Imaging of the Patient with Thoracic Outlet Syndrome[1]

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Abbreviations: GRE = gradient-echo, MIP = maximum intensity projection, TOS = thoracic outlet syndrome

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe the anatomy of the thoracic outlet and the pathophysiology involved in development of TOS.
- Recognize the imaging appearance of vascular forms of TOS.
- Identify complications of thoracic outlet decompression in relation to the timing of presentation.

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Patients with symptoms from compression of the neurovascular bundle in the thoracic outlet are described as having thoracic outlet syndrome (TOS), which is best thought of as three conditions classified according to which structures are involved. The purpose of this article is to review the role of imaging in evaluation of patients with TOS, beginning with diagnosis and extending through postoperative management. While diagnosis of TOS still rests on the patient's presenting history and physical examination, imaging examinations are helpful in supporting the diagnosis, delineating abnormal anatomy, determining which structures are compressed, identifying the site of compression, and excluding other diagnoses. Magnetic resonance imaging is the noninvasive imaging modality of choice in evaluating patients with suspected TOS, but computed tomography also plays an important role, particularly in delineating bone anatomy. Evidence of vascular damage is required to make the diagnosis of TOS at imaging. Dynamic compression of the axillosubclavian vessels at the thoracic outlet can be a finding supportive of the diagnosis of TOS but is not a stand-alone diagnostic criterion, as it can be seen in patients without TOS. As diagnosis and treatment of TOS increase, radiologists will increasingly encounter the TOS patient after decompression surgery. Recognition of the expected postoperative appearance of these patients is critical, as is an understanding of the imaging findings of potential short- and long-term complications.

Introduction

The thoracic outlet extends from the cervical spine and superior border of the mediastinum to the lateral border of the pectoralis minor muscle. The brachial plexus, subclavian vein, and subclavian artery traverse the thoracic outlet, and when compressed, can result in upper extremity symptoms. Patients with symptoms from compression of the neurovascular bundle in the thoracic outlet are described as having thoracic outlet syndrome (TOS), which is best thought of as three conditions classified according to which structures are involved: neurogenic TOS (from compression of the brachial plexus nerves), venous TOS (from compression of the subclavian vein), and arterial TOS (from compression of the subclavian artery).

Owing to lack of objective reporting criteria and inconsistent reporting standards, the incidence and prevalence of TOS are poorly understood. However, TOS is an important clinical entity because it typically affects younger individuals and can have a significant effect on a working cohort, with disability and lost productivity if unrecognized and untreated. As of 2008, approximately 2000–2500 first rib resections were performed in the United States for TOS, mostly at referral centers by vascular or thoracic surgeons (1–3).

TEACHING POINTS

- TOS is best thought of as three separate conditions, depending on the structure causing the symptoms: neurogenic TOS, venous TOS, and arterial TOS.
- Although the main cornerstones of diagnosis are the patient's presenting history and physical examination, imaging studies can be helpful to confirm the diagnosis or site of involvement, delineate abnormal anatomy, evaluate for other potential causes of the patient's symptoms, and appropriately classify the patient's condition as neurogenic TOS, arterial TOS, or venous TOS.
- It is critically important to recognize that the diagnosis of vascular TOS is not made by identifying positional changes in the vessel caliber alone. Both symptomatic and asymptomatic patients can have arterial and/or venous compression at the thoracic outlet at cross-sectional imaging. Venous compression is much more common, however, and occurs in over 50% of asymptomatic patients.
- MR imaging is the noninvasive cross-sectional imaging test of choice in patients with suspected TOS. Given that the evaluation for positional narrowing requires imaging acquisitions in multiple positions, MR imaging has an inherent advantage over CT due to its lack of ionizing radiation, an advantage of particular benefit in the generally young patient population affected by TOS.
- Many surgeons will intentionally violate the apical pleura to provide a means for decompression of postoperative fluid into the pleural space. Thus, small or even moderate pleural effusions or a small pneumothorax are not unexpected findings.

While the diagnosis of TOS typically rests on a careful history and physical examination, imaging can play an important role in identifying the underlying cause and supporting the diagnosis. Imaging can also exclude other conditions or provide alternative explanations for the patient's symptoms. The purpose of this review is to explore the use of imaging in the patient with TOS. We focus not only on the role of imaging in diagnosis of TOS but also on evaluation of patients after thoracic outlet decompression surgery, with a review of important complications that need to be recognized promptly. Our experience is drawn from a large cohort of patients seen at a referral center for patients with TOS.

Anatomy of the Thoracic Outlet

Understanding the anatomy and compartments of the thoracic outlet is essential for accurate reporting of the location of disease. The thoracic outlet extends from the cervical spine and superior border of the mediastinum to the lateral border of the pectoralis minor muscle. Anatomically, the thoracic outlet can be divided into three compartments.

The most medial compartment, the scalene triangle, lies above and behind the clavicle. The scalene triangle is bordered by the middle scalene muscle posteriorly, the anterior scalene anteriorly, and the first rib inferiorly. The scalene triangle contains only the subclavian artery and brachial plexus; as the subclavian vein lies anterior to the anterior scalene muscle, it is thus outside the triangle.

Moving laterally, the costoclavicular space is bordered by the subclavius muscle anteriorly, the clavicle superiorly, and the first rib and anterior scalene muscle inferiorly and posteriorly. The costoclavicular space contains the entire neurovascular bundle. The most lateral compartment of the thoracic outlet lies inferior to the clavicle and is known as the subcoracoid pectoralis minor space. In the pectoralis minor space, the neurovascular bundle courses between the pectoralis minor muscle tendon anteriorly and the ribs and intercostal muscles posteriorly (2,4,5).

The anatomy of the thoracic outlet is dynamic, with abduction of the ipsilateral arm potentially resulting in narrowing of the thoracic outlet at all three spaces. This narrowing can be seen in symptomatic and asymptomatic patients and plays a role in development of neurovascular compression in patients with TOS (4–8) (Fig 1).

Thoracic Outlet Syndrome

TOS is the constellation of symptoms produced when the neurovascular structures that traverse the thoracic outlet are compressed. TOS is best thought of as three separate conditions, depending on the structure causing the symptoms: neurogenic TOS, venous TOS, and arterial TOS (2,9,10). The symptoms produced by compression of the components of the neurovascular bundle as it passes through the thoracic outlet are variable, and some patients may have compression of one or more of these structures.

Anatomic abnormalities, such as cervical ribs, are often present in patients with TOS but do not define TOS, as many patients without TOS may have these abnormalities as well (11–14). Most patients with TOS also have a history of trauma or a repetitive movement as part of a job or activity. Thus, TOS is a product of an anatomic predisposition and an exacerbating factor (2,9). TOS is more common in patients less than 40 years old. While some authors report a greater incidence in women, our experience has been a male-to-female ratio of near 1:1 (2,9).

Neurogenic TOS is typically thought to account for over 90% of cases of TOS, but this may be an overestimate due to referral bias (4). The most common cause of neurogenic TOS is a hyperextension neck injury with a whiplash mechanism, but falls on an outstretched arm or repetitive motion injuries from work or sporting activity may also cause neurogenic TOS (9,15). Anatomic abnormalities, including hypertrophied scalene musculature, bone abnormalities, and fibrous

bands, are also often present and likely predispose to development of neurogenic TOS (16).

The classic patient with neurogenic TOS has shoulder, neck, head, chest, or arm pain or paresthesias, especially with arm activity, elevation, or dangling. The location of the pain depends on the level at which the brachial plexus is compressed. Some patients may demonstrate Raynaud phenomenon–like symptoms of hand coldness, finger swelling, or color changes, which can overlap with features of arterial TOS. It should be emphasized that the Raynaud phenomenon–like symptoms seen in patients with neurogenic TOS are due to an overactive sympathetic nervous system and not ischemia (10).

At examination, the patient is typically tender to palpation in the supraclavicular scalene triangle and/or at the subcoracoid pectoralis minor tendon. The end stage of neurogenic TOS can result in the Gilliatt-Sumner hand, in which there is atrophy of the hand muscles, as well as hypesthesia in the ulnar and medial antebrachial cutaneous distributions with normal median nerve sensation (2,9).

Venous TOS is the second most common cause of TOS. The typical site of subclavian vein compression is at the level of the costoclavicular space between the first rib, subclavius muscle, and clavicle. Most patients present with spontaneous axillosubclavian thrombosis (also known as "effort thrombosis" or Paget-Schroetter syndrome), although some can present with symptoms of intermittent venous compression in the absence of thrombosis (McCleery syndrome) (2,17,18).

The mechanism that predisposes patients to venous TOS is the anterior-based costoclavicular shears mechanism, which is present in normal individuals at the extreme of abduction. Bone anatomy is frequently normal, but there is often surrounding muscular hypertrophy due to a sporting or occupational activity. These hypertrophied muscles can result in narrowing of the costoclavicular space and raise the potential for repetitive damage to the vein, which can lead to wall thickening, fibrosis, and thrombosis. A high frequency of subtle or occult medial first rib fractures has also been noted in venous TOS, especially in patients over 30 years of age (19).

