Acute management of subclavian vein thrombosis

Subclavian vein thrombosis is a rare disorder of multiple aetiology. Its incidence appears to be increasing, and it may now account for around 4 per cent of all deep venous thrombosis. Its aetiology is also changing. A large review by Hughes in 1949 reported 83 per cent primary and 17 per cent secondary thromboses; more recent data suggest that only one-quarter are now primary and three-quarters secondary (one-third caused by indwelling subclavian catheters).

Patients commonly present with swelling (74 per cent), discolouration (68 per cent) and aching (26 per cent) of the limb. There may be distended collateral veins over the shoulder girdle, a palpable axillary vein (32 per cent) and, in women, ipsilateral breast oedema (22 per cent). Venography confirms the clinical diagnosis and delineates the extent of the thrombus. The basilic vein is preferred for venography as contrast agent entering the cephalic vein bypasses the subclavian vein via collaterals, resulting in poor resolution of the morphology of the thrombus. Digital subtraction techniques improve resolution and decrease the dose of contrast agent.

Morbidity is related to aetiology rather than treatment. Conventionally, patients are managed with elevation of the limb, bed rest, intravenous heparin, and warfarin for 3–12 months. Initiation of heparin therapy within 7 days of onset is necessary if organization of the thrombus is to be avoided. Although little dissolution of thrombus occurs with heparin, maintenance of collaterals is facilitated and propagation of thrombus decreased. It used to be thought that heparin protected against pulmonary emboli but, in a recent series, 41 per cent of patients who had pulmonary emboli were already receiving heparin. The results of conventional management are variable. Donayre et al. and Campbell et al. have shown, in patients with thrombosis secondary to subclavian cannulation, that there is complete resolution of both thrombus and symptoms after conventional management and removal of the cannula. However, up to 70 per cent of those with primary thrombosis have residual symptoms after conventional management. Such a form of treatment may not be appropriate for this latter group.

Thrombolysis may be superior to conventional management; streptokinase, urokinase and tissue plasminogen activator have all been used. As with arterial thrombolysis, doses have been reduced without loss of efficacy and with decreased side-effects by infusing directly into the thrombus. The thrombolytic agent is administered through a catheter placed via the ipsilateral antecubital fossa and advanced into the thrombus. Heparin is given simultaneously to reduce the risk of pericatheter thrombosis. During lysis the limb is re-examined with venography every 12 h and the infusion discontinued once lysis is complete; anticoagulation is continued. Venography after lysis often reveals stenosis of the vein at the site of the semilunar valve.

Given the high proportion of patients with primary subclavian vein thrombosis who have persistent symptoms after conventional management, correction of any underlying venous stenosis or external compression from structures at the thoracic inlet appears to be justified. The timing of such intervention is debatable. Traditionally, patients are managed conventionally with anticoagulants and offered surgery only if symptoms persist or the vein rethromboses. However, immediate correction of any underlying anatomical lesion may be desirable.

Correction of subclavian vein stenosis by percutaneous balloon venoplasty has been reported in small numbers. In a series by Glanz et al., all patients had an ipsilateral arteriovenous fistula and were on dialysis. The initial success rate was 77 per cent, with 50 per cent patent at 6 months and 30 per cent at 1 year; the secondary patency rate was higher because of repeat venoplasty. The present authors have successfully dilated eight primary stenoses of the subclavian vein, with long-term patency in six. The stenosis recurred immediately on balloon deflation in two patients and an endoluminal stent was placed in both with patency at 6 and 18 months respectively.
A wide variety of surgical procedures has been described for the treatment of primary subclavian vein thrombosis but most series are small and historical cases are used as controls. Many authors believe that the aetiology of primary thrombosis is extrinsic compression of the vein as it passes over the first rib, which produces endothelial damage, fibrosis around the vein, stasis and thrombosis. Surgery is directed at releasing the tissues around the vein at the thoracic outlet, for example resection of the first or cervical rib (if present). The results of surgical decompression of the thoracic outlet are difficult to interpret because many patients have simultaneous surgical exploration of the vein with thrombectomy, removal of an intrinsic venous lesion, patch venoplasty or thrombolysis. Success is often subjective rather than objective but there seems to be an overall 'success' rate of approximately 80 per cent. Endovenectomy with venoplasty or venous bypass has been performed for patients with intrinsic venous stenosis10. However, these procedures are often combined with thoracic outlet decompression, again making interpretation of results difficult.

In summary, subclavian vein thrombosis is a rare disorder of multiple aetiology. Treatment must be started early to reduce long-term morbidity. The diagnosis is confirmed by venography via the ipsilateral basilic vein. Secondary thrombosis may be adequately managed by heparin therapy followed by warfarin for up to 1 year. Local low-dose thrombolysis is the treatment of choice for primary thrombosis. Successful lysis reveals an intrinsic venous lesion in a high proportion of cases. There is still debate about the place and timing of subsequent intervention; some authors favour immediate correction of any intrinsic lesion and/or decompression of the thoracic outlet. If patients are managed with anticoagulation for 3 months and then reviewed, those who are asymptomatic may avoid surgery. However, if rethrombosis occurs, intervention is less successful. Intrinsic lesions may be treated by balloon venoplasty, stenting or patch venoplasty, while extrinsic compression at the thoracic outlet requires surgical release. The durability of thrombolysis alone or combined with balloon venoplasty and stenting has yet to be established.

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