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Assessment of the Incidence and Predictors of Revision Surgery Following Carpal Tunnel Release

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A B S T R A C T

Introduction: Carpal tunnel release is highly effective; however, a subset of patients develops persistent or recurrent symptoms requiring revision surgery. Failure may reflect incomplete decompression, perineural scarring, technical injury, or patient-related systemic and occupational factors. This study aimed to assess the incidence of revision surgery and identify its independent predictors following primary carpal tunnel release.

Material and methods: This retrospective, descriptive cross-sectional study was conducted at Imam Reza Hospital, Tabriz, Iran, and included 300 adults who underwent primary carpal tunnel release. Eligible records were selected through convenience sampling. Demographic, clinical, electrophysiological, surgical, postoperative, and revision-related data were extracted from medical records using a standardized form and analyzed statistically to determine the incidence and predictors of revision surgery.

Results: Revision carpal tunnel surgery occurred in 8.0% of patients. Multivariable logistic regression identified preoperative severe electrodiagnostic grade (adjusted OR=3.86, 95% CI:1.31-11.38; P=0.014), persistent postoperative pillar or scar pain (adjusted OR=3.41, 95% CI:1.18-9.85; P=0.023), primary endoscopic release (adjusted OR=3.25, 95% CI:1.04-10.15; P=0.043), preoperative thenar atrophy (adjusted OR=3.12, 95% CI:1.08-8.99; P=0.035), and diabetes mellitus (adjusted OR=2.94, 95% CI:1.05-8.24; P=0.040) as independent predictors of revision surgery.

Conclusion: Secondary revision following carpal tunnel release is primarily driven by advanced baseline neural degeneration, metabolic comorbidity, endoscopic decompression, and persistent postoperative pillar pain. Comprehensive preoperative electrodiagnostic risk stratification, careful surgical technique selection, and targeted glycemic management are essential to mitigate surgical failure rates and optimize long-term clinical recovery.

Introduction

Carpal tunnel syndrome represents the most prevalent compressive neuropathy encountered in peripheral nerve surgery, accounting for substantial individual morbidity, physical impairment, and healthcare expenditures across industrial and developing populations globally. Arising from mechanical entrapment and sustained ischemic compromise of the median nerve as it traverses the fibro-osseous canal bordered by the carpal bones and the tough transverse carpal ligament, this disabling condition manifests characteristically with acroparesthesia, nocturnal burning pain, digital numbness, loss of fine motor dexterity, and thenar muscular atrophy. While conservative and non-operative interventions including wrist immobilization splints, local corticosteroid infiltration, ergonomics adjustments, and targeted

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oral pharmacotherapy serve as effective initial therapeutic strategies for mild to moderately symptomatic presentations, recalcitrant symptoms or progressive electrophysiological axonal denervation invariably mandate surgical intervention to halt irreversible neural degeneration [1].

Primary carpal tunnel release, accomplished either via traditional open incision, minimally invasive mini-open techniques, or modern endoscopic approaches, serves as the definitive gold-standard surgical treatment for decompressing the confined anatomical compartment. By completely dividing the transverse carpal ligament and investing fibrous antebrachial fascia, surgical decompression immediately abolishes elevated intraneural compartment pressures, restores endoneuria microvascular capillary perfusion, and initiates functional neuroregeneration within compressed median nerve fascicles. Contemporary surgical series universally report outstanding subjective and objective clinical success rates following primary decompression, with up to ninety percent of operated individuals achieving complete pain mitigation, sensory recovery, and substantial restoration of manual functional performance within the initial postoperative year [2].

Despite the profound efficacy and technical reproducibility of primary operative decompression, a recalcitrant and surgically challenging subgroup of patients experiences either persistent, recurrent, or novel postoperative neuropathic symptoms requiring secondary operative intervention, widely designated as revision carpal tunnel release. Persistent manifestations, characterized by an absence of symptomatic quiescence or immediate worsening following the index procedure, are most frequently attributable to incomplete surgical division of the transverse carpal ligament, unaddressed anatomical variations, or iatrogenic nerve injury. In contrast, recurrent presentations emerge after an extended disease-free interval of genuine clinical improvement and are predominantly driven by circumferential perineural scar formation, robust fibrotic adhesions tethering the epineurium to adjacent gliding flexor tendons, or progressive tenosynovial proliferation within the reconstructed carpal canal [3].

The pathophysiological cascade underlying persistent compressive neuropathy frequently points directly toward technical intraoperative failures during the index surgical attempt. Incomplete transection of the distal margin of the transverse carpal ligament, failure to fully divide the deep distal investing antebrachial fascia proximally, or overlooking anomalous bifid median nerve branches and persistent median arteries constitute prominent anatomical pitfalls that prevent biological decompression. Under these conditions, the compressed nerve remains subject to sustained focal mechanical shearing forces and elevated chronic tissue pressure, perpetuating mechanical demyelination, disruption of the blood-nerve barrier, endoneuria edema, and progressive intrafascicular fibrosis that resist spontaneous postoperative restoration [4].

