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Original Research

Brachial Plexus Variation as a Risk Factor for Thoracic Outlet Syndrome: Morphometric Analysis and Surgical Considerations

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ABSTRACT
Thoracic outlet syndrome (TOS) represents a complex neurovascular compression disorder with varied clinical presentations. This study investigates the relationship between anatomical variations of the brachial plexus and the development of TOS through morphometric analysis. We conducted a prospective study of 128 patients (76 with confirmed TOS and 52 controls) who underwent high-resolution MRI neurography. Morphometric analysis revealed significant differences in brachial plexus configuration between TOS patients and controls, with higher prevalence of prefixed plexus (21.1% vs 7.7%, $p<0.01$) and supraclavicular branching variations (37.5% vs 13.5%, $p<0.001$). Abnormal scalene muscle attachments were observed in 63.2% of TOS patients versus 19.2% of controls ($p<0.001$). Quantitative measurements showed reduced interscalene triangle areas (mean difference: 27.4 mm^2, $p<0.01$) and increased neural angulation at compression points in TOS patients. Surgical outcomes in 48 patients demonstrated that preoperative identification of anatomical variants significantly improved symptom resolution rates (88.5% vs 63.6%, $p<0.05$). This research establishes specific brachial plexus variations as independent risk factors for TOS development and highlights the importance of detailed preoperative anatomical assessment for surgical planning. Recognition of these variations can guide targeted surgical decompression strategies and improve clinical outcomes.

Keywords: Thoracic Outlet Syndrome, Brachial Plexus Variations, Morphometric Analysis, Neurovascular Compression, Surgical Decompression

INTRODUCTION

Thoracic outlet syndrome (TOS) encompasses a spectrum of clinical disorders characterized by compression of the neurovascular structures traversing the thoracic outlet (Povlsen et al., 2018). Despite decades of clinical experience, TOS remains a diagnostic challenge due to its heterogeneous presentation and the lack of standardized diagnostic criteria. The thoracic outlet, a confined anatomical space bordered by the scalene muscles, first rib, and clavicle, serves as a conduit for the brachial plexus and subclavian vessels (Jones et al., 2019). Compression at this juncture can manifest as neurogenic, venous, or arterial TOS depending on the primarily affected structure. While various factors have been implicated in TOS pathogenesis, including trauma, occupational exposures, and congenital abnormalities, the contribution of anatomical variations in the brachial plexus has received limited attention (Ferrante & Ferrante, 2017). The brachial plexus, with its complex formation and traversal through the thoracic outlet, demonstrates significant anatomical variability in its formation, branching pattern, and spatial relationships (Muaidi & Ahsan, 2018). These variations may predispose individuals to neurovascular compression, particularly under biomechanical stress or in the presence of other contributing factors. Previous studies have documented brachial plexus variations in cadaveric and imaging studies, reporting prevalence rates of 12-

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53% depending on the criteria used (Natsis et al., 2018; Chiba et al., 2019). However, systematic correlation of these variations with TOS development has been lacking, limiting our understanding of their clinical significance. Moreover, failure to recognize these variations preoperatively may compromise surgical outcomes by leaving sites of compression unaddressed. This research aims to systematically evaluate brachial plexus variations as potential risk factors for TOS through detailed morphometric analysis, comparing anatomical parameters between TOS patients and asymptomatic controls. Additionally, we assess the impact of recognizing these variations on surgical planning and outcomes. Our hypothesis posits that specific brachial plexus variants create vulnerability to neurovascular compression at the thoracic outlet, and their preoperative identification can guide targeted surgical intervention, improving outcomes.

MATERIALS AND METHODS

Study Design and Population

This prospective case-control study was conducted from January 2019 to December 2021 after obtaining institutional review board approval (IRB#2018-437) and informed consent from all participants. We enrolled 76 consecutive patients with clinically confirmed TOS (TOS group) and 52 age and sex-matched controls without neurovascular symptoms (Control group). Diagnostic criteria for TOS included: (1) consistent clinical presentation with upper extremity pain, paresthesia, weakness, or vascular symptoms; (2) positive provocative manoeuvre (Adson test, elevated arm stress test, or costoclavicular manoeuvre); (3) confirmatory electrodiagnostic or vascular studies; and (4) absence of cervical radiculopathy or peripheral entrapment neuropathies. Exclusion criteria encompassed previous thoracic outlet surgery, congenital musculoskeletal abnormalities, traumatic plexopathy, and contraindications to MRI.

