Axillary–Subclavian Venous Thrombosis

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Axillary–subclavian venous thrombosis (ASVT) is classified as either primary, which is often associated with active use of the upper extremities and underlying anatomic abnormalities, or secondary to recognized thrombotic risk factors. Although there is a risk of pulmonary embolism with both, late outcome varies with etiology. Primary ASVT occurs in young healthy patients, and up to 80% may have significant chronic symptoms. Secondary ASVT occurs in older patients with medical comorbidities, high mortality rates, and few late manifestations. Although anticoagulation is appropriate for most patients with secondary ASVT, a multidisciplinary approach including catheter-directed thrombolysis, thoracic outlet decompression, and correction of intrinsic venous lesions more effectively prevents late symptoms in selected patients with primary ASVT.
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Key words: Axillary–subclavian venous thrombosis • Upper extremity deep venous thrombosis • Thrombolysis

Thrombosis of the axillary and subclavian veins occurs in only 2 per 100,000 individuals and has historically accounted for only 1%–2% of deep venous thrombi.[1–3] This low incidence has been attributed to infrequent immobilization as well as the absence of venous sinuses, increased fibrinolytic activity, higher venous flow velocities, fewer valves, and decreased hydrostatic forces in the upper extremity.[4,5] However, largely owing to increased central venous instrumentation, the incidence of deep venous thrombosis (DVT) involving the axillary and subclavian veins is increasing.

Table 1
Demographics of Axillary–Subclavian Venous Thrombosis (ASVT)

	Primary ASVT	Secondary ASVT
Age	Young	Older
Comorbidity	Healthy	Sick
Activity	High	Low
Lower extremity DVT	_	38%[10]
6-month mortality	_	48%[12]
Late symptoms	47%–93%[21,22]	6%[10]
Treatment	Multidisciplinary	Anticoagulation

DVT, deep vein thrombosis.

Consideration of the natural history of upper extremity thrombosis requires some appreciation of the underlying etiology. Although there is no uniform system of classification, axillary–subclavian venous thrombosis (ASVT) is commonly regarded as primary or secondary.[5,6] Primary thrombosis occurs in the absence of recognized risk factors and includes effort thrombosis, or the Paget-Shroetter syndrome. Effort thrombosis is associated with repetitive or strenuous exercise, usually in the presence of underlying anatomic abnormalities. A spectrum of activities, including painting, chopping wood, typesetting, golf, tennis, and baseball, associated with hyperabduction and external rotation of the arm, have been implicated in the pathophysiology of primary ASVT.[7,8] In contrast, secondary thrombosis occurs in the setting of recognized thrombotic risk factors. The underlying etiology is not always clear, and traumatic and idiopathic thrombosis have sometimes been classified as primary ASVT. However, coagulation abnormalities have been identified in 42% of patients with idiopathic upper extremity thrombosis and many are more appropriately regarded as secondary in nature.[9]

The increased use of central venous instrumentation has caused a shift from primary to secondary causes as the most common type of ASVT. Subclavian vein cannulation is now responsible for 39%–63% of cases, with as few as 1%–9% attributable to effort thrombosis.[3,4,10,11] Correspondingly, the patient population has shifted toward older patients with underlying medical problems (Table 1).[12] Patients with secondary ASVT are significantly more ill and have a higher prevalence of metastatic malignancies than those with lower extremity DVT. As many as 38% of patients with secondary upper extremity DVT will have a concomitant lower extremity thrombosis,[10] and 6-month mortality may be as high as 48%.[12]

