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An Unusual Case of Unilateral Upper Limb Deep Vein Thrombosis

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ABSTRACT

Subclavian vein thrombosis is rare upper extremity deep vein thrombosis that comprises 1- 4% or all DVT; it has a potential for considerable morbidity in the form of pulmonary embolism, persistent upper extremity pain and swelling, superior vena cava syndrome and loss of vascular access. Here we report a case of an active, healthy 60-year-old male who presented with left upper limb pain and swelling, his hypercoagulability profile was with in normal limit, but CT angiography revealed left subclavian vein thrombosis, he was successfully managed with anticoagulation, antiplatelet and statin therapy.

Keywords: Deep vein thrombosis, CT Angiography, Anticoagulation therapy.

INTRODUCTION

Upper extremity deep vein thrombosis (UEDVT) is an increasingly significant clinical condition with a high risk of significant morbidity. Up to one-third of individuals with UEDVT have a pulmonary embolism (PE). A UEDVT accounts for approximately 2% of all cases of deep vein thrombosis. Initially described by Paget in 1875 and Van Schroetter in 1884, it long felt to be a disorder associated with either trauma or spontaneous thrombogenesis after strenuous exercise or profound hyperabduction of the arm. Recently there has been an increasing incidence directly related to using central venous catheters for cancer chemotherapy, bone marrow transplantation, dialysis and parenteral nutrition.

CASE REPORT

A 60-year-old male presented to the emergency department with complaints of left upper limb pain, and swelling of left arm for 5 days, which started after strenuous exercise. No history of trauma or injury to upper extremities, no complaints of chest pain, dyspnea, cough, fever, bleeding manifestation, paresthesia, tingling sensation of upper extremities. He had no comorbidities or significant past medical history for coagulopathies, venous thromboembolism, pulmonary embolism. He is a nonsmoker and non-alcoholic. Vitals were BP- 120/700 mm Hg, pulse-84/ min, SpO2 -98%, afebrile, RBS-120 mg. On local examination, left arm of the patient was swollen, both the limbs were warm, and radial pulse was felt equally and capillary refill time was within normal limit in both upper limb. The range of motion of his shoulders, elbows and wrists was normal bilaterally, and deep tendon reflexes at elbows and wrists were intact. Routine blood investigations comprising of complete blood count, standard hypercoagulable screen available at our center was ordered. The screen showed he was negative for lupus anticoagulant, and that his levels of antithrombin, protein C and protein S were

all normal; neither did he carry Factor V Leiden or prothrombin allele mutations. The chest x ray was normal and no evidence of cervical rib or space occupying lesion at thoracic outlet was present, ECG and echocardiogram was normal, the venous doppler of the left upper limb showed thrombus in left subclavian vein. CT angiography of the upper extremity also showed thrombosis of left subclavian vein. Thus, a diagnosis of Paget Schroeder syndrome was made. He was treated initially with recommended dose of low molecular weight heparin and antiplatelet therapy with aspirin and statin therapy was instituted. Significant improvement in swelling of the affected limb was observed after a week of initiating the therapy with anticoagulant and antiplatelet drugs.

Fig 1: CT angiogram demonstrating left subclavian vein thrombosis

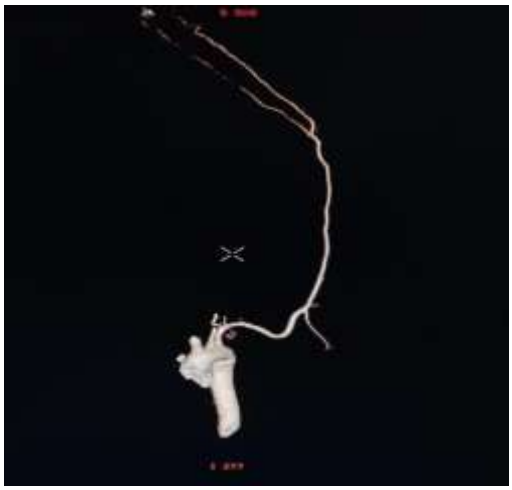

Fig 2: CT angiogram demonstrating left subclavian vein thrombosis.

CASE DISCUSSION

A rare primary thrombosis of the axillary-subclavian vein, Paget-Schroetter syndrome (PSS), is also called as "effort thrombosis." Paget and von Schroetter first described it in 1875 and 1884, respectively. In 1948, Hughes named the condition "Paget-Schroetter syndrome" [1]. One to two cases are thought to occur for every 100,000 people per year [2] Based on the pathogenesis, the upper extremity deep vein thrombosis (UEDVT) can be classified as primary or secondary. A rare condition, primary UEDVT affects two people per 100,000 annually [3]. When engaging in physical activities that involve abduction or hyperabduction of the upper extremity, such as rowing, wrestling, weight lifting, or painting, patients with Paget Schroetter syndrome typically have spontaneous UEDVT in the dominant arm. The pathogenesis includes the activation of the