The classic presentation of a patient with axillosubclavian thrombosis is rapid onset of discoloration, swelling, congestion, and pain. Collateral vessels can form and be engorged across the shoulder and lateral pectoral areas. There may be associated paresthesias or weakness, but the overall clinical presentation is usually very different from that of neurogenic TOS. The majority of patients with axillosubclavian thrombosis will report a history of recent vigorous upper extremity use with development of symptoms the next day. There may be an additional history of "herald" events with symptoms suggestive of previous subocclusive thrombus and resolution. Pulmonary emboli may be seen in up to 10% of patients with occlusive venous TOS (17).

Arterial TOS is the rarest form of TOS, accounting for less than 3% of cases. Arterial TOS consists of two components: damage to the subclavian artery at the level of the first rib and distal embolic phenomena. Arterial TOS is almost always associated with underlying bone abnormalities, either a cervical rib, anomalous first rib, or first rib or clavicle fracture (2,9,16,20).

The classic patient with arterial TOS presents with symptoms of distal ischemia of the upper extremity including weakness, a cold limb, and pain. Some patients may have an asymptomatic pulsatile mass in the supraclavicular space due to an underlying aneurysm. Rarely, retrograde embolism can take place, resulting in a cerebrovascular event (21,22). Most patients with arterial TOS are young and otherwise healthy and report a history of vigorous use of the arm in occupational or recreational activities. The pain may be restricted to exercise or overhead positioning due to collateralization.

Imaging in Diagnosis of TOS

The radiologist plays an important role in initial evaluation of patients with suspected TOS. Although the main cornerstones of diagnosis are the patient's presenting history and physical examination, imaging studies can be helpful to confirm the diagnosis or site of involvement, delineate abnormal anatomy, evaluate for other potential causes of the patient's symptoms, and appropriately classify the patient's condition as neurogenic TOS, arterial TOS, or venous TOS (23). Chest radiography is typically performed in all patients with suspected TOS, as it can help delineate bone abnormalities. In patients with suspected vascular TOS, either angiography or cross-sectional imaging with MR imaging or CT is often performed, depending on the acuity of the presentation and potential need for immediate intervention. Ultrasonography (US) can also be used to evaluate patients with suspected vascular TOS, but in our practice, US is recognized to have many limitations and is typically reserved for the clinic setting.

Diagnosis of vascular TOS with imaging studies requires the presence of findings of vascular damage. For venous TOS, this involves identification of axillosubclavian vein thrombosis, focal fixed subclavian vein stenosis at a site of dynamic compression, or expanded collateral vessels. In the setting of arterial TOS, supportive

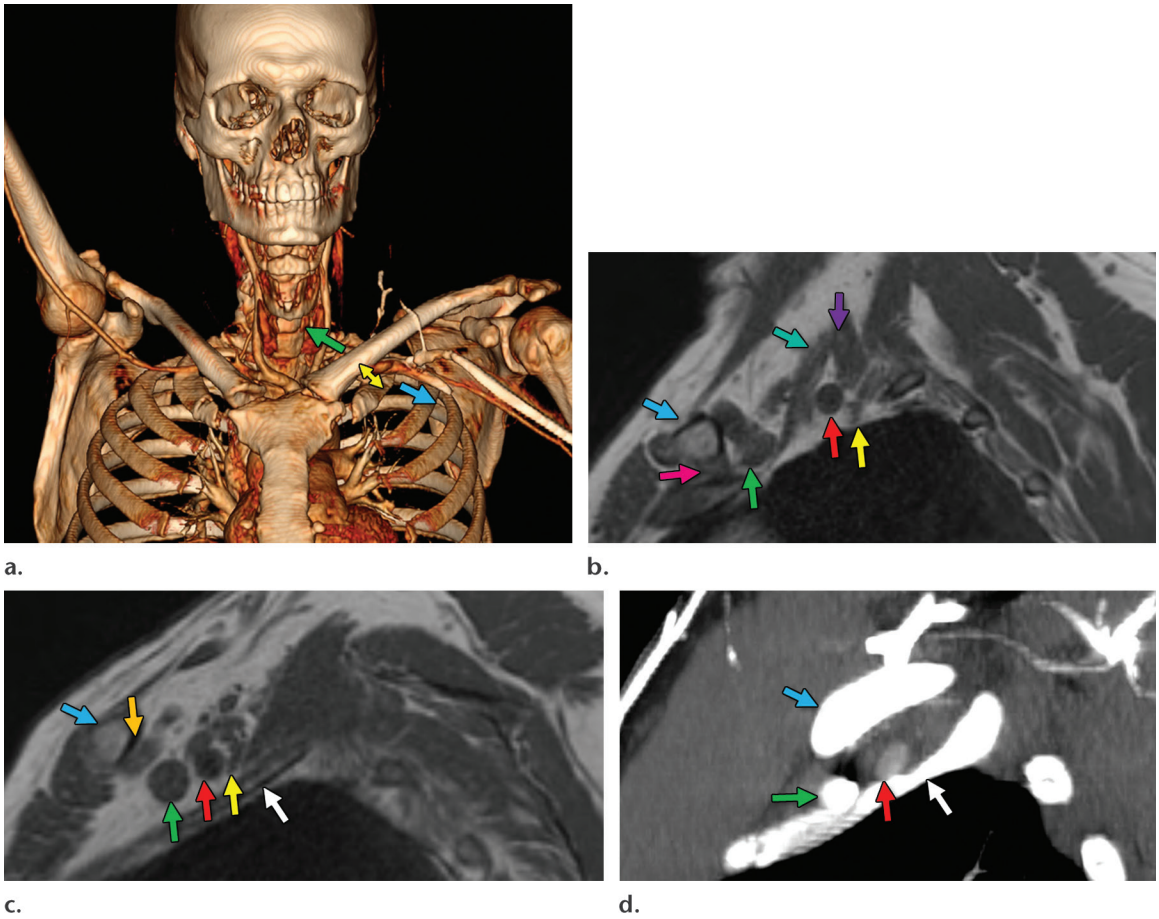


Figure 1. Cross-sectional anatomy of the thoracic outlet at computed tomography (CT) and magnetic resonance (MR) imaging. **(a)** Volumetric CT reconstruction shows the locations of the three compartments of the thoracic outlet. The scalene triangle lies above the clavicle (green arrow). Moving laterally, the costoclavicular space lies between the clavicle and first rib (double-headed yellow arrow). The most lateral compartment, the pectoralis minor space, lies inferior to the clavicle (blue arrow). **(b)** Sagittal T1-weighted MR image at the level of the scalene triangle with the arms adducted shows key structures at this level, including the middle scalene muscle (purple arrow), anterior scalene muscle (teal arrow), clavicle (blue arrow), costoclavicular ligament (pink arrow), subclavian vein (green arrow), subclavian artery (red arrow), and brachial plexus (yellow arrow). **(c)** Sagittal T1-weighted MR image at the level of the costoclavicular space with the arms adducted shows the clavicle (blue arrow), subclavius muscle (orange arrow), subclavian vein (green arrow), subclavian artery (red arrow), brachial plexus (yellow arrow), and first rib (white arrow). **(d)** Sagittal CT image of a different patient at the level of the costoclavicular space with the arms abducted shows how the clavicle (blue arrow) moves posteriorly over the neurovascular bundle, resulting in crowding of these structures. Green arrow = subclavian vein, red arrow = subclavian artery, white arrow = first rib.

findings include axillosubclavian arterial aneurysm or pseudoaneurysm, arterial thrombus, focal fixed subclavian artery stenosis at a site of dynamic compression, distal emboli, or enlarged collaterals (2). Separate image acquisitions are often performed with the patient's arms adducted and abducted (with external rotation) to elicit positional vascular narrowing, as this can affect surgical management by identifying the precise location of vascular compression.

It is critically important to recognize that the diagnosis of vascular TOS is not made by identifying positional changes in the vessel caliber alone. Both symptomatic and asymptomatic patients can have arterial and/or venous compression at the thoracic outlet at cross-sectional imaging. Venous compression is much more common, however, and occurs in over 50% of asymptomatic patients (7,24,25). Similar findings have been reflected in US studies of normal patients, which have demonstrated variability of vessel caliber and flow patterns in the axillosubclavian vessels with positional changes (26–28). Labeling patients with vascular compression alone as having TOS can lead to misdiagnosis and potentially overtreatment with surgical intervention (Fig 2).

