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Case Report
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Thrombosis in a Bleeding Disorder - A Paradox

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Abstract

A 24-year-old female presented with swelling left thigh following a 5 days history of pain left hip and thigh region. She was diagnosed with left proximal deep venous thrombosis secondary to factor VII deficiency. She also had pus collection in left thigh region which got complicated by left hip osteomyelitis for which she underwent drainage of pus with orthotomy of left hip and received multiple broad spectrum antibiotics. Her deep venous thrombosis was managed with therapeutic doses of low-molecular-weight heparin with good tolerance. Factor VII deficiency being a rare bleeding disorder can present with wide spectrum of clinical manifestations especially the bleeding-thrombosis dyad. Our case report suggests that Factor VII deficiency has a paradoxical thrombotic predisposition.

Keywords: Thrombosis, Factor VII deficiency, DVT, Prothrombin time, Clotting factor

Introduction

Congenital factor VII deficiency is a rare bleeding disorder affecting 1 in 500000 individuals. It has a variable presentation with symptoms ranging from asymptomatic to a life-threatening bleeding episode [1,2]. Factor VII deficiency although being a bleeding disorder can paradoxically present with thrombosis (arterial or venous). We report a case of DVT in a patient who was found to have Factor VII deficiency after she was evaluated for an abnormal coagulation profile.

Case Presentation

A 24 years female, unmarried with no underlying comorbidities presented to us with 10 days history of pain and swelling left thigh. To start with she had insidious onset pain left hip, thigh region and after 5 days of onset of pain, she noticed swelling of left thigh followed by sensation of heaviness of left leg. Her pain was aggravated by walking and weight bearing and relieved by analgesics. She reported no history of trauma, insect bite, recent surgery or any drug intake (whether prescribed or over-the-counter). Her past history was unremarkable for any such episode. On examination she was afebrile with normal vitals and had a discrepancy in limb girth of 5 cm in left thigh compared to opposite side. She was diagnosed to have deep venous thrombosis. Her left thigh was warm and significantly tender to touch but there were no features suggestive of compartment syndrome. Later on she revealed that she was given some analgesic intramuscularly in left gluteal region by some local practitioner.

On admission her investigations were significant for normocytic normochromic anemia (hemoglobin 10.1; mean corpuscular volume 91 fl), neutrophilic leucocytosis (total leucocyte count 11.9k, differential leucocyte counts 88% neutrophils, 5% lymphocytes) and isolated prolongation of prothrombin time (39 seconds) with nomal activated partial thromboplastin time. Renal, liver function tests and electrolytes were within normal range. Blood gas analysis revealed respiratory alkalosis and her blood sugars and serum lactate levels were normal. Electrocardiogram, chest x-ray and ultrasound abdomen/pelvis were unremarkable. Ultrasound left thigh showed a filling defect in external iliac and common femoral vein. In view of isolated prolongation of prothrombin time with normal aPTT, factor VII deficiency was suspected which on confirmation came out to be deficient (25.2%). Prothrombin time mixing studies were significant for 100% correction of prothrombin time (after adding normal plasma, PT normalised from 39 to 13 seconds). USG left thigh revealed multiple small collections in anterolateral aspect of left thigh. MRI pelvis with left thigh showed features suggestive of myofascitis with left hip osteomyelitis (Figure 1 and 2). Her inflammatory markers at admission were significantly raised [erythrocyte sedimentation rate= 45mm/hr; c-reactive protein=68] which over the course of illness showed a downward trend. Pus for Gram-stain & culture sensitivity showed growth of multidrug resistant Escherichia.coli sensitive to ampicillin-sulbactam, tigecycline and polymixin-B.

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Figure 1 & Figure 2: MRI picture showing features suggestive of Myofascitis with left hip osteomyelitis (Arrows).

She was managed with therapeutic doses of low-molecular-weight heparin which she tolerated well and was discharged from hospital with uneventful recovery.

For lower limb DVT the patient received therapeutic doses of LMWH during the course of hospitalization and broad spectrum IV antibiotics for 8 weeks in view of her skin, soft-tissue and bone infection (left thigh myofasciitis with left hip osteomyelitis). She also underwent orthotomy of left hip joint with drainage of collection in view of left hip osteomyelitis.

On follow-up she was afebrile with normal hemodynamics with significant improvement in her appetite and well-being. Her low molecular weight heparin was changed to unfractionated heparin because of her financial constraints and she was shifted on oral antibiotics for two more weeks. Radiological assessment was repeated which showed mild stranding in anterolateral compartment of left thigh with no evidence of any collection. Her CBC showed normal TLC with normal differentials with improvement in Hb from initial of 10.9 g/dl to 12.6 g/dl and her inflammatory markers were significantly improved (ESR= 12; CRP= 7.5).

Discussion

Factor VII (proconvertin) is a clotting factor of human coagulation cascade synthesized by liver which is required for the conversion of prothrombin to thrombin in presence of thromboplastin. It is the only clotting factor with a small proportion of activated form (FVIIa) in the absence of activation of coagulation cascade [3,4]. Of all the vitamin K dependant clotting proteins, it has the shortest half-life (4-6 hours) and it's normal plasma level ranges between 0.35 to 0.6 mg/L. Plasma levels of factor VII are poorly correlated with the severity of clinical manifestations and clinical manifestations rarely appear when FVII:C is more than 30% [5-7].

Factor VII deficiency can be inherited or acquired. Among the rare inherited bleeding disorders, Inherited Factor VII deficiency is the most widespread one with an estimated incidence around 1 per 500000 in the general population and has autosomal recessive pattern of inheritance [2]. Inherited Factor VII deficiency was first described by Alexander et al. in 1951 [8]. Factor VII deficiency is characterized by prolongation of prothrombin time with normal aPTT [9-11]. It can present with a myriad of clinical manifestations ranging from a pure asymptomatic presentation to a mild mucocutaneous bleed to a life-threatening haemorrhage [12]. Rarely patients have presented with paradoxical arterial or venous thrombosis [13-16]. As per earlier case reports, thrombosis can occur in usual sites such as the deep veins of lower limbs or pulmonary embolism, or in atypical sites such as the sinusal veins [17,18]. The first case series of FVII deficiency associated with thrombosis included seven cases of venous thrombosis, localized primarily in typical sites (lower limbs and pulmonary embolism) [19]. Arterial thrombosis can occur in cardiac or cerebral territories or in large arteries like aorto-iliac axis [20].

The mechanism of thrombosis in Factor VII deficiency is poorly understood but some assumptions have been put forward regarding the same. An interesting assumption was based on a defective binding between the mutated FVII molecule and the Tissue Factor Pathway Inhibitor (TFPI) [4]. Thrombosis can occur without any triggering factor but majority of episodes have been reported to occur in the background of factor VII replacement therapy or any surgical procedure [21,22].

Conclusion

Factor VII deficiency is a rare bleeding disorder which can present with a broad range of clinical manifestations from being asymptomatic to a life-threatening bleeding episode. One of the rare presentations of this clinical entity is a bleeding-thrombosis dyad. Factor VII deficiency, though a bleeding disorder can even have a thrombotic predisposition, as suggested by our case report.

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