ejpmr, 2016,3(7), 347-349.

SJIF Impact Factor 3.628

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Report

ISSN 2394-3211

EJPMR

MAY THURNER SYNDROME – UNUSUAL CAUSE OF LEFT LOWER LIMB VARICOSITIES.

[1]Dr. Harshwardhan Shrotri, [2]Dr. Anubhav Kamal, [3]Dr. Rajesh Kuber and [4]Dr. Vigyat Kamal*

[1]Asst. Professor, Department of Radiology, Dr. DY Patil Medical College & Research Center, Pimpri, Pune.
[2,4]PG Student, Department of Radiology, Dr. DY Patil Medical College & Research Center, Pimpri, Pune.
[3]Professor, Department of Radiology, Dr. DY Patil Medical College & Research Center, Pimpri, Pune.

***Corresponding Author: Dr. Vigyat Kamal**

PG Student, Department of Radiology, Dr. DY Patil Medical College & Research Center, Pimpri, Pune.

Article Received on 03/05/2016 **Article Revised on 23/05/2016** **Article Accepted on 13/06/2016**

ABSTRACT

Deep venous thrombosis of lower extremity is a well-known entity having multiple causes. Apart from usual thrombotic causative factors, atypical sources do exist and pose a challenge to both clinicians and radiologists. It is essential to diagnose anatomical vascular variations such as May Thurner syndrome to suggest further diagnostic and treatment options for younger age group and prevent morbid complications like pulmonary embolism.

KEYWORDS: May-Thurner Syndrome (MeSH unique ID: D062108), Venous Thrombosis (MeSH unique ID: D020246), Thrombectomy (MeSH unique ID: D017131), Thrombophilia (MeSH unique ID: D019851).

INTRODUCTION

Iliac vein compression syndrome is also known as May-Thurner syndrome, iliocaval compression syndrome or Cockett syndrome. It is the compression of the left iliac vein by the right sided iliac artery, thereby increasing the chances of deep vein thrombosis in the left lower limb. Usual affection of younger age group females of approximate age 20 to 40 years has been observed. Suspicion due to the clinical presentation of persistent painless left lower extremity swelling is an indicator for CT and iliac venography.[1]

CASE REPORT

A 22 year old young female presented with left sided pedal oedema. She was treated with debridement of non-healing ulcer on left sided medial malleolus and endovenous laser ablation of left sided great saphenous vein 3 years back. Left lower limb venous Doppler showed varicosities around left ankle.

CT inferior venacavography with bilateral lower limb venography was performed which revealed normal inferior vena cava in terms of calibre and patency.

Right common iliac, right internal iliac and right external iliac veins were normal. Left common iliac vein was markedly stenosed from its origin due to overlying right sided common iliac artery (**Figure 1, 2**). A few cross over collateral veins were seen in the pelvis and on the anterior abdominal wall (**Figure 3**).

Bilaterally, the infra-inguinal deep veins were patent and appeared normal. Few varicosities were seen along lateral aspect of the left leg and distal thigh. Sub-cutaneous oedema was noted in the distal leg on left side.

Figure 1

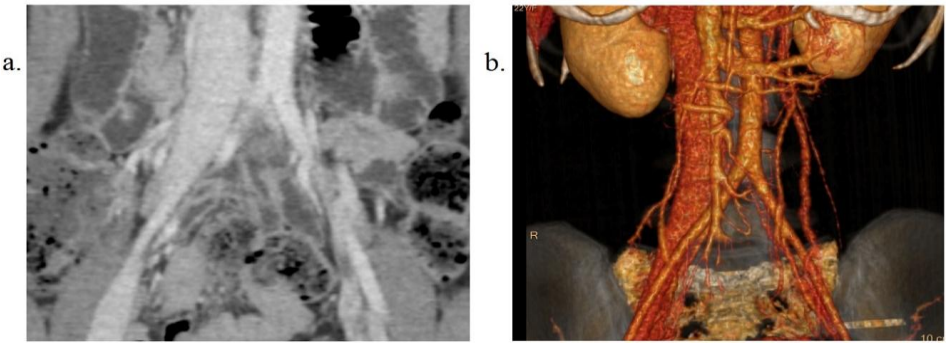

Figure 1: Coronal contrast CT and volume rendered image showing compressed left common iliac vein due to overlying right common iliac artery.