In contrast, patients with primary axillary–subclavian thrombosis are characteristically younger and healthier. These patients have an average age of 31, and over 70% are male, have involvement of the dominant hand, and have a history of unusual upper extremity activity or positioning.[13] Primary thrombosis is frequently associated with underlying anatomic abnormalities and perivascular fibrosis that is presumed secondary to recurrent venous compression at the junction of the first rib and clavicle.[14,15] A variety of compressive elements have been identified, including anomalous anterior scalene and subclavius muscles, fibromuscular bands, and a narrow costoclavicular space.[7] The subclavian vein lies in the space bounded by the first rib inferiorly, the clavicle and subclavius muscle anterosuperiorly, and the anterior scalene muscle posteriorly (Figure 1). During abduction of the arm, the anterior scalene muscle contracts, drawing the first rib superiorly and narrowing the costoclavicular space.[16] In such cases, positional venography will demonstrate venous compression on abduction of the arm to 90 degrees and full external rotation. Notably, approximately half of patients will demonstrate asymptomatic positional occlusion of the contralateral subclavian vein.[16]

Complications of Axillary–Subclavian Venous Thrombosis

Complications of ASVT include recurrent thrombosis, pulmonary embolism, and chronic symptoms of pain, easy fatigability, and edema. Although there is a notable lack of methodologically sound data, the literature does provide some insight into the natural history of upper extremity venous thrombosis. Symptomatic recurrent thrombosis has been noted in 7%–17% of patients with ASVT.[1,2] Perhaps not surprisingly, given the prevalence of underlying anatomic abnormalities, recurrent thrombosis appears to be particularly common among patients with primary ASVT. Multiple venographically documented episodes of thrombosis have been reported in 34% of such patients.[17] Furthermore, although often underemphasized,

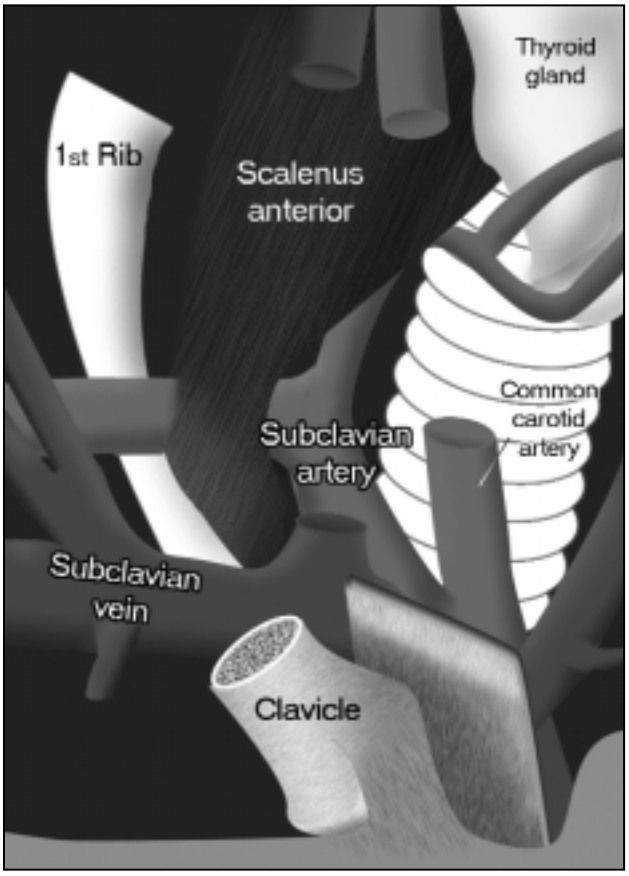

Figure 1. *Anatomic relationships in the costoclavicular space. The subclavian artery passes posterior to the scalenus anterior muscle while the vein pass anterior. The subclavian vein lies in the space bounded by the first rib inferiorly, the clavicle and subclavius muscle anterosuperiorly, and the anterior scalene muscle posteriorly. The costoclavicular space is narrowed and the vein compressed during abduction of the arm as the anterior scalene contracts and the first rib rises. Figure by Zachary Meissner.*

