THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Clinical and Electrophysiological Assessment of Peripheral Nerve Function After Transradial Angiography: A Prospective Study

ABSTRACT

Background: Transradial angiography (TRA) is widely used in contemporary coronary procedures. Although clinically apparent peripheral nerve injury after TRA is uncommon, subclinical nerve involvement may go unrecognized. This study aimed to objectively assess peripheral nerve function after TRA using neurological examination, standardized neuropathic pain questionnaires, and nerve conduction studies (NCS).

Methods: This prospective, single-center observational study included consecutive patients undergoing transradial coronary angiography. A total of 107 patients were analyzed. Neurological examination was performed within 24-48 hours after the procedure. Neuropathic symptoms were evaluated using the Douleur Neuropathique en 4 (DN4) and Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) questionnaires. Bilateral nerve conduction studies of the median, ulnar, and radial nerves were performed 1 month after TRA, and side-to-side comparisons were conducted.

Results: Side-to-side differences were observed in selected nerve conduction parameters. These electrophysiological changes predominantly involved the radial nerve on the procedure side, characterized by lower sensory amplitude, reduced conduction velocity, and decreased motor amplitude ($P < .05$). Median and ulnar nerve conduction findings were largely comparable between sides. Douleur Neuropathique en 4 and LANSS scores were in normal ranges and not associated with nerve conduction parameters. No clinically evident local or neurological complications were detected during follow-up.

Conclusion: Transradial angiography may be associated with mild, procedure-side predominant radial nerve conduction changes detectable by NCS, without clinically evident neuropathy. Objective electrophysiological assessment may therefore help identify underrecognized subclinical nerve involvement following TRA.

Keywords; Nerve conduction studies, peripheral nerve injuries, radial nerve, transradial angiography

ORIGINAL INVESTIGATION

INTRODUCTION

In recent years, the transradial approach (TRA) has become the preferred access route for coronary angiography. Its use is associated with fewer access site bleeding events and vascular complications than the transfemoral approach, resulting in better clinical outcomes, particularly in patients with acute coronary syndrome. As a result, international guidelines now recommend TRA as the default strategy for invasive coronary procedures.[1-5]

Transradial access may be associated with complications including radial artery spasm, catheter kinking, arterial dissection or perforation, radial artery occlusion, hematoma, pseudoaneurysm, arteriovenous fistula, and, less commonly, peripheral nerve injury. Although most TRA-related complications are mild and self-limited, some may lead to patient discomfort, limb dysfunction, prolonged hospitalization, and in rare cases, significant morbidity.[6-10]

Peripheral nerve injury after transradial access is rare and its true incidence is likely underestimated, as mild neurological symptoms are frequently overlooked. Proposed mechanisms include local hematoma, contrast extravasation,

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Received: February 2, 2026
Accepted: February 24, 2026
Available Online Date: May 11, 2026

Cite this article as: Dandinoğlu T, Kozan H, Güner A, Deniz O, Kaya E. Clinical and electrophysiological assessment of peripheral nerve function after transradial angiography: A prospective study. *Anatol J Cardiol.* 2026;XX(X):1-7.

DOI: 10.14744/AnatolJCardiol.2026.6273

compression-related ischemia, compartment syndrome, and complex regional pain syndrome, all of which may affect adjacent neural structures. In this setting, electrophysiological abnormalities may still be identified even in patients without clinically apparent neuropathic symptoms.[11-13]

Nerve conduction studies (NCS) provide an objective method for assessing peripheral nerve function and are capable of identifying subclinical or transient nerve involvement that may not correlate with patient-reported symptoms or neurological examination.[14,15] However, data regarding systematic electrophysiological evaluation of peripheral nerves following TRA remain limited, and existing studies have reported inconsistent findings.

This prospective study objectively evaluated the presence and characteristics of peripheral nerve involvement following TRA through comprehensive neurological examination, standardized neuropathic pain assessment, and nerve conduction studies.

