How I treat nutcracker syndrome

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ABSTRACT

Anatomic compression of the left renal vein in the angle between the aorta and superior mesenteric artery may be asymptomatic or may result in symptoms, including flank pain, hematuria, or pelvic pain and/or congestion. Patients can be referred to a vascular surgeon due to symptoms and/or radiologic findings. Because symptoms of nutcracker syndrome can be vague and/or nondiagnostic, careful evaluation, assessment, and counseling with patients are required before undertaking intervention, which is often an open surgical procedure. The definitive diagnosis is ideally confirmed with diagnostic venography, including pressure measurements from the left renal vein and inferior vena cava. The optimal treatment includes open decompression of the left renal vein with renal vein transposition or gonadal vein transposition, with or without concomitant management of pelvic varicosities if symptomatic. Because most patients with nutcracker syndrome are young, long-term follow-up with scheduled ultrasound examinations should be maintained. (J Vasc Surg Cases Innov Tech 2023;9:101344.)

Keywords: Flank pain; Hematuria; Inferior vena cava; Nutcracker; Renal vein

The anatomic appearance of the left renal vein (LRV) as it passes between the narrow angle between the aorta and superior mesenteric artery (SMA), resulting in LRV compression, was described in 1937 by Grant.[1] Although the radiologic appearance (or "nutcracker phenomenon") will be present in many patients, its presence does not imply clinical symptoms (ie, "nutcracker syndrome" [NCS]). A study reviewing 1000 contrast-enhanced computed tomography images reported that LRV compression can occur in ≤4.1% overall; however, of those with compression, only 8.8% will have unexplained hematuria or proteinuria.[2] Typically, young women present with left flank pain and hematuria most often. The venous hypertension caused by LRV outflow obstruction can cause pain in the flank and progress to pelvic discomfort due to enlarged varicosities in the perirenal space and pelvis. Treatment ranges from conservative measures to open, hybrid, or endovascular venous reconstruction, depending on the duration and degree of symptoms. In the present report, I discuss my clinical practice assessment and treatment for patients referred with possible NCS.

PATIENT EVALUATION

Patients most commonly present with unexplained hematuria and flank or pelvic pain, which can be severe at times. Symptoms can worsen with physical activity. Often, patients have had several emergency room visits for pain. The etiology of hematuria is unknown but theorized to be the result of venous hypertension from the dilated veins adjacent to the renal pelvis and ureter, resulting in microhematuria or macrohematuria. Macrohematuria can be severe enough to warrant blood transfusions, although this is rare for patients with NCS and might suggest loin pain hematuria syndrome.[3] The most common symptoms include abdominal and left flank pain (estimated frequency, 43%-65%), macroscopic hematuria (39%-69%), microscopic hematuria (9%-22%), proteinuria (4%-26%), and varicocele (9%-22%).[4,5] Less common symptoms include pelvic congestion syndrome, dysmenorrhea, and dyspareunia.[3,6] Fatigue symptoms and possible chronic fatigue syndrome have been noted in patients with a high LRV to inferior vena cava (IVC) pressure gradient.[7]

Routine blood test results are typically normal and nondiagnostic, although anemia can occur from severe hematuria. Urinalysis can demonstrate micro- or macrohematuria or proteinuria. Shin et al[8] found that the etiology of isolated hematuria could not be identified in 69% of cases, and, of those, 40% were found to have NCS by ultrasound. In addition, orthostatic proteinuria has been noted, theoretically thought to be caused by positional increased pressure in the LRV resulting in mild immune injury and altered release of angiotensin II and norepinephrine.[9,10]

Exclusion of other etiologies of hematuria must be considered and evaluated by blood tests, urinalysis, urine culture, urethrocystoscopy, computed tomography (CT) urography, and/or renal biopsy. Other causes of hematuria and/or flank pain should be included in the differential diagnosis such as pyelonephritis, renal calculi, pelvic congestion syndrome, genitourinary malignancy, protein-losing nephropathy, and loin pain hematuria syndrome.[11] NCS can occur concomitant with other conditions such as pregnancy, Henoch-Schönlein purpura, IgA or membranous nephropathy, idiopathic hypercalciuria with urolithiasis, a median arcuate ligament, or

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May-Thurner syndrome.[3] The presentation of NCS in combination with other conditions can confound the diagnosis and delay intervention. Often, patients also have symptoms consistent with pelvic compression due to iliac or IVC venous stenoses or will have already been treated for pelvic congestion syndrome with coil embolization of pelvic veins at the time of presentation.

