physiologically relevant risk state (29).

Epidemiologically, GJH is reported in approximately 2-57% of the general population, with prevalence influenced by age, sex, and ethnicity; making its marked enrichment in our EDX-confirmed UNE cohort unlikely to be coincidental (30).

Comparable associations between joint laxity and conduction impairment have been reported in other entrapment neuropathies, such as wrist neuropathies in hEDS, and our EDX-confirmed UNE findings echo this cross-site pattern by translating anatomical susceptibility into measurable electrophysiological change (31).

Clinically, recognizing hypermobility as a modifier of UNE risk highlights the value of routine Beighton screening, which can help identify patients who may benefit from early stabilization strategies and tailored follow-up (25,32). Collectively, these mechanistic and epidemiologic signals support treating GJH as a true disease modifier rather than a coincidental comorbidity.

This study's key strength is that it links a clear host phenotype to objective nerve physiology in an EDX-confirmed UNE cohort. It shows the effect across two independent markers: prolonged DML, and reduced across-elbow conduction velocity. A matched-control design, prespecified adjustment for age, sex, symptom duration, and BMI, and signal stability in sensitivity analyses collectively support strong internal validity.

Study Limitations

This study has several limitations that should be acknowledged. First, its single-center design may restrict the generalizability of the findings to broader populations. Second, the cross-sectional nature of the study precludes any inference of causal relationships between GJH and the development of UNE. Third, although the sample size was sufficient to detect significant associations, it remained relatively modest, which may have limited the statistical power to identify subtler effects. Finally, advanced imaging modalities such as dynamic ultrasound or MRI, were not incorporated to complement the electrophysiological assessments, which could have provided additional insights into structural mechanisms underlying nerve instability.

CONCLUSION

GJH appears to be a significant host-related susceptibility factor for UNE, with potential implications for clinical assessment and management. Recognition of hypermobility in patients presenting with ulnar-distribution symptoms may support earlier EDX evaluation and guide targeted preventive and rehabilitative strategies, such as ergonomic counseling and stabilization-focused physiotherapy. Future multicenter prospective studies integrating electrophysiology and imaging are warranted to

J Acad Res Med 2025;15(3):146-51

confirm causality, clarify underlying mechanisms, and evaluate whether tailored interventions can reduce risk in hypermobile individuals.

Ethics

Ethics Committee Approval: The study protocol was approved by the Başkent University Institutional Ethics Committee (decision no: 16/05, project no: KA15/382, date: 12.01.2016).

Informed Consent: Written informed consent was obtained from all participants prior to enrollment.

Footnotes

Author Contributions: Surgical and Medical Practices - Ö.F.B.; Concept - Ö.F.B., P.Ö.Ç., D.O.; Design - Ö.F.B., P.Ö.Ç., E.E.Ö.E., D.O.; Data Collection and/or Processing - Ö.F.B., P.Ö.Ç., E.E.Ö.E., D.O.; Analysis and/or Interpretation - Ö.F.B., P.Ö.Ç.; Literature Search - Ö.F.B., P.Ö.Ç., E.E.Ö.E., D.O.; Writing - Ö.F.B., P.Ö.Ç.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors report that no financial support was received for this study.