METHODS

Study Design and Population

This prospective, single-center, observational cohort study was conducted at a tertiary referral hospital. Eligibility screening was performed consecutively in patients undergoing transradial coronary angiography. A total of 132 patients were initially assessed to reflect routine clinical practice and to allow a comprehensive evaluation of potential electrophysiological changes related to intervention.

At baseline, all patients underwent a detailed medical history review and comprehensive neurological examination. One month after the TRA procedure, peripheral NCS were performed on both the procedure side and the contralateral upper extremity.

Inclusion and Exclusion Criteria

Patients undergoing transradial coronary angiography were evaluated for eligibility. Individuals with peripheral polyneuropathy (PNP), a history of nerve injury or upper-extremity surgery/injury involving the peripheral nerves, and those with other neurological conditions known to affect nerve functions were not included. Patients unable to adequately cooperate with electrophysiological testing were also excluded. Carpal Tunnel syndrome (CTS) cases were retained in the primary analysis to reflect routine practice and to avoid unnecessary loss of sample size for non-median nerve comparisons. A prespecified secondary analysis was subsequently performed excluding patients with CTS to reduce potential confounding.

HIGHLIGHTS

- Peripheral nerve involvement after transradial angiography may remain clinically unrecognized.
- Nerve conduction studies revealed mild, procedure-side–predominant radial nerve changes.
- Electrophysiological findings were not associated with neuropathic pain scores.
- Objective assessment may help identify subclinical nerve involvement after transradial access.

Transradial Angiography Procedure

All angiographic procedures were performed by an experienced interventional cardiologist using a standardized transradial approach. Only patients who had undergone diagnostic coronary angiography for standard clinical indications, without additional interventional procedures, were included in the study. A standardized 6-French radial sheath was used in all cases. The average procedural duration was approximately 30 minutes, depending on anatomical complexity. The radial sheath was removed immediately after completion of angiography, and hemostasis was achieved using a dedicated radial compression device according to institutional protocol.

Clinical and Neurological Assessment

The neurological examination included assessment of muscle strength, superficial sensation, deep tendon reflexes, and neuropathic pain–related signs such as allodynia, hyperalgesia, and hyperpathia. This examination was carried out within 24-48 hours after the transradial procedure. Nerve conduction studies were performed separately at the 1-month follow-up visit.

Neuropathic complaints were evaluated using the Douleur Neuropathique en 4 (DN4) and Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) questionnaires as screening tools for neuropathic pain. A DN4 score of 4 or higher and a LANSS score of 12 or higher were considered indicative of neuropathic pain.[16,17]

Procedure-related local complications were systematically assessed during follow-up, and medication histories were reviewed for agents that could affect neuropathic pain assessment.

Electrophysiological Evaluation

Nerve conduction studies were performed 1 month after transradial angiography, as electrophysiological manifestations of peripheral nerve injury often require several weeks to become detectable and may be underestimated if assessed earlier. All recordings were performed in a dedicated electromyography laboratory using a commercially available system. Motor nerve studies used supramaximal stimulation with compound muscle action potentials recorded from standard target muscles. Sensory nerve studies were performed using routine antidromic techniques. Standard filter settings were used throughout the study (motor studies: 2 Hz-10 kHz; sensory studies: 20 Hz-2 kHz). Skin temperature was maintained at ≥32°C and monitored before each examination to minimize temperature-related variability. All recordings were performed in accordance with established electrodiagnostic guidelines. All electrophysiological studies were performed and interpreted by an experienced physiatrist.

Nerve conduction studies parameters were obtained from both the procedure side and the contralateral side, and side-to-side comparisons within the same patient were used to reduce inter-individual variability. All studies were

performed and interpreted by the same physician to ensure methodological consistency.