Conversely, genuine recurrent median neuropathy involves intricate molecular and biological fibroproliferative processes that transpire months or even years following an ostensibly successful, technically flawless initial decompression. Following surgical division of the transverse carpal ligament, the resulting inflammatory wound-healing cascade precipitates excessive extracellular matrix deposition, transforming the surgical bed into an unyielding fibrotic scar tissue envelope. As this hyperplastic cicatricial mass contracts, it exerts secondary circumferential constriction upon the median nerve trunk, immobilizing the nerve and precluding normal longitudinal excursion during wrist flexion and digital articulation, which provokes severe dynamic traction neuropathy and debilitating mechanical hyperalgesia [5].

A distinct and catastrophic mechanism necessitating revision intervention is direct iatrogenic injury sustained by the median nerve proper, its recurrent motor branch, or critical sensory communicating fascicles during the primary operative endeavor. Unrecognized transection, laceration, thermal coagulative necrosis from bipolar electrocautery, or inadvertent traction injury can produce neuroma-in-continuity, terminal amputation neuroma formation, or total motor defunction of the thenar musculature. Such catastrophic structural compromises precipitate excruciating neuropathic pain, phantom hypersensitivity, dysesthesia, and catastrophic loss of thumb opposition, introducing complex reconstructive dilemmas that mandate microsurgical exploration, neuroma excision, cable nerve autografting, or vascularized soft-tissue coverage [6].

Beyond localized anatomical anomalies and procedural misadventures, a broad spectrum of patient-specific metabolic, endocrine, and systemic comorbid conditions fundamentally impairs peripheral nerve biology and elevates susceptibility to primary decompression failure. Chronic poorly regulated diabetes mellitus induces microvascular angiopathy, endoneuria ischemia, and advanced glycation end-product deposition within peripheral nerve stroma, fundamentally blunting intrinsic regenerative capacity and rendering the median nerve extraordinarily susceptible to double-crush entrapment syndromes. Similarly, untreated hypothyroidism, end-stage renal disease requiring hemodialysis, inflammatory arthropathies, high body mass index, and chronic systemic inflammatory states promote aggressive soft-tissue remodeling and recurrent canal stenosis, precipitating early structural relapse [7].

Socioeconomic parameters, occupational demands, behavioral habits, and psychological profiles also exert profound modulatory influences on the clinical outcomes of carpal tunnel surgery and subsequent revision trajectories. Prolonged occupational exposure to high-frequency vibratory tools, repetitive manual forceful gripping, awkward wrist posturing, and heavy manual labor significantly impede local tissue recovery and exacerbate micro traumatic shear stresses upon the freshly decompressed nerve bed. Furthermore, active tobacco smoking accelerates microcirculatory vasoconstriction and inhibits tissue oxygenation, while unaddressed psychiatric comorbidities such as chronic depression, somatic symptom disorders, and major anxiety profoundly lower systemic pain thresholds, contributing to perceived failure and driving patients to seek repeated surgical consultations [8].

The clinical presentation of a failed primary carpal tunnel release presents a formidable diagnostic and therapeutic challenge for the orthopedic hand surgeon and reconstructive microsurgeon alike. Unlike the pristine virgin anatomy encountered during primary intervention, the revision surgical field is distorted by dense architectural scarring, obliterated tissue planes, loss of soft-tissue envelopes, and unpredictable positioning of vital neurovascular structures, thereby elevating the risk of inadvertent secondary nerve injury exponentially. Furthermore, re-decompression alone is frequently insufficient to restore long-term function in revision settings, commonly necessitating specialized adjunct reconstructive procedures such as external neurolysis, internal intrafascicular neurolysis, hypothenar fat-pad transposition flaps, vascularized pedicle coverage, or nerve wrapping matrices to prevent recurrent cicatricial tethering [9].

Despite the substantial clinical burden, immense physical distress, and severe economic consequences associated with failed median nerve decompression, wide discrepancies and ambiguities persist across current orthopedic literature concerning the true epidemiological incidence of revision carpal tunnel release. Reported revision rates fluctuate widely from less than one percent to more than twelve percent across published single-center cohorts, large healthcare registry databases, and specialized hand surgery centers worldwide. This striking disparity reflects significant heterogeneity in diagnostic criteria, varying definitions of surgical failure, disparities in surgeon subspecialty training and caseload volumes, divergent durations of prospective follow-up, and inconsistencies in data capture methodologies across different healthcare infrastructures [10].

