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Clinical Research

Arterial Thoracic Outlet Syndrome: A 32-year Experience

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Background: Clinical manifestations of thoracic outlet syndrome (TOS) differ depending on the compromised anatomic structure. Arterial TOS is the least common (1−5% of all cases of TOS), yet the most threatening, due to the risk of limb loss.
Methods: We conducted a retrospective review of consecutive patients treated for arterial TOS between January 1979 and June 2012. Medical records and diagnostic images were reviewed, and follow-up was obtained.
Results: Nineteen procedures were performed in 18 patients for symptomatic arterial TOS. The average age was 34 years (range 16−69 years), and 12 patients were female (63.2%). Surgical indications were upper limb critical ischemia in 8 (acute in 5 cases and acute-on-chronic in 3 cases) and claudication in 11. Imaging studies revealed a subclavian aneurysm in 7 patients, stenosis in 4 patients, and 2 patients with subclavian artery occlusion. The 6 remaining cases had symptoms caused by arterial compression in dynamic studies without arterial wall damage at rest. All limbs underwent surgery with outlet decompression; in addition, 13 underwent arterial reconstruction, and 7 were treated for distal embolic complications. There were no deaths, amputations, or early reoperations; 1 patient was readmitted 2 weeks after surgery for chylothorax, which resolved with conservative measures. During a mean follow-up of 155.8 ± 103.1 months, 1 patient underwent successful reintervention at 4 months for bypass occlusion.
Conclusions: Arterial TOS is an infrequent but relevant manifestation of TOS. An accurate and early diagnosis allows for timely surgery and adequate results, as shown in this group of patients.

INTRODUCTION

Thoracic outlet syndrome (TOS) comprises a series of signs and symptoms relating to compression of vascular and nerve structures of the upper limb while crossing from the neck and thorax through a compact anatomic space, surrounded by bone, ligaments, and muscle structures.[1,2]

There are 3 forms of TOS depending on the predominating compression of the brachial plexus roots, the subclavian vein or the artery, with neurogenic TOS the most frequent clinical expression. Despite the fact that arterial TOS (TOS-A) is the least frequent (1−5% of all TOS cases[3,4]), it is the most severe due to damage to the arterial wall by repetitive local trauma leading to a stenosis and/or poststenotic aneurysmatic dilation, eventually causing distal embolization and limb-threatening secondary ischemia.[1,5]

TOS-A must be suspected in all patients with upper limb ischemia. Therefore, necessary imaging studies must be requested for early diagnosis and

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prompt treatment to avoid its unfavorable outcome.[6] We present our experience with symptomatic patients undergoing treatment for TOS-A.

METHODS

This is a retrospective study of consecutive patients who underwent surgical treatment for TOS-A between January 1979 and June 2012. Clinical records and diagnostic images were reviewed and follow-up was performed. TOS was diagnosed by clinical assessment and a functional study with arterial plethysmography (PVR) of the upper limbs with patients at rest and when performing outlet compression maneuvers, consisting of forced abduction and external limb rotation. Functional TOS-A findings were confirmed with dynamic imaging studies of the anatomy. Subclavian aneurysm was defined as an increase of more than double the usual diameter of the underlying subclavian artery.[7] Severe stenosis was defined as a reduction of the arterial lumen of $\geq$70%.

Treatment was planned after considering both type and extension of arterial damage. Clinical success was defined by improvement of initial symptoms and clinical perfusion by hemodynamic assessment of the affected limb.

During the study period, a total of 82 TOS patients were treated using decompressive surgery in 90 limbs. Neurogenic TOS predominated in 50 (55.6%) cases, with venous TOS in 21 (23.3%) and TOS-A in 19 (21.1%) cases.

The average age of TOS-A patients was 34.0 ± 15.4 years (range 16–69 years), and there were slightly more females ($n = 12$; 63.2%). The majority of patients had no comorbidities (73.7%). The remaining patients presented with previous history of tobacco use (6 cases), hypertension (2), dyslipidemia (2), hypothyroidism (1), and arrhythmia (1). The right limb was the most frequently compromised (12 patients; 63.2%). Three patients presented with bilateral TOS-A: 2 had contralateral subclavian occlusion, asymptomatic due to adequate collateral network, and with no surgical indication for this reason; the last case presented with symptomatic arterial stenosis at rest, and was the only patient treated for bilateral TOS-A.