Laboratory Evaluation

Laboratory parameters known to influence peripheral nerve function were recorded, including vitamin B12, ferritin, thyroid function tests (TSH and free T4), glycemic status (HbA1c), and hematological indices (hemoglobin and mean corpuscular volume). These parameters were evaluated to identify potential metabolic or systemic confounders.

Statistical Analysis

Statistical analyses were performed using standard statistical software. The normality of continuous variables was assessed prior to analysis. Variables with a normal distribution are presented as mean ± standard deviation, whereas non-normally distributed variables are reported as median (interquartile range). Categorical variables are expressed as number and percentage. Side-to-side comparisons within the same patient were performed using paired *t*-tests for normally distributed data and Wilcoxon signed-rank tests for non-normally distributed data. Associations between neuropathic pain scores (DN4 and LANSS) and nerve conduction parameters were evaluated using Spearman's rank correlation coefficient. A *P* value <.05 was considered statistically significant. Given the exploratory nature of the analyses, no formal correction for multiple comparisons was applied.

Ethics

The study was approved by the Local Ethics Committee (15.10.2025-2025-KAEK-47) and was conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS

A total of 132 patients who underwent TRA were initially referred for post-procedural neurological and NCS. Among these, 25 patients were excluded based on predefined criteria, including poorly controlled diabetes (HbA1c > 7%; n=7), vitamin B12 deficiency (< 250 pg/mL; n=5), and use of medications that could influence neuropathic pain assessment (n=5). In addition, electrophysiological findings consistent with PNP were identified during NCS in 8 patients. After these exclusions, 107 patients constituted the final study population for the primary analysis. This analysis was performed to observe all potential electrophysiological changes associated with the procedure before excluding conditions that could independently affect peripheral nerve function. Subsequently, patients with CTS (n=16) were excluded, and a predefined secondary analysis was conducted in the remaining 91 patients.

Table 1 summarizes the demographic, laboratory and clinical characteristics of the final study population following exclusion of patients with CTS (n=91). Douleur Neuropathique en 4 and LANSS neuropathic pain scale scores were within normal ranges in the 91 patients included in the secondary analysis.

Side-to-side nerve conduction findings observed in the primary analysis of the entire cohort (n=107) are summarized in Tables 2 and 3. In this analysis, significant side-to-side differences were identified in selected nerve conduction parameters, including median motor nerve latency, amplitude, and conduction velocity, as well as median sensory nerve conduction velocity. Significant differences were also observed in radial nerve motor amplitude and in radial sensory nerve amplitude and conduction velocity. In contrast, ulnar motor and sensory nerve conduction measures were comparable between sides.

After exclusion of patients with CTS (n=91), side-related differences were limited to the radial nerve (Tables 4 and

Table 1. Demographic and Clinical Characteristics of the Study Population After Exclusion of CTS Cases (n=91)

Characteristics	Values
Age, years	56 ± 11
Sex (female/male) (%)	41 (45)/50 (55)
Vitamin B12 (pg/mL)	412 (318-546)
Ferritin (ng/mL)	74 (38-146)
TSH (μIU/mL)	1.92 (1.21-2.68)
Free T4 (ng/dL)	1.12 (0.98-1.26)
HbA1c (%)	5.8 ± 0.8
Hemoglobin (g/dL)	13.7 ± 1.4
MCV (fL)	89.6 ± 4.8
DN4 score	2 (0-3)
LANSS score	5 (2-8)

Values are presented as mean ± standard deviation, median (interquartile range), or number (percentage), as appropriate. DN4, Douleur Neuropathique en 4 Questions; HbA1c, glycated hemoglobin; LANSS, Leeds Assessment of Neuropathic Symptoms and Signs; MCV, mean corpuscular volume; TSH, thyroid-stimulating hormone.