Moreover, while several individual demographic, systemic, and surgical risk factors have been independently implicated in primary decompression failure, robust multivariable models capable of elucidating the complex synergistic interactions among these clinical determinants remain exceedingly scarce. Identifying modifiable host factors, characterizing procedural technical pitfalls, and delineating objective preoperative electrodiagnostic predictors are of paramount clinical importance for developing accurate pre-procedure risk-stratification algorithms. Establishing refined predictive tools would empower hand surgeons to optimize patient selection, adjust primary surgical techniques, provide tailored informed consent, and institute vigilant postoperative monitoring protocols to mitigate the risk of failure [11].

Addressing these crucial epidemiological and clinical knowledge gaps requires rigorous, systematic evaluation within well-characterized, contemporary surgical cohorts using standardized diagnostic definitions and comprehensive clinical metrics. A refined understanding of patient-specific risk profiles alongside procedural determinants will elucidate the underlying mechanisms leading to median nerve entrapment relapse and inform evidence-based algorithmic strategies designed to minimize surgical morbidity. Therefore, the primary objective of this prospective clinical investigation was to determine the overall incidence of revision surgery following primary carpal tunnel release and to identify the independent demographic, clinical, electrophysiological, and procedural predictors that drive secondary operative intervention.

Material and methods

Study Design

This retrospective, descriptive cross-sectional study was conducted at Imam Reza Hospital, affiliated with Tabriz University of Medical Sciences, Iran. The study assessed the frequency of revision surgery after primary carpal tunnel release and examined demographic, clinical, electrophysiological, and surgical characteristics potentially associated with the need for secondary intervention. Medical records of eligible patients who had undergone primary carpal tunnel release during the defined study period were reviewed retrospectively. Revision surgery was defined as any subsequent operative procedure performed to address persistent, recurrent, or newly developed symptoms attributable to inadequate decompression, recurrent median nerve compression, perineural fibrosis, or complications related to the initial procedure.

Sample Size and Sampling Method

The sample size was estimated using the Cochran formula for estimating a population proportion: $n=Z2p(1-p)/d2$. A 95% confidence level was assumed, corresponding to $Z=1.96$. Because a reliable local estimate of the proportion of patients requiring revision surgery was unavailable, $p=0.50$was selected to produce the maximum

required sample size, with an accepted margin of error of approximately 5.7%. Based on these assumptions, the minimum sample size was estimated at approximately 300 participants. To achieve the planned sample size, eligible medical records were selected using a convenience sampling method. Records were reviewed consecutively according to availability in the hospital archive and clinical documentation system until data from 300 eligible patients were obtained.

Eligibility Criteria

Patients were eligible if they were at least 18 years of age; had a documented diagnosis of carpal tunnel syndrome established on the basis of compatible clinical manifestations, physical examination findings, and, when available, electrodiagnostic evidence; had undergone primary carpal tunnel release at Imam Reza Hospital during the study period; and had sufficient medical, operative, and postoperative documentation to determine the occurrence or absence of revision surgery. Patients were included regardless of sex, laterality, surgical technique, disease duration, or the presence of controlled chronic comorbidities. Patients were excluded if they had undergone revision surgery before the documented index operation; had a history of previous wrist, hand, forearm, or median nerve surgery on the affected side that could substantially alter postoperative outcomes; had carpal tunnel syndrome secondary to acute traumatic injury, fracture, dislocation, tumor, or a known space-occupying lesion; had concomitant proximal median nerve compression, cervical radiculopathy, brachial plexopathy, or another neurologic disorder capable of explaining the postoperative symptoms; had incomplete or internally inconsistent records; lacked adequate follow-up information regarding postoperative symptoms or reoperation; or had undergone the primary procedure at another institution. Records were also excluded when the indication for a subsequent operation could not be distinguished from unrelated hand or wrist pathology.

Study Variables and Data Collection

Data were extracted from hospital records using a structured data-collection form developed for this study. Demographic variables included age at the time of primary surgery, sex, place of residence when documented, occupation, and laterality of the affected hand. Clinical variables included the duration and severity of symptoms, nocturnal paresthesia, numbness, pain, hand weakness, impaired fine motor function, dropping objects, sensory disturbance in the median nerve distribution, thenar muscle wasting, thumb opposition weakness, and the results of provocative examinations, including Tinel's, Phalen's, and Durkan's compression tests. The presence of bilateral disease, previous conservative treatment, symptom persistence after surgery, symptom-free interval, and the clinical pattern of postoperative failure were also recorded. Relevant medical characteristics included diabetes mellitus, hypothyroidism, rheumatoid or other inflammatory arthritis, chronic kidney disease, dialysis dependence, obesity, smoking status, pregnancy-related symptoms when applicable, and other documented systemic disorders. Medication history, including corticosteroid use and treatment for metabolic or endocrine disease, was recorded when available.

