# The spectrum of arterial compression at the thoracic outlet

**Enrique Criado, MD, Ramon Berguer, MD, PhD,** and **Lazar Greenfield, MD,** *Ann Arbor, Mich*

***Objective:*** **In the absence of ischemic events, arterial pathology at the thoracic outlet (TO) is rarely identified because findings of chronic arterial pathology may be masked by symptoms of neurogenic compression. This study describes the clinical presentations and significance of arterial compression at the TO.**

***Methods:*** **This was a retrospective analysis of the clinical records and imaging studies of 41 patients with objective findings of arterial compression at the TO. Sixteen were diagnosed from 1990 to 2003, during which 284 patients underwent surgery for TO decompression with selective arterial imaging; 25 were diagnosed from 2003 to 2009, and 62 underwent TO surgical decompressions.**

***Results:*** **Subclavian artery stenosis, with or without poststenotic dilatation (PSD), was found in 26 patients (63%), subclavian artery aneurysms in 12 (29%), chronic subclavian occlusion in 1(2.4%), and axillary artery compression in 2 (5%). Chronic symptoms difficult to discern from neurogenic compression were present in 27 patients (66%; 24 had subclavian stenoses or PSD, or both, 1 had subclavian occlusion, and 2 had axillary artery compression); 13 (32%) presented with acute ischemia (11 had aneurysms and 2 had PSDs), and 1 asymptomatic patient had a subclavian aneurysm. Osteoarticular anomalies were found in 27 patients (66%), including 19 cervical ribs, 4 first rib anomalies, and 4 clavicular or first rib fractures, or both. Among 27 patients with subclavian aneurysms or PSD, 21 (78%) had a bone anomaly. Arterial pathology was deemed significant in 30 patients (73%) and mild or moderate in 11 (21%). Symptoms in 23 of these patients were compatible with neurogenic compression without clinical suspicion of arterial pathology, but 13 (56%) harbored a significant arterial anomaly.**

***Conclusions:*** **The incidence of arterial pathology secondary to compression at the TO may be underestimated, and in the absence of obvious ischemia, significant arterial pathology may not be suspected. Two-thirds of patients with arterial compression have associated bone anomalies. Therefore, routine arterial imaging seems advisable for patients evaluated for TO syndrome in the presence of a bone anomaly at the TO or an examination that shows an arterial abnormality. In the absence of these signs, however, arterial pathology may be overlooked in patients with symptoms suggestive of neurogenic compression. Further study is needed to elucidate the incidence, natural history, and clinical relevance of arterial compression and PSD at the TO. (J Vasc Surg 2010;52:406-11.)**

The most commonly recognized clinical presentation of arterial compression at the thoracic outlet (TO) is upper extremity arterial embolization, mostly in young, otherwise healthy individuals. Arterial complications at the TO represent the end stage of an undiagnosed condition in which the subclavian artery has been chronically compressed and wall changes develop, including intimal injury with or without poststenotic dilatation (PSD) or aneurysm formation. This presentation is usually limb threatening, often requires emergency surgery, and sometimes results in significant functional impairment of the arm.

It is difficult to discern whether these patients might have had subtle symptoms that would have enabled an earlier diagnosis. Ideally, the identification of early anatomic arterial changes secondary to compression at the TO would allow treatment before further chronic injury results in limb-threatening arterial pathology.

Patients with significant subclavian or axillary artery stenosis, in the absence of acute ischemic complication, may present with typical symptoms of upper extremity claudication. However, patients with chronic positional arterial compression at the TO may be asymptomatic or, more commonly, have associated symptoms of neurogenic compression. In the absence of acute or chronic ischemic signs or symptoms, it is difficult to determine the contribution of chronic arterial compression to the symptoms of neurogenic thoracic outlet syndrome (TOS). For these reasons, many patients who are evaluated and treated for neurogenic compression may have unrecognized arterial pathology. In addition, we cannot predict at what stage PSD or other chronic arterial changes may become potentially limb threatening.

Postural or fixed arterial stenosis at the TO, with or without PSD, is the obvious precursor of aneurysm formation. Although the genesis of PSD by wall vibration secondary to turbulent flow dynamics has been well studied, the clinical outcome of this chronic process is poorly known, but certainly not benign. Experimental evidence suggests that PSD is a reversible phenomenon after relief of the stenotic or compressive process,[1,2] but the natural history and clinical relevance of arterial compression resulting in PSD in patients with TOS is not well understood.