All patients complained of ischemic pain: at rest in 8 cases and intermittent claudication in the remaining 11 cases. Other symptoms included paresthesias in 12 (63.2%) and limb weakness in 2 (10.5%). Physical examination revealed abnormal distal pulses in 15 (78.9%; diminished in 9 and absent in 6), positive Adson's signs in 13 (68.4%), arterial pressure asymmetry >20 mm Hg in the upper extremities in 6 (31.6%), coldness in 6 (31.6%), pallor in 5 (26.3%), supraclavicular murmur at rest in 4 (21.1%), and pulsatile mass in 2 (10.5%). No patients presented with tissue loss.

Table I. Imaging studies and pathologic findings

Thoracic and cervical spinal radiographs, *n* (%)	17 (89.5%)
Cervical rib (bilateral 4)	9
Collarbone fracture	2
Normal	6
Arterial duplex scanning, *n* (%)	3 (15.8%)
Subclavian aneurysm	2
Subclavian stenosis	1
Computed tomographic angiography, *n* (%)	5 (26.3%)
Aneurysm and embolism	3
Subclavian compression at rest	2
Magnetic resonance angiography, *n* (%)	2 (10.5%)
Aneurysm and embolism	1
Subclavian occlusion	1
Arteriography, *n* (%)	13 (68.4%)
Aneurysm and embolism	4
Subclavian stenosis alone	3
Stenosis and embolism	1
Subclavian occlusion	2
Compression without lesions	3
Contralateral occlusion	2

Treatment indications included critical ischemia of the upper limb at rest in 8 cases (42.1%), which presented either as acute onset (5 cases) or chronically reintensified over months (3 cases), and as intermittent claudication in 11 (57.9%).

Imaging studies and their findings are shown in Table I; a cervical rib was present in 9 cases (Fig. 1). Correlation between the findings of the imaging studies and forms of presentation are presented in Table II. The 7 patients with subclavian aneurysm presented with a distal embolism as the primary clinical manifestation (Fig. 2); of these, 6 presented with critical ischemia. The seventh patient with subclavian aneurysm was referred for intermittent claudication that evolved over months; she was the oldest in the series (69 years), positive for hypertension, and diagnosed with subclavian aneurysm by computed tomographic angiography (CTA).

One patient with critical ischemia had partial thrombosis associated with subclavian stenosis, and secondary distal emboli. Three patients with significant subclavian stenosis at the outlet and 2 additional patients with complete occlusion presented with intermittent claudication. Six cases showed no apparent lesions of the arterial wall: 4 presented with dynamic occlusion when raising

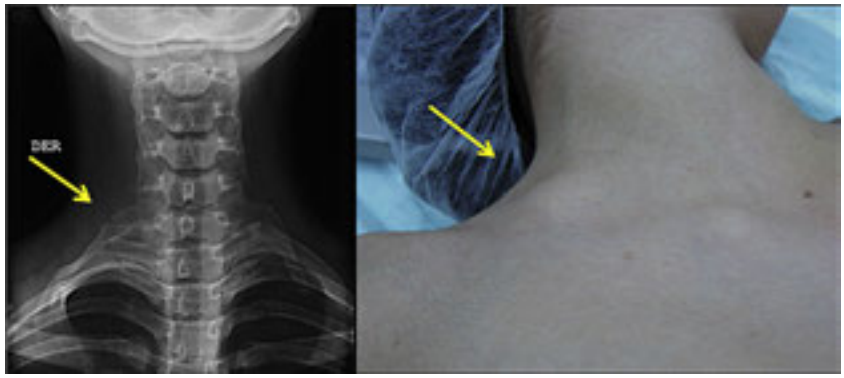

Fig. 1. Anteroposterior cervical X-rays (*left*) and image (*right*) showing the presence of a cervical rib (*arrow*).

Table II. Correlation of pathologic findings in subclavian artery and clinical presentation (number of patients)

	Aneurysm	Stenosis	Occlusion	Compression alone
Acute critical ischemia	3	1	0	1
Acute-on-chronic critical ischemia	3	0	0	0
Intermittent claudication	1	3	2	5
Total	7	4	2	6

a limb during arteriography and 2 had distal embolism.

The remaining patient was referred for intermittent claudication; CTA revealed bilateral subclavian stenosis at rest (Fig. 3), and the patient was treated for both cervical ribs.

