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Case Report

May Thurner Syndrome (MTS), A Rare Cause of Recurrent Lower Extremity Deep Venous Thrombosis (DVT)

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ABSTRACT

This case report highlights a rare cause of Deep Venous Thrombosis (DVT) of lower extremities. We report a case of 32 years old young male, who presented with recurrent history of unprovoked DVT. Initially treated as a case of idiopathic DVT, after extensive laboratory and radiological investigations diagnosis of May Thurner syndrome was established. It is essential for young physicians to keep such rare diagnosis in differentials while dealing with patients with unprovoked recurrent DVT.

Keywords: Deep Vein Thrombosis, May Thurner Syndrome, Thrombosis.

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INTRODUCTION

May Thurner Syndrome is a rare anatomical and pathological condition causing obstruction in flow of venous blood in iliocaval territory leading to DVT. Obstruction in flow is secondary to iliac artery overriding the iliac vein against the lumbar vertebrae.

DVT is defined as blood clot in venous system, commonly involved veins are pelvis and lower extremities. It is most common in lower extremities as compared to upper extremities. It can present as mild symptoms to life and limb threatening conditions. Pain in affected limb along with swelling, redness and enlarged superficial veins are amongst common presentations. If this clot dislodges it may cause pulmonary embolism, often fatal and life threatening if not diagnosed and managed timely.[1]

In 1856 Virchow proposed the pathogenesis of deep vein thrombosis as 'Virchow's triad' which includes venous stasis, injury to vessel walls and hyper coagulable states. Its incidence has been increased in last decade from 1/1000 to 1.5/1000 probably because of improved diagnostics. It is more common in male population than female.[2] As per literature many factors contribute in pathogenesis of DVT including trauma, surgical intervention, cancers, pregnancy, obesity, elderly population, immobility for longer period of time, hypercoagulable blood disorders like protein C and S deficiencies however anatomical defect was seen in our case of May Thurner Syndrome (MTS).[3]

A good clinical history, physical examination and well's score helps in diagnosis of this condition. Management includes treatment of underlying etiology and precipitating factors. Anticoagulants for 3-6 months are given for management. Prolonged or even lifelong anticoagulation may be required in cases with recurrent / unprovoked or ongoing risk factors for DVT.[4]

We are reporting this unique case as a precipitating factor for recurrent DVT.

CASE PRESENTATION

A patient 34 years married male, Government servant by profession admitted in our hospital from outpatient department with presenting complaints of shortness of breath and acutely increasing left leg swelling from last few days. He was diagnosed as a case of unprovoked proximal DVT in 2016 for which he took Warfarin for 6 months. His work up for secondary cause of DVT was unremarkable and DVT recovered completely without any residual disease. In 2017 he had another episode of unprovoked proximal DVT on same site. Again, he underwent extensive workup for thrombophilia screening, age appropriate tumor search, workup for APLA, and autoimmune profile including ANA, RA, Anti ENA without any positive workup. Detailed family history was noncontributory. He was advised to take lifelong anticoagulation with Vitamin K antagonist. He took Warfarin for 4 years without any adverse events and then stopped taking Warfarin on his own. DVT was partially recanalized with some residual swelling and leg discomfort.

He presented to us with acutely increasing left leg swelling and shortness of breath. On physical examination he was pale, and his left leg was swollen

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suggestive of DVT. Doppler USG lower limbs confirmed proximal DVT involving femoral and popliteal vessels. So, diagnosis of DVT with anemia was made and work up for any underlying malignancy was started. Extensive work up was done.

Table: Hematological and Radiological Workup of the Patient

Investigation(s)	Results
Complete blood counts	
Hemoglobin	8.30(13.00-17.00 g/dl)
Red Blood Cells	5.27(4.50-6.50 x 1012/L)
Total Leucocyte Count	6.30(4.00-11.00 x 109/L)
Platelets	200.00(150.00-400.00 x 109/L)
Peripheral smear	Hyper segmented neutrophils
Serum ferritin	117.80 (20.00-250.00 ng/ml)
Vitamin B 12	75.00(176.00-686.00 pmol/L)
Antithrombin	97.00(75.00-125.00%)
Factor V Leiden screening	0.95(>0.80)
ProC Global (Thrombophilia screening)	
Normalize Ratio PCAT PCAT/O	1 86.00(85.00-200.00 Sec) 44.00(35.00-55.00 Sec) Protein S and C deficiency not detected
PCR for JAK-2 Mutation	Negative
Anticardiolipin antibodies IgM, IgG	Negative
Homocysteine	15.00(5.00-15.00mol/L)
ENA profile	Negative
Anti Ds Antibodies	Negative
Anti B-2 microglobulin	2.80(1.00-2.80 mg/L)
HLA B51/5	Negative
Anti transglutaminase ab IgA, IgG	Negative
Gastric parietal cell antibodies	Positive ++
CD55/59	Deficiency not detected
Hemoglobin electrophoresis Hb A Hb A2	Bands of Hb A and Hb A2 seen 93.40(>96.00%) 6.60(2.00-3.30%)
Bone Marrow	Erythroid hyperplasia
CTV Lower limbs	Non Opacification of left common iliac, external iliac, common femoral, deep femoral and superficial femoral, suggestive of deep venous thrombosis Prominent superficial vessel lower abdomen, pelvis and left leg representing collateral formation
CECT NCAP	Compressed left common iliac vein against vertebral body by right common iliac artery and attenuated caliber of left common, external, internal iliac veins suggestive of Chronic DVT and May Thurner Syndrome as shown in Image-I

