

Endovascular management of compression syndromes associated with pelvic venous disease

Marald Wikkeling¹ , Houman Jalaie² ¹Department of Vascular Surgery, Heelkunde Friesland, Locations MCL and Nij Smellinghe Ziekenhuis, Friesland, The Netherlands²Department of Vascular Surgery, University Hospital Aachen, Aachen, Germany

ABSTRACT

Compression syndromes play an important role in pelvic venous disease (PeVD). Although compressive lesions of the common iliac and renal veins are common on non-invasive imaging studies, they are usually asymptomatic and the factors associated with the development of symptoms remain poorly understood. The prevalence of pelvic or lower-extremity symptoms and varicose veins caused by compression of an iliac or left renal vein is unknown. However, a high index of suspicion is needed in the work-up of PeVD, particularly in patients who are refractory to the initial treatment. Once a symptomatic compression syndrome is discovered in a PeVD patient, this should be treated first. After treating compression syndromes, there should be a waiting period to notice if the complaints and symptoms diminish.

Keywords: May-Turner, Nutcracker, PeVD, venous compression syndromes.

Since the publication of the important body of work by Meissner et al.,^[1] a new “helicopter view” approach and classification has been published in May 2021 on the topic of pelvic venous disease (PeVD). Herein, we refer to the Chapter by Tobias Hirsch and Suat Doganci in this publication.

The pathophysiology of the venous disorders can be caused by reflux, obstruction, compression, or a combination of all. In this chapter, we focus on endovascular management of compression syndromes associated with PeVD.

ANATOMY

There are two crucial areas of the venous pelvic and abdominal system where compression can be located, leading to venous insufficiency and causing disabling symptoms.

1. Compression of the left common iliac vein: May-Thurner (Figure 1).

There is compression of the left iliac vein by the right iliac artery, which crosses over it. This is normal anatomy, but the artery presses on the vein enough to thicken the vein wall over time in selected individuals. This condition, in due time, can cause mechanically induced compression, which results in an intraluminal fibrotic ribbon- or mesh-shaped vessel change, obstructing the venous outflow.

Isolated left lower extremity swelling secondary to left iliac vein compression was first described by James Playfair McMurrich (1859-1939), Professor of Anatomy at the University of Michigan, in 1908, and defined anatomically by May and Thurner in 1956 and clinically by Cockett and Thomas in 1965. This condition is

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Correspondence: O.R.M. Wikkeling, MD, MBA, Heelkunde Friesland Locations: Hospital Nij Smellinghe Drachten and MCL, Leeuwarden. The Netherlands.
e-mail: owikkeling@heelkundefriesland.nl

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called May-Thurner syndrome after the Austrian pathologist's May and Thurner.^[2-6] Therefore, the term May-Thurner syndrome should be reserved for patients with typical clinical symptoms associated with demonstrable May-Thurner morphology.

2. *Compression of the left renal vein (LRV): Nutcracker (Figure 2).*

Compression of the LRV causes impeded outflow from the LRV into the inferior vena cava due to the extrinsic LRV compression, often accompanied by

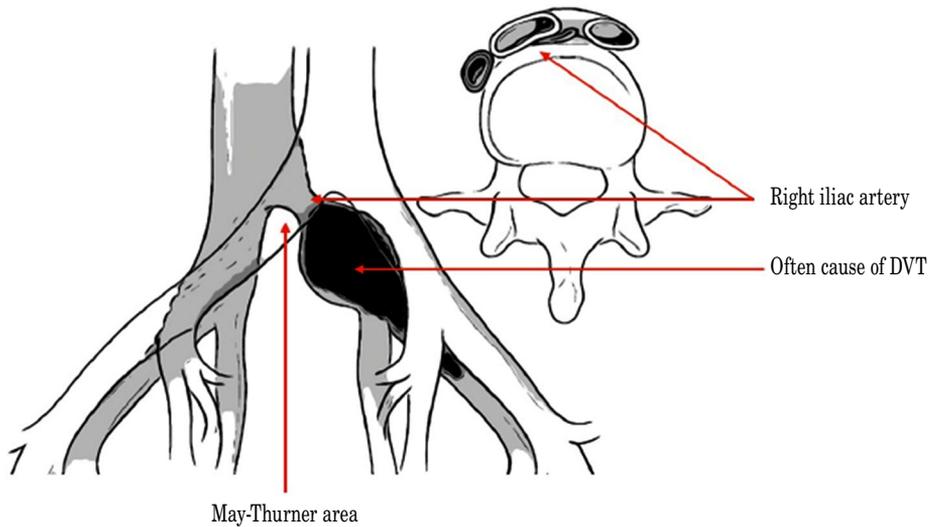


Figure 1. May-Thurner.