Use of MR imaging for diagnosis of patients with neurogenic TOS has also been described, but it has not yet been shown to affect management of patients with suspected neurogenic TOS (2,4,7,29,30). Findings at MR imaging that can be suggestive of neurogenic TOS include edema in the brachial plexus or loss of fat surrounding

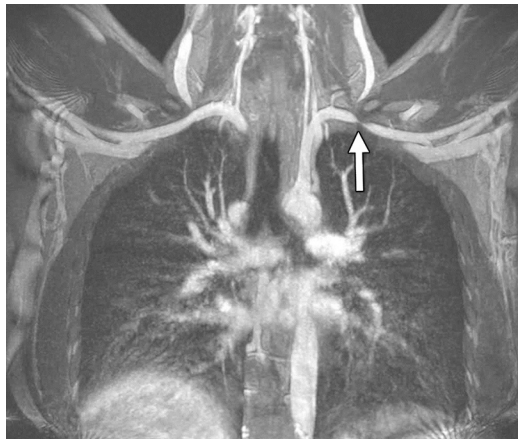


Figure 2. Isolated vascular narrowing at MR imaging. Coronal venous phase gradient-echo (GRE) image with the arms abducted shows narrowing of the left subclavian artery (arrow) at the costoclavicular interval. The patient had normal left subclavian artery caliber with the arms adducted. There was no evidence of vascular damage or thrombus, and the patient had clinical evidence of neurogenic TOS but not arterial TOS. In cases such as this, where there is isolated vascular narrowing, we report the dynamic vascular compression, but do not label the patient as having vascular TOS to prevent overdiagnosis and possible unnecessary treatment.

Table 1: Imaging Findings in TOS

Venous TOS
Bone and soft-tissue anatomic abnormalities
Axillosubclavian vein thrombosis
Enlarged collaterals
Fixed axillosubclavian vein stenosis at site of dynamic narrowing
Axillosubclavian vein narrowing with abduction*
Arterial TOS
Bone and soft-tissue anatomic abnormalities
Axillosubclavian artery aneurysm or pseudoaneurysm
Arterial thrombus
Distal emboli
Enlarged collaterals
Fixed axillosubclavian artery stenosis at site of dynamic narrowing
Axillosubclavian artery narrowing with abduction*
Neurogenic TOS
Bone and soft-tissue anatomic abnormalities
Loss of fat about brachial plexus with abduction†
Edema in brachial plexus†
Diagnosis typically made clinically without imaging

*Not sufficient as a stand-alone criterion for diagnosis of TOS.
†MR imaging findings that can be suggestive of neurogenic TOS.

the brachial plexus with abduction. Patients with suspected neurogenic TOS still often undergo plain radiography or cross-sectional imaging examinations to evaluate for anatomic abnormalities or concurrent vascular TOS. A summary of the imaging findings seen in the setting of TOS is given in Table 1.

The underlying cause of symptoms of neurogenic or vascular TOS is not always compression by bone and soft-tissue structures about the thoracic outlet. TOS should remain a diagnosis of exclusion. Superior sulcus lung malignancies, which are a prime concern in older patients with TOS symptoms and a smoking history, can invade the thoracic outlet and produce symptoms of neurovascular compression. Superior sulcus tumors can often be identified at chest radiography, CT, or MR imaging. Other tumors, including lipomas or neurogenic tumors in the supraclavicular region, can also produce symptoms that overlap with those of TOS (31,32).

Primary conditions affecting the vasculature can produce symptoms similar to those of TOS. A large-vessel vasculitis such as Takayasu arteritis can manifest as intermittent arm and/or hand pain and simulate embolic phenomena from TOS. At imaging studies, Takayasu arteritis will typically manifest as areas of vascular wall thickening with regions of vascular stenosis or dilatation. The pulmonary arteries may be involved (33,34).

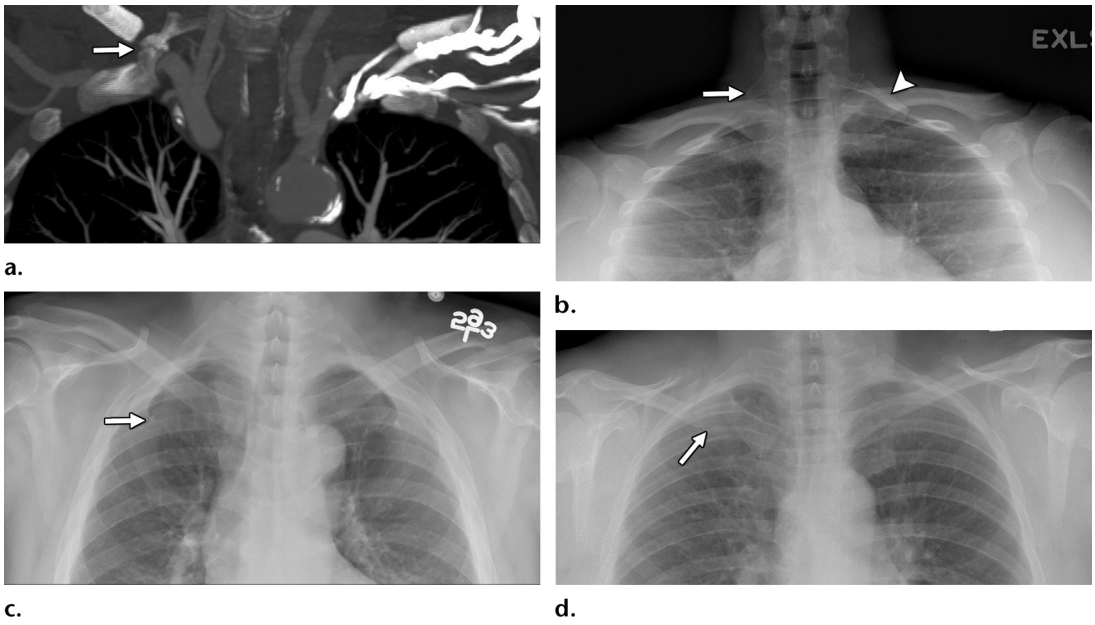


Figure 3. Bone abnormalities that can be associated with TOS. **(a)** Coronal maximum intensity projection (MIP) from CT shows a complete right cervical rib that forms an articulation with the right first rib (arrow). The patient was found to have incidental narrowing of the right subclavian artery due to the cervical rib on this pulmonary embolism protocol CT image, underscoring that not all patients with vascular narrowing at imaging have TOS. **(b)** Lordotic frontal chest radiograph shows an elongated C7 transverse process on the right (arrow) and an incomplete cervical rib on the left (arrowhead). **(c)** Frontal chest radiograph shows bilateral anomalous first ribs (arrow) that articulate with the second ribs. **(d)** Radiograph of a patient with a clinical diagnosis of neurogenic TOS shows an abnormal right clavicle (arrow) due to a bulky ununited fracture.

Finally, patients with severe atherosclerotic disease can have symptoms of upper extremity weakness or embolic phenomena that overlap with those of TOS. The key distinguishing factors in these cases are to determine that the occlusion or plaque is not at the thoracic outlet and is not directly associated with vascular compression. It is incumbent on the radiologist to identify these alternative explanations for symptoms simulating those of TOS, as they may not be suspected clinically.

Conventional Radiography

Conventional radiography should be performed in all patients with suspected TOS and serves as a low-cost means of identifying a bone anatomic abnormality (23). Even if a patient with suspected TOS is scheduled to undergo MR imaging, conventional radiography is still indicated, as cervical ribs and other bone abnormalities can be missed at MR imaging (35). In addition, conventional radiography allows evaluation for other causes of right upper extremity symptoms, including an underlying tumor. The major bone abnormalities that are relevant to TOS and can be detected at plain radiography include cervical ribs, elongated C7 transverse process, anomalous first rib, and abnormal first rib or clavicle (Fig 3).

Cervical Ribs.—Cervical ribs occur in up to 6% of individuals and are twice as common in women. Cervical ribs can be classified as complete or incomplete (2,4,12,36–38). Complete cervical ribs, which account for 30% of cervical ribs, attach to the normal first rib by fusion or by forming a joint. The more common incomplete cervical ribs can be of varying length, but usually have a thick ligament extending from their tip to the first rib. Cervical ribs lie in the plane of the middle scalene muscle and make the scalene triangle narrower than in normal individuals.

Most people with cervical ribs are asymptomatic, but an increased prevalence of cervical ribs has been noted in patients with TOS (4,11,12). In the context of the patient with suspected TOS, cervical ribs often need to be resected if decompression surgery is performed. The best way to identify cervical ribs at radiography or CT is to identify the contralateral first rib and confirm that the cervical rib arises from the C7 transverse process. In the case of bilateral cervical ribs, counting the total number of inferior ribs may also be helpful.

Elongated C7 Transverse Process.—An elongated C7 transverse process is defined as extending laterally beyond the transverse process of T1. Elongated C7 transverse processes are more common than cervical ribs, occurring in up to 23% of people, depending on the population studied (37,39,40). While elongated C7

transverse processes and short incomplete cervical ribs may be difficult to distinguish and have a common embryologic pathway for development, elongated C7 transverse processes do not demonstrate a costovertebral articulation while cervical ribs do. Similar to incomplete cervical ribs, an elongated C7 transverse process can be a causative factor in TOS due to a ligamentous band extending from its apex to the first rib. As with cervical ribs, most patients with elongated transverse processes are asymptomatic, although there is an increased incidence of elongated C7 transverse processes in patients with TOS (41).