From the Department of Surgery, Vascular Surgery Section, University of Michigan School of Medicine.

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Correspondence: Enrique Criado, MD, Section of Vascular Surgery, University of Michigan School of Medicine, CVC 5463, 1500 E Medical Center Dr, SPC 5867, Ann Arbor, Michigan 48109-5867 (e-mail: ecriado@umich.edu).

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Recognition of arterial pathology at the TO in young patients with a long life expectancy may be in itself an indication for surgical arterial decompression, and its treatment has been shown to produce satisfactory long-term results.[3] Our study explores the incidence and potential clinical significance of arterial pathology in patients evaluated for TOS.

## METHODS

This study was approved by the University of Michigan School of Medicine Institutional Review Board for research on human subjects (HUM00025641).

**Patients.** A database of patients treated for TOS by vascular surgeons at the University of Michigan School of Medicine was used to identify patients with objective findings of arterial pathology diagnosed by digital subtraction arteriography, computed tomography arteriography (CTA), magnetic resonance arteriography (MRA), or surgical exploration, or a combination of these.

Sixteen patients were diagnosed from 1990 to 2003, and 25 were diagnosed from 2003 to 2009. Among the 284 patients operated on during the first period, arterial imaging (arteriography or MRA) was done only in the presence of ischemic complications and identified 16 patients (6%) with objective arterial pathology at the TO. During the second period, 62 patients underwent surgery for TOS. Arterial imaging studies were done in 56 of these patients, and an arterial anomaly was diagnosed in 25 (40%) by dynamic CTA or digital subtraction arteriography, or both. During the second period, dynamic CTA was adopted for the routine evaluation of positional changes in the costoclavicular space or other causes of TO compression.

As part of the study, arterial images are rendered that allow evaluation of the subclavian and axillary arteries. Dynamic CTA was conducted with the patient supine and with the arms in external rotation, abducted, and elevated above the head. Patients presenting with subclavian vein thrombosis did not undergo arterial imaging. Subclavian aneurysms were defined as arterial dilatations ≥50% than the diameter of the adjacent normal artery. Arterial dilatation was defined as an enlarged arterial segment <50% greater than the adjacent proximal segment. Moderate stenosis was defined as a ≤50% diameter reduction compared with the adjacent normal artery and severe stenosis as a ≥70% diameter reduction. The severity of arterial pathology found intraoperatively was estimated by the surgeon.

## RESULTS

Arterial anomalies at the TO were identified in 41 patients, including 12 (29%) subclavian artery aneurysms, 26 (63%) arterial stenoses with or without PSD, 1 (2.5.%) chronic subclavian artery occlusion, and 2 (5%) axillary artery compressions (Table I). The affected side was the left in 23 patients (56%), the right in 17 (41%), and was bilateral in 1. These patients were a mean age of 37 years (range, 14-74 years) and 25 (61%) were females. The method of diagnosis, clinical presentation, presence of bone anomalies, and treatment of these patients were analyzed.

**Table I.** Arterial pathology associated with thoracic outlet syndrome

| *Finding* | *No.* |
|---|---|
| Subclavian artery aneurysm | 12 |
| Postural stenosis with poststenotic dilatation | 10 |
| Postural stenosis without dilatation | |
| Subclavian artery | 5 |
| Axillary artery | 2 |
| Fixed subclavian stenosis | 5 |
| Fixed stenosis with poststenotic dilatation | 4 |
| Luminal filling defect | 2 |
| Subclavian artery occlusion | 1 |
| Total | 41 |

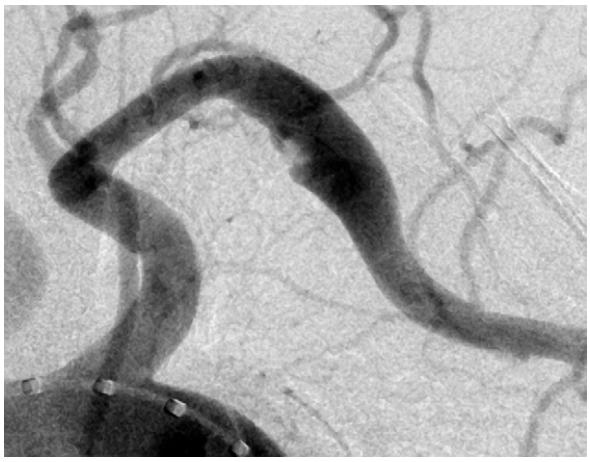

**Fig 1.** Arch arteriogram depicts a left subclavian aneurysm with a luminal filling defect. This patient presented with upper extremity embolization without preexisting symptoms.