Surgical treatment consisted of at least two repairs: outlet decompression and arterial reconstruction. In some patients, additional distal embolism treatment was also required. Decompression was achieved through a supraclavicular approach in 11 cases and the transaxillary approach was used in 8 instances. Bone resection was done in 17 cases: first rib resection in 15 cases and cervical rib in 7 cases; simultaneous anterior scalenectomy and/or fibrous band resection was performed when vascular or nervous structures were being compromised. In 2 cases, isolated scalenectomy and soft tissue removal without rib resection was conducted.

The subclavian artery was repaired in 13 cases via a supraclavicular approach, either exclusively or in combination with an infraclavicular approach in 10 cases. The details of these arterial reconstructions are described in Table III, with subclavian−axillary bypass being the most frequent (Fig. 4). The conduit used in arterial bypass was a saphenous vein in 6 cases and a 6- or 8-mm polytetrafluoroethylene ring-reinforced graft in 7 cases. In the 6 cases that presented only with dynamic arterial compression but no arterial wall lesions, rib resection was performed without arterial reconstruction. In all aneurysmal cases, the dilated segment was removed and sent for pathology examination that reported nonspecific arterial dilation with mural thrombus in 4 cases. Patients with critical ischemia due to associated embolism were treated as described in Table III.

RESULTS

In this study we report no hospital mortality and no cases of amputation nor early reintervention, with clinical success being achieved in all patients. Three cases (15.8%) required transitory pleural drainage due to a pleural tear while performing rib resection, and pneumothorax was found in 1 case during the postoperative period. The median length of stay was 6 days (range 3−13 days), and patients were discharged with antiplatelet drugs in 63.2% of cases, whereas 1 patient was kept on oral anticoagulant treatment after thrombolysis for distal embolism. Follow-up was performed for an average of 155.8 ± 103.1 months (range 13−334 months). One female patient underwent early intervention for contralateral neurogenic TOS without complications. Another female patient was readmitted 2 weeks after surgery for significant ipsilateral

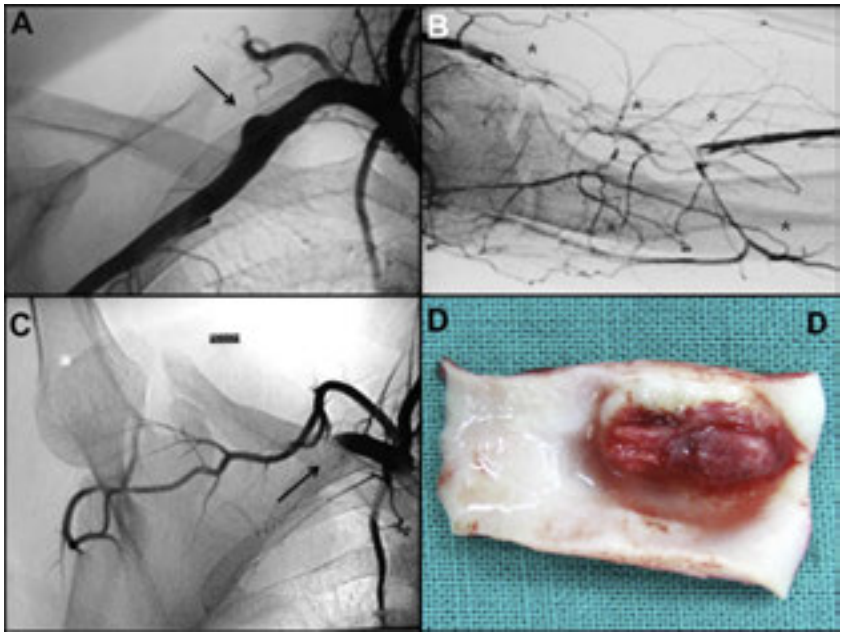

Fig. 2. Selective digital subtraction arteriography in an 18-year-old man with critical ischemia of his right upper extremity. A right subclavian aneurysm can be seen **(A)** (*arrow*), along with distal emboli in the right forearm **(B)** (*asterisks*) and dynamic occlusion of the subclavian artery when raising the extremity **(C)** (*arrow*). During surgery the resected segment of the artery was opened, showing aneurysm degeneration with associated mural thrombus **(D)**.