Table 2. Primary Analysis (Overall Cohort, n=107): Side-to-Side Motor Nerve Conduction Comparisons

	Right	Left	*P*
Median motor nerve latency	3.33 (2.45-7.24)	3.31 (2.31-7.4)	**.035***
Median motor nerve amplitude	8.4 (3.3-14.9)	7.45 (1.4-16.5)	**<.001****
Median motor nerve velocity	50 (33-74)	51 (46-71)	**.036***
Ulnar motor nerve latency	2.59 ± 0.36	2.41 ± 0.42	.1
Ulnar motor nerve amplitude	7.65 (5.4-12.7)	7.8 (4.7-14)	.57
Ulnar motor nerve velocity	57.1 ± 7.6	57.9 ± 7.3	.23
Radial motor nerve latency	2.29 (1.25-3.28)	2.38 (1.25-3.75)	.19
Radial motor nerve amplitude	3.1 (0.6-7.4)	5.3 (3.2-7.9)	**.001****
Radial motor nerve velocity	63 (37-76)	64 (41-76)	.95

Values are presented as mean ± standard deviation or median (interquartile range), as appropriate. Right and left sides correspond to the procedure side and contralateral side, respectively. *P* values were obtained using paired statistical tests. Statistically significant *P* values are indicated with an asterisk (*$P < .05$, **$P < .001$).

Table 3. Primary Analysis (Overall Cohort, n = 107): Side-to-Side Sensory Nerve Conduction Comparisons

	Right	Left	*P*
Median sensory nerve latency	2.71 (2.15-5.05)	2.74 (2.08-4.1)	.12
Median sensory nerve amplitude	17.7 (2.86-48.60)	21.1 (1.96-52)	.42
Median sensory nerve velocity	49.78 ± 7.43	51.30 ± 6.98	**.009****
Ulnar sensory nerve latency	2.14 (1.77-3.75)	2.23 (1.18-4.27)	.44
Ulnar sensory nerve amplitude	20 (16.40-56.90)	21 (15.5-59)	.47
Ulnar sensory nerve velocity	54 (42-64)	56.5 (46-68)	.062
Radial sensory nerve latency	1.51 (0.73-3.96)	1.46 (0.94-3.78)	.26
Radial sensory nerve amplitude	20.15 (14.5-66)	23.5 (14.45-64)	**.004****
Radial sensory nerve velocity	55.18 ± 7.21	64.82 ± 7.24	**.027***

Values are presented as mean ± standard deviation or median (interquartile range), as appropriate. Right and left sides correspond to the procedure side and contralateral side, respectively. *P* values were obtained using paired statistical tests. Statistically significant *P* values are indicated with an asterisk (*$P < .05$, **$P < .001$).

5). Lower sensory amplitude and conduction velocity were observed on the procedure side, along with reduced radial motor amplitude ($P = .03$, $P = .042$, and $P = .04$). Radial latencies and median and ulnar NCS findings were similar on both sides.

Despite these procedure-side differences in radial nerve conduction parameters, correlation analyses demonstrated no significant association between DN4 or LANSS scores and radial sensory or motor nerve conduction findings (Table 6).

Table 4. Predefined Secondary Analysis Excluding CTS (n = 91): Side-to-Side Motor Nerve Conduction Comparisons

	Right	Left	*P*
Median motor nerve latency	3.10 (2.45-4.24)	3.05 (2.31-4)	.055
Median motor nerve amplitude	9.7 (5.3-14.9)	8.80 (5.4-16.5)	.061
Median motor nerve velocity	58 (49-74)	58 (52-71)	.054
Ulnar motor nerve latency	2.45 ± 0.38	2.35 ± 0.43	.81
Ulnar motor nerve amplitude	8.6 (5.6-12.7)	8.2 (5.8-14)	.66
Ulnar motor nerve velocity	55.3 ± 5.6	56.7 ± 6.3	.35
Radial motor nerve latency	2.20 (1.25-2.98)	2.30 (1.25-3.60)	.24
Radial motor nerve amplitude	3.2 (1.5-7.4)	5.4 (3.69-7.9)	**.04***
Radial motor nerve velocity	66 (44-76)	62 (41-70)	.65