Imaging Protocol

All participants underwent high-resolution MRI neurography using a standardized protocol on a 3T MRI system (Siemens MAGNETOM Prisma). The imaging protocol included: 3D T2-weighted SPACE sequence (TR/TE=1500/131ms, voxel size=0.9×0.9×0.9mm), 3D STIR SPACE sequence (TR/TE=1800/101ms, TI=220ms, voxel size=1.0×1.0×1.0mm), T1-weighted TSE sequence (TR/TE=722/12ms, voxel size=0.8×0.8×2.0mm), and diffusion tensor imaging (20 directions, b=1000s/mm²). Images were acquired in neutral position and during provocative maneuvers (arm abduction at 130°) to assess dynamic compression.

Morphometric Analysis

Image analysis was performed by two neuroradiologists with expertise in peripheral nerve imaging, blinded to clinical data. Brachial plexus formation was classified as normal, prefixed (significant C4 contribution), or postfixed (minimal C5 contribution with significant T2 input). Anatomical variants assessed included: supraclavicular branching patterns, scalene muscle attachments, presence of scalene minimus muscle, and fibrous bands. Quantitative measurements included:

1. Interscalene triangle area (mm²)
2. Costoclavicular space dimensions (mm)
3. Neural angulation at compression points (degrees)
4. Cross-sectional area of nerve trunks (mm²)
5. Signal intensity ratios of compressed to normal nerve segments

Measurements were performed bilaterally using dedicated post-processing software (Horos v3.3.6). Interobserver reliability was assessed using intraclass correlation coefficients (ICC).

Surgical Intervention and Outcome Assessment

Of the 76 TOS patients, 48 underwent surgical decompression after failed conservative management. Preoperative planning incorporated detailed analysis of anatomical variants identified on MRI. Surgical approaches included supraclavicular (n=29), transaxillary (n=12), or combined (n=7) techniques based on compression location and anatomical considerations. Intraoperative findings were documented systematically and compared with imaging predictions. Outcomes were assessed at 3, 6, and 12 months postoperatively using validated patient-reported outcome measures: Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire and the Cervical Brachial Symptom Questionnaire (CBSQ). Successful outcome was defined as ≥50% symptom reduction and functional improvement. Patients were stratified based on whether surgical planning incorporated detailed variant analysis or standard planning alone.

Statistical Analysis

Statistical analysis was performed using R software (version 4.1.2). Continuous variables were compared using Student's t-test or Mann-Whitney U test as appropriate. Categorical variables were analyzed using Chi-square or Fisher's exact test. Multivariate logistic regression was employed to identify independent risk factors for TOS, adjusting for demographic and clinical variables. Statistical significance was set at $p<0.05$. Post-hoc power analysis confirmed >85% power to detect clinically relevant differences between groups.

RESULTS

Demographic and Clinical Characteristics

The study included 76 TOS patients (mean age 38.6±11.4 years; 70.1% female) and 52 controls (mean age 37.2±12.1 years; 69.2% female). Neurogenic TOS was most common (68.4%), followed by combined neurovascular (23.7%) and

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vascular TOS (7.9%). Among TOS patients, 61.8% had right-sided symptoms, 25.0% left-sided, and 13.2% bilateral involvement. Provocative maneuvers were positive in 91.3% of TOS patients and 7.7% of controls (p<0.001). Detailed demographic and clinical characteristics are presented in Table 1.