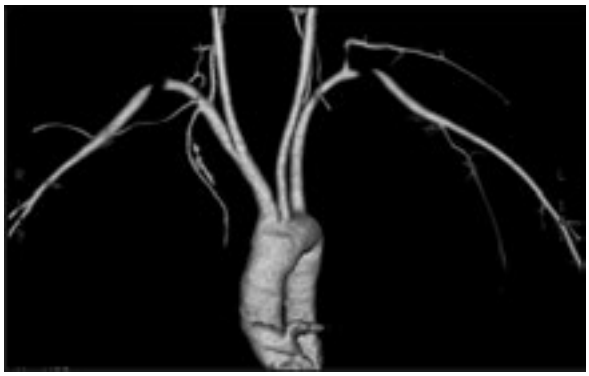

Fig. 3. Computed tomographic angiography with 3-dimensional reconstruction in anteroposterior view and with upper extremities in a passive position (at rest). A compressive occlusion of both subclavian arteries at the thoracic outlet without presenting parietal damage can be seen. This image is from a 22-year-old woman with intermittent claudication and bilateral cervical ribs who underwent surgery on both sides via a supraclavicular rib resection without arterial repair.

chylothorax, which was resolved with percutaneous drainage and a low-fat diet. Yet another female patient, a 43-year-old woman with critical ischemia initially caused by subclavian stenosis and distal embolism, underwent a carotid–subclavian bypass with a polytetrafluoroethylene graft and distal thrombolysis but without concomitant rib resection. Four months later she was readmitted with pain and coldness in her hand and absence of left upper extremity pulses; arteriography revealed an occluded bypass. She underwent a transaxillary resection of the first rib and a new carotid–axillary bypass with saphenous vein and brachial thrombectomy, which has now been patent for 12 years. The remaining patients have remained asymptomatic and required no additional procedures.

DISCUSSION

TOS-A occurs because the subclavian artery is repeatedly compressed in the scalene triangle at the level of the first rib and in the costoclavicular space, damaging its wall.[2] Its incidence is low, affecting <5% of TOS cases.[3,4] The higher proportion of TOS-A cases in this series (21.1%) can be explained by our conservative approach for neurogenic TOS and by the fact that our institution is a national referral center for arterial pathology.[8] Most publications have described symptomatic TOS cases in active young adults with an average age of 37 years, with men and women found in similar proportions.[6,7,9,10] This distribution is similar to that found in this series, but differs from neurogenic TOS, which is considerably more frequent in women.

Table III. Arterial reconstructive procedures in accordance with pathologic findings of subclavian artery

Subclavian aneurysm	7 cases
Resection and subclavian–axillary bypass	7
With vein	4
With PTFE	3
Subclavian stenosis	4 cases
Carotid–axillary bypass	2
With vein	1
With PTFE	1
Carotid–subclavian bypass	1
With PTFE	
Subclavian–axillary bypass	1
With vein	
Subclavian occlusion	2 cases
Carotid–axillary bypass	2
With PTFE	
Distal embolism	7 cases
Embolectomy	4
Remote	1
Local	3
Thrombolysis	3
Upon hospital admission	2
During surgery	1
Anticoagulation	3
Distal bypass with vein	2

PTFE, polytetrafluoroethylene.

Repetitive trauma and chronic compression of the subclavian artery damages its wall and can cause local stenosis or poststenotic dilation and/or thrombosis.[7] The most frequent trauma is caused by abnormal bone structures, most commonly cervical ribs, which are present in two thirds of TOS-A cases[6,9,10] and were present in 50% of the TOS-A cases of this series. Muscular hypertrophy[11] or compression at the humeral head level are more common in competitive athletes and were not present in this series.

Arterial wall damage becomes progressive: initially, an intimal lesion is responsible for stenosis and poststenotic dilation, which progresses to a fusiform aneurysm with mural thrombosis that can eventually progress to local occlusion or distal embolization from the aneurysm or from a stenosis. Embolization and secondary ischemia occurred in all the aneurysms of this series and in a complicated stenosis case. Subclavian aneurysms are present in half of the patients diagnosed with TOS-A,[7,10] but they correspond only to 1.2% of all the ischemic syndromes affecting the upper limb.[9] They must be specifically searched in patients with cervical ribs, as they are frequently complicated by ischemia. In this series, 5 of 7 cases (71.4%) of subclavian aneurysms were associated with a cervical rib.