studies employing routine ventilation–perfusion lung scanning have clearly shown that upper extremity thrombi may be responsible for pulmonary emboli.[18–20] Symptomatic pulmonary emboli may complicate up to 17% of these thrombi.[4,10,12,21,22] Autopsy series of hospitalized patients have similarly identified arm thrombosis in 13% of patients with pulmonary embolism.[3] Although some[18] have reported a higher incidence of pulmonary embolism (25%) with catheter-related thromboses, others have found pulmonary embolism to be independent of etiology.[10] Pulmonary embolism has been documented in 26% of patients with primary ASVT, although only 36% of these were symptomatic.[23] Approximately 10% of upper extremity–associated emboli are fatal, similar to the mortality rate for emboli arising from the lower extremities.[24]

The pathophysiology of chronic symptoms after axillary–subclavian thrombosis differs from that in the lower extremities.[13] Whereas late lower extremity symptoms are primarily due to valvular incompetence, with obstruction having a secondary role, upper extremity symptoms are entirely the result of venous obstruction. As preservation of valve function is not relevant, the prevention of chronic symptoms is theoretically simpler after ASVT. Furthermore, as the hydrostatic effects of gravity are less important in the upper extremity, late symptoms tend to be limited to pain, swelling, and cyanosis. Such symptoms frequently occur only with muscular activity. Stasis dermatitis and ulceration are distinctly unusual.[25]

The prevalence of chronic symptoms after ASVT has varied, but appears related to the underlying etiology. Overall, 34% of the patients reported in the literature have had some symptoms during follow-up.[24] Older series, including a significant number of patients with superior vena caval occlusions and recurrent thromboses, have noted persistent symptoms in up to 74% of extremities, regardless of the underlying etiology.[2] However, the frequency of late manifestations is lower in contemporary reports,[1,21] and it is clear that severe chronic symptoms are unusual after secondary ASVT. Series consisting largely of inpatients have reported the prevalence of late symptoms to be as low as 6%.[10] In contrast, some degree of long-term disability has been noted in 47%–93% of patients with effort thrombosis.[21,22] Among patients reported in the literature, Rutherford[13] found only 18% to be asymptomatic, whereas 47% had severely disabling late symptoms. Both the presence of underlying anatomic abnormalities and the increased activity levels of these patients likely contribute to the increased frequency of late symptoms among patients with primary thrombosis.

Treatment of Axillary–Subclavian Thrombosis

In contrast to the management of lower extremity thrombosis, which has been well defined by randomized clinical trials, the treatment of upper extremity thrombosis is not standardized and remains controversial. Consideration of the natural history of ASVT and the risks and benefits of treatment in an individual patient is therefore appropriate. Based on such an approach, anticoagulation is appropriate for most patients in whom the risk of symptomatic recurrent thrombosis and

Table 2
Results of Thrombolytic Therapy for Axillary–Subclavian Venous Thrombosis

Author	Drug	Route	Patients	Complete Lysis	Partial Lysis
Lindblad et al[3]	SK	?	5	0%	_
Wilson et al[14]	SK	Systemic	8	62.5%	25%
AbuRahma et al[7]	SK/UK	Systemic	4	75%	25%
Machleder[17]	UK	Catheter-directed	23	74%	_
Kunkel & Machleder[28]	SK/UK	Catheter-directed	11	82%	_
Lee et al[27]	UK	Catheter-directed	11	82%	18%
Beygui et al[26]	UK	Catheter directed	31	84%	_

SK, streptokinase; UK, urokinase.

pulmonary embolism exceeds that of major hemorrhage. Few patients with secondary ASVT will have chronic symptoms when treated with anticoagulation, and most authorities agree that there is little role for more aggressive treatment in these patients.[26]

The Multidisciplinary Approach

In contrast, anticoagulation alone may not adequately prevent late disability in patients with primary thrombosis. In addition to preventing recurrent thrombosis and pulmonary embolism, the goals of treatment in these patients include restoration of venous patency, relief of any underlying anatomic compression, and correction of intrinsic venous pathology. A multidisciplinary approach, including thrombolytic therapy, surgery, balloon angioplasty, and anticoagulation may be required to attain these goals. Selection of patients for such treatment should focus on young, active patients with primary thrombosis who use their upper extremities in their job or avocation.[13]