Electrophysiological variables included the presence of median nerve compression on nerve-conduction studies, sensory and motor distal latencies, sensory and motor conduction velocities, compound muscle action potential and sensory nerve action potential amplitudes, electrodiagnostic severity category, and evidence of axonal loss or denervation. Surgical variables included the date of the index operation, operative technique, open or endoscopic approach, anesthesia type, surgeon-related documentation when available, laterality, concomitant procedures, intraoperative complications, and postoperative complications. The postoperative course was evaluated for wound infection, hematoma, pillar pain, scar tenderness, complex regional pain syndrome, iatrogenic nerve injury, and other adverse events. Outcome variables included persistent symptoms, recurrent symptoms after an initial symptom-free period, worsening or new symptoms, postoperative functional limitation, referral for specialist reassessment, and performance of revision surgery. For patients who underwent reoperation, the interval between the primary and revision procedures, documented indication, operative findings, extent of residual ligament release, perineural adhesions, nerve injury, tenosynovial changes, and additional reconstructive procedures were recorded whenever these details were available.

Statistical Analysis

All statistical analyses were performed using a standard statistical software package, and the data were initially examined for completeness, plausibility, and outliers. Continuous variables were assessed for distribution using the Shapiro–Wilk test and visual inspection of histograms and quantile–quantile plots; the data were considered normally distributed and are therefore presented as mean ± standard deviation. Categorical variables are reported as frequencies and percentages. Continuous variables were compared between patients who did and did not undergo revision surgery using the independent-samples Student's t-test, whereas categorical variables were analyzed using the chi-square test or Fisher's exact test when expected cell counts were small. The incidence of revision surgery

was calculated as the proportion of patients undergoing secondary intervention among the total study cohort, with a corresponding 95% confidence interval. Univariate logistic regression was used to estimate crude odds ratios and 95% confidence intervals for potential predictors of revision surgery. Variables with clinical relevance or a univariate P-value below 0.20 were considered for entry into the multivariable logistic regression model. Adjusted odds ratios with 95% confidence intervals were reported, and model performance was evaluated using the Hosmer-Lemeshow goodness-of-fit test and the area under the receiver operating characteristic curve. Multicollinearity was assessed before final model construction. All tests were two-sided, and a PPP-value below 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Ethics Committee of Tabriz University of Medical Sciences under approval code IR.TBZMED.FMD.REC.1404.136. Because the investigation used retrospectively collected medical-record data and involved no direct intervention, biological sampling, or alteration of patient management, the requirement for written informed consent was waived in accordance with institutional regulations. Patient confidentiality was protected throughout the study by restricting access to the research dataset, removing personally identifying information from the analytical file, and reporting the findings only in aggregate form. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and applicable national regulations governing retrospective medical research.

Results

The baseline demographic and clinical characteristics of the study cohort stratified by revision surgery status are summarized in Table 1. Among the 300 analyzed patients, 24 individuals (8.0%) required secondary operative intervention following primary carpal tunnel release. Comparative bivariate analyses demonstrated that patients undergoing revision surgery were significantly older (54.3 ± 11.1 vs. 48.1 ± 10.2 years; P=0.006), presented with a substantially higher body mass index (30.6 ± 4.5 vs. 27.5 ± 4.1 kg/m²; P=0.001), and exhibited an increased prevalence of obesity (54.2% vs. 23.6%; P=0.001) and active tobacco use (37.5% vs. 16.3%; P=0.012). Furthermore, prolonged preoperative symptom duration (22.4 ± 10.9 vs. 14.1 ± 8.1 months; P<0.001), high-demand repetitive manual occupation (70.8% vs. 44.9%; P=0.014), and pre-existing diabetes mellitus (50.0% vs. 17.4%; P<0.001) were significantly more frequent in the revision cohort. From a clinical and functional standpoint, marked baseline neuromuscular impairment was disproportionately observed among revision cases, evidenced by higher rates of preoperative thenar muscle atrophy (50.0% vs. 13.0%; P<0.001), impaired grip performance (58.3% vs. 28.6%; P=0.003), and advanced electrodiagnostic staging (66.7% vs. 19.2%; P<0.001), whereas classical provocative physical diagnostic maneuvers including the Phalen, Tinel, and Durkan tests did not demonstrate statistically meaningful intergroup discrepancies.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population Stratified by Revision Surgery Status

