Current management approach for left renal vein entrapment syndrome: the so-called 'Nutcracker' syndrome

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Introduction: James T McPhee1 & Matthew T Menard1 1Brigham & Womens Hospital, Division of Vascular and Endovascular Surgery, 75 Francis St., Bost (SMA) and the abdominal aorta, as it traverses between these two structures toward the inferior vena cava was first described in 1937 [1]. In certain instances, compression of the vein may lead to clinically relevant dilatation of its distal aspect (Figure 1). The important features associated with this unique anatomic relationship were recognized and ultimately given the moniker the 'nutcracker' phenomenon in the subsequent decades [2–4]. An analogous syndrome has been described in patients with an anomalous retroaortic LRV, whereby the vein is compressed between the abdominal aorta and the vertebral column [3]. With this new description, it has become necessary to describe the SMA compression of an ante-aortic LRV as 'anterior nutcracker syndrome' and the compression of a retroaortic LRV as 'posterior nutcracker syndrome'. With just ten known cases of the latter syndrom, first described in 1986 [5], the anterior configuration remains far more commonly observed [6].
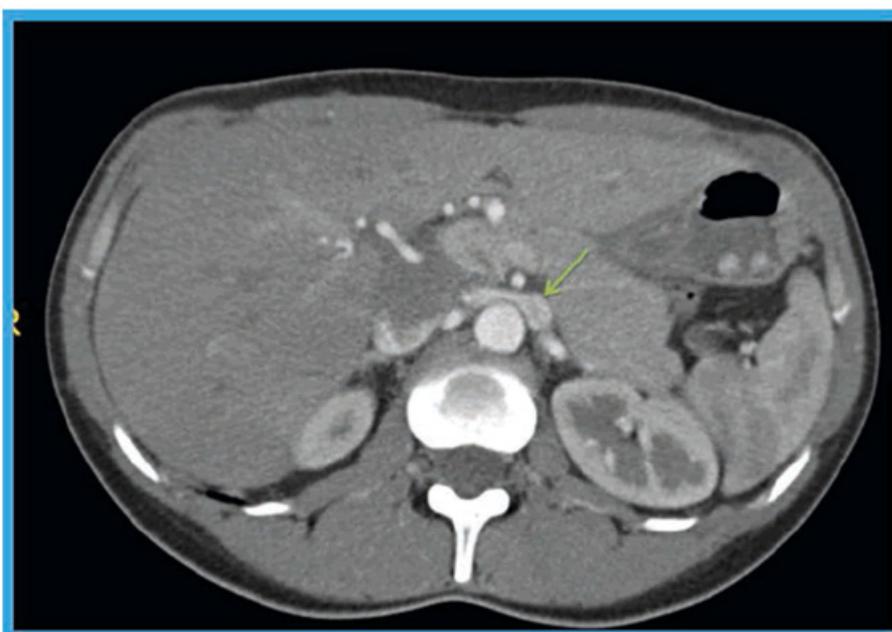

Figure 1. Axial computed tomography angiogram demonstrates left renal vein compression with dilatation of the distal renal vein.

The first description of surgical repair of the venous ouflow obstruction emerged in 1974 [7], and since that time, a logical approach to the evaluation and diagnosis of suspected LRV entrapment has evolved. In part given its relatively rare incidence, what remains unexplained is the variable clinical penetrance of a somewhat common radiographic abnormality. As up to 50–75% of asymptomatic children and adults may demonstrate some degree of LRV entrapment and dilatation on axial imaging surveys without clinical sequelae, clearly an unknown variable exists that predisposes some patients to manifest symptoms of renal venous hypertension while others do not [8,9]. Further to this point, it is well-known that elective ligation of the LRV during abdominal aortic aneurysm repair is well-tolerated in most circumstances [10], as is the elective procurement of the LRV for use as a bypass conduit in abdominal vascular reconstructions [11,12].