Figure 2

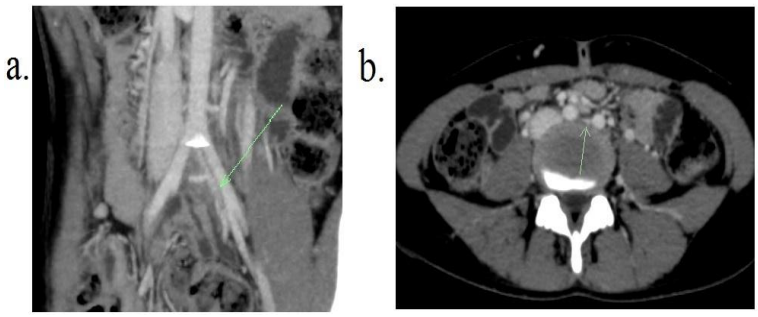

Figure 2: Coronal and axial contrast CT showing compressed and stenosed left common iliac vein (arrow) due to overlying right common iliac artery.

Figure 3

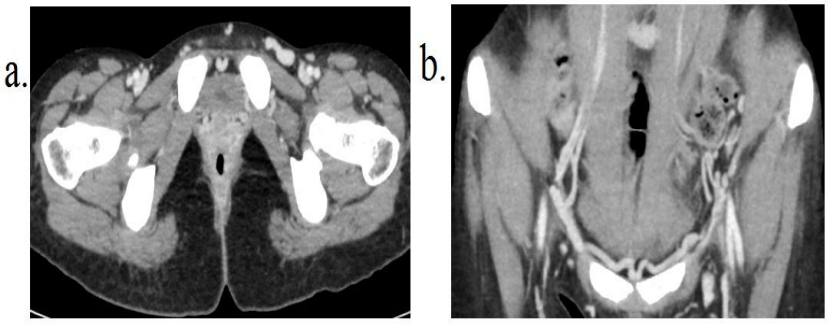

Figure 3: Axial and coronal contrast CT showing collateral veins in the anterior abdominal wall and in the pelvis.

DISCUSSION

Iliac vein compression syndrome is a clinical entity in which there is extrinsic compression of the left common iliac vein by the right common iliac artery. Patients present with complaints of pain and diffuse swelling in the left lower limb or as left sided iliofemoral venous thrombosis.[2]

Virchow first described the condition in 1851 due to an observation of the left sided prevalence of iliofemoral deep venous thrombosis secondary to venous stasis. Left iliac vein compression with anatomical descriptive details was later described by May and Thurner.[3]

Accurate incidence is not known, however, it is noted from 22-32% of autopsy studies. Prevalence of May Thurner syndrome related deep venous thrombosis is 2-3% of all lower extremity deep venous thrombosis and is seen in females between 20-40 years of age.[4]

It is noted in 2-5% patients with venous diseases of the lower limbs.[5]

Pathogenesis

Two accountable factors have been postulated to cause this condition. First being intrinsically formed internal webs or bands (spurs) secondary to local intimal hypertrophy due to the chronic pulsatile force of the overlying right sided common iliac artery and second being extrinsic physical compression of left common iliac vein between right sided common iliac artery and the underlying lumbar vertebral body.[6]

Repeated transmitted arterial pulsation causes scrubbing of the walls of the vein against each other leading to endothelial irritation. Over the course of time, the irritation causes proliferation and formation of the band (spur) as well as partition of the lumen.[7]

It is a type of partial obstruction which has varying consequences like leg swelling, varicosities, deep venous thrombosis and chronic venous stasis with ulcers or more grave complications like pulmonary embolism, phlegmasia cerulea dolens which is characterized by acute limb ischaemia later leading to gangrene.[7]

There are cases where patients remain asymptomatic and no treatment is needed. Terminology of May Thurner syndrome is reserved for cases of venous stasis due to compression, repetitive intimal endothelial injury resulting from arterial pulsations.[8]

May Thurner syndrome consists of three morphological variations: focal extrinsic compression by overlying iliac arteries, diffuse atrophy and cordlike obliteration. Knowledge of morphological types may be helpful for analysing endovascular technical challenges.[8]

May Thurner syndrome has been found to be an infrequent cause of varicose veins as well as the formation of intra-abdominal collaterals. These collaterals may be retroperitoneal and pudendal veins which develop as a counter regulatory response to venous stasis shunting distal iliofemoral venous system to contralateral deep venous vessels.[8]