Variable	Overall Cohort (N=300)	No Revision Surgery (N=276)	Revision Surgery (N=24)	P-value
Age (years), Mean ± SD	48.6 ± 10.4	48.1 ± 10.2	54.3 ± 11.1	0.006
Female Sex, n (%)	207 (69.0%)	193 (69.9%)	14 (58.3%)	0.238
Body Mass Index (kg/m²), Mean ± SD	27.8 ± 4.2	27.5 ± 4.1	30.6 ± 4.5	0.001
Obesity (BMI ≥ 30 kg/m²), n (%)	78 (26.0%)	65 (23.6%)	13 (54.2%)	0.001
Current Smoking, n (%)	54 (18.0%)	45 (16.3%)	9 (37.5%)	0.012
Repetitive Manual Activity, n (%)	141 (47.0%)	124 (44.9%)	17 (70.8%)	0.014
Bilateral Carpal Tunnel Syndrome, n (%)	114 (38.0%)	100 (36.2%)	14 (58.3%)	0.033
Dominant Hand Affected, n (%)	198 (66.0%)	181 (65.6%)	17 (70.8%)	0.601
Symptom Duration (months), Mean ± SD	14.8 ± 8.6	14.1 ± 8.1	22.4 ± 10.9	< 0.001
Nocturnal Paresthesia, n (%)	261 (87.0%)	241 (87.3%)	20 (83.3%)	0.547
Thenar Muscle Atrophy, n (%)	48 (16.0%)	36 (13.0%)	12 (50.0%)	< 0.001
Diminished Grip Strength, n (%)	93 (31.0%)	79 (28.6%)	14 (58.3%)	0.003
Positive Phalen Test, n (%)	249 (83.0%)	230 (83.3%)	19 (79.2%)	0.584
Positive Tinel Sign, n (%)	216 (72.0%)	198 (71.7%)	18 (75.0%)	0.731

Positive Durkan Test, n (%)	255 (85.0%)	236 (85.5%)	19 (79.2%)	0.395
Diabetes Mellitus, n (%)	60 (20.0%)	48 (17.4%)	12 (50.0%)	< 0.001
Hypothyroidism, n (%)	33 (11.0%)	29 (10.5%)	4 (16.7%)	0.339
Rheumatoid Arthritis, n (%)	18 (6.0%)	15 (5.4%)	3 (12.5%)	0.161
Chronic Kidney Disease, n (%)	12 (4.0%)	9 (3.3%)	3 (12.5%)	0.063
Prior Local Steroid Injection, n (%)	102 (34.0%)	90 (32.6%)	12 (50.0%)	0.088
Preoperative Severe Electrodiagnostic Grade, n (%)	69 (23.0%)	53 (19.2%)	16 (66.7%)	< 0.001

The operative details, electrophysiological parameters, and postoperative outcomes categorized by revision surgical status are summarized in Table2. Patients undergoing revision intervention demonstrated a distinct primary operative profile, characterized by a higher utilization rate of endoscopic decompression (33.3% vs. 10.1%; P=0.038), elevated involvement of junior trainees as the operating surgeon (45.8% vs. 23.2%; P=0.042), longer initial operative times (28.7 ± 8.4 vs. 21.8 ± 6.2 minutes; P<0.001), and a higher rate of intraoperative complications (12.5% vs. 2.2%; P=0.027). Furthermore, baseline electrodiagnostic evaluations revealed significantly more severe median nerve dysfunction in the revision cohort, evidenced by prolonged distal motor latencies (6.2 ± 1.4 vs. 4.7 ± 1.1 ms; P<0.001), slower sensory conduction velocities (28.8 ± 5.9 vs. 37.1 ± 6.3 m/s; P<0.001), and marked reductions in sensory nerve action potential amplitudes (7.9 ± 3.8 vs. 15.4 ± 5.3 μV; P<0.001). Postoperatively, the revision group experienced higher incidences of surgical site infections (16.7% vs. 4.0%; P=0.024) and persistent scar tenderness or pillar pain (41.7% vs. 11.6%; P<0.001). The primary indications prompting revision decompression performed at a mean interval of 13.8 ± 6.4 months following the index procedure were recurrent symptoms following an initial asymptomatic period (45.8%), persistent unrelenting symptoms indicative of incomplete retinacular release (41.7%), and new-onset iatrogenic neurological deficits (12.5%), with secondary re-exploration and external neurolysis representing the predominant salvage surgical modality (54.2%).

Table 2. Operative Details, Electrodiagnostic Parameters, and Postoperative Outcomes According to Revision Carpal Tunnel Surgery Status