A careful clinical assessment is essential to suspect TOS-A. The spectrum of clinical presentations is broad, ranging from asymptomatic patients with compensatory collateral circulation to critical ischemia in patients with aneurysm or stenosis with distal embolization.[12] It is necessary to stress the high prevalence of critical ischemia in this and other published series.[6,7,10] Intermittent claudication is an intermediate presentation in patients with significant stenosis or subclavian occlusion.[6] Moreover, the high frequency of critical ischemia observed in this series (40%) is similar to that seen in earlier studies,[6,7,10] with arterioarterial embolization from subclavian aneurysms being the main and most serious cause of critical ischemia—as embolism of the digital arteries may lead to early necrosis. Retrograde embolization toward the carotid or vertebral arteries has been described less frequently[13] and was not present in the current series.

A detailed physical examination may help diagnose TOS-A; the presence of a pulsatile mass in the supraclavicular space, the palpation of a cervical rib, and the absence of other embolic sources, such as atrial fibrillation or heart valvulopathy, suggest TOS-A as the etiology of an upper limb ischemia. The detection of supraclavicular murmurs increases the chance of diagnosing arterial stenosis and the risk of subsequent arterioarterial embolism, and decreases the possibility of an embolism from a different origin.

According to previous publications, the presence of Adson's sign (disappearance of radial pulse when raising the arm, with contralateral cervical rotation, hyperextension, and deep inhalation) is reported in 10–20% of the asymptomatic population.[14] Clinical assessment must be supplemented with an arterial pulse volume recording or Doppler ultrasonography, and segmentary pressure measurement under baseline conditions and during the outlet compression maneuvers to confirm diagnosis and localize the embolic obstruction level. Imaging studies should be performed in all patients with TOS and ischemia and/or rib anomalies.[7] Duplex scanning may help diagnose aneurysms or stenosis of the subclavian artery, although adequate exposure is limited by the clavicle.[5] CTA scans in the arterial phase[15] and magnetic resonance angiogram (MRA)[16] with thoracic outlet protocols are more helpful for diagnosis, as they identify the site and cause of arterial damage along with the presence of associated emboli. Traditionally, angiography has been the ''gold standard'' and still can be useful