In addition to the evolution of diagnostic maneuvers that have helped to clarify the clinical relevance of radiographic findings in a given patient, the surgical management of LRV entrapment has similarly evolved. Approaches have ranged from a creative array of major open abdominal vascular reconstructions to, more recently, less invasive approaches such as external stenting, SMA elevation techniques and laparoscopic decompression. Concurrent advancements in endoluminal therapy in general and endovenous interventions for left iliac vein compression and venous thoracic outlet obstruction in particular have paved the way for percutaneous treatment of this anatomically analogous external mechanical compression of the LRV.

Clinical presentation

The symptom complex associated with nutcracker syndrome relates to relative venous hypertension of the LRV. The syndrome is most commonly observed in thin young men and middle-aged women. It typically manifests as left-sided flank pain and hematuria, which may range in severity from a microscopic finding observed on urinalysis to gross hematuria with an ongoing transfusion requirement [13]. The hematuria is likely due to microvascular rupture of thin-walled renal pelvic and periureteral varices [14,15] and may be intermittent in nature, perhaps related to a relative increase of the venous hypertension, which may worsen symptoms after periods of physical exertion or while in an upright position [16], which has been shown radiographically [17]. Additional symptoms that have an underlying anatomic explanation relate to collateral drainage venous hypertension, but are not universally present in patients with LRV compression. Most notably, elevated

pressure in the gonadal venous circuit may ultimately lead to reflux, dilatation (Figure 2), collateralization and variceal formation. The presence of gonadal vein involvement is a hallmark feature of the so-called 'pelvic congestion syndrome' (PCS), which manifests as left-sided varicocele in young men and chronic lower abdominal or pelvic pain and dyspareunia in middle-aged women (Box 1) [18,19]. A minority of patients with PCS will have overlapping symptoms attributable to LRV compression. A study by Scultetus et al. reviewed a series of 51 female patients with symptoms of PCS (gluteal, vulvar and lower extremity varices), nine of which (17%) had additional symptoms of hematuria and left flank pain [19]. They concluded that patients that have PCS in addition to hematuria may benefit from a diagnostic evaluation of possible LRV compression.
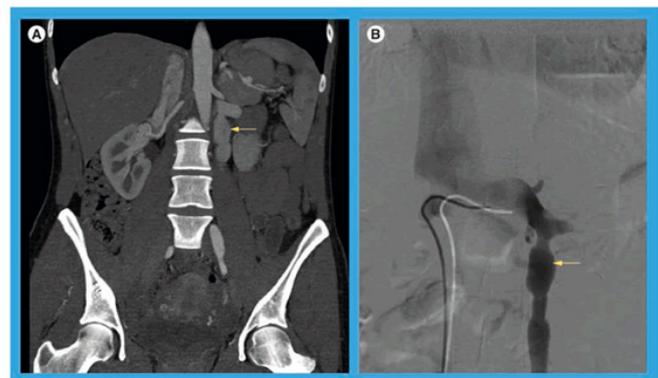
Figure 2. Left gonadal vein dilatation as demonstrated by (A) coronal computed tomography reconstruction and (B) digital subtraction selective left renal venogram.

Box 1. Symptoms of pelvic congestion syndrome.

Women
- Vulvar varices
- Chronic pelvic pain
- Dyspareunia/postcoital ache
- Dysmennorrhea
- Dysuria
- Left lateral vaginal tenderness
- Lower limb varices

Men
- Left-sided variocele
- Scrotal varices
- Lower limb varices

LRV entrapment syndrome is also welldescribed in children, although it is typically self-limited in nature in this population. Based on reports of long-term success when early intervention has been deferred, management is usually expectant [20]. Etiologic explanations for this phenomenon in developing children relate to a relative paucity of retroperitoneal fat;

this more posterior position of the left kidney leads to 'bow-stringing' of the LRV across the aorto-mesenteric angle. Subsequent development resulting in increased height and body mass index serve to create a more favorable hemodynamic situation for the LRV and typically lead to symptom resolution [21].

Diagnosis

A reliable diagnosis of LRV compression is predicated on a comprehensive evaluation of patients with suggestive symptoms. A suggested management algorithm is provided in Figure 3. When a young man or middle-aged woman presents with symptoms concerning LRV entrapment, a directed evaluation aimed at excluding multiple other diagnostic possibilities is paramount. It should be noted that the successful identification of LRV entrapment, as with other uncommon intra-abdominal compression disease entities such as median arcuate ligament syndrome or vascular compression of the duodenum, is often preceded by protracted periods of diagnostic confusion; not infrequently, the correct diagnosis is not considered until months or years after initial symptom development.