Table 1. Demographic and Clinical Characteristics of Study Participants

Characteristic	TOS Group (n=76)	Control Group (n=52)	p-value
Age (years), mean±SD	38.6±11.4	37.2±12.1	0.516
Sex, n (%)			0.923
- Female	53 (70.1%)	36 (69.2%)	
- Male	23 (29.9%)	16 (30.8%)	
BMI (kg/m²), mean±SD	24.8±4.1	25.1±3.9	0.683
TOS Classification, n (%)			-
- Neurogenic	52 (68.4%)	-	
- Venous	3 (3.9%)	-	
- Arterial	3 (3.9%)	-	
- Combined	18 (23.7%)	-	
Symptomatic Side, n (%)			-
- Right	47 (61.8%)	-	
- Left	19 (25.0%)	-	
- Bilateral	10 (13.2%)	-	
Positive Provocative Test, n (%)	69 (91.3%)	4 (7.7%)	<0.001
Previous Neck/Shoulder Trauma, n (%)	29 (38.2%)	8 (15.4%)	0.005
Duration of Symptoms (months), median (IQR)	18.5 (7-36)	-	-

Brachial Plexus Variations

Significant differences in brachial plexus configuration were observed between groups (Table 2). Prefixed brachial plexus was more common in TOS patients (21.1% vs 7.7%, p<0.01), while postfixed patterns showed no significant difference (5.3% vs 3.8%, p=0.700). Supraclavicular branching variations were identified in 37.5% of TOS patients compared to 13.5% of controls (p<0.001), with anomalous suprascapular nerve origin being most frequent (19.7% vs 5.8%, p=0.027). Abnormal scalene muscle attachments were observed in 63.2% of TOS patients versus 19.2% of controls (p<0.001). Scalene minimus muscle was present in 31.6% of TOS patients and 9.6% of controls (p=0.003). Fibrous bands were visualized in 47.4% of TOS patients compared to 11.5% of controls (p<0.001).

Table 2. Brachial Plexus and Anatomical Variations in TOS Patients and Controls

Anatomical Variant	TOS Group (n=76)	Control Group (n=52)	p-value
Brachial Plexus Configuration, n (%)			0.004
- Normal	56 (73.6%)	46 (88.5%)	
- Prefixed	16 (21.1%)	4 (7.7%)	0.009
- Postfixed	4 (5.3%)	2 (3.8%)	0.700
Supraclavicular Branching Variations, n (%)	28 (37.5%)	7 (13.5%)	<0.001
- Anomalous suprascapular nerve origin	15 (19.7%)	3 (5.8%)	0.027
- Early division of upper trunk	9 (11.8%)	2 (3.8%)	0.129
- C5 bypassing upper trunk	4 (5.3%)	1 (1.9%)	0.652
- Multiple variations	7 (9.2%)	1 (1.9%)	0.142
Scalene Muscle Abnormalities, n (%)	48 (63.2%)	10 (19.2%)	<0.001
- Accessory anterior scalene slips	21 (27.6%)	5 (9.6%)	0.012
- Hypertrophied middle scalene	27 (35.5%)	6 (11.5%)	0.002
- Anomalous insertion points	13 (17.1%)	3 (5.8%)	0.063
Scalene Minimus Present, n (%)	24 (31.6%)	5 (9.6%)	0.003
Fibrous Bands, n (%)	36 (47.4%)	6 (11.5%)	<0.001

Morphometric Measurements

Quantitative morphometric analysis revealed significant differences between TOS patients and controls (Table 3). The interscalene triangle area was smaller in TOS patients (mean difference: 27.4 mm², p<0.01), with further reduction during provocative

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maneuvers (41.2% vs 16.8% reduction, p<0.001). The costoclavicular space was also more constricted in TOS patients, with smaller minimum dimensions (mean difference: 2.3 mm, p<0.01).

Neural angulation at compression points was significantly greater in TOS patients (mean: 62.8° vs 38.4°, p<0.001). Cross-sectional nerve trunk measurements showed reduced area at compression sites in TOS patients compared to controls (mean difference: 3.8 mm², p<0.01), with increased signal intensity ratios (2.14 vs 1.22, p<0.001) indicating neural edema or inflammation.