IMAGING EVALUATION

In my practice, CT venography is most helpful in supporting the need for further evaluation. If the CT findings suggest NCS, the patient would then undergo direct contrast-enhanced venography with concomitant intravascular ultrasound (IVUS) and pressure measurements. These imaging modalities, combined with the patient's symptoms and history, provide a diagnosis that allows for a definitive decision (either perform open surgery or search for other causes of symptoms) for >90% of my patients. An overview of all imaging modalities that I might use in an evaluation is provided.

Duplex ultrasound. Duplex ultrasound (DUS) has 69% to 90% sensitivity and 89% to 100% specificity and is often used as the first diagnostic imaging examination when NCS is suspected.[3] The DUS criteria for LRV compression varies and could be different for adults than for children.[12] Measurements of the LRV diameter and peak flow velocity through the LRV might provide the most accurate DUS diagnosis. The criteria for adults include those proposed by Kim et al[13] to correlate the DUS and venography findings: ratio of the anteroposterior diameter of the compressed LRV to renal hilum or peak velocity of maximum flow in the compressed LRV in relation to a renal hilum >5.[14] In contrast, the criteria for the diagnosis of NCS in children are proposed to be a ratio of >4.2 for the anteroposterior diameter and >4.0 for the peak velocity.[15] Other DUS findings, such as the presence of collateral veins, can support the diagnosis.[12] DUS can be limited by patient positioning or compression from the transducer in thin patients. Ideally, the examination should be performed with the patient in several positions (ie, supine, upright, prone, Fowler) to accurately document the images and velocities.[7]

CT or CT venography. Although DUS is noninvasive and could be adequate for screening patients with suspected NCS, more detailed vascular imaging might be required for a definitive diagnosis. In children, contrast-enhanced imaging is typically avoided. In the study by Nalcacioglu et al,[12] only 8 of 44 children required CT after DUS because of DUS images inadequate for diagnosis. Some investigators have proposed that CT venography or magnetic resonance venography could replace the reference standard of contrast-enhanced venography.[12] CT imaging can identify (1) LRV compression between the aorta and SMA; (2) a "beak sign" of a triangular shape at the narrowing of the LRV at the aortomesenteric space, conferring 91.7% sensitivity and 88.9% specificity[16]; (3) LRV diameter ratio (comparing the hilar diameter to the compressed LRV) of >4.9; (4) an angle between the SMA and aorta of <41°; and (5) the presence of venous collateral vessels around the renal hilum and retroperitoneum.[16]

Magnetic resonance imaging or magnetic resonance venography. Compared with CT, magnetic resonance imaging (MRI) does not require radiation nor carries the risk of contrast allergy. However, MRI can provide anatomic data similar to that obtained with CT, with similar sensitivity and specificity.[17] In addition, noncontrast-enhanced MRI with specialized sequences can be used, further decreasing the examination risk. Many clinicians prefer MRI to CT for children, specifically, to reduce the cumulative radiation risk. Also, many use MRI combined with history and physical examination findings, with DUS used to make a definitive diagnosis.

Direct contrast-enhanced venography. Contrast-enhanced venography is considered the reference standard to diagnose LRV compression. Phlebography via femoral access can demonstrate venous compression at the site of the SMA crossing, enlarged venous tributaries and reflux into pelvic varicosities or perirenal varicosities.[9] Because the normal gradient of venous pressure from the LRV to the IVC is considered to be <1 mm Hg, a gradient of >1 mm Hg, but typically >3 mm Hg, is considered to support a diagnosis of NCS.[18] However, it is possible for symptomatic NCS patients to have a gradient of <3 mm Hg if robust collateral vessels exist to decompress the LRV venous hypertension[9] (Fig 1).