Anomalous First Rib.—Anomalous first ribs, sometimes referred to as hypoplastic first ribs, are first ribs that articulate with the second rib rather than the sternum. Anomalous first ribs are often thinner, tend to lie more cephalad than normal first ribs, and may have a more vertical "J" shape in comparison with a normal first rib. Anomalous first ribs can cause narrowing at the thoracic outlet due to associated fibrous bands or a bulky articulation with the second rib (20,42–44).

Abnormal First Rib or Clavicle.—In the context of a patient with possible TOS, an abnormal first rib or clavicle is defined as having either an old fracture or exostosis. Old fractures can often heal with bulky callus or with offset alignment that can narrow the thoracic outlet and result in vascular or nerve compression. Nonunited fractures of the first rib or clavicle can also often have extensive surrounding callus or heterotopic ossification. An abnormal first rib or clavicle in a patient with suspected TOS is often removed with decompression surgery (19,45,46).

Ultrasonography

In the patient with suspected TOS, US allows portable and noninvasive imaging without use of ionizing radiation. US also allows imaging of the patient in any body position (sitting, standing) or degree of arm abduction. At our institution, its main use is in the clinic setting, where a US evaluation of the axillosubclavian vessels can be performed to confirm patency during an outpatient visit. One important limitation of US for TOS is its limited acoustic window; specifically, it may be difficult or impossible to directly image the costoclavicular interval in many patients. US is also operator dependent and can be technically challenging in muscular patients or those with extensive adipose tissue.

The findings of vascular occlusion at US include increased echogenicity of the vascular lumen on gray-scale images, lack of Doppler signal, and noncompressibility. Vascular collaterals may be identified with US; in the setting of chronic thrombosis, care must be taken to not misidentify a large collateral as the subclavian vein or artery. Owing to limited visualization of the central subclavian vein, US should not be used to exclude the presence of venous TOS when there is clinical suspicion.

An abrupt change in vessel contour should raise suspicion for vascular damage, particularly when arterial, as it could represent a poststenotic aneurysm or pseudoaneurysm. As discussed earlier, positional changes in vascular caliber or abnormal or even absent flow can be seen in the axillosubclavian vessels in normal patients and should not alone trigger a diagnosis of vascular TOS (26–28).

Catheter Angiography

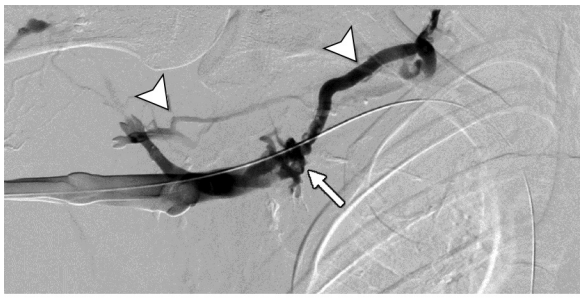
In the era of CT and MR angiography, catheter angiography is still useful in initial management of patients with acute symptoms or known thrombosis. In patients with venous TOS who present with acute axillosubclavian thrombosis, catheter venography allows treatment via infusion of a thrombolytic agent or pharmacomechanical thrombolysis. Indeed, thrombolysis is the usual first step in management of patients with venous TOS before definitive surgical treatment.

While thrombolysis is typically effective and demonstrates success rates of 62%–84%, success rates can approach 100% in patients who present within 2 weeks of the onset of symptoms (47–49). Stent placement is not recommended, as stent placement alone without surgical intervention generally fails and the presence of a stent can limit options for vein reconstruction after stent failure (50,51). Thrombolysis alone is not adequate to treat patients with venous TOS, as a majority of patients who do not undergo definitive surgical decompression will demonstrate persistent symptoms or recurrent thrombosis (52,53).

Catheter angiography can also be used in patients with findings of underlying arterial thrombosis or distal ischemia (Fig 4). Again, thrombolysis with an infusion catheter or pharmacomechanical thrombolysis can be performed before definitive decompression surgery. Angiography is also useful in postoperative care of TOS patients, as it can be used to evaluate for residual stenoses that can be managed with angioplasty or, in cases refractory to angioplasty, with stent placement.

MR Imaging

MR imaging is the noninvasive cross-sectional imaging test of choice in patients with suspected TOS (23). Given that the evaluation for positional narrowing requires imaging acquisitions in multiple positions, MR imaging has an inherent advantage



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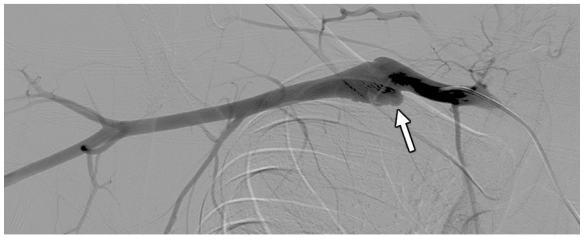

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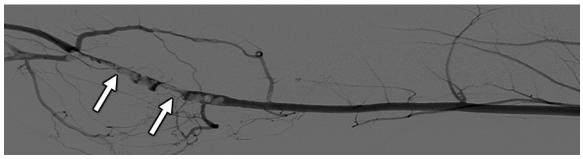


c.

Figure 4. Angiography of patients with vascular TOS. **(a)** Digital subtraction image from a right-sided venous injection in a baseball player with acute right arm swelling shows abrupt occlusion of the right subclavian vein (arrow) with filling of venous collaterals proximal to the site of obstruction (arrowheads). These findings are consistent with venous TOS, and the patient went on to thrombectomy followed by definitive decompression surgery. **(b)** Digital subtraction image from selective injection of the right subclavian artery in a 28-year-old patient with ischemic ulcers of the right digits shows a lobular pseudoaneurysm of the right subclavian artery (arrow). **(c)** Digital subtraction image of the downstream brachial artery in the same patient shows evidence of embolic occlusion (arrows). The patient also had occlusion of multiple digital arteries (not shown). These findings are consistent with arterial TOS, as there is direct evidence of trauma to the subclavian artery resulting in an aneurysm with associated emboli seen in distal vessels.

Table 2: MR Imaging Protocol for TOS

Phased-array body coil, center at aortic arch
Contrast material
Preferred: single dose, single injection of gadofosveset trisodium at 1 mL/sec
Alternate: split dose of extracellular agent at 2 mL/sec*
Place intravenous line in arm contralateral to symptoms
Imaging protocol
1. Variable scout with arms adducted
2. Coronal high-resolution T1W TSE
3. Axial high-resolution T1W TSE
4. Sagittal high-resolution T1W TSE
5. Precontrast axial 3D volume-interpolated GRE (breath-hold)
6. Precontrast coronal 3D MRA (breath-hold)
7. Test bolus: timed acquisition off aortic arch
8. Postcontrast coronal 3D MRA (breath-hold)
a. Acquisition 1 at test bolus
b. Acquisition 2 at 12-sec delay
c. Acquisition 3 at 40-sec delay
9. Postcontrast axial 3D volume-interpolated GRE (breath-hold)
10. Change patient position to arm abducted, external rotation, repeat variable scout
11. Postcontrast coronal 3D MRA (two acquisitions, breath-hold)
12. Postcontrast coronal 3D MRA (isotropic voxels, reduced voxel size)
13. Postcontrast axial 3D volume-interpolated GRE (breath-hold)
14. Optional: sagittal high-resolution T1W TSE (for assessment of brachial plexus compression)

Note.—MRA = MR angiography, 3D = three-dimensional, T1W = T1-weighted, TSE = turbo spin-echo.
*For extracellular contrast agent, split dose and perform steps 1–9 with arms adducted. Then change position to arms abducted and use second half of dose and repeat steps 4–9.

over CT due to its lack of ionizing radiation, an advantage of particular benefit in the generally young patient population affected by TOS.

Our MR imaging protocol for TOS is tailored to the goal of evaluating the vasculature and is performed with gadolinium-based intravenous contrast material. The intravenous catheter should be placed on the side opposite the symptoms to prevent T2* artifact due to concentrated gadolinium contrast material from obscuring the ipsilateral axillosubclavian vasculature. We prefer to use an intravascular or "blood pool" contrast agent (gadofosveset trisodium) in our protocol to allow only a single injection for both arm positions and the potential for higher-spatial-resolution imaging with isotropic voxels (54,55). Multiple series are performed, with the arms adducted and abducted, allowing evaluation of positional narrowing of the axillosubclavian vessels. If a blood pool agent is not available, an extracellular gadolinium-based contrast agent can be used, with the dose divided for the adducted and abducted positions.