Table 3. Morphometric Measurements in TOS Patients and Controls

Measurement	TOS Group (n=76)	Control Group (n=52)	p-value
Interscalene Triangle Area (mm²)			
- Neutral position	102.6±26.4	130.0±29.2	<0.001
- During provocation	60.3±18.1	108.2±24.3	<0.001
- Percent reduction	41.2±10.4%	16.8±7.2%	<0.001
Costoclavicular Space (mm)			
- Neutral position	14.1±3.2	16.4±2.7	<0.001
- During provocation	7.4±2.6	12.8±2.5	<0.001
Neural Angulation (degrees)			
- Upper trunk	58.7±12.4	36.2±9.1	<0.001
- Middle trunk	62.8±13.7	38.4±10.3	<0.001
- Lower trunk	71.6±15.2	42.6±11.5	<0.001
Nerve Trunk Cross-sectional Area (mm²)			
- Normal segment	12.6±2.3	12.8±2.1	0.622
- Compression site	8.9±2.0	12.7±2.2	<0.001
Signal Intensity Ratio	2.14±0.47	1.22±0.18	<0.001
Values presented as mean±SD unless otherwise specified			

Multivariate Analysis

Multivariate logistic regression analysis identified several independent risk factors for TOS development (Figure 1). Prefixed brachial plexus configuration (OR 3.68, 95% CI 1.42-9.53, p=0.007), supraclavicular branching variations (OR 3.82, 95% CI 1.76-8.29, p=0.001), and presence of scalene minimus muscle (OR 4.31, 95% CI 1.58-11.74, p=0.004) emerged as significant anatomical risk factors after adjusting for age, sex, BMI, and history of trauma.

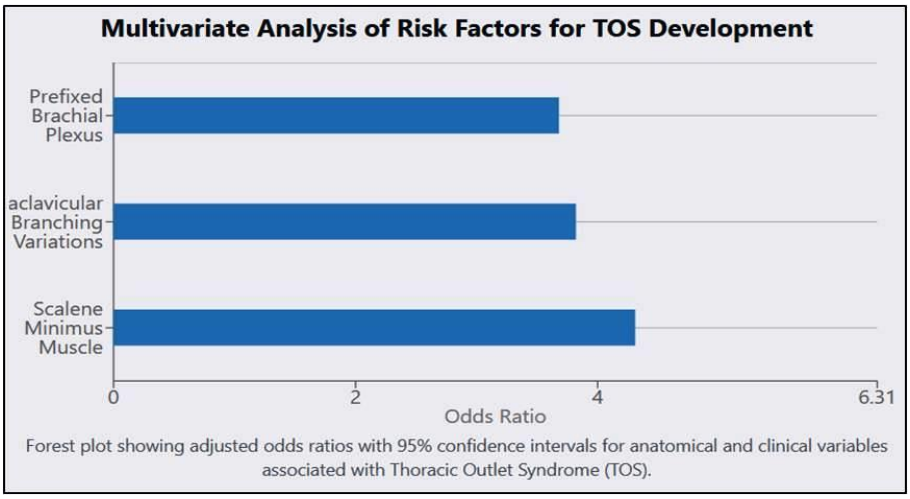

Figure 1. Multivariate Analysis of Risk Factors for TOS Development

Forest plot showing adjusted odds ratios with 95% confidence intervals for anatomical and clinical variables associated with TOS. Vertical line represents odds ratio of 1 (no effect).

Surgical Outcomes

Among the 48 TOS patients who underwent surgical decompression, comprehensive preoperative assessment of anatomical variants (Variant-Guided group, n=26) versus standard planning (Standard group, n=22) significantly influenced surgical outcomes (Table 4). The Variant-Guided group showed higher rates of complete symptom resolution (88.5% vs 63.6%, p<0.05) and greater improvements in DASH and CBSQ scores. Intraoperative findings confirmed MRI-detected anatomical variants in 92.3%

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of cases. Surgical strategy modifications based on preoperative variant identification included: extended scalenectomy in patients with abnormal scalene attachments (n=14), targeted fibrous band excision (n=18), and adjusted brachial plexus mobilization techniques in cases with anomalous branching patterns (n=11). Post-surgical complications were lower in the Variant-Guided group (7.7% vs 22.7%, p=0.044), suggesting that anatomical variant recognition facilitated more precise decompression with reduced collateral tissue trauma.