Intravascular ultrasound. IVUS can be an important adjunct to contrast-enhanced venography. An 0.035-in. Volcano Visions PV catheter (Philips) is inserted through a femoral sheath, and cine-loop images are taken from the renal vein at the hilum of the kidney as the catheter is withdrawn into the IVC. These images are recorded to document the degree of compression. Using IVUS, direct measurement of the LRV diameter along its length and the size of the IVC can be taken.[19,20] This technique can also be used for possible intervention if endovascular treatment is indicated. IVUS has a high specificity (90%) for the diagnosis of NCS compared with 62% for contrast-enhanced venography[9] (Fig 2).

MANAGEMENT

My overall algorithm is depicted in Fig 3, with more in-depth explanations in the next sections.

INDICATIONS FOR TREATMENT

There is no indication for treatment of asymptomatic NCS (or nutcracker phenomenon). Because NCS is uncommon, other more common causes of symptoms such as renal colic or infection, must be excluded before

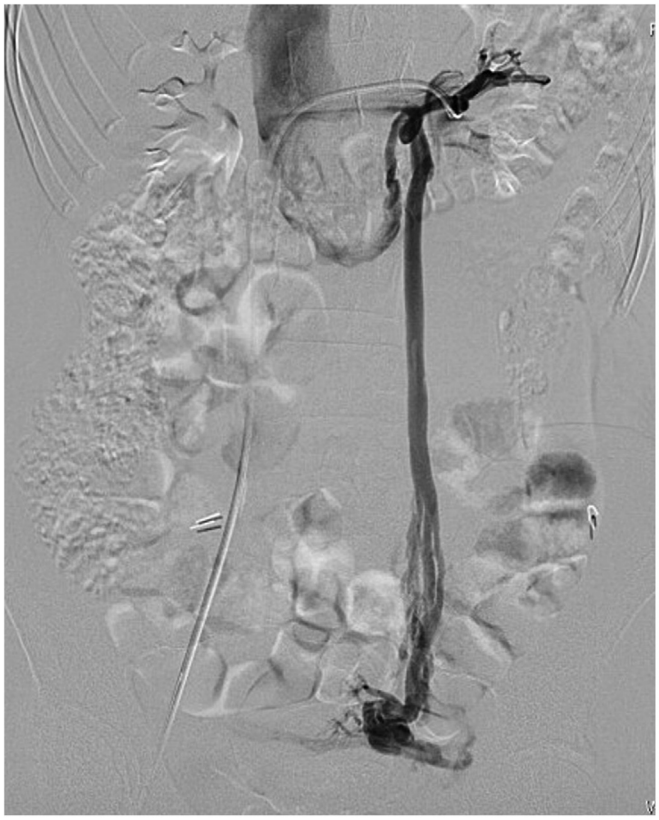

Fig 1. Diagnostic contrast venogram via right femoral vein demonstrating compression of the left renal vein (LRV) and reflux through the left ovarian vein with pelvic varicosities.