Figure 3. Suggested management algorithm for suspected left renal vein entrapment.
CTA: Computed tomography angiography; LRV: Left renal vein; MRA: Magnetic resonance angiography; PCS: Pelvic congestion syndrome; SMA: Superior mesenteric artery.
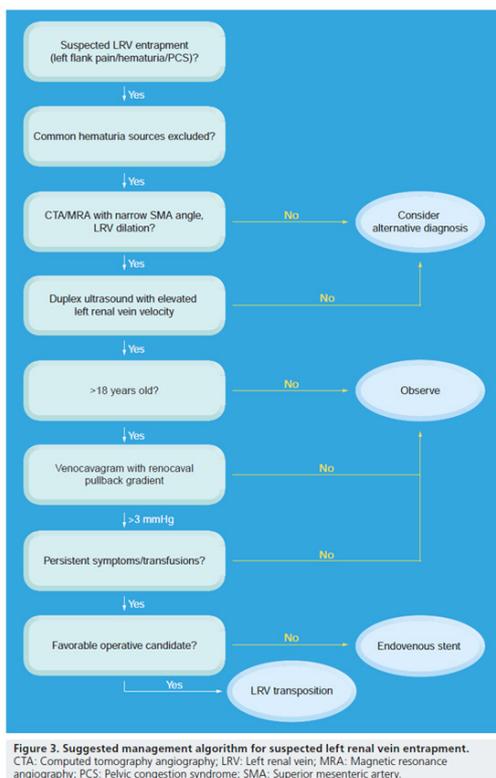
If hematuria is present, a full urologic workup to rule out other possible causative factors is indicated. Cystoscopic confirmation of isolated left ureteral hematuria suggests left-sided venous hypertension, but is an insensitive test due to the intermittent and potentially microscopic nature of the hematuria. Axial imaging, including computed tomography angiography or magnetic resonance angiography, serve both to reveal the necessary inclusion criteria of LRV compression/ dilatation and exclude other nephrogenic or abdominal etiologies (Figure 4). In addition, evaluation of the aortomesenteric angle may be discerned via sagittal reconstructions of axial imaging. Typically, this angle should be >35° in normal subjects, but has been shown to be as narrow as 16° or less in affected individuals [22]. As previously alluded to, cross-sectional imaging alone by no means secures the diagnosis, as renal vein compression may be a normal variant in a substantial portion of the adult and pediatric population [8]. Duplex ultrasonography has recently emerged as a sensitive and accurate means of identifying elevated left renal venous velocities across the aorto-mesenteric angle [22,23]. A ratio of venous peak systolic velocity (PSV) at the narrowed segment as compared with the noncompressed hilar segment has been used to characterize the syndrome. While varying PSV ratio data are reported in the literature, in general a venous PSV ratio >4.7 is very sensitive and specific (>90% for both) to the diagnosis of the venous compression syndrome [24]. The main limitation of duplex imaging remains its lack of universal reproducibility secondary to significant inter-institutional and operator variance. Its primary appeal relates to its safety and non-invasive nature and its utility in serially tracking patients in an expectant fashion or pre- and post-intervention.
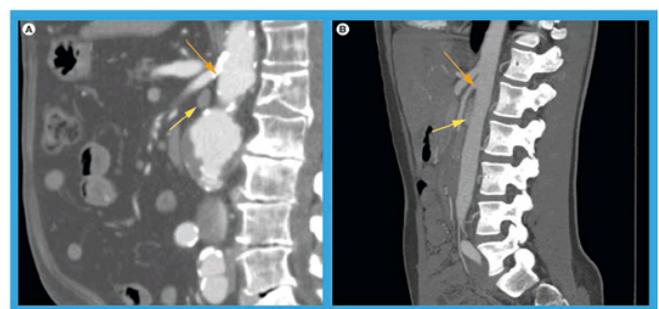
Figure 4. Computed tomography venogram sagittal reconstruction demonstrates (A) normal aortomesenteric angle (top arrow) and normal left renal vein (bottom arrow), and (B) a narrow aortomesenteric angle (top arrow) with compressed left renal vein (bottom arrow).