PCAT: Protein C Activation Time, PCATO: Protein C Activation Time without Activator, PCR: Polymerase Chain Reaction, ENA: Extractable Nuclear Antigen, HLA: Human Leucocyte Antigen, CTV: Computed Tomography Venography, CECT NCAP: Contrast-Enhanced Computed Tomography of the Neck, Chest, Abdomen, and Pelvis

Based on laboratory and radiological investigations as given in Table final diagnosis of May Thurner syndrome and pernicious anemia was made.

Interventional radiologist and vascular surgeon consultation was done for possible intervention to correct the anatomical defect. An MDT involving Vascular surgeon, interventional radiologist, clinical hematologist and pulmonologist was carried out. Due to extensive DVT and formation of collateral circulation, no intervention was possible by IR team or vascular surgeons. Due to in correctable nature of risk factor, he was advised to continue lifelong anticoagulation and replacement of Vit B-12 (Cobalamin) on monthly basis.

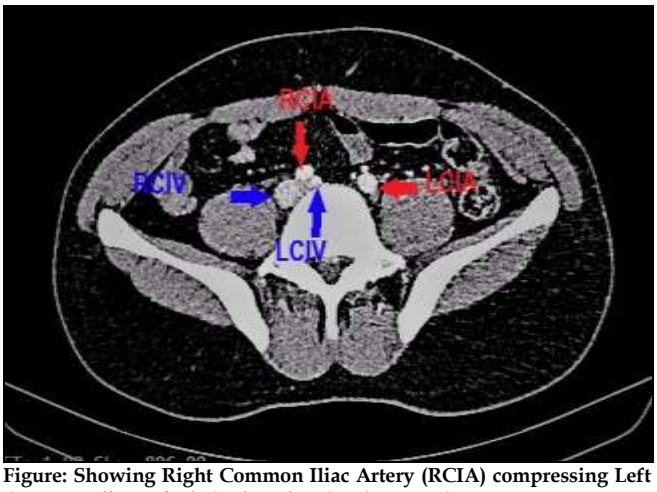

Figure: Showing Right Common Iliac Artery (RCIA) compressing Left Common Iliac Vein (LCIV) against lumbar vertebra

Currently patient is tolerating anticoagulation well and under regular follow-up.

DISCUSSION

MTS is one of the rare causes of DVT. Interestingly, MTS is 3-5 times more common in female genders compared to male population. In MST right common iliac artery overrides and compress left common iliac vein against the lumbar vertebral spine. It was first described by two researchers May and Thurner in 1957. This pulsatile compression of artery to vein leads to constant disturbance of blood flow leading to abnormal proliferation of endothelium of vein and development of spur within common femoral vein causing stasis and thrombosis.5 Patients may be asymptomatic and sometimes present with complaints of claudication and and swelling without underlying DVT.[6] A study was conducted by Negus et al. on cadavers in which 14% of 100 cadavers which showed this anatomical variant and formation of fibrous band secondary to compression of venous results in stasis and thrombosis.[7]

In 1965 the two researchers Cockett and Thomas first time studied alive patients with DVT secondary to this anatomical variant (MTS). According to them once the spur has developed, the condition becomes irreversible and most of the patients remain asymptomatic due to formation of collaterals.8The modalities of choice in diagnosis are doppler ultrasound as initial investigation for DVT. Contrast venography is gold standard for diagnosis. Noninvasive techniques including computed

tomography (CT) and magnetic resonance (MR) venography also helps in diagnosing cases of MTS. Intravascular ultrasonography of involved vessels along with pressure measurements are confirmatory.[9]

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Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

AR & KA: Data acquisition, data analysis, critical review, approval of the final version to be published.

AK & ARA: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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