**Diagnosis of arterial anomaly.** Arterial pathology was diagnosed by arteriography in 22 patients, by CTA in 12, by MRA in 2, and during surgery, without arterial imaging, in 5. These 5 patients were operated on during the first part of the study, and the artery was found to be pathologically compressed during the surgical exposure of the TO. The identified arterial pathology was considered clinically significant in 30 patients (73%), comprising 12 aneurysms, 13 positional arterial stenoses >70%, 2 fixed subclavian stenoses, 2 luminal filling defects, and 1 subclavian occlusion. The arterial pathology was mild to moderate in 11 patients (27%), all with <70% positional stenosis, 7 with PSD, 2 with fixed stenoses, and 2 with positional compression.

**Clinical presentation.** Acute ischemia secondary to arm/hand embolization was present in 13 patients (32%). Among them, 11 (85%) had subclavian artery aneurysms, (Fig 1) and 2 (15%) had mild PSDs. Chronic upper extremity symptoms in 27 patients (66%) were compatible with neurogenic compression; of these, 24 had subclavian ste-

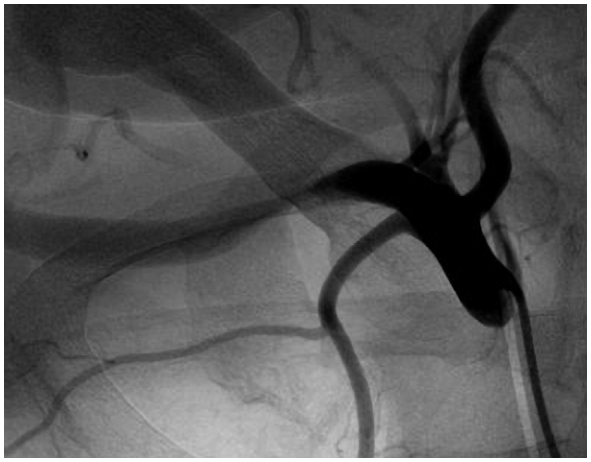

**Fig 2.** A selective right subclavian arteriogram demonstrates an almost complete arterial occlusion with arm abduction, without a cervical rib or other bone anomaly. The arteriogram was normal in the neutral position. The patient had chronic upper extremity symptoms difficult to differentiate from neurogenic thoracic outlet syndrome. He underwent first rib resection and neurolysis of the brachial plexus, with complete resolution.

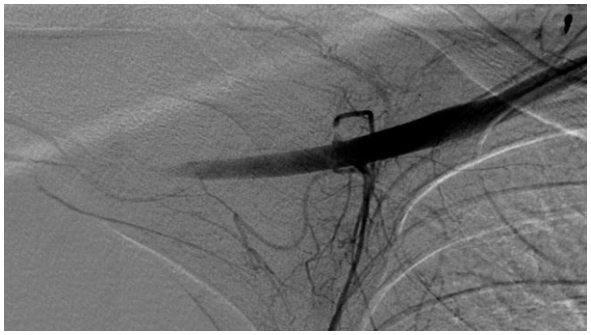

**Fig 3.** A selective right subclavian artery arteriogram shows a complete axillary artery occlusion with the arm abducted 90° and externally rotated. The humeral head is compressing the axillary artery. The patient presented with tingling and numbness of his hand and arm that interfered with his work as an automobile mechanic. The patient underwent mobilization of the artery, freeing it from multiple fibrotic attachments to the aponeurotic tissues overlying the humeral head. This was followed by complete resolution of the symptoms.

nosis (Fig 2) or PSD, or both, 1 had chronic subclavian occlusion, and 2 had axillary artery compression (Fig 3). One patient with a subclavian aneurysm was asymptomatic (Table II).

In 23 of the patients presenting with symptoms compatible with neurogenic compression, arterial pathology was not suspected and most likely would not have been identified without CTA or angiography. Among these 23 patients, 13 (56%) were harboring significant pathology, including 6 dynamic arterial stenoses >70%, 3 dynamic subocclusions, 1 subclavian artery chronic occlusion, 1 subclavian fixed stenosis, 1 luminal filling defect, and 1 subclavian aneurysm.