coagulation cascade by exertion, microtrauma of the vascular intima, and stoppage of venous flow due to a reflex Valsalva manoeuvre.[4] Idiopathic UEDVT, in contrast to Paget Schroetter syndrome, may not have a recognised cause or visible underlying condition. Thoracic outlet blockage is the compression of the subclavian vein at its exit point at the thoracic inlet. This intermittent positional extrinsic vein compression can be brought on by the long transverse process of the cervical spine, the cervical ribs, musculoskeletal bands, and anomalies of the clavicle or first rib.[5] The prevalence of hypercoagulable disorders in UEDVT patients is unknown. These tests yield the highest in individuals with idiopathic disease, a favourable family history, recurrent DVT, or pregnancy loss. This is true, claims a recent study on the subject. In contrast to Paget Schroetter syndrome, idiopathic UEDVT may have no known trigger or obvious underlying disease. In one study, one-fourth of patients with occult cancer developed lung or lymphoma within one year of follow-up.[6] Uncertainty exists regarding the prevalence of hypercoagulable states in UEDVT patients. These tests yield highest in patients with idiopathic disease, a favourable family history, recurrent DVT, or pregnancy loss. According to a recent study, the most common finding was the presence of lupus anticoagulant or anticardiolipin antibodies (27%). In addition to these abnormalities, hyperhomo-cysteinemia, factor V Leiden, prothrombin gene mutations, protein C, protein S, and antithrombin III deficiency have also been reported on occasion.[7] The majority of thrombosis cases are secondary to UEDVT. Fewer than 3% of patients with pacemakers and central venous catheters who have UEDVT develop it in a clinically evident manner. The majority of UEDVT patients (33–60%) are asymptomatic. In a recent series, 63% of patients with known DVT of the upper extremities had malignant disease. [3] Patients with cancer with central venous catheters experienced increased DVT incidence. In these situations, the predisposing factor may be catheter-induced vessel wall damage and obstruction of blood flow through veins. [7] Patients who previously used temporary pacemakers, had low left ventricular ejection fractions (less than 40%), and had permanent pacemakers implanted had a significantly higher incidence of venous thrombosis.[8]

PSS's cause is unknown, but it is thought to be related to chronic subclavian vein compression near the thoracic outlet.[9] Venous distension, arm swelling, and agonising pain in the affected extremities comprise a typical symptom triad. Other symptoms include bluish discolouration and subcutaneous venous collaterals in the shoulder (Urschel's sign).[10] The first rib, the clavicle, and the costoclavicular ligament form the borders of the costoclavicular space, where the subclavian vein is compressed. This may cause scar tissue to form inside the vein, subsequently decreasing blood flow through the vein. The decreased blood flow allows for the production of blood clots, creating symptoms for the patient.[11] Doppler ultrasound has a sensitivity range of 78%–100% and a specificity range of 82%–100%, making it the first-line imaging modality of choice.[4] In patients with negative ultrasound results but a high index of suspicion for PSS or to rule out a structural obstructive cause like a cervical rib, computed tomography and magnetic resonance venography are helpful. Our patient's left subclavian vein displayed a thrombus on the venous doppler.[12] Recent research has shown that upper extremity deep venous thrombosis complications are common [9]. Most times, pulmonary embolism (8–36%) is asymptomatic. Compared to effort-related thrombosis of the subclavian veins, acute presentation is more frequently observed in DVT related to a catheter with underlying cancer. Recurrent thromboembolism can occasionally cause fatal right heart failure and pulmonary hypertension. In some circumstances, catheter removal and fibrinolytic therapy

may make fibrin emboli more likely to fragment, resulting in pulmonary embolism. Venous hypertension, which causes post-thrombotic syndrome in up to 50% of cases, can cause anything from minor oedema with little discomfort to severe limb swelling accompanied by pain and ulceration. The most common cause of recurrent thrombosis (2–15%) after stopping anticoagulation therapy is Paget Schroetter syndrome, and a recent series[13] has demonstrated that the risk of recurrence can be significantly decreased by using catheter-directed thrombolysis as soon as possible (within seven days) in conjunction with a first rib resection. Untreated UEDVT has a case fatality rate that can reach 10%, comparable to the case fatality rate for pulmonary embolisms from the lower extremities. A multidisciplinary approach to treatment is required, incorporating medical and surgical treatments. The cornerstone of treatment is anticoagulation. It promotes the patency of venous collaterals and prevents the thrombus from spreading. Lytic treatment minimises endothelial damage to vessels in the upper limbs, restores early venous patency, and lowers the incidence of post-thrombotic syndrome. According to a recent study, 92% of patients who underwent catheter-directed thrombolysis with recombinant tissue plasminogen activator (rtPA) as a continuous infusion of 1-2 mg/hour for at least 8 hours or reteplase at a dose of 1.0 unit/hour experienced thrombolytic success. Percutaneous mechanical thrombectomy using tools like the angiojet [14], surgical removal of a portion of the first rib or clavicle [9], residual stricture after surgery and thrombolytic therapy requires balloon angioplasty and stenting (52% in a recent study) to ensure maintenance of patency and superior vena cava filters can also be used.

Our patient presented with a five days history of left arm pain, followed by an acutely swollen arm; initially, our differential diagnosis included cellulitis, lymphedema, and neoplastic compression of the subclavian vein. He had no fever or obvious site of infection to suggest cellulitis; he had had no previous surgery or episodes of swelling to suggest lymphedema; and he had no risk factors and displayed no constitutional symptoms to suggest a malignancy. A CT angiogram and duplex ultrasound confirmed subclavian thrombosis, and an X-ray and CT ruled out both a tumour and a cervical rib as causes for the swelling. Our patient's thrombosis is likely related to his strenuous exercise, and he was advised frequent postural changes in the prevention of recurrence or contralateral involvement. He was treated with anticoagulants, antiplatelets and stains and a significant improvement in the symptoms was observed.

Declaration by Authors

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