Our MR imaging protocol for TOS is shown in Table 2. In rare cases in which an intravenous contrast agent cannot be used due to severe renal failure and the patient cannot undergo CT due to a severe allergy, a modified protocol using time-of-flight noncontrast imaging can be performed. MIP and volume-reconstructed images can be

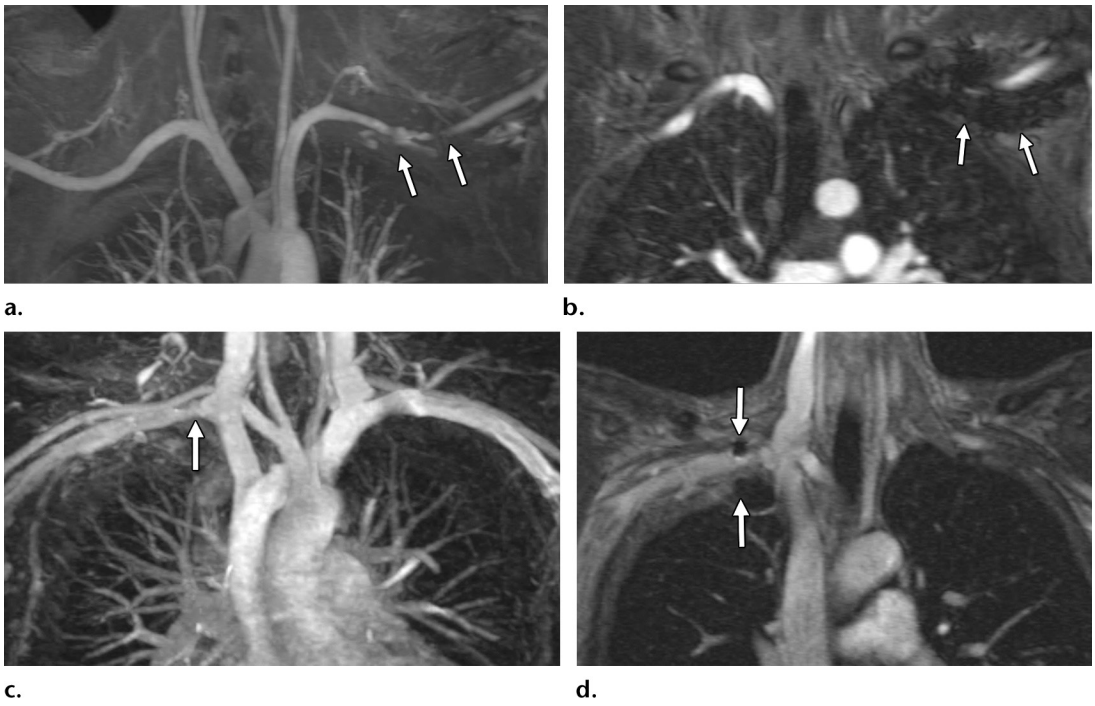


Figure 5. Technical considerations in MR imaging for TOS. **(a)** Arterial phase MIP GRE image with the arms abducted shows extensive irregularity in the mid left subclavian artery (arrows), suspicious for vascular narrowing. (The examination used an extracellular agent with a split bolus.) **(b)** Source image shows that this appearance is secondary to T2* artifact related to concentrated gadolinium contrast material in the left subclavian vein (arrows), which obscured the left subclavian artery. This case illustrates the importance of placing the intravenous catheter on the patient's asymptomatic side and of verifying findings on MIP or reconstructed images on the source images. **(c)** Coronal venous phase MIP GRE image with the arms down shows mild focal narrowing of the central right subclavian vein (arrow). **(d)** Source image shows that this narrowing is artifactual and due to T2* artifact from surgical clips (arrows), the presence of which was confirmed at plain radiography.

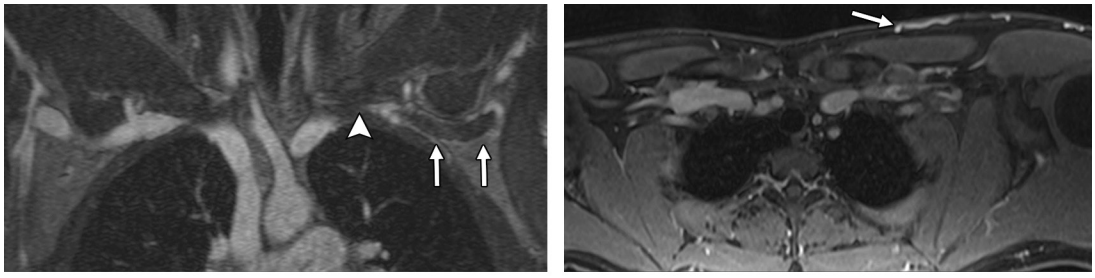
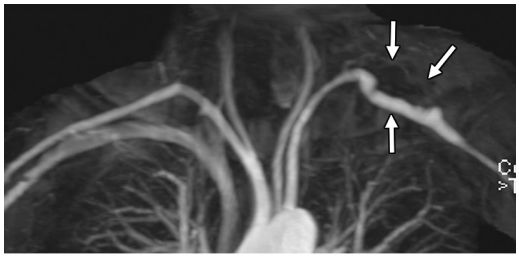


Figure 6. Venous TOS in a baseball pitcher with acute arm swelling. **(a)** Coronal GRE image with the arms abducted shows extensive left axillosubclavian vein thrombosis (arrows) and venous narrowing at the costoclavicular space (arrowhead). **(b)** Axial volume-interpolated breath-hold image shows a collateral vessel (arrow). Collateral vessels are often difficult to identify on coronal GRE images but often shown to advantage on axial images.

created as needed and are often helpful in depicting disease for referring physicians. Because of the potential for reconstruction artifacts, findings identified on postprocessed images need to be confirmed on the source images (Fig 5).

Interpretation of MR imaging results in a patient with suspected TOS should begin with a description of any potential findings of vascular damage. For venous TOS, this includes identification of thrombosis or a fixed stenosis. In cases of chronic venous TOS, a normal subclavian vein may not be identified and collateral vessels may be seen extending around its expected location (Fig 6). If venous thrombosis is found, a brief investigation of the central pulmonary vasculature is warranted to evaluate for emboli.

In arterial TOS, findings of vascular damage include arterial aneurysm or pseudoaneurysm formation with or without arterial thrombus (Fig 7). Enlarged collaterals may be present, but are less often seen than in venous TOS. Evaluation for downstream emboli in the arms should be performed even in the event that no central thrombus is seen (Fig 8).



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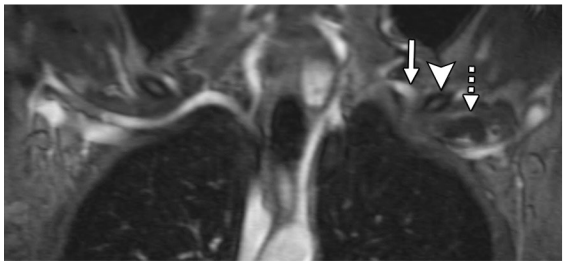

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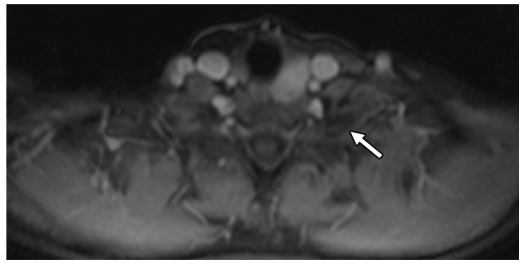
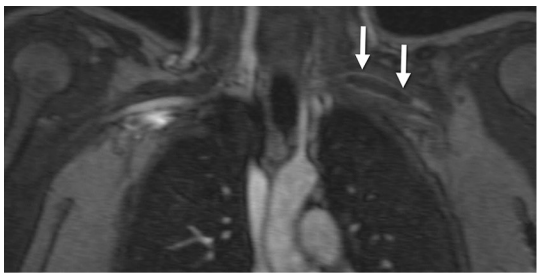


c.

Figure 7. Arterial TOS in a female patient with signs of left hand ischemia after prolonged overhead use of the left upper extremity. **(a)** Coronal arterial phase MIP GRE image with the arms adducted shows a lobular partially thrombosed pseudoaneurysm (arrows) just distal to the costoclavicular space. **(b)** Coronal GRE image with the arms abducted shows narrowing of the left subclavian artery (solid arrow) at the level of the clavicle (arrowhead) with the pseudoaneurysm seen distally (dashed arrow). **(c)** Volume-interpolated breath-hold image shows a cervical rib (arrow) as a subtle finding. Cervical ribs can be difficult to identify at MR imaging but are best identified on noncontrast T1-weighted high-resolution and axial volume-interpolated breath-hold images.

Figure 8. Arterial TOS in a patient with symptoms of left-sided arterial TOS and known bilateral cervical ribs (not shown). **(a)** Coronal venous phase GRE image with the arms adducted shows complete thrombosis of the left subclavian artery (arrows). Thrombosed vessels are often shown to advantage in the venous phase, as the vascular wall is distinguishable from the nonenhancing thrombus. **(b)** Coronal GRE image with the arms abducted shows no dynamic stenosis, underscoring the point that dynamic vascular narrowing is not required to make the diagnosis of vascular TOS at imaging. **(c)** Arterial phase MIP with the arms adducted shows the thrombus in the left subclavian artery (solid arrow) and a distal embolus in the brachial artery (arrowhead) as well as a large collateral (dashed arrow).