Table 5. Predefined Secondary Analysis Excluding CTS (n = 91): Side-to-Side Sensory Nerve Conduction Comparisons

	Right	Left	*P*
Median sensory nerve latency	2.6 (2.15-4.15)	2.51 (2.08-3.1)	.26
Median sensory nerve amplitude	27 (20-48.60)	29 (22-52)	.56
Median sensory nerve velocity	54.58 ± 5.23	53.5 ± 5.54	.068
Ulnar sensory nerve latency	2.2 (2.23-3.75)	2.05 (1.18-3.70)	.56
Ulnar sensory nerve amplitude	23 (20.15-54)	23.45 (20-59)	.68
Ulnar sensory nerve velocity	54 (42-63)	55 (46-63)	.07
Radial sensory nerve latency	1.65 (1.63-3.96)	1.60 (1.74-3.78)	.32
Radial sensory nerve amplitude	22 (19.5-66)	24.1 (16.5-62)	**.03***
Radial sensory nerve velocity	54.28 ± 7	62.56 ± 6.84	**.042***

Values are presented as mean ± standard deviation or median (interquartile range), as appropriate. Right and left sides correspond to the procedure side and contralateral side, respectively. *P* values were obtained using paired statistical tests. Statistically significant *P* values are indicated with an asterisk.

No clinically evident procedure-related local complications, including hematoma, extravasation, compartment syndrome, or complex regional pain syndrome, were observed during postprocedural follow-up.

DISCUSSION

This prospective clinical study provides objective evidence of peripheral nerve conduction changes following transradial angiography. By combining detailed neurological examination, standardized neuropathic pain assessment scales, and comprehensive NCS, procedure-side–predominant radial nerve electrophysiological alterations were demonstrated, despite the absence of motor or sensory deficits on clinical examination. This study represents one of the few prospective investigations systematically evaluating potential peripheral nerve involvement after transradial angiography using objective electrophysiological measures.

Table 6. Correlation Between Neuropathic Pain Scores and Radial Nerve Conduction Parameters (n = 91)

	DN4 (*r*, *P*)	LANSS (*r*, *P*)
Radial sensory amplitude	$r = -0.14$, $P = .18$	$r = -0.11$, $P = .27$
Radial sensory velocity	$r = -0.09$, $P = .36$	$r = -0.13$, $P = .21$
Radial motor amplitude	$r = -0.17$, $P = .10$	$r = -0.15$, $P = .14$

Correlation analyses were performed using Spearman's rank correlation.
DN4, Douleur Neuropathique en 4 Questions; LANSS, Leeds Assessment of Neuropathic Symptoms and Signs.

In this study, patients with CTS (n=16) were not excluded from the primary analysis in order to preserve the statistical power of the ulnar and radial nerve conduction assessments. As these conditions predominantly affect the median nerve or reflect generalized neuropathy, their inclusion in the primary analysis enabled a more comprehensive evaluation of non-median nerve conduction findings. To minimize potential confounding effects, a predefined secondary analysis excluding these patients was subsequently performed. After the second analysis, side-to-side differences in non-radial nerves were no longer observed, whereas radial nerve abnormalities persisted, supporting a radial nerve–specific effect rather than a generalized or systemic neuropathic process.