Unfortunately, there are no randomized trials comparing anticoagulation to thrombolytic or surgical management, and treatment is largely guided by inadequate data from small retrospective reports. Data using historic controls suggests that whereas 25 of 35 (71%) extremities remained symptomatic after anticoagulation alone, 32 of 36 (89%) treated with a multidisciplinary approach were asymptomatic.[8] Consensus opinion also favors such an approach. Among vascular surgeons participating in a panel sponsored by the American Venous Forum, 70% believed intervention was appropriate for patients requiring active use of their arm.[25]

Catheter-Directed Thrombolysis

Interventional management of primary ASVT historically consisted of thrombectomy and thoracic outlet decompression. However, catheter-directed thrombolysis has now largely replaced surgical thrombectomy. In comparison to thrombectomy, catheter-directed thrombolysis is less morbid, can be performed with fluoroscopic guidance, and allows precise definition of underlying anatomic lesions using provocative positioning. A variety of catheter-directed thrombolysis protocols have been suggested, most undertaken using an antegrade approach from the arm and a bolus or pulse spray of 125,000–250,000 IU urokinase, followed by a urokinase infusion of 50,000–120,000 IU/hr into the thrombus.[26–28] Concurrent anticoagulation with heparin is usually recommended. Complete thrombolysis has often required a 24–72-hour infusion as guided by the progress on serial venograms. Provocative venograms with the arm abducted and externally rotated should be obtained on completion of lytic treatment to disclose any underlying anatomic lesions.

Despite the absence of randomized trials or even large series evaluating catheter-directed thrombolysis for ASVT, the results of small series are consistent and suggest that a catheter-directed approach achieves complete thrombolysis in approximately 80% of cases (Table 2). Although optimal results are obtained within 7 days,[14] consensus opinion favors consideration of thrombolysis within 10–14 days after the onset of symptoms.[25] Thrombolysis also appears to be durable when used as part of a multidisciplinary approach. After a mean follow-up of 29 months, Machleder[17] reported patent veins in 74% of patients treated with urokinase, compared to only 25% of those treated with heparin alone. As thrombolysis is usually offered