Currently, a definitive diagnosis of LRV entrapment in symptomatic individuals relies on venocavography and demonstration of a LRV pull-back pressure gradient. Demonstration of such a gradient in the right clinical setting suggests a causative relative hypertensive state in the LRV. It has been conclusively shown that normal individuals will have

a renocaval pressure gradient of <3 mmHg, and that in fact, the vast majority of individuals will have a gradient <1 mmHg [25,26]. These historical studies form the underpinning for the declarative statements throughout the nutcracker syndrome literature that the demonstration of an elevated gradient of ≥3 mmHg in symptomatic individuals is imperative to confirming the suspected diagnosis. Of note, variability in pressure measurements with patient positioning has been demonstrated by some groups [17].

Treatment options

Treatment options for LRV entrapment syndrome consist of expectant management, open surgical reconstruction, minimally invasive surgical reconstruction or endovascular stenting. The decision to intervene and by which method should be tailored to the individual patient and their particular demographic circumstances. As previously mentioned, in the pediatric population, symptoms are often self-limited and normal development will usually obviate the need for intervention [27]. As such, in the absence of severe, persistent symptoms or transfusiondependent hematuria in a developing child, these authors would advocate for an observational approach. Moreover, the slender build of pediatric patients with LRV entrapment syndrome render them good candidates for serial monitoring with advanced duplex imaging, obviating the need for more invasive imaging that would be unlikely to alter treatment recommendations.

Some adult patients may also experience spontaneous symptom resolution if managed conservatively, as was shown in one series with intermediated follow-up [24]. Certainly, for patients with prohibitive anatomic or prohibitive co-morbid risk factors, observation likely remains the safest approach. However, for adult patients with persistently debilitating symptoms and a demonstrated elevation in the renocaval pressure gradient, early intervention has the potential to be curative. For better risk patients, the decision becomes surgical reconstruction versus endovascular repair. It should be noted that a discussion of welldescribed endovenous techniques for isolated symptoms of pelvic congestion in the absence of flank pain and hematuria was considered beyond the scope of this review [28–30].

Open surgical treatment options

The earliest report of surgical intervention to relieve LRV entrapment ('anterior nutcracker syndrome') was in 1974 and described extensive venolysis to successfully free a constricted LRV [7]. Since that initial description, a variety of approaches have been utilized and modified over time to address different aspects of this obstructive process. Early approaches included direct attempts at addressing the narrowed SMA angulation by way of transposing the SMA to an alternative origin [31]. Similarly, attempts were made at increasing the aortomesenteric angle by elevating the SMA with a prosthetic wedge to relieve the compression [32]. While these procedures have largely been abandoned due to attendant morbidity, the concept was later modified in the form of an external stent structure (ringed PTFE graft) placed around the LRV to alleviate the venous hypertension. This was initially described as an open surgical technique [33], but has more recently been accomplished in a laparoscopic fashion [18]. More radical open surgical options have included autotransplantation of the left kidney to the pelvis [34] and even a left nephrectomy [35]. Techniques more directly targeting surgical decompression of the hypertensive venous circuit include left renocaval [18] and gonadocaval bypass [36] and, more recently, a laparoscopic splenorenal bypass [37]. Although no prospectively randomized studies have been performed, the aggregate cumulative experience represented by case reports and retrospective series suggests that transposition of the LRV remains the most common approach and has very good long-term results [24,34,38]. Technically, transposition involves a laparotomy and division of the LRV at its junction with the inferior vena cava (IVC). The renal vein is then transposed several centimeters caudad to create a neo-renocaval junction, thereby relieving compression by the SMA (Figure 5). This approach has been performed with low rates of morbidity even when performed through shorter incisions of <15 cm in length [24,34,37,39]. In a recent report by Reed et al. from the Mayo Clinic, symptoms of flank pain and hematuria resolved in >80% of patients treated with LRV transposition. Of note, the longest follow-up in this series was 12 years [24].