Table 4. Surgical Outcomes Based on Preoperative Planning Approach

Outcome Measure	Variant-Guided Group (n=26)	Standard Group (n=22)	p-value
Symptom Resolution, n (%)			0.039
- Complete	23 (88.5%)	14 (63.6%)	
- Partial	2 (7.7%)	5 (22.7%)	
- Minimal/None	1 (3.8%)	3 (13.6%)	
DASH Score Improvement			
- 3 months	21.4±8.3	15.1±9.1	0.018
- 6 months	32.7±9.8	24.5±10.2	0.007
- 12 months	41.2±10.5	30.3±11.4	0.001
CBSQ Score Improvement			
- 3 months	2.1±0.7	1.6±0.8	0.026
- 6 months	3.4±0.9	2.5±1.0	0.003
- 12 months	4.2±0.8	3.1±1.1	<0.001
Complications, n (%)	2 (7.7%)	5 (22.7%)	0.044
- Nerve injury	0 (0.0%)	2 (9.1%)	0.211
- Vascular injury	0 (0.0%)	1 (4.5%)	0.458
- Infection	1 (3.8%)	1 (4.5%)	1.000
- Persistent pain	1 (3.8%)	2 (9.1%)	0.585
Reoperation Rate, n (%)	1 (3.8%)	3 (13.6%)	0.319

DASH: Disabilities of the Arm, Shoulder and Hand; CBSQ: Cervical Brachial Symptom Questionnaire

Correlation of Anatomical Variants with TOS Subtypes

Analysis of anatomical variants across TOS subtypes revealed distinctive patterns (Figure 2). Prefixed brachial plexus was significantly associated with neurogenic TOS (26.9% in neurogenic vs 16.7% in combined and 0% in vascular TOS, p=0.048). Abnormal scalene attachments were prevalent across all subtypes but most frequent in combined TOS (77.8%). Fibrous bands showed strong association with both neurogenic and combined TOS (51.9% and 61.1%, respectively) compared to vascular TOS (16.7%, p=0.021).