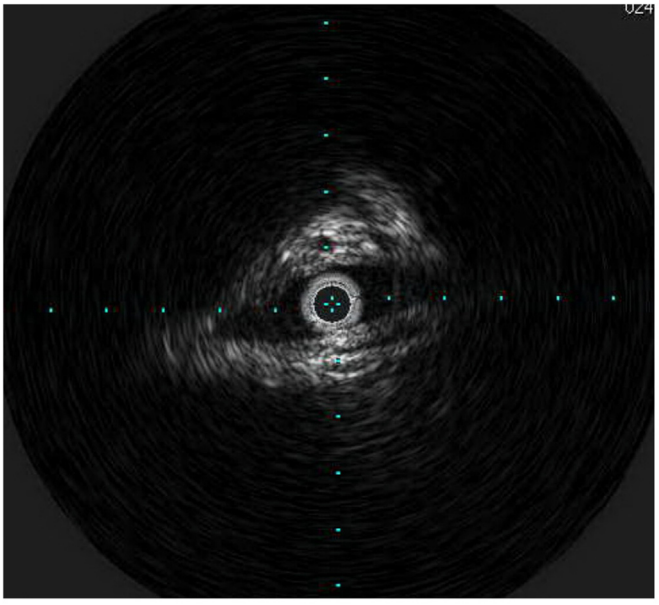

Fig 2. Intravascular ultrasound (IVUS) confirming compression of the left renal vein (LRV) as it crosses the aorta.

the diagnosis is considered definitive and intervention can be considered. The indications for treating symptomatic NCS are dependent on the degree and type of symptoms.

Nonoperative treatment. Patients with NCS aged <18 years are typically treated nonoperatively, because the changes that occur with growth during childhood and adolescence could change the anatomic configuration of the SMA to the aorta angle and relieve LRV compression.[21] In addition, weight gain in thin patients has been shown to resolve symptoms in ≤30% of patients.[22] Weight gain can cause an increase in retroperitoneal fat, which, in turn, can alleviate the compression on the LRV by increasing the space between the SMA and aorta. For patients with mild symptoms, the development of venous collateral vessels over time can increase. The combination of physical growth and collateral formation over time can explain why nearly 75% of young patients and children with hematuria will spontaneous improve completely ≤2 years.[3]

When to proceed to intervention. Operative intervention should be considered for adults with gross hematuria, especially for patients requiring transfusion or with recurrent episodes, and for patients with such severe flank or abdominal pain that narcotic pain medication or hospitalization is required. Other indications include failed conservative measures after 2 years for patients aged <18 years and after 6 months for adults. Patients with anemia due to hematuria, autonomic dysfunction, severe proteinuria, and varicocele should also be offered intervention.[16,21,23,24] Patients are counseled to understand that although the resolution of flank pain and/or hematuria are excellent with LRV transposition (80%-100%),[9,24] symptoms and/or venous hypertension can recur in ≤30% to 40% of patients over time. If the pain does not resolve, other sources of venous hypertension, such as pelvic congestion syndrome or pelvic varicosities, could be the culprit.

APPROACH TO INTERVENTION

I believe that open surgical repair remains the reference standard of treatment. Many methods are available to manage NCS surgically, including LRV transposition, gonadocaval transposition, LRV to IVC bypass, nephropexy, laparoscopic-assisted procedures, renal autotransplantation, and nephrectomy, depending on the patient's anatomy. Although translocation of the SMA has been discussed in the literature, I have never considered that modality in my practice. I preferentially choose LRV transposition, with gonadal vein transposition my second most common procedure, depending on the size of the gonadal vein (ie, best for a vein >8-10 mm diameter). The adjunctive use of vein patch angioplasty or a vein cuff

