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Case Report

Lower Limb Deep Vein Thrombosis in a Young Ambulant Teenager: An Uncommon Case with Multiple Etiological Causes

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Abstract

Deep vein thrombosis (DVT) is a type of venous thromboembolism with diverse clinical and environmental risk factors. Very few cases of DVT with multiple high risk factors have been reported. Here, we report an uncommon case of DVT in a 19-year-old female with multiple etiological causes. She was extensively evaluated and the etiological cause for DVT was found to be antiphospholipid antibody positivity with associated protein S deficiency and Factor V Leiden mutation. Anticoagulation was started with low dose warfarin. The International Normalized Ratio (INR) was monitored daily to adjust warfarin dose. Multiple etiological factors present in our patient may have contributed to her lower-limb DVT. Therefore, it is important to follow the complete workup for hypercoagulable states. This can help with diagnosis and therapy, and also give insight into the pathogenicity, which can help with prevention of recurrence and severe complications of DVT.

Keywords: Deep vein thrombosis; Antiphospholipid

Introduction

Deep vein thrombosis (DVT) causes chronic lower limb venous insufficiency which results in chronic leg swelling and ulceration. It can also be the cause of death through pulmonary embolism or may contribute to the genesis of pulmonary hypertension. Cellulitis, lymphangitits or ruptured Baker's cyst mimic deep venous thrombosis, hence requires a high index of clinical suspicion. Doppler and duplex ultrasonography remains the investigative modalities for deep vein thrombosis. Doppler ultrasound detects flow in the veins when they are compressed or when the patient performs a vasalva manoeuvre. Venogram is the gold standard in the diagnosis of DVT. We report a young, non-obese ambulant female who presented with features of deep vein thrombosis.

Case Presentation

A 19-year-old hypothyroid female, presented with pain and swelling in the left popliteal region and calf muscles of 2 weeks duration. There was no pain in any other joints. She denied skin lesions, recurrent urinary tract infections, oral/genital ulcers, chest pain, cough, syncope, palpitation, hemoptysis, photosensitivity or shortness of breath. There was no history of diabetes, hypertension, dyslipidemia, asthma or tuberculosis.

On examination, she was hemodynamically stable with blood pressure of 110/80mmHg; heart rate of 96beats/ minute and respiratory rate of 24/minute. Left lower limb was warm and swollen. Left popliteal region was tender. Left lower limb movements were restricted. There was no significant synovitis in any of the joints. Chest was clear. Air entry was bilaterally equal. She was tachycardic. Abdomen was soft and non-tender. She had normal power in all limbs. There were no focal neurological deficits.

Hemogram showed hemoglobin of 8.9g/dl with a total count of 6210/µL and platelets 2,03,000/µL. ESR was 34mm/hr. Liver and kidney functions were within normal limits. Pro-thrombin time was 14.8s. INR was 1.12. Chest imaging was normal. There was no evidence of any infiltrative lesions or hilar prominence. ECG showed sinus tachycadia. There was no evidence of any ischemic lesions or atrial fibrillation. Ultrasound doppler was suggestive of deep vein thrombosis involving popliteal and superficial femoral veins. Ultrasound abdomen was normal except for left follicular ovarian cyst. MRI left knee was normal apart from deep vein thrombosis. In regard to her deep vein thrombosis, she was anticoagulated with 80mg of low molecular weight heparin once daily. Later on, warfarin was initiated at low dose 5mg daily. The International Normalized Ratio (INR) was monitored daily to adjust warfarin dose until INR was stabilized at between 2 and 3.

Deep vein thrombosis in a young female of 19 years was looked into and she was worked up further for etiology. Both IgG and IgM antiphospholipid antibodies were more than 100 [strongly positive]. She had associated Protein S deficiency and Factor V Leiden mutation. Thus her deep vein thrombosis was predisposed by multiple factors – protein S deficiency, Factor V Leiden mutation and strongly positive antiphospholipid antibodies.

Discussion

The interplay of 3 processes resulting in venous thrombosis are known as Virchow's triad: 1) venous stasis or immobilization – e.g., caused by hospitalization for acute medical illness, nursing-home residence, long-haul travel, and paresis or paralysis; 2) hypercoagulability – e.g., caused by older age, active cancer, obesity, hormonal therapy, pregnancy, positive antiphospholipid, and personal or family history of venous thromboembolism [VTE]; and

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3) vascular damage – e.g., caused by surgery, trauma and central venous catheter or pacemaker [1,2].

Other factors that may promote DVT are factor V Leiden mutation, prothrombin G20210A gene mutation, deficiencies of anti-thrombin III, proteins C deficiency, protein S deficiency, and hyperhomocysteinemia [3]. The presence of antiphospholipid and lupus antibodies is also associated with an increased risk of venous thromboembolism [3].

Protein C and protein S, vitamin K dependent proenzymes synthesized in the liver, regulates hemostasis. They interact with thrombin- thrombomodulin complex on the surface of endothelial cells. Protein C becomes activated (activated protein C) after binding to these complexes. Protein S acts as a cofactor in this process. Activated protein C inhibits factor VIIIa and factor Va thus exhibiting its anticoagulant property and also enhances fibrinolysis through the inhibition of plasminogen activator inhibitor.

History of immobilization and an added risk factor such as the above-mentioned conditions is sufficient to make the diagnosis of DVT and start anticoagulation. It is rather a complex task to identify conditions promoting hyper-coagulability, such as factor V Leiden and prothrombin gene mutations, homocysteinemia, antiphospholipid syndrome, and congenital deficiency of factor S, factor C and anti-thrombin 3, as well as some of rare conditions such as plasminogen inhibitors. If these situations are present in a recessive fashion, a second risk factor is necessary to promote thrombogenesis.

Our patient had multiple factors. She was positive for antiphospholipid, antinuclear and anti ds DNA antibodies. She had protein S deficiency along with Factor V Leiden mutation.

Mutation of factor V Leiden makes factor V resistant to inactivation by activated protein C. This moderately increases risk for VTE by 3 to 8 times [4,5]. Deficiency of factor S, factor C, and anti-thrombin 3 normally prevent blood from clotting. This increases the risk of VTE by about 10 times in patients with factor V Leiden mutation [5].

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