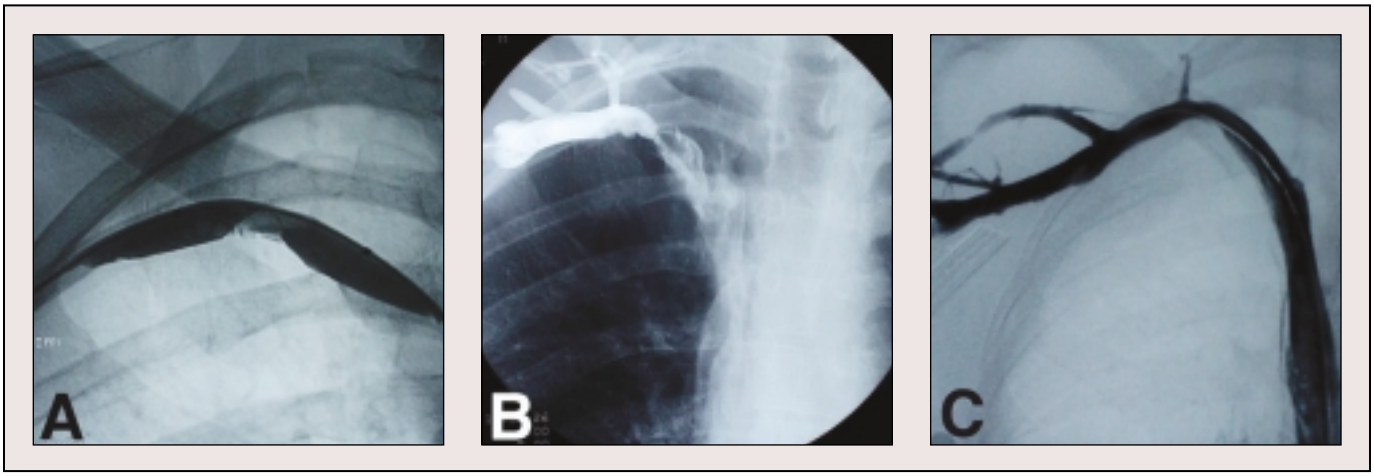

Figure 2. *Venograms of a 26-year-old housepainter who underwent catheter-directed thrombolysis for a first episode of right axillary–subclavian deep vein thrombosis.* **(A)** *Balloon angioplasty failed to completely resolve a compressive lesion at the first rib crossing of the subclavian vein. The patient was discharged on warfarin.* **(B)** *He presented 1 year later with recurrent thrombosis, again treated with catheter-directed urokinase. Follow-up venograms again demonstrate stenosis at the first rib.* **(C)** *The patient underwent transaxillary first resection with venolysis. A post-operative venogram shows minimal residual stenosis of the subclavian vein. The patient has had no further thrombotic episodes 4 years after completing a 3-month course of post-operative anticoagulation.*

only to young, otherwise healthy patients, most series have reported no associated bleeding complications.[25]

Surgical Intervention

Primary axillary–subclavian venous thrombosis is associated with an underlying anatomic abnormality, most commonly an element of costoclavicular compression, in 75%–100% (average 92%) of cases.[13] Thoracic outlet decompression should therefore be considered after successful thrombolysis if there is extrinsic compression of either the recanalized vein or venous collaterals with arm abduction.[17] Surgical intervention likely is not required in the few patients without evidence of extrinsic compression or intrinsic venous stenosis. Wilson and colleagues[14] reported no recurrent thrombosis in 7 patients treated with thrombolytic therapy alone, and Machleder[17] noted that 5 of 23 patients successfully treated with thrombolytic therapy required no further intervention. However, without decompression, approximately three quarters of patients will remain symptomatic,[28] and one third will develop recurrent thrombosis.[17]

First rib resection, either by a transaxillary or supraclavicular approach, is the most widely used means of decompression. The scalenus anterior and any other compressive elements are also frequently divided or resected.[8] Although excellent results have been reported for transaxillary rib resection,[8,17,28] many surgeons advocate a supraclavicular approach as providing optimal exposure for first rib resection, scalenectomy, and circumferential venolysis.[16,27] This approach may be supplemented by an infraclavicular incision to achieve complete first rib resection to the costosternal junction.

The appropriate timing of surgical intervention after thrombolysis continues to be debated. A 3- to 6-month interval was previously recommended to allow resolution of inflammation and assessment of residual symptoms.[13] However, as the majority of patients remain symptomatic, and 10% will develop recurrent thrombosis during this period,[17,28] shorter delays have more recently been advocated. Although some have performed decompression during the same hospitalization,[27] most surgeons currently defer operation for 3–4 weeks.[25]

Management of Intrinsic Venous Lesions

Despite high rates of success for thrombolysis and costoclavicular decompression, results for the adjunctive treatment of intrinsic venous lesions have been more variable.[26] Venolysis at the time of rib resection may effectively relieve stenosis in more than half of cases with perivascular fibrosis (Figure 2).[27] Alternatives for managing persistent intrinsic venous lesions include balloon angioplasty with or without stents, patch angioplasty, and venous bypass.