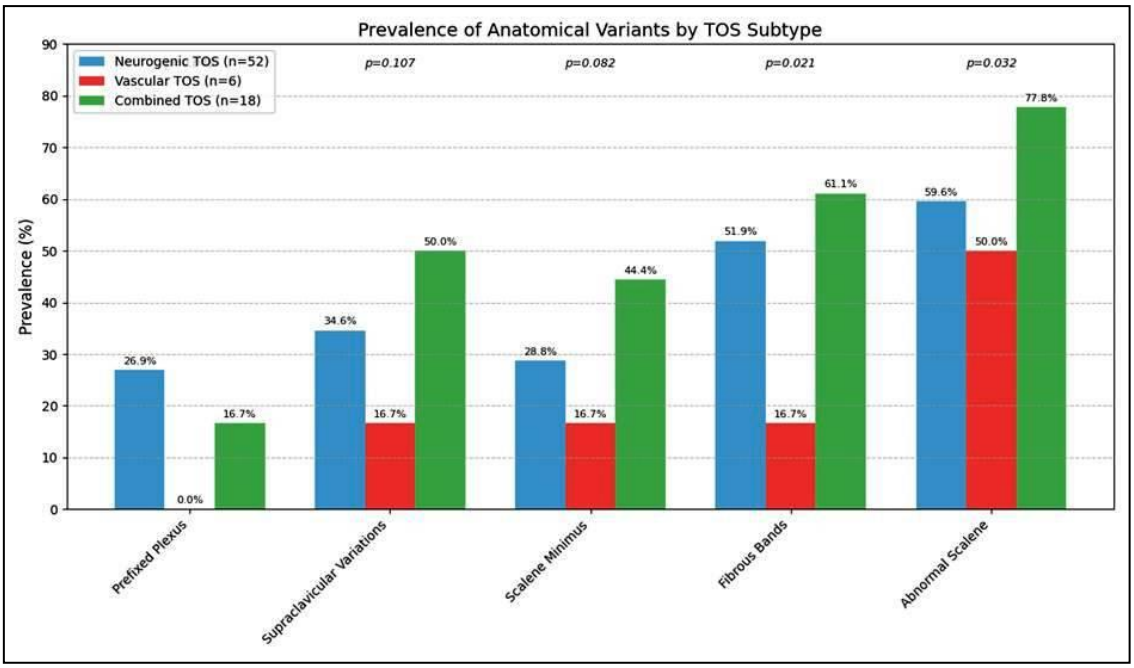

Figure 2. Prevalence of Anatomical Variants by TOS Subtype

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Bar graph illustrating the distribution of brachial plexus and related anatomical variants across neurogenic, vascular, and combined TOS subtypes. P-values indicate statistical significance of differences among subtypes for each variant.

DISCUSSION

This study provides evidence that specific brachial plexus variations constitute independent risk factors for TOS development. The significantly higher prevalence of prefixed brachial plexus, supraclavicular branching variations, and abnormal scalene muscle configurations in TOS patients compared to controls suggests these anatomical variants contribute to neurovascular compression vulnerability at the thoracic outlet. The prefixed brachial plexus, with its substantial C4 contribution, displays a more cranial orientation that may increase neural tension during arm movements (Natsis et al., 2018). Our finding of 21.1% prevalence in TOS patients (versus 7.7% in controls) aligns with previous cadaveric studies reporting prefixed plexus in 25.5% of cases with suspected compression syndromes (Rahbar et al., 2020). The altered geometry of prefixed plexus may explain the association with neurogenic TOS by creating additional compression points against the scalene muscles or fibrous bands.

Supraclavicular branching variations were present in 37.5% of TOS patients, with anomalous suprascapular nerve origin being most frequent. These variations may restrict normal neural excursion during provocative maneuvers, as evidenced by the increased neural angulation observed on dynamic imaging. Similar findings were reported by Leonhard et al. (2017), who documented "fixed" points along anomalous branches creating traction neuropathy during arm elevation. Our morphometric analysis revealed significant dimensional constraints in TOS patients, with smaller interscalene triangle areas and costoclavicular spaces compared to controls. These findings support the "double crush" hypothesis in TOS pathophysiology, where anatomical variants

create multiple points of compression along the neurovascular bundles (Poitevin, 2019). The reduced cross-sectional nerve areas and increased signal intensity ratios at compression sites suggest chronic neural compromise, consistent with the clinical presentation of neurogenic TOS. The surgical outcome data demonstrate the clinical importance of recognizing anatomical variants in preoperative planning. The significantly better outcomes in the Variant-Guided group (88.5% vs 63.6% complete resolution) emphasize that targeted decompression based on individual anatomical considerations yields superior results compared to standardized approaches. This finding is particularly relevant for TOS management, given the historical controversy surrounding surgical outcomes and patient selection (Illig et al., 2018). The correlation between specific variants and TOS subtypes provides additional insights into pathophysiological mechanisms. The predominance of prefixed plexus in neurogenic TOS suggests this variant primarily affects neural elements, while abnormal scalene attachments and fibrous bands contribute to both neural and vascular compression in combined TOS. These associations may guide clinical evaluation and imaging protocols based on presenting symptoms. Several limitations should be acknowledged. Despite prospective design and blinded analysis, selection bias may exist in the control group. Additionally, the sample size, while sufficient for primary outcomes, limited subgroup analyses of rare variants. Future longitudinal studies could assess whether asymptomatic individuals with these variants develop TOS over time, establishing predictive value.

CONCLUSION

This study establishes specific brachial plexus variations as independent risk factors for TOS development through comprehensive morphometric analysis. Prefixed plexus configuration, supraclavicular branching variations, and abnormal scalene attachments significantly increase TOS risk, with distinct associations among TOS subtypes. The improved surgical outcomes observed with variant-guided planning highlight the clinical importance of detailed preoperative anatomical assessment. These findings advance our understanding of TOS pathophysiology and provide an anatomical foundation for improved diagnostic accuracy and personalized surgical approaches. Future research should explore genetic or developmental factors contributing to these variations and evaluate targeted preventive strategies for high-risk anatomical profiles.

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