**Table II.** Symptoms associated with arterial anomaly at the thoracic outlet

| *Symptoms* | *No.* |
|---|---|
| Acute ischemia | 13 |
| Arm, shoulder, and neck pain increasing with activity | 26 |
| Arm/hand paresthesias | 15 |
| Arm/hand numbness | 10 |
| Hand color changes | 3 |
| Hand coldness | 2 |
| No symptoms | 1 |
| Hand weakness | 1 |

The radial pulse of the affected arm disappeared during the Adson or modified Wright maneuver in 24 patients (58%), and 17 (71%) had significant arterial pathology. The radial pulse did not change in two patients. The pulse was absent or weak in the anatomic position in 11 patients, and the extremity pulses were not recorded in 4 patients.

**Associated bone anomalies.** Osteoarticular anomalies at the TO were encountered in 27 patients (66%), including 19 cervical ribs, 4 first rib anomalies, and 4 old clavicular or first rib fractures, or both. Among 30 patients with significant arterial findings, 19 (63%) had a cervical rib or other bone anomaly. Of the 27 patients with subclavian aneurysms or PSDs, 22 (81%) had a cervical rib or other bone anomaly at the TO (11 [92%] of 12 aneurysms and 11 [73%] of 15 PSDs).

During the first part of this study, 284 patients underwent surgery for TOS. A cervical rib was found in 56 patients (20%), 9 (15%) of whom had arterial anomalies. In comparison, during the second period of this study, 11 (18%) of 62 patients undergoing surgery for TOS had a cervical rib, and an associated arterial anomaly was found in 9 (82%).

**Treatment.** No deaths or arterial complications occurred in 37 patients who underwent surgical treatment. The TO procedure in 32 patients was a first rib or cervical rib resection through a supraclavicular approach, and 5 underwent medial claviculectomy. Among the 37 operations, 13 patients had simultaneous vascular reconstruction, all for subclavian aneurysms. The vascular reconstructions included 10 subclavian-to-axillary interposition grafts (5 with polytetrafluoroethylene [PTEF] and 5 with reversed saphenous vein), 1 subclavian-to-carotid transposition, 1 common carotid-to-axillary bypass with PTFE, and 1 aneurysm resection with end-to-end arterial anastomosis. Two patients remained under surveillance because of the moderate symptoms and mild vascular changes. Two patients declined treatment, one because of synchronous malignancy, and one for health insurance reasons. No imaging follow-up studies of the arteries were conducted in operated-on patients.

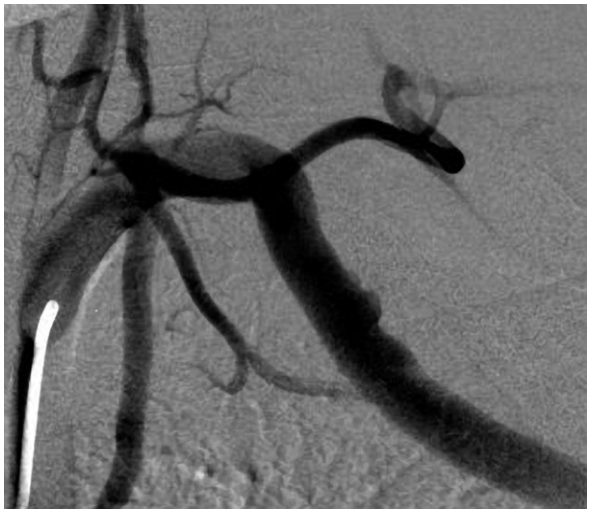

**Fig 4.** A selective left subclavian arteriogram reveals subtle luminal filling defects without poststenotic dilatation. This patient presented with limb-threatening acute ischemia from embolization. In retrospect, the patient recognized a history of ill-defined preexisting arm symptoms.

## DISCUSSION

The diagnosis of neurogenic TOS remains one of exclusion and is based on the subjective interpretation of signs and symptoms by the physician and patient.[4] The clinical diagnosis of neurogenic TOS is based on standardized provocative signs and is rather inaccurate.[5] In some patients, it may be difficult to decide whether the signs or symptoms of neurogenic TOS may be derived in part from positional arterial compression at the TO.

