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Pictorial Review

Central venous obstruction in the thorax

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Central venous stenosis and occlusion can occur secondary to a spectrum of conditions ranging from aggressive malignancy to benign extrinsic anatomical compression in otherwise healthy individuals. Irrespective of aetiology, significant morbidity in the acute setting and long term can occur unless prompt accurate diagnosis and appropriate management is initiated, the radiologist being central to both. The present review will provide radiologists with a thorough illustration and explanation of the range of central venous conditions in the thorax (including deep vein thrombosis, thoracic outlet syndrome, haemodialysis, and malignancy related causes), the salient imaging findings and interventional management using case examples from the authors' practice.

Introduction

Blood from the arms, head, and neck is drained via the central veins to the heart. For the purposes of this article, we consider the central veins to consist of the subclavian veins, innominate/brachiocephalic veins, and superior vena cava (SVC). The iliac veins, and inferior vena cava (IVC) comprise the central veins for drainage of the legs and abdominal structures and will not be discussed in this article. Central veins differ from peripheral veins in that they are larger, and have high flow rates with few or no valves. When functioning normally central veins have a single route of passage of blood. Collateral routes of flow only become apparent in the presence of disease in contrast to peripheral veins where blood can drain via several parallel routes. Disease in the central veins results in impairment of venous drainage. The clinical presentation of central venous disorders varies with the site, rate of onset, and aetiology of the disease. There are various anatomical variants of central veins in the thorax which are rarely associated with impaired function and are beyond the scope of this article.

An obstruction to flow of the central veins can be classified in a number of ways including aetiology of disease, anatomical location in relation to dependant area, or anatomical location in relation to lumen. For the purposes of this article we consider venous disease in relation to aetiology of disease.

Benign extrinsic compression

The most common site of extrinsic anatomical compression of central veins occurs in the subclavian vein as it crosses between the clavicle and the first rib. In its mildest form this can present with a feeling of fullness of the affected limb, often produced on exercise or forced abduction. This can be part of a global thoracic outlet syndrome with compressive symptoms affecting the adjacent artery and nerves. More commonly patients present acutely with Paget–Schroetter syndrome or effort thrombosis of the axillo-subclavian venous system. The aetiology of this is thought to relate to a congenitally small thoracic outlet exacerbated by hypertrophy of the subclavius muscle.

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Repeated movement on exercise causes repetitive trauma to the adjacent subclavian vein wall. Over time the vessel becomes stenosed. Damage to the intima may increase the thrombogenic effect of the stenosis with the end result of occlusive thrombus.

A sudden occlusion as a result of acute thrombosis will cause acute swelling and pain as there is no time for a collateral network to form. In its most extreme examples propagating thrombus may cause phlegmasia caerula dolens[1] (an acute fulminating form of deep venous thrombosis with reactive arterial spasm, pronounced oedema of the extremities, severe cyanosis, purpuric areas, and petechiae) with potential to cause irretrievable long lasting harm to the affected limb.

The mainstay of diagnosis of suspected thrombus in the acute situation is ultrasound. The diagnosis of thrombus within the axillary and deep veins of the arm is formed on the basis of echogenic thrombus causing distension and non-compressibility of the vessel with absent or reduced flow on colour Doppler. In most patients it is possible to image the majority of the subclavian vein with ultrasound. Occasionally anatomy is not conducive to ultrasound assessment of the subclavian vein, in which case cross-sectional imaging or venography may be required. In cases of intermittent venous obstruction without thrombosis, the diagnosis is usually made with a good clinical history and examination. Dynamic ultrasound or contrast-enhanced venography with the patient at rest and in forced abduction can show changes in flow.

The management of the Paget–Schroetter syndrome has been extensively discussed with several different conflicting views expressed. It is certainly best managed within a multidisciplinary team with input from radiologists and surgeons. One suggested course of management is the Exeter protocol.[2] Initial treatment with aspirin is followed by intravenous thrombolysis and heparin. A transaxillary rib resection and release of vein is then performed and followed by either aspirin or warfarin treatment for 3 months. At 2–3 weeks venography and venoplasty is performed. Venoplasty prior to surgical decompression is not advocated on this protocol because of a perceived increase in the risk of thrombosis following surgery. Treatment of subacute subclavian venous obstruction without thrombosis is similarly controversial. Most authorities recommend conservative measures initially to include weight loss, and cessation of activities that promote muscular hypertrophy (e.g., weight lifting, swimming, etc.). If symptoms persist then surgery to decompress the thoracic outlet can be considered. Endovascular intervention has little role to play here.

The case illustrated in Fig 1 is of a patient with a clinical presentation of acute left axillo-subclavian venous thrombosis. There was an underlying stenosis in the subclavian vein at the thoracic inlet and the predisposing event of a central venous catheter placed for an unrelated surgical procedure. The management was exclusively endovascular as the presence of extrinsic compression was felt to be a partial contributing cause.

Dialysis patients

Dialysis patients have a high incidence of venous disease ranging from damage to small peripheral veins from repeated peripheral cannulation, through to stenosis and occlusion of central veins. The rate of central venous disease in this population is thought to range from 25–40%.[3,4] Causes of central venous disease in these patients include (1) trauma to vessels from central venous cannulation, particularly relevant in the dialysis population due to the large calibre of dialysis catheter used; (2) trauma to vessels from small movements of a dialysis catheter tip within a vessel; this can be a constant ongoing process due to patient moving, breathing, and cardiac motion; (3) trauma to the vessel resulting from high flow and turbulent flow around the tip of a catheter during dialysis; and (4) high flow and turbulent flow as a consequence of peripheral AV fistula formation and graft placement.

Central venous disease can be silent, only presenting with symptoms when a new fistula or graft is placed as a result of higher blood flow through the diseased vessel. Line placement can also reveal silent central venous disease, by reducing the lumen through a pre-existing stenosis. Patients can present with a range of symptoms from mild limb swelling to an acutely swollen erythematous tender arm accompanied with head and neck swelling. Breast swelling and pleural effusion have also been described.[5] Dialysis via an arteriovenous (AV) fistula in the presence of central venous stenosis is usually poor with high recirculation rates and prolonged bleeding following dialysis. This is in contrast to the typical difficulties of needle access and poor pump speeds in a patient with a stenosis at the AV anastomosis.

In patients with fistula dysfunction ultrasound assessment of the fistula itself has long been known to be accurate and reproducible.[6] Fistulography is most often used for problem solving where ultrasound has failed to reveal a diagnosis and as a road map to subsequent intervention. Ultrasound is not sensitive or specific for assessment of the central veins, and venography via the fistula is usually the first technique used. It also has the advantage of allowing treatment in the same sitting.

Once the diagnosis of central venous stenoses has been made in a dialysis patient the initial management is usually balloon venoplasty. There appear to be two distinct types of central venous stenosis: non-elastic stenoses, which respond well to venoplasty, and elastic stenoses, which have high rates of immediate recoil and do not respond well.[7] This distinction cannot be made prior to balloon insufflation. Non-elastic stenoses usually respond to standard angioplasty balloons with good immediate result. Occasionally, high-pressure balloons can be required. Elastic stenoses have a high rate of immediate recoil following balloon venoplasty as judged by post-treatment venography. Significant proportions of these lesions are resistant to standard angioplasty balloons and require the use of balloons capable of high-pressure insufflation to 30 atm. Cutting balloons can also be used for resistant stenosis, but these are mainly used in the periphery in our institution