Modern Treatment of Axillary Subclavian Vein DVT

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Modern treatment of axillary subclavian venous thrombosis involves multimodal management including catheter-directed thrombolysis, surgical decompression (first-rib resection), venous reconstruction or angioplasty, and oral anticoagulation.[1,2] Goals of management are to restore patency, remove extrinsic compression, and repair any intrinsic vein stenosis. Early surgical therapy following thrombolysis is prefered since anticoagulation therapy alone is associated with arm disability in the majority (60–70%) of cases. When subclavian vein thrombolysis is successful, a venous defect will be identified in the two-thirds of patients.[2] If the vein defect is subtle, positional venography will often document compression of the subclavian vein with arm abduction–such testing confirms the diagnosis of "venous thoracic outlet syndrome" (TOS). Our group favors immediate operative thoracic outlet decompression during a single hospitalization. The patient's return to normal arm activity is shortened as is the time period of oral anticoagulation (warfarin [Coumadin]; therapeutic INR range: 2–3). The surgical TOS compression procedure involves supraclavicular exposure of the first rib with concomitant excision of the anterior scalene muscle insertion. An infraclavicular approach to resecting the anterior first-rib segment may be required. Intraoperative duplex ultrasonography is recommended to identify residual subclavian thrombus which is typically located at a sclerotic valve site. Repair of the intrinsic vein abnormality may require endovenectomy with vein patch, intraoperative balloon angioplasty, internal jugular vein turn-down, or rarely, interposition vein bypass. Circumferential venolysis should be performed routinely.

During an 8-year period, the University of South Florida vascular surgery division operated on 19 patients (16 men, 3 women) for venous TOS. Etiology was related to recreational sports or work-related activity (n = 16), trauma (n = 2), and in one patient (a car salesman), no precipitating event was identified. Patient ages ranged from 16 to 53 years (mean age: 33 years). All patients were symptomatic with arm swelling and pain. Time to intervention ranged from 1 to 19 days (mean time: 7 days). Following successful catheter-directed thrombolysis using urokinase (n = 2) or tPA (n = 17), a residual subclavian vein stenosis was demonstrated in 14 patients, extrinsic compression at the level of the first rib in two patients, and in two patients, positional venography confirmed subclavian vein occlusion and collateral venous flow with arm abduction. In one patient, thrombolysis was not successful and a venous bypass was performed. Operative approach was via a supraclavicular (n = 14) or paraclavicular (n = 5) exposure. Correction of extrinsic vein compression was accomplished by first-rib resection (n = 18), anterior/middle scalenectomy (n = 18), cervical-rib resection (n = 2), and medial claviculectomy (n = 1). To repair the intrinsic vein defect, the following procedures were performed: circumferential venolysis (n = 18), thrombus extraction/excision of vein valve with vein patching (n = 8), balloon catheter thrombectomy (n = 3), intraoperative balloon angioplasty (n = 2), internal jugular vein turn-down (n = 3), and vein bypass (n = 1). Intraoperative duplex scanning confirmed adequate subclavian vein lumen with phasic venous flow in all patients. Procedural complications occurred in 4 patients including wound hematoma (n = 2) and hemo-pneumothorax requiring tube thoracostomy (n = 2). Duplex ultrasonography confirmed axillo-subclavian vein patency in all patients at the time of hospital discharge.

During a mean patient follow-up of 23 months (range: 5–70 months), recurrent thrombosis occurred in 5 (26%) of the 19 patients—2 patients were asymptomatic and 3 patients had recurrent symptoms. Two symptomatic patients were treated with thrombolysis and PTA or stenting, and 1 patient underwent an internal jugular vein turn-down procedure. Recanalization of a vein segment occurred in the 2 asymptomatic patients during long-term oral anticoagulation therapy. To date, all internal jugular vein turn-down repairs are patent. The time period of oral anticoagulation was typically 6 months, and recommendation to discontinue anticoagulant therapy was based on testing with duplex ultrasonograpy showing a patent axillo-subclavian venous segment with normal phasic flow with respiration. At last follow-up, 17 (89%) of 19 venous segments are patent and 14 of 19 patients are working or performing normal arm activities. One patient remains disabled.

Our experience treating venous TOS supports an aggressive endovascular/surgical management algorithm. Residual subclavian vein stenosis is common after catheter-directed thrombolysis and several strategies can be used to correct the venous abnormality including: direct vein repair, percutaneous balloon angioplasty, and use of the internal jugular vein turn-down procedure. The vascular surgery literature discourages the use of a venous stent.[3] In our experience of one patient treated with a venous stent after thrombolysis for recurrent thrombolysis, stent stenosis was documented by duplex scanning at 1 month and occlusion occurred rapidly thereafter. Our treatment approach consisting of early TOS decompression surgery with venous repair if necessary after successful thrombolysis has been associated with a secondary patency of > 85%.

References

1. Schmacht DC, Back MR, Novotney ML, et al. Primary axillary-subclavian venous thrombosis: is aggressive surgical intervention justified? Vasc Surg 2001;35:353–9.
2. Schneider DB, Dimuzio PJ, Martin ND, et al. Combination treatment of venous thoracic outlet syndrome: open surgical decompression and intra operative angioplasty. J Vasc Surg 2004;40: 599–603.
3. Urschel HC Jr, Patel AN. Paget-Schroetter syndrome therapy: failure of intravenous stents. Ann Thorac Surg 2003;75:1693–6.

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