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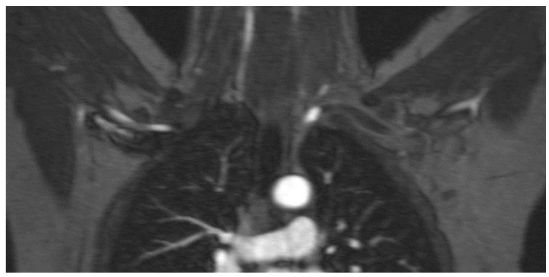

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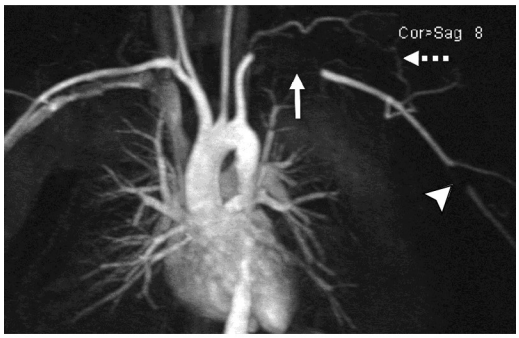


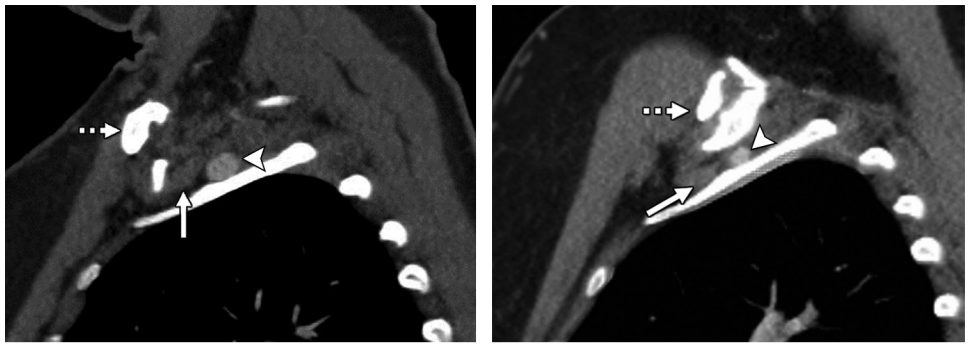
c.

Positional vascular narrowing—arterial or venous—should be reported and can be a supportive finding indicating the location of extrinsic vascular compression, but should not be used as a stand-alone criterion for diagnosing vascular TOS. Attention should also be given to any bone or soft-tissue abnormalities, including cervical ribs, abnormal first ribs, fibrous bands, and asymmetric musculature (Fig 7).

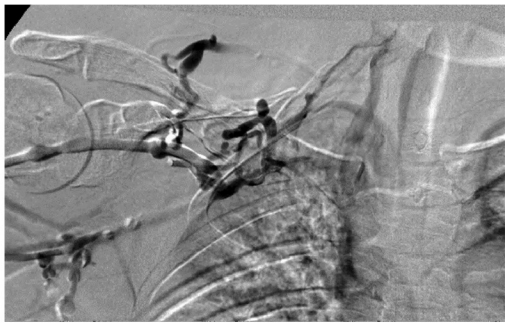
Computed Tomography

While MR imaging is the preferred imaging modality in patients with suspected TOS due to its lack of ionizing radiation, CT is an alternative means of performing noninvasive vascular imaging. Certainly, when performing MR imaging is not possible secondary to severe dialysis-dependent

Figure 9. Venous TOS in a patient with recurrent swelling and treated deep venous thrombosis of the right axillosubclavian vein. **(a)** Sagittal CT image with the right arm adducted shows a nonunited fracture (dashed arrow) anterior to the right subclavian vein (solid arrow) and artery (arrowhead). **(b)** Image with the right arm abducted shows posterior shift of the nonunited fracture (dashed arrow) over the now compressed right subclavian vein (solid arrow) and artery (arrowhead). No thrombus was seen at CT. **(c)** Image from digital subtraction angiography shows multiple collaterals proximal to the narrowing at the level of the nonunited fracture, indicating that this is a hemodynamically significant stenosis and confirming the diagnosis of venous TOS.



a. **b.**



c.

renal failure, claustrophobia, or implanted device incompatibility, CT becomes the study of choice. CT is also a preferred choice for evaluation of patients with suspected anomalous ribs or fractures, as bone anatomy is more easily definable at CT (35) (Figs 9, 10). CT is also useful in postoperative patients in whom there is a question of a remnant first rib. Interpretation of CT studies for TOS is similar to interpretation of MR imaging studies, relying on identification of anatomic abnormalities, findings of direct vascular damage, and positional vascular narrowing.

CT for TOS is performed with intravenous contrast material. As with MR imaging, the intravenous catheter should be placed on the side opposite the symptoms to prevent streak artifact from dense contrast material in the injected vein from obscuring the vasculature. In a standard protocol, we first position the patient with the symptomatic arm adducted and asymptomatic arm abducted. Half of the total contrast material bolus is then injected at 4–5 mL/sec, and bolus tracking software is used to trigger an acquisition off the aortic arch. After the arterial phase acquisition, a second acquisition is performed at 90 seconds for optimal venous enhancement.

The second half of the bolus is then administered with the symptomatic arm abducted and asymptomatic arm adducted. An arterial phase timed off the aorta and an acquisition with a 90-second delay are once again performed, for a total of four imaging sets in the examination. Anatomic coverage is from the midchest to the elbow of the abducted arm. If there is only a clinical question of specifically arterial or venous narrowing, the corresponding unneeded phases of the examination can be omitted to conserve radiation dose. A summary of our CT protocol for TOS is shown in Table 3.

Imaging the Postoperative Patient

As the number of surgical procedures performed for TOS rises, radiologists will increasingly encounter the postsurgical TOS patient. While there are several different approaches for thoracic outlet decompression, most involve resection of the entirety of the first rib and cervical ribs. Resection of muscles about the thoracic outlet is variable. Some procedures, particularly those in patients with vascular TOS, may involve venolysis, vascular reconstruction, vascular grafting, or stent placement (2). Detailed understanding of the various surgical approaches is beyond the scope of this article and probably not necessary even for the specialized cardiothoracic radiologist. However, an understanding of the major postoperative complications is valuable.

Expected Findings in the Immediate Postoperative Period

When evaluating immediate postoperative radiographs in a patient after thoracic outlet decompression surgery, there are several findings that should be expected. The first rib will have been resected, and a drain is typically present in the supraclavicular region. Many surgeons will

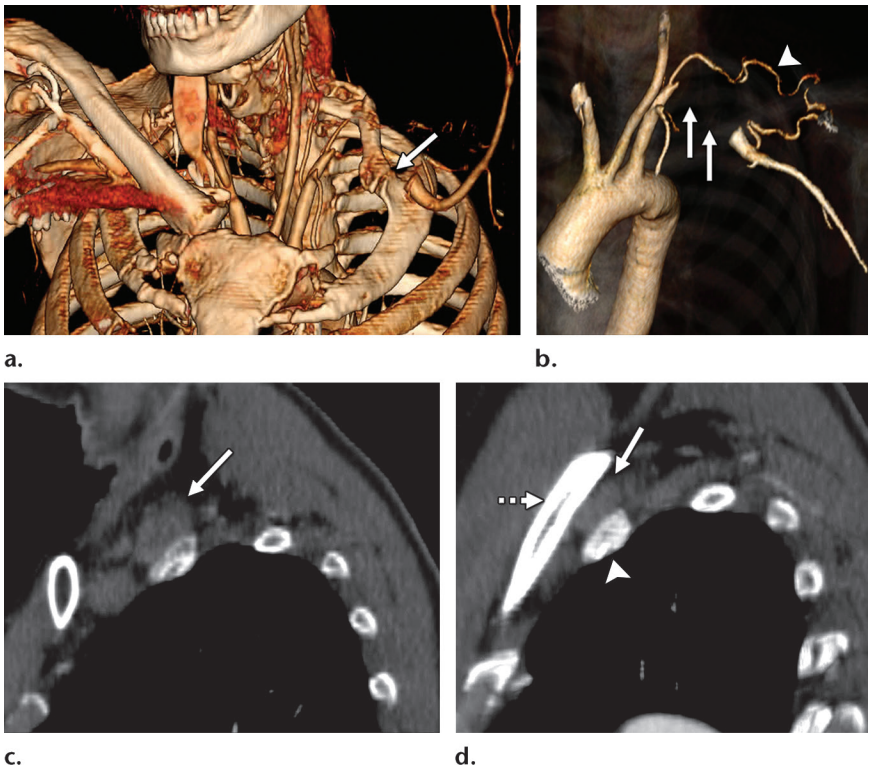


Figure 10. Arterial TOS in a patient with symptoms of arterial insufficiency to the left upper extremity. **(a)** Volumetric reconstruction from CT shows an anomalous left first rib that is hypoplastic and forms a joint with the second rib (arrow). **(b)** Volumetric reconstruction from arterial phase CT with the left arm adducted shows an area of thrombosis of the left subclavian artery (arrows) as well as an enlarged collateral (arrowhead). **(c)** Sagittal arterial phase CT image with the left arm adducted shows the uncompressed left subclavian artery (arrow), which is larger than expected and nonenhancing, consistent with thrombosis. **(d)** Image with the left arm abducted shows that the clavicle (dashed arrow) has moved posteriorly and compresses the left subclavian artery (solid arrow) against the hypoplastic left first rib (arrowhead). The findings of left subclavian artery thrombosis, enlargement, and dynamic compression confirmed the diagnosis of arterial TOS.