Arterial compression at the TO with provocative maneuvers produces a decrease in palpable arterial pulses in up to 60% of healthy, asymptomatic individuals and is found bilaterally in 33%.[6] That this obliteration of the pulse is a common finding in healthy individuals has led to the dismissal of its significance in symptomatic patients, and the possible contribution of arterial compression to neurologic symptoms in patients with TOS is disregarded. It is reasonable to assume that radial pulse disappearance in an individual with postural changes may imply the presence of a tighter TO than in an individual without such a physical finding. We do not know, however, under what circumstances in asymptomatic individuals chronic postural arterial compression may lead to significant arterial pathology and when it may evolve into perilous arterial dilatation or mural injury (Fig 4).

The close anatomic relationship between the subclavian artery and brachial plexus makes it difficult to explain how could these structures be almost always compressed independently, as the TOS literature suggests. In this regard, one of the largest experiences with the surgical treatment of TOS estimates the incidence of arterial compression at $<0.5\%$ of all cases of TOS and separates arterial and neurogenic compression in a distinct way, without considering the possibility of combined neurogenic and arterial compression.[4] However, arterial imaging studies were seldom done in that series of patients.

Because arterial TOS is thought to be rare, patients being evaluated for TOS do not usually undergo imaging studies that may reveal pathologic arterial findings. For this reason, it is possible that many patients with symptomatic or asymptomatic arterial anomalies secondary to arterial compression are diagnosed as having nerve compression without the benefit of an objective arterial evaluation. This is exemplified by a report of a series of 65 operations performed for TOS in the presence of a cervical rib or first rib anomaly, among which 54 patients (83%) were diagnosed with neurogenic TOS without any arterial involvement, 9 (14%) with arterial TOS, and 2 with venous compression.[7] In this experience, however, only a few patients underwent ultrasound arterial evaluation to rule out arterial dilatation (a limited study to investigate arterial pathology in this area). None were evaluated for dynamic arterial compression because the authors argued that all patients had positive signs for neurologic TOS.[7]

This is in sharp contrast with our recent experience, when a liberal use of arterial imaging found an 82% rate of arterial pathology in patients treated for TOS with cervical ribs, first rib, or clavicular anomalies. This suggests that many patients treated for TOS who have not had arterial imaging studies may harbor significant undiagnosed underlying arterial pathology.

Cervical ribs are found in $<0.5\%$ of the general population, but their presence is associated with objective histopathologic changes in the brachial plexus, including epineural and perineural fibrosis, vascular hyalinization, mucinous degeneration, and frequent intraneural collagenous nodules.[8] These changes, which would most likely produce symptoms, are not seen in the nerve specimens of individuals without cervical ribs.[8] This observation suggests that in cases of arterial compression by a cervical rib, nerve compression will very likely be simultaneously present. We believe that nerve compression produces the most common and dominant symptoms in patients with TOS, but we think that arterial compression may be underestimated because it may be asymptomatic and it is not commonly investigated.

In addition, the treatment of neurogenic compression at the TO usually involves a resection of a first or cervical rib, which when properly performed, should resolve any concomitant subclavian arterial compression that may exist. From this fact it could be argued that because the surgical treatment of neurogenic TOS also decompresses the artery, it is not necessary to investigate the presence of arterial pathology. On the other hand, those patients with symptoms of neurogenic TOS who are not surgically treated may harbor early stages of subclavian artery injury that may remain silent and will not be recognized until a limb-threatening complication occurs (Fig 5), as has been suggested.[9] A similar evolution appears to take place in patients with venous TOS, in whom progressive venous injury develops, most of the times without heralding symptoms,

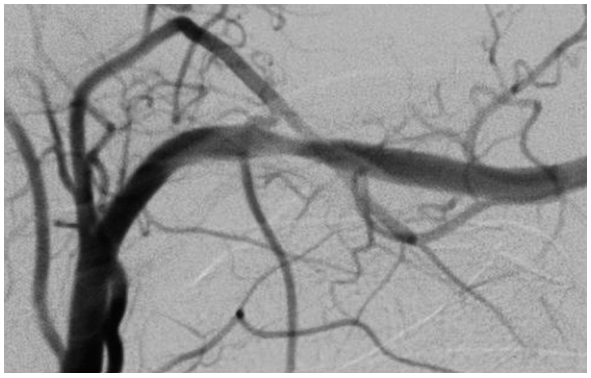

**Fig 5.** A selective left subclavian arteriogram demonstrates moderate compression of the subclavian artery with mild poststenotic dilatation in the presence of a cervical rib. The patient had signs and symptoms that were difficult to differentiate from neurogenic thoracic outlet syndrome. The symptoms resolved after cervical rib resection and neurolysis of the brachial plexus.

and who are often diagnosed after an acute thrombotic episode ensues.