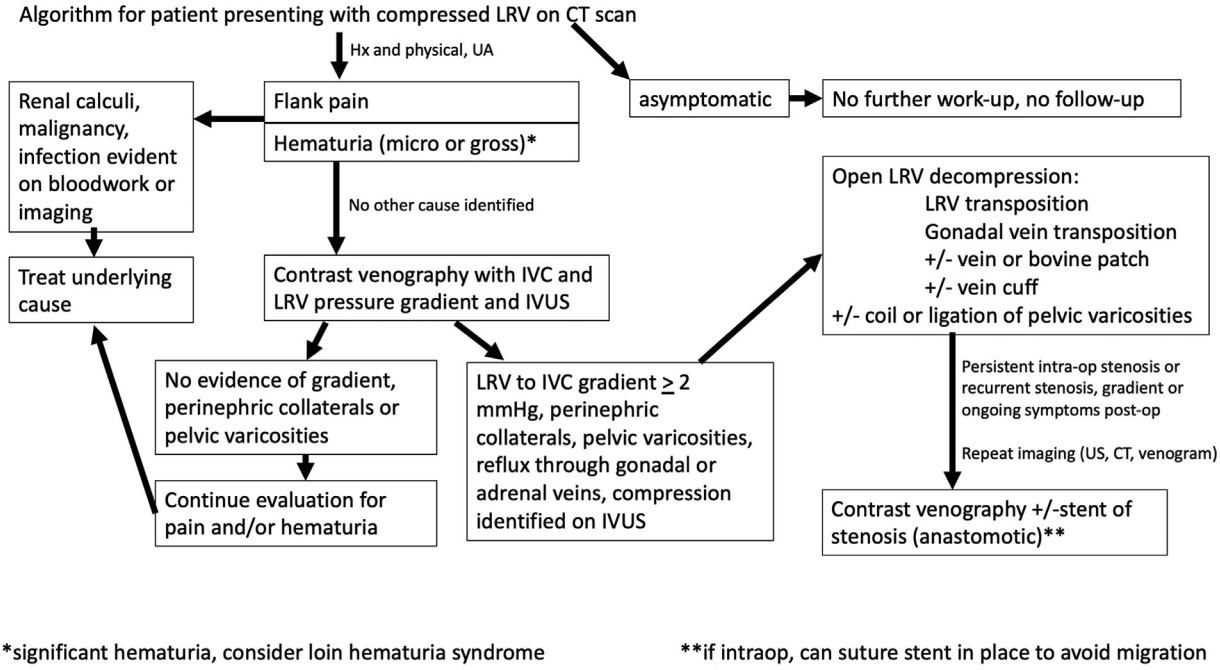

Fig 3. Algorithm for evaluation and management of patients presenting to my practice for possible nutcracker syndrome (NCS). *CT,* Computed tomography; *intra-op,* intraoperative; *intraop,* intraoperative; *IVC,* inferior vena cava; *IVUS,* intravascular ultrasound; *LRV,* left renal vein; *post-op,* postoperative; *US,* ultrasound.

is indicated if tension is created in the transposition of the LRV on the IVC. In extreme cases of pain or hematuria, without appropriate options for venous decompression, nephrectomy could be indicated as a last resort, although I have not yet had to do this in my practice. Additionally, in patients with large pelvic varicosities, I will concomitantly ligate them during open surgery, ligate the gonadal vein (if not used as a bypass graft), or coil embolize them on-table. I typically use endovascular interventions as an adjunct to open intervention for restenosis, rather than as a primary option due to the small, but critical, risk of stent migration, and the unknown long-term use of stents in this young population.

OPEN SURGICAL TREATMENT

LRV transposition. LRV transposition is approached through a midline incision (either vertical or transverse, such as in pediatric cases), with exposure of the LRV as it crosses the aorta behind the SMA. A minilaparotomy can also be used for thin patients. Vein tributaries, including the gonadal vein, adrenal vein, and lumbar renal vein, are double-ligated to mobilize the LRV and allow for tension-free transposition. After heparinization, the IVC is clamped and the LRV excised from the IVC. The proximal IVC venotomy is closed with a single layer suture. Depending on how mobile the LRV is, either the IVC venotomy can be extended from the site of the initial LRV origin or a separate venotomy can be created on the medial IVC 1 to 2 cm caudal to the original LRV origin. The venotomy is typically 8 to 12 mm, depending on the size of the LRV and the degree of tension on the transposition. At least two separate 5-0 polypropylene sutures are used to create the anastomosis to avoid a purse-string effect that can cause narrowing of the anastomosis (Fig 4). When removing the clamps, the anastomosis should be covered with saline, and the patient placed under a Valsalva maneuver to avoid air embolus with venous reperfusion. The completion assessment can include intraoperative ultrasound and assessment of intravenous pressure by insertion of a 22-gauge needle connected to an arterial line into the LRV at the renal hilum and also into the IVC (pressure should be <1 mm Hg).