The lack of correlation between DN4/LANSS scores and nerve conduction parameters is consistent with clinical experience. In carpal tunnel syndrome and other entrapment neuropathies, symptoms may be prominent despite normal or only mildly abnormal nerve conduction findings, whereas electrophysiological abnormalities can also be detected in patients with minimal or no symptoms.[18] Another possible explanation for the lack of correlation between neuropathic symptoms and electrophysiological findings relates to patient reporting behavior. Mild dysesthetic symptoms such as numbness or tingling may be underrecognized or underreported unless accompanied by more prominent complaints like weakness or functional impairment. This tendency may contribute to a dissociation between subjective symptom reporting and objective neurophysiological findings. Consistent with this observation, a systematic review reported a pooled incidence of clinically documented nerve damage after transradial access of 0.16%, while the pooled incidence of sensory symptoms such as numbness or tingling was 1.61%, with only 1 study identifying radial nerve damage (1/488; 0.25%).[19,20]

In a paresthesia-focused cohort, most patients had no pathological NCS findings. However, including patients with diabetic polyneuropathy in the primary analysis may have reduced sensitivity for subtle side-to-side differences because polyneuropathy tends to be bilateral and symmetric.[21]

A systematic review evaluating hand dysfunction after transradial artery catheterization reported a very low incidence of clinically documented nerve damage and sensory symptoms such as numbness or tingling. Analyzed studies mostly relied on symptom-based assessments or functional questionnaires, with limited use of objective neurophysiological testing. Findings of this review suggest that while clinically evident hand dysfunction after transradial access is uncommon, mild or subclinical nerve conduction changes may remain underrecognized without systematic electrophysiological evaluation, as demonstrated in the present study.[11]

The pattern of electrophysiological changes observed in this study—predominantly involving radial sensory nerve amplitude and conduction velocity, as well as radial motor nerve amplitude, with preserved latencies—may provide important clues regarding the underlying mechanism. Latency prolongation is typically associated with focal demyelination or conduction block, whereas reductions in amplitude and conduction velocity are more consistent with axonal dysfunction, impaired axonal recruitment, or transient ischemic effects. In the context of transradial access, prolonged or excessive local compression, edema, or microvascular compromise may preferentially affect axonal function without causing apparent demyelination. Such mechanisms could explain why amplitude- and velocity-based parameters were more sensitive to procedure-related changes than latency measures. This pattern is also in keeping with a mild and potentially reversible form of nerve involvement rather than a fixed structural nerve injury.[12,14,22]

The procedure-side–predominant radial nerve conduction changes observed in this study may be related to local factors inherent to transradial access rather than direct nerve injury. Hemostasis after transradial angiography is commonly achieved using prolonged manual compression, elastic bandaging, or dedicated radial compression devices.[23] While these methods are effective in preventing bleeding and radial artery occlusion, excessive pressure, prolonged compression duration, or suboptimal positioning may lead to transient local nerve compression, microvascular compromise, or perineural ischemia. In addition, access-site–related complications such as local edema, minor hematoma formation, or contrast extravasation may contribute to subclinical radial nerve involvement. Given the superficial course of the radial nerve and its close anatomical relationship to the radial artery at the wrist and forearm, the radial nerve may be particularly vulnerable to these local mechanical and ischemic factors. Radial artery occlusion (RAO) is another recognized vascular complication after transradial access and has been reported most frequently in the early post-procedural period, although delayed cases have also been described. While RAO is often clinically silent due to collateral circulation, it may occasionally present with hand numbness or vague pain, which may overlap clinically with symptoms attributed to peripheral nerve involvement.[23] Sensory complaints of vascular origin generally show a broader and more diffuse distribution, often involving the entire hand and sometimes extending into the forearm. In such cases, numbness or vague pain is typically poorly localized and may be accompanied by additional findings such as color changes or temperature asymmetry of the affected limb. In contrast, sensory symptoms related to peripheral nerve involvement tend to follow the anatomical distribution of the affected nerve and are usually more clearly demarcated distally. Color change or temperature difference is not commonly observed in isolated nerve dysfunction. The predominantly amplitude- and velocity-based changes observed in the study, without corresponding latency prolongation or clinical deficits, are consistent with mild, potentially reversible conduction alterations rather than apparent structural nerve injury.[12,14,23,24,25]

During follow-up, some patients continued to apply tight wrapping or excessive protection to the procedure side beyond the immediate post-procedural period. Insufficient

discharge guidance regarding limb use, together with overly protective patient behavior, may contribute to prolonged local compression and transient radial nerve stress.