Recently, reports have emerged detailing surgical management of retroaortic LRV compression ('posterior nutcracker syndrome'). While the published operative experience to date is limited to <ten cases, the most common approach remains LRV transposition and creation of a neocaval junction in an ante-aortic location [6,34].
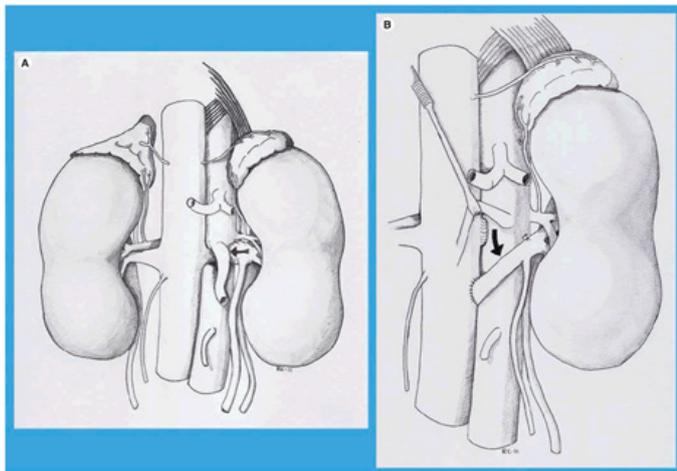
Figure 5. Artist rendition of (A) the 'anterior' nutcracker syndrome prior to surgical decompression and (B) the caudad transposition of the left renal vein with relief of the compression caused by the superior mesenteric artery.

Endovenous stenting

The first description of placement of an intravascular stent for LRV compression emerged in 1996 [40]. In a recent survey of the literature, 44 cases were identified, including three in young children [41]. The clinical details of all reported cases are included in Table 1. Overall, the most common presenting symptoms were hematuria and flank pain. Patient ages ranged from 7 to 58 years and the longest available follow-up period was 3 years. Most reports include detailed pre- and post-procedural renocaval gradient information, with normalization of the gradient after intervention seen in the majority. An exception to this pattern was a series reported by Zhang et al. in which symptom resolution was achieved despite the presence of a persistent gradient following stent deployment in all three patients treated [42].

Table 1. Reported cases of endovenous stenting for left renal vein compression.

No. of cases	Year	Age, gender (M/F)	Hematuria (micro/gross)	Flank pain (yes/no)	Pelvic congestion symptoms	Pre-Rx gradient (mmHg)	Post-Rx gradient (mmHg)	Device type (dimension of stent [mm])	Follow-up interval (months)	Complication	Ref.
1	1996	58, M	Gross	No	No	18	3	WS (10 × 42)	9	NR	[40]
1	1999	40, F	Gross	No	No	5	0	Custom (20 × 70)	6	NR	[49]
1	2000	47, M	Micro	No	Varicocele	3	NR	Niki (18 mm)	4	NR	[50]
1	2001	17, M	Gross	Yes	No	4	<1	WS (16 × 30) MT (20 × 40)	12	NR	[45]
1	2001	40, F	Micro	Yes	Vulvar varices	6	1	Palmaz® (60)	24	NR	[39]
1	2003	37, M	Gross	No	No	NR	NR	WS (NR)	3	NR	[51]
1	2003	23, F	None	Yes	No	7	0	SMART® (14 × 25)	9	NR	[52]
3	2003	24, M	Gross	Yes	No	3.5	3.3	(3) WS (10–12 × 40–60)	NR	NR	[41]
		26, M	Gross	Yes	No	3.7	3.4		NR	NR	
		32, M	Gross	Yes	No	3.4	3.4		NR	NR	
3	2003	Mean 22.7, M	Gross	NR	NR	NR	NR	NR	Mean 24.7	1 stent migration	[53]
1	2004	36, F	Gross	Yes	No	NR	NR	NR	15	NR	[54]
3	2005	10, M	Gross	No	Varicocele	6	NR	Optimed (10 × 40)	36	NR	[44]
		13, M	Gross	No	No	5	NR	Optimed (10 × 40)	27	NR	
		7, M	Gross	No	No	4	NR	Optimed (10 × 40)	24	NR	
1	2005	47, F	Gross	No	No	4	0	WS (10 × 65)	24	NR	[55]
5	2005	Mean 34.7, F	3/5 micro	No	Yes	Mean 4.3	Mean 0	(1) WS (20 × 60) (5) WS (16 × 40)	Mean 14.3	2 stent migrations	[44]
15	2007	Mean 25, NR	NR	NR	NR	NR	NR	(1) Palmaz (15) SMARTCONTROL® (10–14 × 40)	Mean 24	1 stent migration to right ventricle	[43]
3	2007	20, F	Micro	Yes	Dyspareunia	6	0	LX (14 × 40)	18	NR	[46]
		18, M	Protein	Yes	No	7	0	(2) LX (14 × 40)	15	NR	
		18, M	Gross	No	No	7	0	LX (14 × 40)	14	NR	
1	2009	40, M	Gross	No	No	5	1	MT (12 × 40)	18	NR	[56]
1	2009	20, F	Gross	Yes	No	4	NR	Nitinol	24	1 Stent migration	[57]
1	2011	23, M	Gross	Yes	No	5	NR	Protégé (14 × 40)	6	NR	[47]