Overall, the surgical risks of LRV transposition are low, because most patients are young and healthy. The risks include deep vein thrombosis, retroperitoneal hematoma, and ileus. The long-term risks include restenosis of the transposed vein, especially if any tension was present at the anastomosis at the initial operation.[5,24] Resolution of flank pain and hematuria is excellent with LRV transposition (80%-100%)[9,24]; however, the need for reintervention for symptomatic restenosis can be as high as 30% to 40%.[24] In the cases of symptomatic recurrence, endovascular intervention, as indicated in a subsequent section, is considered. The most important technical feature is to create a tension-free LRV–IVC anastomosis. Adjunctive measures to reduce tension on the anastomosis and possibly reduce the incidence of restenosis are discussed.

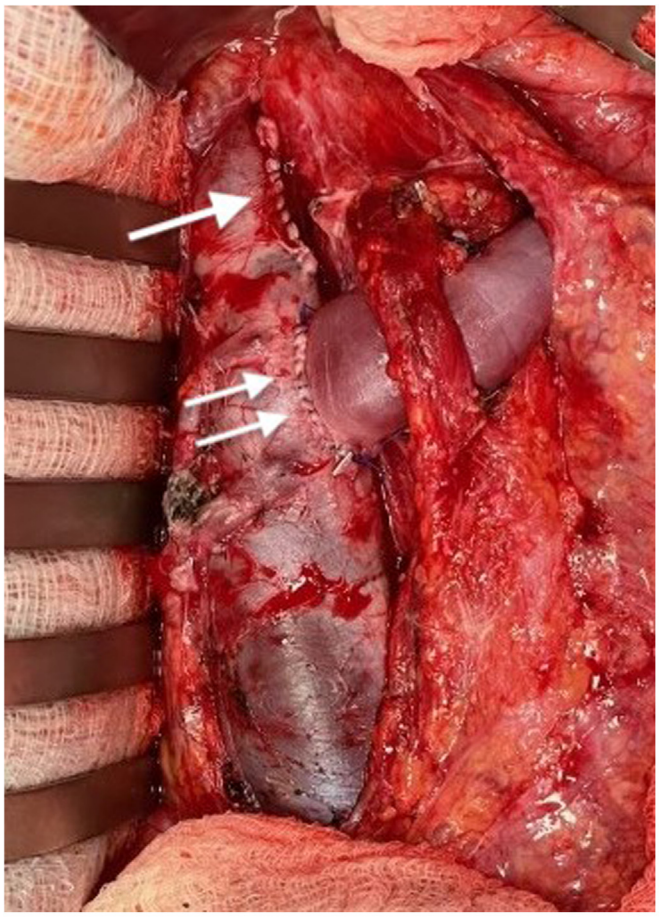

Fig 4. Intraoperative image after left renal vein (LRV) transposition. The LRV is excised from the medial aspect of the inferior vena cava (IVC) and the venotomy sutured in a single layer (*single arrow*). The LRV is then anastomosed 1 cm distal to its original site using two separate sutures to avoid "purse-stringing" (*double arrows*).

Vein patch angioplasty. Venoplasty of the LRV at the LRV–IVC confluence can be performed using a segment of autologous vein, prosthetic material, or bovine pericardium. Patch angioplasty can be used in conjunction with LRV transposition to reduce the tension on the LRV–IVC anastomosis, especially for thin patients without retroperitoneal fat and those with a prominent aorta or ptotic kidney that can create stretch on the anastomosis. In addition, patients can have a persistent gradient present despite transposition due to permanent distortion of the LRV, which can be resolved by placement of a patch.[16] In cases for which distal transposition is not possible, such as a short renal vein, or anatomy for which transposition would not improve external compression, wide venoplasty without transposition can be effective in improving symptoms.[16]

Vein cuff. As an alternative option to patch angioplasty, a vein cuff can be used to reduce tension on the LRV–IVC transposition anastomosis. The saphenous vein is most often used to create the cuff by harvesting the vein segment, ligating any side branches, dividing the vein segment longitudinally, and creating a cuff by anastomosing the short ends together with 6-0 polypropylene suture. Interrupted sutures, or three or four separate running sutures, are necessary when performing the IVC to cuff anastomosis to avoiding "purse-stringing" and narrowing of the anastomosis.