Variable	Overall Cohort (N = 300)	No Revision Surgery (n = 276)	Revision Surgery (n = 24)	P-value
Primary Surgical Technique, n (%)	-	-	-	0.038
Standard Open Carpal Tunnel Release	186 (62.0%)	176 (63.8%)	10 (41.7%)	-
Mini-Open Carpal Tunnel Release	78 (26.0%)	72 (26.1%)	6 (25.0%)	-
Endoscopic Carpal Tunnel Release	36 (12.0%)	28 (10.1%)	8 (33.3%)	-
Primary Anesthesia Modality, n (%)	-	-	-	0.612
Local Infiltration Anesthesia	201 (67.0%)	186 (67.4%)	15 (62.5%)	-
Regional Axillary or Bier Block	81 (27.0%)	74 (26.8%)	7 (29.2%)	-
General Anesthesia	18 (6.0%)	16 (5.8%)	2 (8.3%)	-
Operating Surgeon Seniority, n (%)	-	-	-	0.042
Senior Attending Surgeon	225 (75.0%)	212 (76.8%)	13 (54.2%)	-
Junior Staff or Resident Trainee	75 (25.0%)	64 (23.2%)	11 (45.8%)	-
Intraoperative Complications, n (%)	9 (3.0%)	6 (2.2%)	3 (12.5%)	0.027
Operative Duration (minutes), Mean ± SD	22.4 ± 6.8	21.8 ± 6.2	28.7 ± 8.4	< 0.001
Median Distal Motor Latency (ms), Mean ± SD	4.9 ± 1.2	4.7 ± 1.1	6.2 ± 1.4	< 0.001
Median Sensory Nerve Conduction Velocity (m/s), Mean ± SD	36.4 ± 6.7	37.1 ± 6.3	28.8 ± 5.9	< 0.001
Median Sensory Nerve Action Potential Amplitude (μV), Mean ± SD	14.8 ± 5.6	15.4 ± 5.3	7.9 ± 3.8	< 0.001
Postoperative Superficial Surgical Site Infection, n (%)	15 (5.0%)	11 (4.0%)	4 (16.7%)	0.024
Postoperative Wound Hematoma, n (%)	12 (4.0%)	9 (3.3%)	3 (12.5%)	0.063
Persistent Scar Tenderness or Pillar Pain, n (%)	42 (14.0%)	32 (11.6%)	10 (41.7%)	< 0.001

Primary Symptom Pattern Leading to Evaluation, n (%)	-	--	-	< 0.001
Complete Resolution of Symptoms	243 (81.0%)	243 (88.0%)	0 (0.0%)	-
Persistent Symptoms Without Any Relief	15 (5.0%)	5 (1.8%)	10 (41.7%)	-
Recurrent Symptoms After Initial Improvement	33 (11.0%)	22 (8.0%)	11 (45.8%)	--
New-Onset Iatrogenic Neurological Deficit	9 (3.0%)	6 (2.2%)	3 (12.5%)	
Time from Primary Surgery to Revision (months), Mean ± SD	-	-	13.8 ± 6.4	-
Specific Revision Operative Procedure Performed, n (%)	-	--	-	-
Re-Exploration and Secondary Neurolysis	-	-	13 (54.2%)	-
Hypothenar Fat Pad Transposition Flap	-	-	6 (25.0%)	-
Synthetic Collagen Nerve Wrapping Application	-	-	3 (12.5%)	-
Direct Epineural Microsurgical Repair	-	-	2 (8.3%)	-

The univariate and multivariable logistic regression analyses identifying the independent predictors of revision carpal tunnel release are presented in Table 3. Although multiple clinical, demographic, and perioperative variables demonstrated statistically significant associations on unadjusted univariate evaluation, multivariable penalized logistic regression identified five independent determinants significantly linked to an elevated risk of revision surgery. Specifically, baseline severe electrodiagnostic classification emerged as the strongest independent predictor, conferring a nearly fourfold elevation in the odds of revision decompression (adjusted OR=3.86, 95% CI:1.31-11.38; P=0.014). In addition, primary endoscopic release compared with open decompression (adjusted OR=3.25, 95% CI:1.04-10.15; P=0.043), the presence of persistent postoperative scar tenderness or pillar pain (adjusted OR=3.41, 95% CI:1.18-9.85; P=0.023), preoperative thenar muscle atrophy (adjusted OR=3.12, 95% CI:1.08-8.99; P=0.035), and pre-existing diabetes mellitus (adjusted OR=2.94, 95% CI:1.05-8.24; P=0.040) remained independently associated with secondary operative failure, whereas patient age, obesity, tobacco use, manual occupation, and operating surgeon seniority did not maintain independent statistical significance following multivariable adjustment.

Table 3. Univariate and Multivariate Logistic Regression Analyses Identifying Independent Predictors of Revision Carpal Tunnel Surgery

Candidate Predictor Variable	Crude Odds Ratio (95% CI)	Unadjusted P-value	Adjusted Odds Ratio (95% CI)	Adjusted P-value
Age (per 1-year increase)	1.06 (1.02-1.11)	0.007	1.03 (0.98-1.09)	0.224
Obesity (BMI ≥ 30 kg/m²)	3.84 (1.63-9.04)	0.002	2.14 (0.78-5.86)	0.138
Active Tobacco Smoking	3.08 (1.26-7.53)	0.014	2.45 (0.86-6.98)	0.093
Repetitive Manual Activity	2.98 (1.21-7.35)	0.018	1.88 (0.67-5.28)	0.231
Bilateral Carpal Tunnel Syndrome	2.46 (1.05-5.78)	0.038	1.62 (0.59-4.45)	0.352
Symptom Duration (per 1-month increase)	1.09 (1.04-1.14)	< 0.001	1.05 (0.99-1.11)	0.082
Preoperative Thenar Muscle Atrophy	6.67 (2.73-16.28)	< 0.001	3.12 (1.08-8.99)	0.035
Diminished Preoperative Grip Strength	3.50 (1.49-8.23)	0.004	1.74 (0.61-4.96)	0.301
Diabetes Mellitus	4.75 (2.01-11.23)	< 0.001	2.94 (1.05-8.24)	0.040
Preoperative Severe Electrodiagnostic Grade	8.42 (3.42-20.73)	< 0.001	3.86 (1.31-11.38)	0.014
Primary Endoscopic Surgical Technique	4.43 (1.71-11.48)	0.002	3.25 (1.04-10.15)	0.043
Primary Surgery by Junior Staff or Trainee	2.80 (1.19-6.59)	0.018	1.95 (0.71-5.34)	0.194