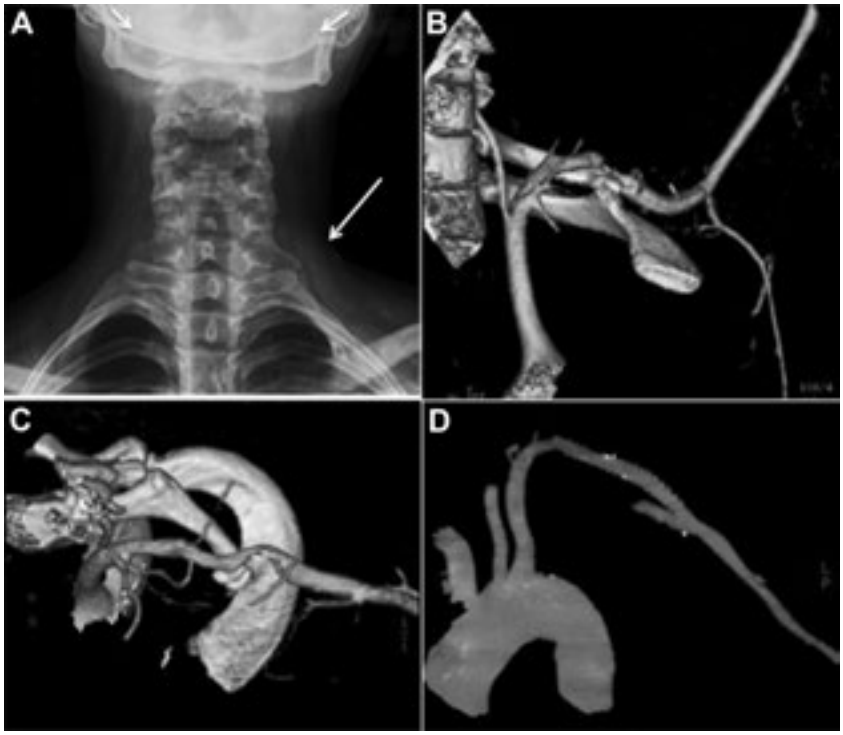

Fig. 4. Imaging studies of a 33-year-old woman with critical ischemia in her left upper extremity. **(A)** A cervical rib can be observed on an anteroposterior cervical X-ray (long white arrow). Using 3-dimensional reconstruction images of computed tomographic angiography, the **(B)** anteroposterior and **(C)** cephalic views reveal compression of the subclavian artery by the left cervical rib with critical reduction of its lumen. The patient underwent surgery with cervical and first rib resection, and arterial repair with subclavian–axillary interposition of a ring-reinforced 6-mm polytetrafluoroethylene graft, which was patent and without lesions on follow-up tomography **(D)**.

when the diagnosis is unclear,[12] if there is need for assessment of forearm and hand arteries (noninvasive studies are less reliable), and, eventually, to plan surgery or to perform intraarterial thrombolysis. Angiography presents well-known morbidity and diagnostic limitations in cases of aneurysm with intraluminal thrombi,[9] and CTA and MRA are good complementary tools. Finally, bone lesions should be searched using the aforementioned methods. Noninvasive imaging studies should be used liberally in TOS, particularly CTA, which has shown a higher accuracy for detecting TOS-A.[6]

Treatment is indicated in asymptomatic TOS patients with a proven arterial lesion and in all symptomatic patients. It involves surgery and implies decompressing the thoracic outlet, repairing the arterial lesion, and/or treating critical ischemia (embolism). Decompression consists of resecting the first and/or cervical rib, division of the anterior scalene muscle, and eventual fibrous bands.[10] It is always recommended to resect the first rib given the high concomitance of neurogenic TOS (up to 30% of patients present with associated neurologic symptoms[7]) and to avoid compression when bypass revascularization is needed, which occurred in 1 case in our series. Revascularization treatment must be adjusted to the pathologic findings; in cases with arterial aneurysm it is important to get complete resection and to perform a terminal to lateral bypass or interposition graft, as was used in this series. Common carotid or ipsilateral subclavian arteries are the preferred inflow vessels, and an autologous vein is the preferred conduit.[9] Moreover, in cases of symptomatic complete occlusion of the subclavian artery, revascularization procedures should be performed to avoid retrograde thrombosis and the subsequent risk of cerebral infarction[13]; in our series, extra-anatomic reconstructions were performed in such cases with a carotid–subclavian bypass.

Secondary embolism was treated similarly to the way described in other series. Fogarty catheter thrombectomy, direct local thrombectomy, anticoagulation, or perioperative thrombolysis was done for recent embolization, whereas chronic embolisms with barely developed compensatory collateral

circulation required a distal bypass with autologous vein.[7] In this series complementary treatments, such as cervicothoracic sympathectomy (CS), were not necessary although CS may be indicated for chronic embolism with unsatisfactory revascularization.[7] Finally, for the 6 symptomatic patients who presented with no apparent arterial wall lesions on the imaging studies, isolated decompression surgery was performed, and it was not necessary to add any revascularization techniques.

The results of the surgically treated TOS-A reported in this series are similar to those published earlier, with symptoms being resolved in >90% of cases and with long-term bypass patency in 90−100% of cases.[7,9] In these series, mortality was not described and surgical morbidity was low (7%),[7] mainly pneumothorax, hemothorax, chylothorax, transitory brachial neuropathy, and vascular injuries.

Contemporary findings have indicated endovascular treatment for TOS-A in isolated cases,[17] mainly aneurysms,[18−22] with the need for decompression to avoid stent fractures, restenosis, and arterial thrombosis,[23] and with unknown long-term patency rates. These drawbacks, along with the compressive nature of the etiologic mechanism, make conventional surgery the first alternative for revascularization.

In conclusion, a careful search of specific signs in a rigorous physical examination and cervical X-rays looking for cervical ribs in all TOS patients allows us to detect TOS-A before the development of ischemic complications. When TOS-A is suspected, arterial imaging studies can lead to early diagnosis and treatment. Likewise, when faced with acute ischemia of the upper limb, TOS must be identified as separate from other more frequent diagnoses, such as an embolism of cardiogenic origin, which is treated differently. Although an endovascular approach has major limitations, open surgery with outlet decompression and eventual revascularization offers a definitive cure for the vast majority of patients with TOS-A.

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