Gonadal vein transposition. Many patients with NCS have an incompetent and enlarged gonadal vein, with associated pelvic congestion. By transposing the gonadal vein on to the IVC, the kidney is decompressed, and the gonadal reflux is eliminated, yet the LRV does not have to be relocated. The left gonadal vein can be approached via the transverse mesocolon and the side branches divided. The caudal end of the gonadal vein is ligated, transected, and tunneled to the IVC, posterior to the inferior mesenteric vessels and anterior to the abdominal aorta. The gonadal vein is then anastomosed to the IVC with interrupted 6-0 polypropylene suture.[16]

ENDOVASCULAR TREATMENT

Stent placement into the LRV was first described in 1996,[25] with a larger series presented by Chen et al[26] in 2011. In an assessment of 61 patients with a median follow-up of 66 months, the results were excellent, with only 2 patients having unchanged symptoms and 1 patient with recurrent symptoms. The ideal stent to use in this procedure is described as having high radial strength and conformable with little change in size to ensure accurate positioning.[16] In most cases, 6- to 8-cm-long self-expanding stents (ie, Wallstents; Boston Scientific) are the best choice and are best positioned adjacent to the first division of the renal vein to minimize migration. Intraoperative complications included a poorly deployed stent requiring open treatment. The postoperative complications reported by Chen et al[26] included stent migration into the renal hilum, migration to the right atrium, and stent erosion into the IVC. Although the overall results seem acceptable, the complications of stent migration or kinking are concerning for this otherwise healthy group. In particular, the risk of migration of the stent to the heart makes this technique high risk compared with open LRV transposition. To reduce the occurrence of stent migration, the stents should be oversized. Other factors that affect stent migration include the distance between the first branch of the LRV and ostium, adjacent aortic pulsations, and, even, early patient mobilization.[16]

Migration or tilt complications can be managed with endovenous techniques in selected patients.[27] A hybrid technique has been described to prevent stent migration by placing the LRV stent and using an open technique to secure the stent to the vein wall with carefully placed polypropylene sutures between the stent struts. Other stent complications such as thrombosis or stenosis are

uncommon; however, high pressure differences or anticoagulation therapy could decrease such morbidity.[26]

Ligation or embolization of collateral veins should be combined with a decompressive procedure (open or endovascular), otherwise the LRV to IVC gradient could actually increase. Ovarian vein coil embolization has been described to relieve pelvic congestion or treat symptomatic pelvic varicoceles with good success of symptom relief in 56% to 98%.[3,28] The risks of ovarian vein coil embolization include pelvic vein rupture and coil migration to the lung.

POSTOPERATIVE MANAGEMENT

After open surgical LRV transposition, other vein reconstruction, or endovascular intervention to decompress the LRV, anticoagulation therapy with a direct-acting oral anticoagulant is considered for ≤3 months. If patients are hypercoagulable, have concomitant deep vein thrombosis, or have a strong family history of hypercoagulable traits, I might consider a longer duration or lifelong anticoagulation therapy, especially if significant venous reconstruction was performed and the final LRV to IVC gradient was 0 mm Hg. Asymptomatic patients postoperatively are evaluated with ultrasound at 3 months, annually for 3 years, and then every 3 to 5 years or sooner if symptoms arise. If patients become or remain symptomatic with flank pain and/or hematuria, we will suggest CT venography or contrast-enhanced venography to assess the patient's anatomy and proceed with intervention according to the findings.

CONCLUSIONS

In my practice, definitive management of young patients with symptomatic NCS with hematuria and pain is best accomplished with open decompression of the venous hypertension. Contrast-enhanced venography with IVUS and pressure measurements is the optimal confirmatory study after initial CT venography identifies vein compression. The most common procedures for decompression in my practice are LRV transposition, followed by gonadal vein transposition with or without pelvic varicosity ligation or embolization. Long-term follow-up is important for this young patient population.

DISCLOSURES

None.

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