It is clear that balloon angioplasty without costoclavicular decompression is almost uniformly unsuccessful[25–28] and may have an adverse effect on venous patency.[16,28] Among 12 patients treated with pre-operative angioplasty, 7 occluded immediately, and there was no effect on the compressive abnormality in the remaining 5.[17] Such procedures may, however,

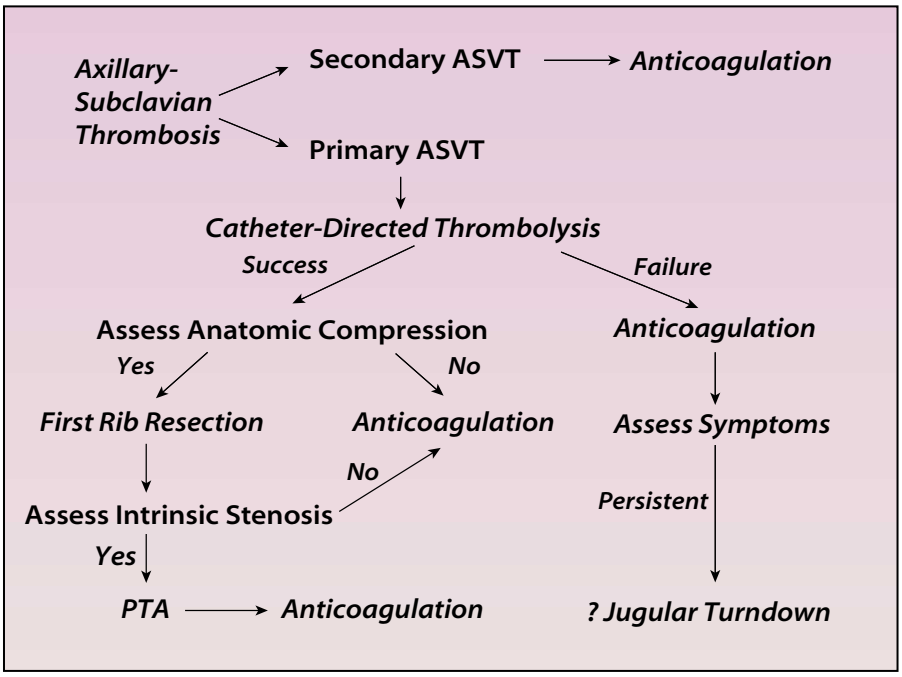

Figure 3. *Algorithm for the management of axillary–subclavian thrombosis (ASVT). PTA, percutaneous transluminal angioplasty.*

have a role after rib resection. Although no well-supported criteria have been established, accepted indications for angioplasty have included a greater than 50% luminal diameter reduction or the presence of significant residual collateral flow.[16] Machleder[17] reported post-operative balloon angioplasty to be successful in 7 of 9 patients.

Similar considerations apply to the use of metallic stents for residual venous lesions. Without decompression, stents are susceptible to the same mechanical compressive forces as the vein.[29] Stent deformation has been reported to occur in virtually all patients with stress abduction, and both crushed and fractured stents have been reported.[29] Even with first rib resection, recurrent stenosis has been reported in 2 of 5 patients within the first year.[29] Venous reconstruction for intrinsic stenosis has been successful in only one half of patients reported in small series.[26] Residual, symptomatic short segment occlusions are most often managed by end-to-side anastomosis of the divided jugular vein to the subclavian vein (jugular vein turndown). Long segment obstructions requiring more extensive bypass operations have often fared poorly, with patency rates as low as 25%.[15]

As emphasized by authors having substantial experience with primary ASVT, some flexibility in the multidisciplinary approach is required to account for differences in disease severity, duration, previous treatment, and underlying anatomic abnormalities. However, a general algorithm for the management of patients with ASVT is illustrated in Figure 3. Unfortunately randomized trials comparing multidisciplinary treatment with anticoagulation are lacking. However, outcome is related to the ultimate status of the axillary and subclavian veins—93% of those with a patent vein and 64% of those with a persistently occluded vein being free of symptoms.[17] Furthermore, in comparison to reports in which only 18% of primary ASVT patients treated with anticoagulation are asymptomatic,[13] 83%–91% of patients have been reported to be without significant symptoms after multidisciplinary treatment (Table 3). Surgical decompression also appears to be very effective in preventing recurrent thrombosis in these patients.[17]