Study Limitations

There were several limitations to this study. This was a single-center study, which may limit the generalizability of the findings. Although electrophysiological changes were observed on the procedure side, there was no clinically apparent motor or sensory neuropathy, which makes it difficult to draw conclusions about their direct clinical relevance. Because pre-procedural baseline nerve conduction studies were not performed, direct intra-individual temporal comparison of the procedure side remains limited. In addition, the findings observed may represent an early and potentially reversible axonal response related to transient mechanical stress, edema, or inflammatory changes following transradial access. However, since long-term follow-up electrophysiological assessments were not performed, it remains unclear whether these changes persist or resolve over time.

Clinical Implications

Although no clinically evident neuropathy was detected, procedure-side–predominant electrophysiological changes suggest that transradial angiography may be associated with mild radial nerve stress. These findings emphasize the importance of careful post-procedural limb care, particularly appropriate hemostasis and avoidance of unnecessarily prolonged or tight compression, and may help clinicians interpret post-procedural upper-extremity symptoms in the absence of objective neurological deficits.

Ethics Committee Approval: The study was approved by the Bursa City Hospital Clinical Research Ethics Committee (15.10.2025-2025-KAEK-47) and was conducted in accordance with the principles of the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Artificial Intelligence (AI) Disclosure: The authors declare that artificial intelligence–assisted tools were used for language editing and clarity enhancement only. The authors take full responsibility for the scientific content, data interpretation, and conclusions of the manuscript.

Author Contributions: Concept – T.D., H.K.; Design – T.D., H.K.; Supervision – H.K., O.D.; Resources – H.K., E.K.; Materials – T.D., H.K.; Data Collection and/or Processing – T.D., A.G., E.K.; Analysis and/or Interpretation – T.D., O.D.; Literature Search – T.D., A.G.; Writing – T.D., A.G.; Critical Review – H.K., O.D., E.K.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declare that this study received no financial support.