Comprehensive clinical details, physical examination and diagnostic work-up including thrombophilia should be performed to evaluate deep venous thrombosis and its risk factors in young patients. Diagnosis with simple Doppler ultrasonography is difficult as iliac vessels are deep seated. Therefore, diagnostic evaluation ultimately relies upon computed tomography venography and magnetic resonance venography.[4]
Multi detector computed tomography localizes the clot with its extent and evaluates iliofemoral anatomy along with lower limb vasculature. Multi detector computed tomography is essential for planning endovascular procedures.[8]

Computed tomography venography and magnetic resonance venography show greater sensitivity for assessing deep venous thrombosis. Magnetic resonance venography shows better analysis of intraluminal thrombus and differentiates it from contrast mixing. These allow localization of sites of venous compression as well as pelvic venous collaterals. Magnetic resonance imaging has the advantage of evaluating haemodynamic significance of venous compression as it shows retrograde flow in the ipsilateral iliac vein and collateral veins.

Left common femoral or iliac venography not only provides details of venous obstruction, but also offers haemodynamic assessment analysis and the treatment to be offered simultaneously.

Reversal of flow in the ipsilateral internal iliac vein, presence of multiple cross pelvis collaterals and enlarged ascending lumbar vein are noted secondary to underlying chronic venous hypertension.[3]

Management is dependent on the presence or absence of deep venous thrombosis. In the absence of thrombosis, compression stockings may be effective. In the situation of acute deep venous thrombosis, anticoagulation is the first line treatment. Treatment of venous compression uses a combined approach of self-expanding stents and endovascular thrombolysis with tissue plasminogen activator.

Surgical treatment involves iliofemoral vein thrombectomy with iliac venous reconstruction. Another alternative is cross femoral venous bypass graft (Palma procedure). Patients are put on long term anticoagulants along with stockings.[3]

CONCLUSION

May Thurner syndrome is an important differential causing unilateral deep venous thrombosis, especially in younger age groups. Diagnosing the condition is essential failing which there are recurrent episodes of thrombosis and pulmonary embolism leading to substantial morbidity and mortality. Treatment entirely depends on precise anatomic localization of the lesion along with clot removal procedure.

Source of support: Nil.

Conflict of interest: Nil.

REFERENCES

1. Duran C, Rohatgi S, Wake N, Rybicki FJ, Steigner M. May-thurner syndrome: a case report. Eurasian J Med., 2011 Aug; 43(2): 129-31. PMID: 25610179.
2. Cavalcante LP, Souza JEDS, Pereira RM, Bernardes MV, Amanajás AMDS, Parisati MH et al. Iliac vein compression syndrome: literature review. J Vasc Bras., 2015 Jan.-Mar.; 14(1): 78-83.
3. Butros SR, Liu R, Oliveira GR, Ganguli S, Kalva S. Venous compression syndromes: clinical features, imaging findings and management. Br J Radiol. 2013 Oct; 86(1030): 20130284. PMID: 23908347.
4. Kalu S, Shah P, Natarajan A, Nwankwo N, Mustafa U, Hussain N. May-thurner syndrome: a case report and review of the literature. Case Rep Vasc Med., 2013; 2013: 740182. PMID: 23509664.
5. Kalu S, Shah P, Natarajan A, Nwankwo N, Mustafa U, Hussain N. May-thurner syndrome: a case report and review of the literature. Case Rep Vasc Med., 2013; 2013: 740182. PMID: 23509664.
6. Lamba R, Tanner DT, Sekhon S, McGahan JP, Corwin MT, Lall CG. Multidetector CT of vascular compression syndromes in the abdomen and pelvis. Radiographics., 2014 Jan-Feb; 34(1): 93-115.PMID: 24428284.
7. Cil BE, Akpinar E, Karcaaltincaba M, Akinci D. Case 76: May-Thurner syndrome. Radiology., 2004 Nov; 233(2): 361-5. PMID: 15516613.
8. Wu WL, Tzeng WS, Wu RH, Tsai WL, Chen MC, Lin PC et al. Comprehensive MDCT evaluation of patients with suspected May-Thurner syndrome. AJR Am J Roentgenol., 2012 Nov; 199(5): W638-45. PMID: 23096209.