REFERENCES

1. Caliandro P, La Torre G, Padua R, Giannini F, Padua L. Treatment for ulnar neuropathy at the elbow. Cochrane Database Syst Rev. 2016; 11: CD006839.
2. Song JW, Waljee JF, Burns PB, Chung KC, Gaston RG, Haase SC, et al.; Surgery for the Ulnar Nerve (SUN) Study Group. An outcome study for ulnar neuropathy at the elbow: a multicenter study by the surgery for ulnar nerve (SUN) study group. Neurosurgery. 2013; 72: 971-82.
3. Reischl S, MacDermid J. A retrospective analysis of quality of life domains impacted by distal radius fracture and ulnar neuropathy. J Hand Ther. 2025; 38: 33-41.
4. An TW, Evanoff BA, Boyer MI, Osei DA. The prevalence of cubital tunnel syndrome: a cross-sectional study in a U.S. metropolitan cohort. J Bone Joint Surg Am. 2017; 99: 408-16.
5. Juratli SM, Nayan M, Fulton-Kehoe D, Robinson LR, Franklin GM. A population-based study of ulnar neuropathy at the elbow in Washington State workers' compensation. Am J Ind Med. 2010; 53: 1242-51.
6. Cambon-Binder A. Ulnar neuropathy at the elbow. Orthop Traumatol Surg Res. 2021; 107: 102754.
7. Albano D, Di Rocco G, Gitto S, Serpi F, Fusco S, Vitali P, et al. Imaging of elbow entrapment neuropathies. Insights Imaging. 2025; 16: 24.
8. Nyman E, Dahlin LB. The unpredictable ulnar nerve-ulnar nerve entrapment from anatomical, pathophysiological, and biopsychosocial aspects. Diagnostics (Basel). 2024; 14: 489.
9. Tinkle BT. Symptomatic joint hypermobility. Best Pract Res Clin Rheumatol. 2020; 34: 101508.
10. Reuter PR, Fichthorn KR. Prevalence of generalized joint hypermobility, musculoskeletal injuries, and chronic musculoskeletal pain among American university students. PeerJ. 2019; 7: e7625.
11. Zhong G, Zeng X, Xie Y, Lai J, Wu J, Xu H, et al. Prevalence and dynamic characteristics of generalized joint hypermobility in college students. Gait Posture. 2021; 84: 254-9.
12. Barbosa C, Gavinha S, Soares T, Reis T, Manso C. Is generalized joint hypermobility associated with chronic painful temporomandibular disorders in young adults? A cross-sectional study. J Clin Med. 2024; 14: 44.
13. Carroll MB. Hypermobility spectrum disorders: a review. Rheumatol Immunol Res. 2023; 4: 60-8.
14. Kawahara Y, Yamaguchi T, Honda Y, Tomita Y, Uetani M. The ulnar nerve at elbow extension and flexion: assessment of position and signal intensity on mr images. Radiology. 2016; 280: 483-92.
15. Tsukada K, Yasui Y, Sasahara J, Okawa Y, Nakagawa T, Kawano H, et al. Ulnar nerve dislocation and subluxation from the cubital tunnel are common in college athletes. J Clin Med. 2021; 10: 3131.
16. Mirza A, Mirza JB, Thomas TL Jr. Classification and treatment of ulnar nerve subluxation following endoscopic cubital tunnel release. J Hand Surg Glob Online. 2020; 2: 232-9.
17. Yadav RL. A retrospective study of electrodiagnostically evaluated ulnar neuropathies with special guidelines for ulnar neuropathies at elbow. Beni-Suef Univ J Basic Appl Sci. 2022; 11: 31.
18. Pelosi L, Arányi Z, Beekman R, Bland J, Coraci D, Hobson-Webb LD, et al. Expert consensus on the combined investigation of ulnar neuropathy at the elbow using electrodiagnostic tests and nerve ultrasound. Clin Neurophysiol. 2021; 132: 2274-81.
19. Campbell WW, Carroll C, Landau ME. Ulnar neuropathy at the elbow: five new things. Neurol Clin Pract. 2015; 5: 35-41.
20. Malfait F, Francomano C, Byers P, Belmont J, Berglund B, Black J, et al. The 2017 international classification of the Ehlers-Danlos syndromes. Am J Med Genet C Semin Med Genet. 2017; 175: 8-26.
21. Yew KS, Kamps-Schmitt KA, Borge R. Hypermobile Ehlers-Danlos syndrome and hypermobility spectrum disorders. Am Fam Physician. 2021; 103: 481-92.
22. Glans M, Humble MB, Elwin M, Bejerot S. Self-rated joint hypermobility: the five-part questionnaire evaluated in a Swedish non-clinical adult population. BMC Musculoskelet Disord. 2020; 21: 174.
23. Werner RA. Electrodiagnostic evaluation of carpal tunnel syndrome and ulnar neuropathies. PM R. 2013; 5: s14-21.
24. Practice parameter for electrodiagnostic studies in ulnar neuropathy at the elbow: summary statement. American Association of Electrodiagnostic Medicine, American Academy of Neurology, American Academy of Physical Medicine and Rehabilitation. Muscle Nerve. 1999; 22: 408-11.
25. Mezian K, Jačisko J, Kaiser R, Machač S, Steyerová P, Sobotová K, et al. Ulnar neuropathy at the elbow: from ultrasound scanning to treatment. Front Neurol. 2021; 12: 661441.
26. Nakano K, Murata K, Omokawa S, Nakanishi Y, Shimizu T, Kira T, et al. Dynamic analysis of the ulnar nerve in the cubital tunnel using ultrasonography. J Shoulder Elbow Surg. 2014; 23: 933-7.
27. Endo F, Tajika T, Kuboi T, Shinagawa S, Tsukui T, Nakajima T, et al. The ultrasonographic assessment of the morphologic changes in the ulnar nerve at the cubital tunnel in Japanese volunteers: relationship between dynamic ulnar nerve instability and clinical symptoms. JSES Int. 2021; 5: 942-7.
28. Choi CK, Lee HS, Kwon JY, Lee WJ. Clinical implications of real-time visualized ultrasound-guided injection for the treatment of ulnar neuropathy at the elbow: a pilot study. Ann Rehabil Med. 2015; 39: 176-82.
29. Kang JH, Joo BE, Kim KH, Park BK, Cha J, Kim DH. Ultrasonographic and electrophysiological evaluation of ulnar nerve instability and snapping of the triceps medial head in healthy subjects. Am J Phys Med Rehabil. 2017; 96: e141-6.
30. Blajwajs L, Williams J, Timmons W, Sproule J. Hypermobility prevalence, measurements, and outcomes in childhood, adolescence, and emerging adulthood: a systematic review. Rheumatol Int. 2023; 43: 1423-44.
31. Granata G, Padua L, Celletti C, Castori M, Saraceni VM, Camerota F. Entrapment neuropathies and polyneuropathies in joint hypermobility syndrome/Ehlers-Danlos syndrome. Clin Neurophysiol. 2013; 124: 1689-94.
32. Bage T, Power DM. Iatrogenic peripheral nerve injury: a guide to management for the orthopaedic limb surgeon. EFORT Open Rev. 2021; 6: 607-17.