The high incidence of arterial findings in our TOS population was derived from a liberal use of arterial imaging studies. During the last few years, we routinely used dynamic CTA of the TO, which offers an objective measurement of the narrowing of the costoclavicular space and also evaluates the artery for mural and intraluminal changes (Fig 6). We are currently evaluating the role of CT imaging in the diagnosis and subsequent treatment of TOS, with the expectation that it may constitute an objective predictor of treatment success after surgical decompression for TOS, as suggested by others.[10]

An unresolved question is how to manage these abnormal arterial findings, because we do not know their natural history very well. Arterial aneurysms and significant subclavian artery PSDs, in the presence of a fixed or postural arterial compression, should be treated because of their tendency to cause embolization. In the same manner, fixed stenotic lesions or complete dynamic arterial occlusion should be treated in the presence of symptoms compatible with arm ischemia. However, it is debatable what to do with more subtle arterial pathology. Two of our patients presenting with acute ischemia from embolization had only subtle PSDs that were previously asymptomatic. We suspect that early arterial changes in TOS patients without arterial signs or symptoms do not necessarily have a benign course.

Our experience suggests that patients presenting with TOS and bone anomalies at the TO should undergo arterial imaging. However, because not all patients with arterial pathology have associated bone anomalies, and the arterial problem may not produce symptoms, it is difficult to determine which patients without bony anomalies should undergo arterial imaging.

Dynamic CTA evaluation of the TO may become a diagnostic standard for TOS, which will include a systematic evaluation of the arterial segment at the TO. In the meantime, we have to use judgment when requesting arterial imaging studies in patients with TOS.

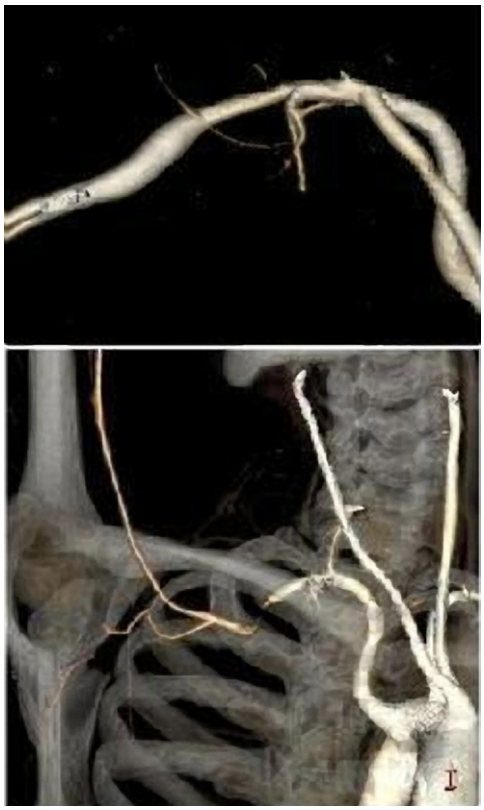

**Fig 6. Top,** A computed tomography (CT) 3-dimensional reconstruction shows left subclavian arterial dilatation without stenosis in the neutral position and without a cervical rib or other bone anomaly. **Bottom,** A repeat CT scan with the arm abducted shows complete occlusion of the subclavian artery. This case illustrates how postural compression of the subclavian artery at the thoracic outlet produces distal dilatation in the same way that fixed arterial stenoses do.

## CONCLUSIONS

Our experience suggests that the incidence of arterial pathology related to compression at the TO may be underestimated. Obvious findings of acute arterial complications may occur only in a minority of patients with arterial TOS, while significant chronic arterial pathology may remain asymptomatic or be masked by a clinical picture consistent with nerve compression at the TO.

## AUTHOR CONTRIBUTIONS

Conception and design: EC
Analysis and interpretation: EC, RB, LG
Data collection: EC, LG
Writing the article: EC
Critical revision of the article: EC, RB, LG
Final approval of the article: EC, RB, LG

Statistical analysis: EC
Obtained funding: Not applicable
Overall responsibility: EC

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