Intraoperative Complication During Index Procedure	6.43 (1.50-27.56)	0.012	2.82 (0.52-15.28)	0.228
Postoperative Surgical Site Infection	4.82 (1.40-16.58)	0.013	2.61 (0.61-11.16)	0.193
Postoperative Persistent Pillar or Scar Pain	5.45 (2.24-13.25)	< 0.001	3.41 (1.18-9.85)	0.023

Discussion

The present investigation comprehensively evaluated the incidence patterns, perioperative risk profiles, and independent predictors of revision surgery following primary carpal tunnel release in a defined cohort of 300 patients. Overall, secondary operative intervention was required in 24 individuals, establishing an institutional revision incidence rate of 8.0%. Bivariate analyses demonstrated that patients requiring revision surgery exhibited significantly greater age, higher body mass index, extended preoperative symptom chronicity, increased rates of active tobacco consumption, and higher frequencies of bilateral disease and manual occupational exposure compared with their non-revision counterparts. Furthermore, baseline functional impairment, marked by thenar muscle atrophy and diminished grip strength, alongside severe electrophysiological median nerve dysfunction, was disproportionately represented in the revision cohort. Operative characteristics also differed substantially, with higher rates of endoscopic decompression, longer operative durations, and increased trainee involvement observed among failed primary cases. Multivariable penalized logistic regression modeling confirmed that baseline severe electrodiagnostic classification, persistent postoperative pillar or scar pain, primary endoscopic surgical technique, preoperative thenar muscle atrophy, and pre-existing diabetes mellitus served as strong, statistically independent determinants of secondary surgical decompression [12,13].

The observed 8.0% revision rate aligns closely with contemporary epidemiological reports across diverse surgical centers, where secondary intervention rates typically range between 3% and 12% depending on follow-up duration, diagnostic criteria, and institutional referral patterns. Carpal tunnel release remains one of the most frequently performed hand surgical procedures worldwide, yet persistent or recurrent symptoms continue to represent a formidable clinical challenge for both reconstructive surgeons and affected patients. Incomplete release of the flexor retinaculum has historically been recognized as the single most prevalent technical cause of persistent median nerve compression, whereas circumferential perineural cicatrization, traction neuritis, and structural reformation of the transverse carpal ligament predominate in delayed recurrent cases. The relatively high frequency of secondary interventions within our cohort highlights the inherent complexity of identifying vulnerable clinical phenotypes prior to the index procedure. By systematically analyzing preoperative physiological factors alongside surgical variables, our findings demonstrate that secondary operative failure is not merely a consequence of isolated surgical inaccuracies, but rather reflects an intricate pathophysiological convergence of biological tissue vulnerability, severe underlying axonopathy, and procedure-specific mechanics [14,15].

Baseline electrodiagnostic severity emerged as the most potent independent predictor of secondary surgical failure in our multivariable analysis, with severely affected individuals exhibiting a nearly fourfold elevation in revision risk. Electrophysiological deterioration characterized by markedly prolonged distal motor latencies, profound sensory conduction slowing, and extinguished or diminished sensory nerve action potentials signifies advanced, chronic mechanical compression accompanied by extensive intra-fascicular endoneuria fibrosis and demyelination. In chronic high-grade entrapment neuropathy, persistent endoneuria ischemia and breakdown of the blood-nerve barrier trigger permanent microvascular remodeling and dense scar formation within the nerve parenchyma. Consequently, simple mechanical transection of the overlying transverse carpal ligament may fail to restore adequate microvascular perfusion or reverse chronic axonal degeneration in these advanced stages. Such biological resistance to spontaneous recovery often leaves patients with refractory dysesthesias or persistent motor weakness that mimics persistent compressive failure, ultimately prompting clinicians and patients toward secondary surgical exploration and aggressive external neurolysis [16,17].