Table 3: CT Protocol for TOS

Place intravenous line in arm contralateral to symptoms
Field of view: center at aortic arch, coverage from midchest to elbow of abducted arm
Imaging protocol
1. Symptomatic arm adducted, asymptomatic arm abducted and externally rotated
a. Timed acquisition off aortic arch
b. 90-second delay*
2. Symptomatic arm abducted and externally rotated, asymptomatic arm adducted
a. Timed acquisition off aortic arch
b. 90-second delay*

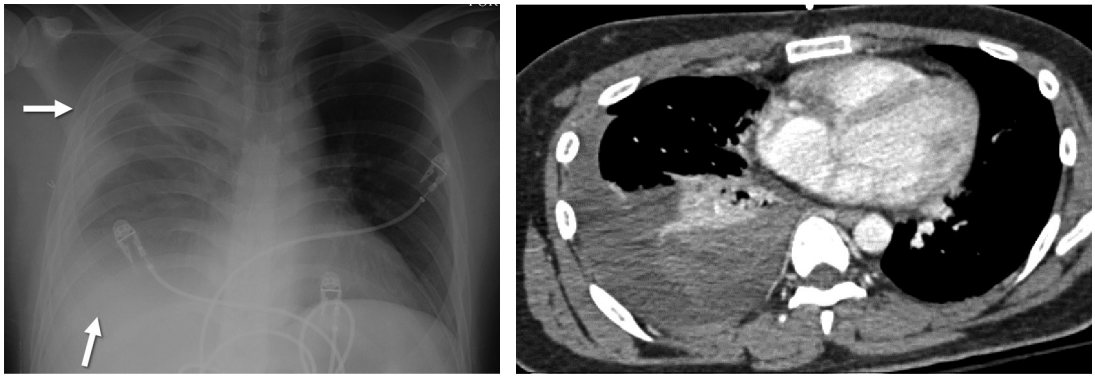
*The 90-second delay acquisitions can be omitted if the venous phase is not clinically relevant.

intentionally violate the apical pleura to provide a means for decompression of postoperative fluid into the pleural space (56). Thus, small or even moderate pleural effusions or a small pneumothorax are not unexpected findings. Associated subcutaneous gas is often seen in the supraclavicular space and chest wall.

In many patients in the immediate postoperative period, there will be respiratory splinting on the side of surgery; consequently, lower lobe atelectasis is a common finding in patients who have recently undergone thoracic outlet decompression. Identification of atelectasis is important, as it will direct the clinical team to encourage incentive spirometry and deeper inspirations to ventilate the atelectatic lung. Elevation of the ipsilateral diaphragm may also be present early after thoracic outlet decompression due to temporary phrenic nerve dysfunction from operative manipulation or infusion of local anesthetics.

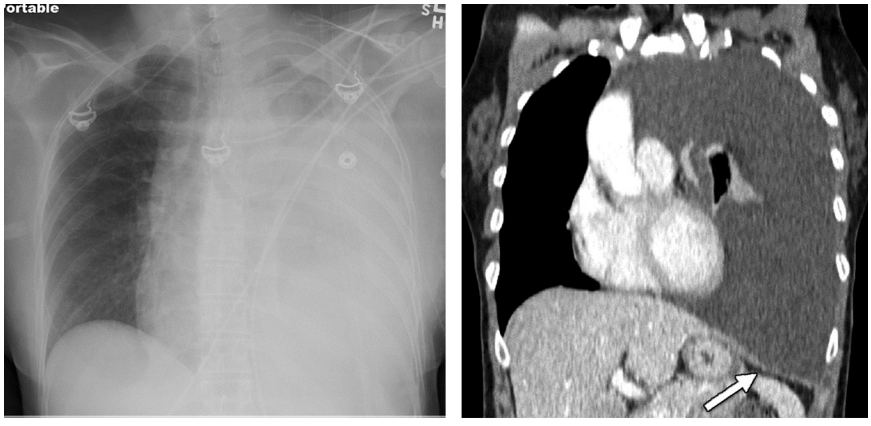
Short-term Complications (Days to Weeks)

Radiologists need to be aware of several important potential short-term complications of thoracic outlet surgery. Given the confined space of the thoracic outlet and the potential need to perform venolysis, unexpected vascular damage can result in a supraclavicular hematoma in the postsurgical bed (56,57). If there is also breach



a. b.

Figure 11. Hemothorax in a patient with neck swelling and dyspnea 8 days after right paraclavicular thoracic outlet decompression surgery. **(a)** Chest radiograph shows a right pleural fluid collection with lateral and subpulmonic components (arrows). This new and relatively rapidly accumulating pleural fluid collection was worrisome for a hemothorax. **(b)** CT image shows high-attenuation material in the right pleural space, consistent with blood products. The patient underwent operative evacuation of the hemothorax.



a. b.

Figure 12. Chylothorax in a patient with chronic dyspnea 6 months after left-sided thoracic outlet decompression surgery. Postoperative radiography showed normal findings (not shown). Chest radiograph **(a)** and coronal CT image **(b)** show a large left pleural effusion. The pleural fluid was simple but exerted tension, displacing the mediastinum to the right and inverting the left hemidiaphragm (arrow in **b**). Chylous fluid was drained from the pleural fluid collection. Initial management consisted of diet modification and octreotide, but medical management ultimately failed and surgical thoracic duct ligation was necessary.

of the apical pleural surface and the bleeding is continuous, a hemothorax can result. A rapidly enlarging pleural fluid collection on the ipsilateral side of a thoracic outlet decompression should raise the possibility of a hemothorax, which can be further characterized with CT and may require operative evacuation (Fig 11). Similarly, while a small pneumothorax is not an unexpected finding after thoracic outlet decompression surgery, an enlarging pneumothorax or one that exerts tension typically requires immediate clinical attention.

The thoracic duct and right lymphatic duct are additional important structures in the thoracic outlet that may be subject to injury during decompression surgery. If there is substantial lymph fluid leak from these major lymphatic ducts postoperatively, a chylothorax can result. A chylothorax can develop from days to months after thoracic outlet decompression surgery and is a key component of the differential diagnosis in post–thoracic outlet decompression patients with dyspnea or increasing pleural fluid collections (58–60). At CT, chylous effusions typically manifest as simple fluid and may contain a fat-fluid level within them (Fig 12). Chyle leaks are often managed nonoperatively with diet management and octreotide. When medical management fails, surgical re-exploration and ligation of the thoracic duct may be required. Patients with large chyle leaks may develop lymphopenia and subsequent immunocompromise if left untreated (61).

As in any postsurgical patient, patients after thoracic outlet decompression surgery are at risk for

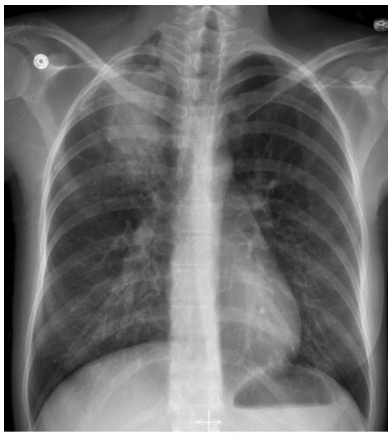


Figure 13. Infection in a patient with fever and clinically evident supraclavicular infection 1 month after right thoracic outlet decompression. **(a)** Chest radiograph shows a confluent right upper lobe airspace opacity with some mottled gas at the right apex. **(b, c)** CT images with lung **(b)** and soft-tissue **(c)** windows show pneumonia as well as an open tract from the pulmonary infection through the pleura and to the skin (arrow in **c**). Patients who have undergone thoracic outlet decompression are at risk for pleural space and lung infections due to communication with surgical bed infections via violations of the apical pleura.

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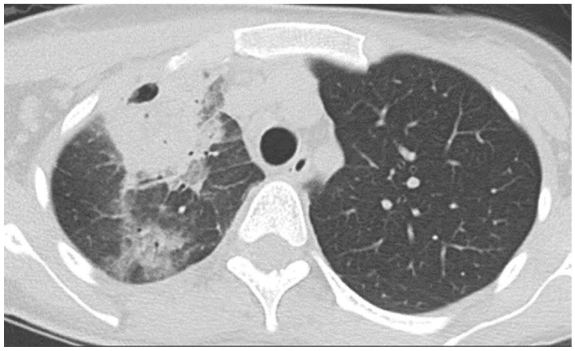

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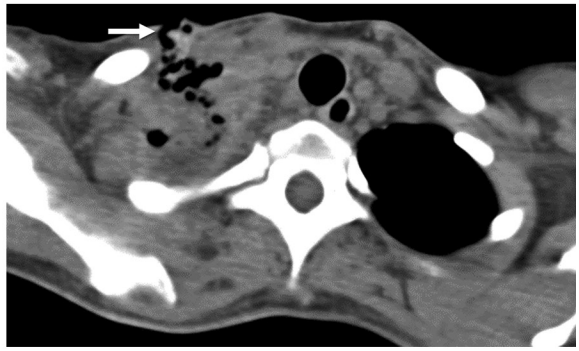

c.

infection (3). Owing to hematoma accumulation and potential inoculation of thrombus, patients undergoing surgery for venous TOS may have a slightly higher rate of infection. Infections after thoracic outlet decompression may be contained in the surgical bed, but due to potential violation of the pleural space, they can result in pleural space or pulmonary infections as well (Fig 13). For better characterization of postoperative infections in thoracic outlet decompression surgery patients, CT is often performed. It is incumbent on the radiologist to carefully examine the ipsilateral pleural space for pleural enhancement as well as loculi of gas or fluid, findings indicative of developing empyema.