F: Female; LX: Luminexx; M: Male; MT: Memotherm; NR: None reported; WS: Wallstent®

A total of five cases of stent migration have been reported, including one traveling to the right ventricle and necessitating a thoracotomy [43]. Four others became lodged within the IVC at various levels, leading to re-stenting [42,44]. While symptom resolution was reported in the vast majority of

patients, microhematuria did persist in some patients despite an adequate radiologic result and normalization of the pressure gradient [42].

Technical considerations

General anesthesia may be the preferred approach in selected cases, as on occasion considerable patient discomfort may be experienced upon dilation of the renal vein [43]. Some authors, on the other hand, have indicated that use of newer nitinol stents may obviate the need for post-dilation of the stent, theoretically lowering the risk of stent migration and rendering the use of local anesthesia a more appealing option. The laterality of venous access appears less critical as successful reports exist with both left and right sided femoral vein access [41–43]. Chiesa and colleagues described a subclavian vein approach after failure of the device to track appropriately with femoral venous access [45]. On rare occasion, angulation of the LRV may preclude easy cannulation from a femoral approach and an upper extremity access may prove necessary. Concurrent arterial access with a catheter in the SMA has been described, but is not considered necessary [44]. Both pre-operative antiplatelet therapy administration and intra-procedural systemic heparinization are recommended.

As with most endovascular procedures, stent selection and sizing are important to achieving technical success. Zhang et al. report the largest series of endovenous stent placement for the LRV entrapment syndrome [43]. In their early experience, they used the stiff balloon-expandable Palmaz® stent (Cordis, Johnson & Johnson, USA), but subsequently favored the self expanding SMART® CONTROL® stent (Cordis, Johnson & Johnson, USA). They found the less rigid stent conformed well to the renal vein anatomy and that delivery proved more reliable. They did note that post-deployment shortening may limit stent visibility, which may be mitigated by using a slightly longer device (>40mm) [42]. Other authors have avoided the self-expanding metal Wallstent® (Boston Scientific, Natick MA, USA) for similar reasons [43,44].

In the absence of established stent sizing guidelines, sizing should be individualized to the encountered anatomy. Zhang et al. advocate 20% stent over-sizing relative to renal vein diameter on initial venography, and most commonly used a 14 mm diameter stent in their series [42]. In instances of stent migration, reporting authors typically upsized the subsequent replacement stent [42,43]. Some authors suggest that optimal stent placement will leave it flush with the ostium rather than

protruding across the renocaval junction [46]. An exception to this was a recent report by Baril et al. in which an endovenous stent was utilized to treat a symptomatic recurrence following prior open LRV transposition. In that instance, the authors were specifically treating an anastomotic stenosis at the neo-renocaval junction and intentionally allowed the stent to protrude into the IVC to adequately traverse the stenotic segment. They also employed intravascular ultrasound to enhance localization of the stenosis [47]. As a final technical consideration to consider, if ovarian vein embolization is being considered, this may be best undertaken prior to renal vein stenting; doing so avoids the unnecessary potential challenge associated with crossing of the stent when accessing the ovarian vein.

