Case report

Subclavian cycle syndrome

P. SEN GUPTA\textsuperscript{1}, C. BOYTON\textsuperscript{2}, S. BAX\textsuperscript{1}, K. KHAN\textsuperscript{1}, Z. SIVARDEEN\textsuperscript{3}, S. ROWE\textsuperscript{4} and J.V. ANDERSON\textsuperscript{1}

From the \textsuperscript{1}Department of Diabetes, \textsuperscript{2}Department of Acute Medicine, \textsuperscript{3}Department of Orthopaedics and \textsuperscript{4}Department of Radiology, Homerton University Hospital, London E9 6SR, UK

Address correspondence to Dr P. Sen Gupta, Department of Diabetes, Homerton University Hospital, Homerton Row, London E9 6SR, UK. email: psengupta79@doctors.net.uk

Case report

A 37-year-old lady was admitted with a 5-day history of spontaneous swelling and pain affecting her right arm associated with skin tightening and discoloration. There was no recent trauma although she had undertaken 3-mile runs on the preceding 3 days. One year previously she sustained a comminuted right clavicular fracture following a bicycle accident, requiring internal fixation surgery. She regained full function of the arm subsequently. There was no other previous medical history or family history of thrombotic disease and her weight was stable. She smoked five cigarettes daily and took no regular medication.

On examination, she was slim, apyrexial, pulse 55/min regular, blood pressure 108/65 mmHg, respiratory rate 18/min. There was a general purple tinge and mottled appearance to the skin throughout her arm, which was swollen and had distended superficial veins (Figure 1). There was no arterial insufficiency, radio-radial delay or neurological deficit. There was no jaundice, anaemia, cervical or axillary lymphadenopathy, and breast, respiratory and abdominal examinations were normal.

Initial blood tests revealed: haemoglobin 14.0 g/dl, white cell count $8.1 \times 10^9/l$, platelets $147 \times 10^9/l$, normal electrolytes, international normalised ratio 1.0 and fibrin D-dimer 107 ng/ml (normal range 0–218 ng/ml). Electrocardiogram was normal and chest radiograph (Figure 2) showed normal cardiac and pulmonary appearances with a plate attached to the right clavicle. Doppler ultrasound imaging confirmed a hyperechoic thrombus within the subclavian vein with no Doppler colour flow, but normal axillary and brachial vein compressibility.

She was anticoagulated with low-molecular weight heparin followed by warfarin. A thrombophilia screen prior to anticoagulation was negative. It remained unclear why this subclavian vein thrombosis had occurred in a young woman with so few risk factors. For this reason, we performed focused computed tomography (CT; Figure 3). This revealed a clavicular screw extending beyond the inferior cortical margin of the clavicle causing right subclavian compression between it and the first rib. Her arm symptoms improved with anticoagulation, and corrective orthopaedic surgery is planned following the anticoagulation term.

Discussion

Upper limb deep vein thromboses (DVT) are important to diagnose and treat as serious sequelae
include disabling post-thrombotic symptoms. As with lower limb DVT, a cause must be sought to identify serious underlying pathology and prevent thrombosis recurring after anticoagulation cessation.

Subclavian vein thromboses can be classified as primary (20%) or secondary (80%) depending on pathogenesis.\(^1,2\) Primary effort-induced thrombosis is also termed ‘Paget-Schröetter syndrome’;\(^3\) secondary causes include thrombophilia, malignancy and local intervention, e.g. central vein catheters, pacemakers or orthopaedic devices, as in this case. That this patient’s clavicle fracture, fixation surgery and thrombosis were all right sided, suggested a possible association warranting investigation. Our report describes a case of subclavian thrombosis of likely combined primary and secondary aetiology, being effort induced from the patient’s repetitive running arm movements and secondary to clavicular screw impingement.

This case also highlights that clinical decision making should not hinge on the result of one test.\(^4\) The D-dimer-automated laboratory report stated ‘excludes venous thromboembolic disease (sensitivity 100%)’. However, the strong clinical suspicion of arm DVT justified definitive imaging. Other imaging modalities to employ if ultrasound had been negative or limited due to metal or bone proximity include venography, CT or magnetic resonance imaging.\(^1\) Ultrasound remains less invasive and more specific than venography, though also less sensitive and operator dependent.

Upper limb DVT accounts for an increasing proportion of all DVT cases,\(^2\) partly attributable to escalating central vein catheter usage. There are three reports in the literature of subclavian venous thromboses caused by clavicular fractures\(^5\) by bone fragment impingement mechanisms. However, this is the first case caused by a screw from the fixation device. It is important for clinicians to be aware of this pathology, as it may be a correctable cause of subclavian vein thrombosis.

**Conflict of interest:** None declared.
Figure 3. CT images. (A) Coronal image demonstrating the opacified cephalic vein draining into the subclavian vein, a short segment of which is not opacified due to thrombus. (B) Sagittal image demonstrating the unopacified vein inferior and anterior to the artery. (C) Sagittal image demonstrating the screw and 1st rib impinging upon the subclavian vein resulting in decrease in its calibre. Streak artefact from the metalwork is seen.

References