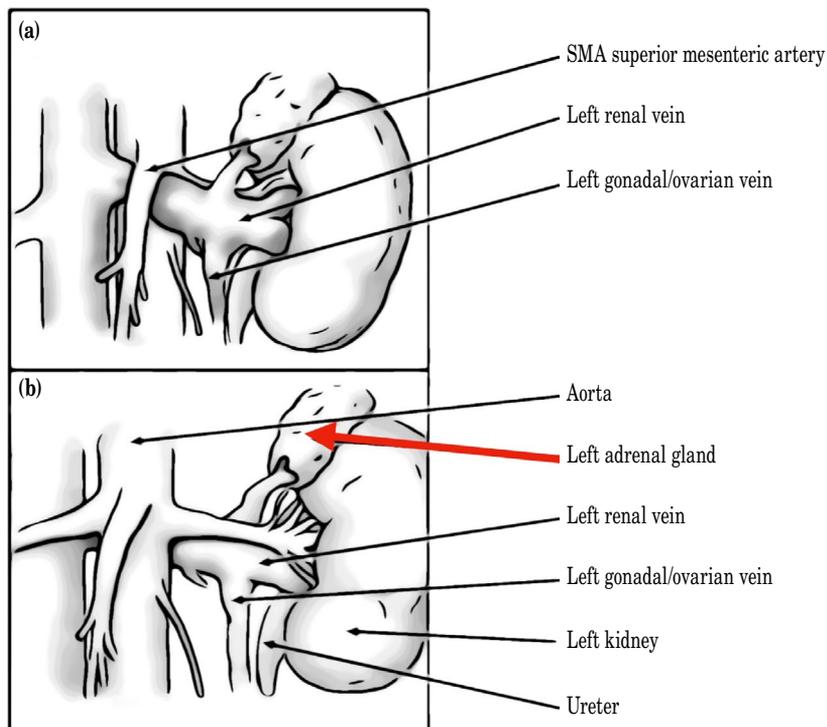


Figure 2. Nutcracker. (a) Left renal vein underneath SMA anterior Nutcracker syndrome. (b) Left renal vein posterior aorta posterior Nutcracker syndrome.

demonstrable lateral (hilar) dilatation and medial (meso-aortic) narrowing. However, the terms Nutcracker syndrome and Nutcracker phenomenon are sometimes used interchangeably in the literature.^[7,8] Emphasize that the Nutcracker anatomy is not always associated with clinical symptoms and that some of the anatomic findings suggestive of Nutcracker may represent a standard variant or be accounted for by other conditions. Therefore, Nutcracker syndrome should be reserved for patients with typical clinical symptoms associated with demonstrable Nutcracker morphology.

There are two types of Nutcracker compression:

B1. Entrapment of the LRV underneath the superior mesenteric artery; anterior Nutcracker syndrome (Figure 2a).

B2. Entrapment of the LRV posterior to the aorta. Posterior Nutcracker syndrome (Figure 2b).

Both compressions (A and B) can cause entrapments of the LRV leading to a higher venous pressure upstream to the narrowing. This can result in venous insufficiencies and higher flow in the collateral veins; e.g., the vena ovarica on the left side and associated symptoms and complaints.

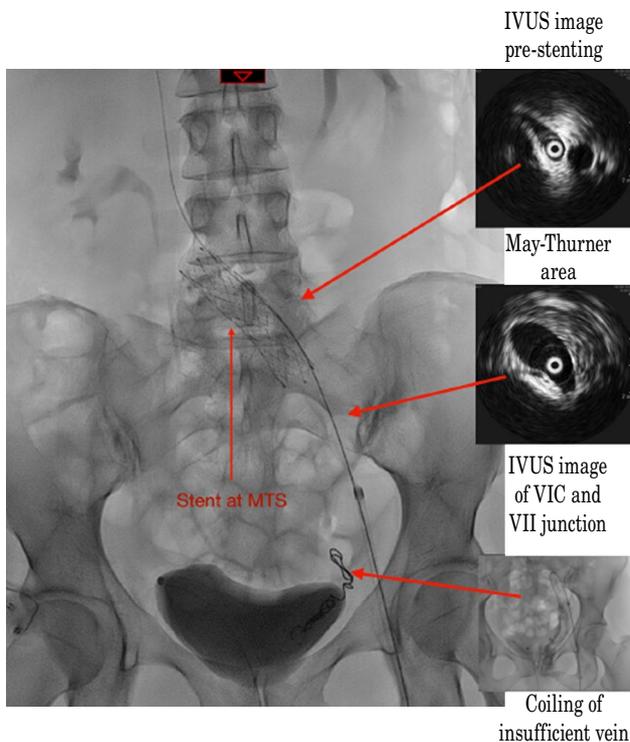


Figure 3. May-Thurner and pelvic vein insufficiency. IVUS: Intravascular ultrasonography; VIC: Common iliac vein.