Long-term Complications (Weeks to Months to Years)

Patients can also develop more delayed complications after thoracic outlet decompression surgery. Given their relationship to the thoracic outlet, brachial plexus or phrenic nerve dysfunction are potential complications of thoracic outlet decompression surgery, but are usually temporary with spontaneous resolution. While the exact incidence is unknown, phrenic nerve dysfunction probably occurs in less than 5% of patients (60). In the early postoperative period, using hemidiaphragm elevation as a finding suggestive of phrenic nerve injury is not advised, as atelectasis, small pleural effusions, respiratory splinting, and temporary paralysis can all lead to real or apparent hemidiaphragm elevation. If a hemidiaphragm is elevated in a patient who is outside the initial postoperative period, phrenic nerve palsy should be queried, and this can be further evaluated with a dedicated fluoroscopic examination to evaluate diaphragmatic motion.

Another rare potential long-term complication of thoracic outlet decompression is apical lung herniation. This occurs due to violation of the apical pleura, allowing lung to herniate into the supraclavicular space and cause patient discomfort (62). While this finding may be suggested at chest radiography, CT with coronal reconstructions is typically needed to confirm the finding and determine the extent of the hernia (Fig 14). Patients with apical lung herniation after thoracic outlet decompression may require a wedge resection to treat the hernia.

Patients who have thoracic outlet decompression surgery may have recurrent or persistent symptoms. Evaluation of the vasculature in these patients is often needed to determine patency of native vessels or grafts and is typically performed with MR imaging or CT. To best interpret these examinations, it is necessary for the radiologist to have access to the patient's operative history. Without knowledge of the operative history, it is possible for the radiologist to miss occluded

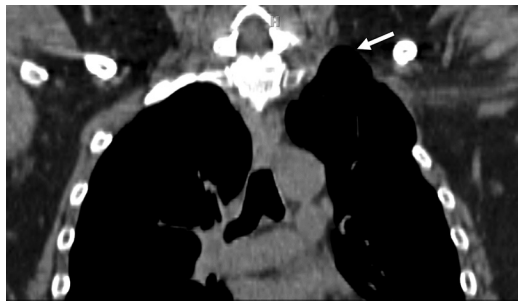

Figure 14. Apical lung herniation in a patient with a painful bulge in the left supraclavicular region 6 months after left thoracic outlet decompression. Coronal CT image shows a wide-mouthed herniation of the apical left upper lobe (arrow) into the left supraclavicular region. The patient underwent wedge resection of the left upper lobe hernia and did well.

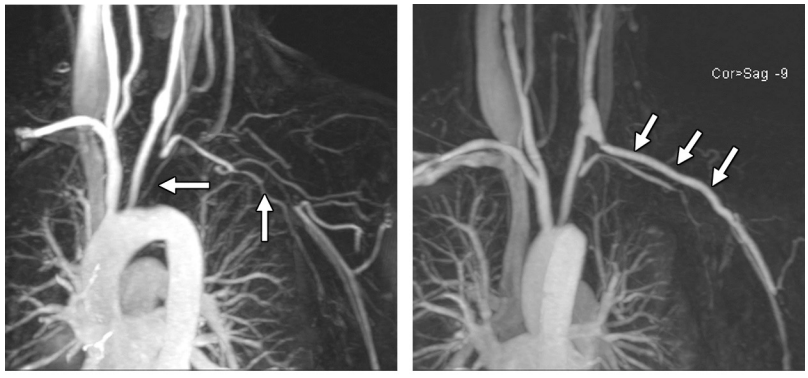

Figure 15. Graft occlusion in a patient with recurrent symptoms after left thoracic outlet decompression with left carotid-to-subclavian artery bypass. **(a)** Coronal arterial phase MIP from subtracted phase GRE imaging with the arms down shows occlusions of the proximal and mid left subclavian artery (arrows). The left carotid-to-subclavian artery graft was occluded, but does not appear on this image and could easily be missed if the patient's operative history was not known. **(b)** MR image after thrombolysis of the graft shows restored flow (arrows).

a. **b.**

grafts, particularly at MR imaging, where identification of graft material can be difficult (Fig 15).

Finally, a remnant or regrown first rib can cause persistent symptoms or recurrent thrombosis after thoracic outlet decompression surgery. Complete resection of the first rib involves resection of the sternum anteriorly and costovertebral junction posteriorly. In patients with an incomplete resection, there may be a remnant anterior first rib still attached to a residual portion of the anterior scalene muscle. This remnant first rib is often close to the traversing axillosubclavian vessels and can cause recurrent vascular complications. In cases in which there is suspicion of a remnant first rib, CT is the imaging study of choice, as it best defines the bone anatomy at the thoracic outlet (Fig 16). It is important to report not only the presence of a remnant first rib but also the size of the remnant rib, given that larger remnant ribs likely have a higher probability of causing recurrent symptoms (63–66).

Conclusion

The radiologist plays an important role in management of patients with TOS. While it is still largely a diagnosis of exclusion, imaging examinations are routinely used in diagnosis of TOS to define anatomic abnormalities and determine if findings suggestive of vascular compromise are present. After chest radiography, MR imaging and CT are the most commonly used noninvasive imaging modalities in patients with suspected vascular TOS, while patients with acute symptoms who may require urgent thrombolysis are often managed with angiography.

To prevent misdiagnosis, it is important for the radiologist to understand that vascular compression alone is not sufficient for diagnosis, as findings of vascular damage or thrombosis must be present in cases of vascular TOS. As surgical intervention in TOS patients increases, the radiologist also plays a role in care of patients after thoracic outlet decompression surgery. Identification of short- and long-term postoperative complications via imaging examinations is critical to providing prompt management, given that some of these complications may not be suspected on clinical grounds alone.

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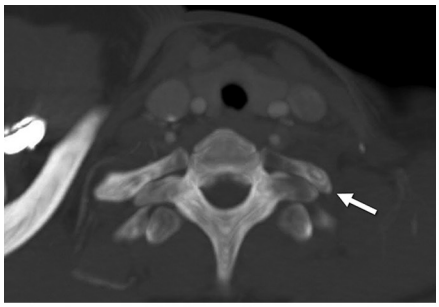

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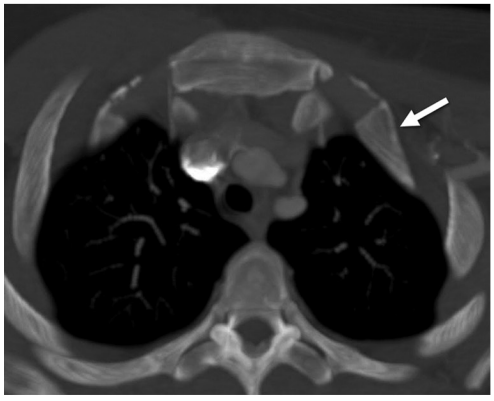

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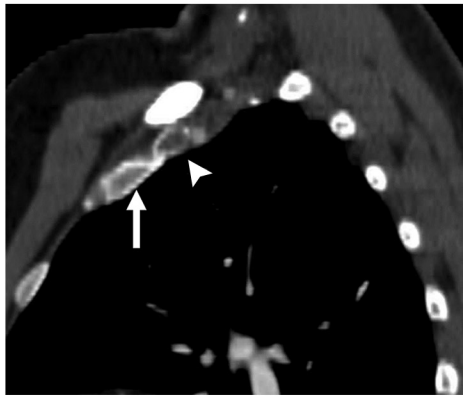

c.

Figure 16. Remnant first rib in a patient who underwent left thoracic outlet decompression surgery but experienced multiple repeat thromboses of the left axillosubclavian vein. Ultimately, a stent was placed. **(a, b)** CT images at presentation to our institution show large posterior (arrow in **a**) and anterior (arrow in **b**) remnant portions of the left first rib. **(c)** Sagittal CT image with the left arm in abduction shows the anterior left first rib remnant (arrow) contacting the undersurface of the mildly compressed left subclavian vein stent (arrowhead). The patient underwent complete resection of the left anterior and posterior first rib remnants and did well without recurrent thrombosis. Identification of remnant portions of the resected first ribs is best performed with CT and may provide an explanation for recurrent symptoms.

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