Conclusion

The natural history of axillary–subclavian venous thrombosis is dependent on the underlying etiology. The incidence of secondary ASVT has increased with more frequent central venous instrumentation. Although the thromboembolic complications of secondary ASVT cannot be ignored and warrant anticoagulation in most patients, late manifestations are unusual in this older population

Table 3
Results of Multidisciplinary Treatment for Primary Axillary–Subclavian Venous Thrombosis

Author	Patients	Symptom-Free
Urschel & Razzuk[8]	36	32 (89%)
Molina[15]	24	20 (83%)
Machleder[17]	35	29 (83%)
Lee et al[27]	11	10 (91%)

with multiple medical problems and a high mortality. In contrast, primary ASVT affects young, active patients with underlying anatomic abnormalities and may be associated with a high incidence of recurrent thrombosis and late symptoms. Given that up to 80% of patients remain symptomatic after anticoagulation alone, a more aggressive multidisciplinary approach including catheter-directed thrombolysis, thoracic outlet decompression, and correction of intrinsic venous lesions is warranted in selected patients. In contrast to the results with anticoagulation alone, resolution of symptoms and a return to normal activities can be expected in over 80% of patients treated with such an approach.

Unfortunately, the treatment of ASVT, even with anticoagulation, is guided largely by imperfect data from largely retrospective reviews. Effort thrombosis is, in particular, a relatively uncommon disorder with no single center seeing sufficient patients for a large series, much less a randomized clinical trial comparing different treatment approaches. Even multicenter trials would be difficult, given the time intervals required to accumulate the patients reported by centers with expertise in managing ASVT. Although treatment is best determined by homogenous data from randomized clinical trials, this is currently unrealistic for primary ASVT. Treatment recommendations can, however, be guided by an understanding of the natural history of the disease, which does suggest that a multidisciplinary approach has benefits for selected patients with primary ASVT. ■

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Main Points

- Largely because of increased central venous instrumentation, the incidence of deep venous thrombosis involving the axillary and subclavian veins is increasing.
- Axillary–subclavian venous thrombosis (ASVT) is commonly regarded as primary or secondary; primary thrombosis occurs in the absence of recognized risk factors and includes effort thrombosis, or the Paget-Shroetter syndrome, whereas secondary thrombosis occurs in the setting of recognized thrombotic risk factors.
- Given the prevalence of underlying anatomic abnormalities, recurrent thrombosis appears to be particularly common among patients with primary ASVT. Multiple venographically documented episodes of thrombosis have been reported in 34% of such patients.
- Pulmonary embolism has been documented in 26% of patients with primary ASVT, although only 36% of these were symptomatic. Approximately 10% of upper extremity–associated emboli are fatal, similar to the mortality rate for emboli arising from the lower extremities.
- Anticoagulation alone may not adequately prevent late disability in patients with primary thrombosis, for whom a multidisciplinary approach, including thrombolytic therapy, surgery, balloon angioplasty, and anticoagulation may be required.
- Data using historic controls suggests that whereas 25 of 35 (71%) extremities remained symptomatic after anticoagulation alone, 32 of 36 (89%) treated with a multidisciplinary approach were asymptomatic.
- Catheter-directed thrombolysis has now largely replaced surgical thrombectomy, because it is less morbid, can be performed with fluoroscopic guidance, and allows precise definition of underlying anatomic lesions using provocative positioning. The results of a small series suggest that a catheter-directed approach achieves complete thrombolysis in approximately 80% of cases.
- Thoracic outlet decompression should be considered after successful thrombolysis if there is extrinsic compression of either the recanalized vein or venous collaterals with arm abduction.

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