REFERENCES

1. Rao SV, O'Donoghue ML, Ruel M, et al. 2025 ACC/AHA/ACEP/NAEMSP/SCAI guideline for the management of patients with acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2025;85(22):2135-2237. [CrossRef]
2. Fazel R, Rao SV, Cohen DJ, et al. Radial vs femoral access for percutaneous coronary intervention: temporal trends and outcomes in the USA. *Eur Heart J*. 2025;46:426.
3. Chiarito M, Cao D, Nicolas J, et al. Radial versus femoral access for coronary interventions: an updated systematic review and meta-analysis of randomized trials. *Eur Heart J*. 2021;42(14):1387-1396.
4. Özyüncü N, Tan Kürklü TS, Özcan ÖU, Turan N, Hüseynova S. Pectoral muscle hematoma: a rare complication of transradial cardiac catheterization. *Anatol J Cardiol*. 2016;16(12):E22-E23. [CrossRef]
5. Lalani K, Devasia T, Paramasivam G. Can distal radial access replace conventional radial access for coronary catheterization? A study comparing puncture time, attempts, patient and operator comfort. *Anatol J Cardiol*. 2024;28(9):454-460. [CrossRef]
6. Roy S, Kabach M, Patel DB, Guzman LA, Jovin IS. Radial artery access complications: prevention, diagnosis and management. *Cardiovasc Revasc Med*. 2022;40:163-171. [CrossRef]
7. Sadler M, Lawson C. Cardiac catheterisation: avoiding common pitfalls with transradial vascular access. *Br J Cardiol*. 2023;30(3):21. [CrossRef]
8. Dwivedi SK, Sharma AK, Nayak GR, et al. Factors influencing radial artery occlusion after transradial coronary intervention in the Indian population. *Anatol J Cardiol*. 2022;26(2):105-111. [CrossRef]
9. Huyut MA, Yamaç AH. Comparison of the transradial and transfemoral approach in treatment of chronic total occlusions with similar lesion characteristics. *Anatol J Cardiol*. 2018;19(5):319-325. [CrossRef]
10. Soydan E, Kış M, Akın M. Evaluation of radial artery endothelial functions in transradial coronary angiography according to different radial access sites. *Anatol J Cardiol*. 2021;25(1):42-48. [CrossRef]
11. Ul Haq MAU, Rashid M, Kwok CS, Wong CW, Nolan J, Mamas MA. Hand dysfunction after transradial artery catheterization for coronary procedures. *World J Cardiol*. 2017;9(7):609-619. [CrossRef]
12. Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. *J Bone Joint Surg Am*. 1999;81(11):1600-1610. [CrossRef]
13. Harden RN, Oaklander AL, Burton AW, et al. Complex regional pain syndrome: practical diagnostic and treatment guidelines, 4th edition. *Pain Med*. 2013;14(2):180-229. [CrossRef]
14. Preston DC. *Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations*. 3rd ed. Philadelphia: Elsevier; 2013.
15. England JD, Gronseth GS, Franklin G, et al. Practice parameter: evaluation of distal symmetric polyneuropathy: role of autonomic testing, nerve biopsy, and skin biopsy (an evidence-based review) [RETIRED]. Report of the American Academy of Neurology, American Association of Neuromuscular and Electrodiagnostic Medicine, and American Academy of Physical Medicine and Rehabilitation. *Neurology*. 2009;72(2):177-184. [CrossRef]
16. Bouhassira D, Attal N, Alchaar H, et al. Comparison of pain syndromes associated with nervous or somatic lesions and development of a new neuropathic pain diagnostic questionnaire (DN4). *Pain*. 2005;114(1-2):29-36. [CrossRef]
17. Bennett M. The LANSS Pain Scale: the Leeds assessment of neuropathic symptoms and signs. *Pain*. 2001;92(1-2):147-157. [CrossRef]

18. Dandinoğlu T, Karadeniz M, Yılmaz V, Tekin L, Dinçer Ü. Evaluating neuropathic complaints by DN4 and LANSS scales after local corticosteroid therapy in carpal tunnel syndrome. *J Back Musculoskelet Rehabil*. 2016;29(3):575-580. [CrossRef]
19. Sandoval Y, Burke MN, Lobo AS, et al. Contemporary arterial access in the cardiac catheterization laboratory. *J Am Coll Cardiol*. 2017;69(22):2233-2241.
20. Ayyaz Ul Haq MAU, Rashid M, Gilchrist IC, et al. Incidence and clinical course of limb dysfunction post cardiac catheterization: a systematic review. *Circ J*. 2018;82(11):2736-2744. [CrossRef]
21. Eğilmez Sarıkaya CE, Salkın FÖ, Sarıkaya C. Electrophysiological assessment of paresthesia in patients following radial angiography: a prospective study. *Anatol J Cardiol*. 2024;28(7):363-366. [CrossRef]
22. Lundborg G. Nerve injury and repair: a challenge to the plastic brain. *J Peripher Nerv Syst*. 2003;8(4):209-226. [CrossRef]
23. Bernat I, Aminian A, Pancholy S, et al. Best practices for the prevention of radial artery occlusion after transradial diagnostic angiography and intervention: an international consensus paper. *JACC Cardiovasc Interv*. 2019;12(22):2235-2246. [CrossRef]
24. Tang DT, Barbour JR, Davidge KM, Yee A, Mackinnon SE. Nerve entrapment: update. *Plast Reconstr Surg*. 2015;135(1):199e-215e. [CrossRef]
25. Moore KL, Dalley AF, Agur AMR. *Clinically Oriented Anatomy*. 7th ed. Philadelphia (PA): Wolters Kluwer Health/Lippincott Williams & Wilkins; 2014.