Preoperative thenar muscle atrophy was similarly identified as a significant independent predictor of revision surgery, mirroring the pathophysiological ramifications of end-stage compression. Thenar wasting represents motor axon loss and irreversible motor endplate degeneration following prolonged denervation of the abductor pollicis brevis muscle. When patients present with visible thenar wasting, the chronological window for rapid and complete neuromuscular recovery following simple decompression has typically elapsed. Even after complete, anatomically verified retinacular transection, functional motor restoration remains incomplete due to persistent intrinsic muscle fibrosis and irreversible loss of motor units. The persistent subjective functional deficit, characterized by

impaired key pinch and diminished cylindrical grasp, frequently leads to patient dissatisfaction and repeated clinical presentations. In many instances, the inability of standard decompression to alleviate motor deficits prompts revision surgery aimed at extensive neurolysis or secondary reconstructive adjuncts such as hypothenar fat pad transposition or opponensplasty to restore functional biomechanics [18,19].

Pre-existing diabetes mellitus conferred a substantial independent risk for secondary carpal tunnel intervention, underscoring the metabolic vulnerability of peripheral nerves under chronic hyperglycemic stress. Diabetic peripheral neuropathy renders peripheral nerve fibers exceptionally susceptible to mechanical compression through the well-established double-crush mechanism, wherein impaired axonal transport and microangiopathy diminish the nerve's tolerance to extrinsic physical entrapment. Chronic hyperglycemia promotes the accumulation of advanced glycation end-products within the endoneuria and epineurial collagen matrices, leading to structural stiffening, microvascular sclerosis, and impaired regenerative capacity of peripheral axons. Furthermore, diabetic patients frequently exhibit exuberant, dysregulated fibroblastic proliferation and abnormal wound healing cascades, predisposing the decompressed median nerve to aggressive perineural scar tethering within the newly opened carpal canal. The combination of blunted intrinsic regenerative capacity and heightened susceptibility to compressive microenvironments explains why diabetic individuals experience elevated rates of both persistent post-decompression dysesthesias and true structural recurrence [20,21].

The surgical technique utilized during the index procedure demonstrated a significant correlation with subsequent revision rates, with primary endoscopic release carrying an independent threefold increase in the odds of revision compared to open approaches. While endoscopic carpal tunnel release offers well-documented early advantages, including accelerated rehabilitation and reduced early incision tenderness, it entails a recognized learning curve and limited direct intraoperative visualization of anatomical variants. Incomplete transection of the distal or proximal margins of the flexor retinaculum, aberrant median nerve branches, or anomalous muscle bellies can be more readily overlooked through narrow endoscopic portals, resulting in persistent unrelenting nerve compression. Moreover, inadequate visualization in difficult anatomical settings increases the risk of traction neuropraxia or structural iatrogenic trauma to the palmar cutaneous or recurrent motor branches. When persistent postoperative symptoms emerge following endoscopic intervention, hand surgeons frequently lower their threshold for secondary open exploration to verify retinacular continuity, excise dense scar tissue, and perform comprehensive median nerve decompression under direct wide visualization [22,23].

Persistent scar tenderness and pillar pain represented an additional powerful postoperative determinant associated with secondary operative procedures. Pillar pain defined as localized, deep-seated tenderness over the thenar and hypothenar eminences flanking the surgical incision arises from altered carpal arch compliance, transection of cutaneous nerve branches, and biomechanical stress concentrations across the carpal ligaments. Although pillar pain is frequently regarded as a self-limiting postoperative phenomenon, its persistence beyond several months can induce severe functional disability and exacerbate regional myofascial hypersensitivity. Severe, unyielding pain over the volar carpal region frequently mimics recurrent entrapment neuropathy, generating substantial diagnostic confusion and psychological distress. In refractory cases, persistent pain combined with localized scar tethering leads surgeons to perform secondary re-exploration to excise painful neuromas, mobilize adherent cutaneous nerve twigs, or provide soft-tissue vascularized padding over the scarred carpal floor [24,25].

The clinical implications of our findings emphasize the necessity of rigorous preoperative risk stratification and refined patient counseling before embarking on carpal tunnel release. Patients presenting with advanced electrodiagnostic impairment, thenar wasting, active diabetes mellitus, or chronic symptom durations must be explicitly informed regarding their heightened biological risk of incomplete functional recovery and potential revision requirements. In such high-risk clinical subsets, hand surgeons should carefully weigh the technical nuances between wide open decompression and minimally invasive endoscopic approaches, ensuring that complete retinacular visualization is achieved. Postoperatively, the early emergence of persistent neuropathic symptoms or unrelenting pillar pain demands structured diagnostic vigilance, including high-resolution neuromuscular ultrasound and repeat electrophysiological testing, to distinguish irreversible baseline axonal loss from correctable mechanical compression before planning secondary salvage operations [26,27].

Conclusion

Secondary revision following carpal tunnel release is primarily driven by advanced baseline neural degeneration, metabolic comorbidity, endoscopic decompression, and persistent postoperative pillar pain. Comprehensive preoperative electrodiagnostic risk stratification, careful surgical technique selection, and targeted glycemic management are essential to mitigate surgical failure rates and optimize long-term clinical recovery.

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