While post-procedural intravenous heparin is sometimes utilized, most investigators believe antiplatelet therapy should be instituted prior to and following discharge [45]. No clear consensus exists on class and duration of a post-procedure medication regimen. Whether duel aspirin and plavix antiplatelet therapy versus solo treatment with either aspirin or plavix alone is preferable remains unclear. Notably, low rates of in-stent thrombosis exist in the reported series to date. This has been attributed by some to physiologic levels of urokinase present in the renal vein, which may confer intrinsic protection [48]. Post operatively, patients may be followed with interval duplex scanning to survey for in-stent stenosis that may potentially require secondary therapy.

Future perspective

The rarity of renal vein entrapment syndrome as it relates to symptomatic individuals is such that a prospectively designed trial evaluating treatment options will likely not be undertaken in the foreseeable future. Most agree that clear diagnostic criteria with regard to symptoms and appropriate radiographic findings should be present, in order to reliably make the diagnosis. Left renal venography demonstrating a pullback pressure gradient across the compressed segment of vein is a further key feature of the diagnostic standard. Venous duplex serves as an important adjunct as it represents a non-invasive way to serially monitor patients that forego intervention; at present it remains institution- and operator-dependent and may be adversely affected by individual patient body habitus.

In terms of treatment, experiential reports and small case series reflect a wide array of historical techniques, ranging from the potentially dangerous and now rarely utilized manipulations of the SMA to a growing experience with LRV transposition. Newer minimally invasive surgical techniques, such as laparoscopic extravascular stenting or laparoscopic bypass techniques, may have a future role, but remain largely untested. Most recently, endovascular stenting has been shown to be technically feasible and has emerged as an enticing option with acceptable short and mid-term results. The available literature on this therapeutic strategy, however, is currently limited to case reports and small series with limited long-term follow-up. The aggregate experience reviewed in this summary demonstrated that up to 11% of stents ultimately migrated from the LRV into the IVC and/or heart. To be fair, this figure presumably reflects the learning curve for clinicians exploring alternative treatment options for this rare condition.

In summary, while primary endovascular stenting of the LRV in an otherwise healthy young person with LRV entrapment syndrome is an acceptable treatment option, it remains of questionable long-term durability. It may be the preferred therapy, on the other hand, for patients with prohibitively hostile anatomic constraints (e.g., those with an abdominal stoma or a history of prior abdominal operations or intra-abdominal radiation) or in the setting of a symptomatic recurrence following prior open surgical repair.

Executive summary

Clinical presentation

▪▪ The hallmark symptoms of nutcracker syndrome include

left flank pain and hematuria (microscopic or gross). The possibility of concomitant pelvic congestion syndrome exists.

Diagnosis

▪▪ Axial imaging will demonstrate left renal vein compression/dilatation with a narrowed aortomesenteric angle.

▪▪ Venography will demonstrate a renocaval pullback gradient ≥3 mmHg and represents the current gold standard for diagnosis.

Treatment options

▪▪ Children and developing minors should be treated conservatively as it is usually a self-limited process.

▪▪ Adult patients with persistent symptoms may be candidates for repair.

▪▪ High-risk surgical patients are best treated expectantly.

Open versus endovascular management

▪▪ Open surgical reconstruction with left renal vein transposition is the standard open surgical approach based on available favorable 12-year follow-up data.

▪▪ Endovenous stenting remains the preferred approach for recurrent disease or patients at higher risk for open abdominal operations.

▪▪ Stent migration rates remain >10% in the available reported literature.

Endovenous stent technical considerations

▪▪ The anesthetic approach will be dictated by the clinical scenario and physician preference.

▪▪ Left or right femoral venous access is acceptable. Occasionally central or upper extremity venous access may be necessary for left renal vein cannulation.

▪▪ A self-expanding stent should be used, with stent postdilation only in necessary cases.

▪▪ A 14–16 mm stent diameter is the most commonly reported with desired 10–20% oversizing to the renal vein.

▪▪ Antiplatelet therapy is the mainstay of pharmacotherapy. Routine peri-procedure systemic heparinization is also recommended.

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