Treatment of Compression Syndromes:

Treatment should be based on the severity of symptoms and their expected reversibility concerning the patient's age and stage of the syndrome. Both open surgery and endovascular treatments are feasible. However, in this day and age, the paradigm has shifted to endovascular treatment options as the first-line choice.

Compression syndromes associated with PeVD. Which should be treated first? Compression syndrome or venous insufficiencies?

In the beginning of the 20th century, the paradigm shifted to treating the compression syndrome first and, after a waiting period (at least three months) notice whether venous insufficiencies complaints subside. If not, in the second procedure, treat venous insufficiencies.^[9,10]

May-Thurner

With endovascular treatment of May-Thurner syndrome, a durable primary patency can be achieved.^[11] Self-expanding stents are preferred that should allow for strength and flexibility with high radial force. However, foreshortening or precise placement can be challenging and requires experience. Nitinol stents can be precisely delivered, as there is no foreshortening during deployment.

Hager et al.^[12] evaluated mid- and long-term patency rates of endovascular stents in patients treated with symptomatic and non-thrombotic May-Thurner syndrome. Their results were concordant with previous reports showing favorable patency rates: 91% at 36 months in patients who presented determinants of

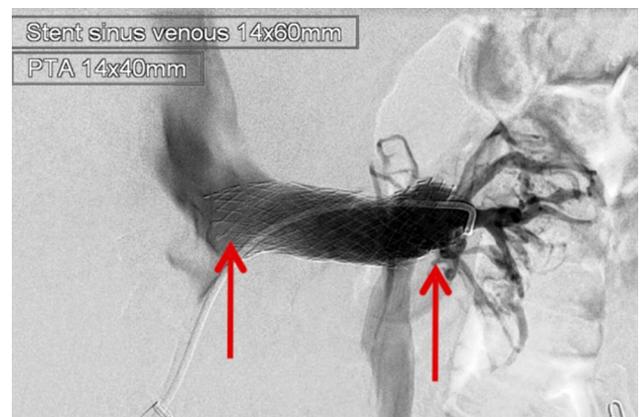


Figure 4. Nutcracker, endovascular treatment of left renal vein. Courtesy of Prof. de Haan, Dept. of Radiology MUMC+.

long duration of pain and swelling, but no deep vein thrombosis and 91% at 36 months for patients who presented in a post-thrombotic state.^[12,13] Stenting was found to be favorable in both groups. These findings demonstrate that stent patency is not affected by the ongoing extrinsic compression. Hypercoagulability, residual thrombus affecting overall flow, and anticoagulation may be determinants of long-term patency in certain individuals (Figure 3).^[12,13]

Nutcracker

Nutcracker syndrome is a very common condition in >40%^[14] of women with other forms of pelvic vein disease, such as May-Thurner syndrome and pelvic congestion. Both conditions are anatomic variants in the structure of abdominal veins that predispose the patient to all three issues.

When to treat Nutcracker:

In the guidelines, these five factors should be addressed:^[15]

1. At least three episodes of hematuria.
2. A significant reno-caval gradient (measured at venogram) demonstrates a significant pressure difference between the left kidney vein and vena cava.
3. Severe narrowing of the left kidney vein on venogram and ultrasound.
4. Absence of other causes of hematuria such as high blood pressure and diabetes.
5. Patient desire to treat his/her Nutcracker syndrome.

How to treat:

As previously mentioned, there are three main strategies for the treatment of Nutcracker syndrome:

- Open surgery
- Laparoscopic and robotic surgery
- Endovascular approach

Currently, the latter is the first option of treatment. Endovascular stenting is preferentially used in the treatment of Nutcracker syndrome which is associated with pelvic congestion.^[16] However, endovascular treatment is not without risks, with complications such as incorrect stent placement requiring a surgical intervention,^[17] stent migration (e.g., to the right atrium), partial stent dislodgement into the infrarenal cava, and stent migration into the hilar region of the LRV.^[18] This highlights the importance of adequate stent (little over-) sizing (Figure 4).

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