Renal Vein Stenting for Nutcracker Syndrome

A potentially less-invasive treatment for a rare but debilitating condition.

BY JAMES L. GUZZO, MD, AND HEITHAM T. HASSOUN, MD, FACS

Entrapment of the left renal vein (LRV) between the abdominal aorta and superior mesenteric artery (SMA) causing the classic clinical triad of hematuria, varicocele, and left abdominal or flank pain was first described in 1950.[1] Later, this array of symptoms was termed "the nutcracker syndrome" by De Schepper in 1972.[2] Endovascular technology has evolved as a potential minimally invasive therapy for relief of symptoms caused by this compression syndrome. We report a case of successful percutaneous LRV stenting for treating nutcracker syndrome and review the current literature.

CASE REPORT

A healthy 26-year-old man was referred for a second opinion regarding nutcracker syndrome. He described a several-month history of intermittent left flank pain, progressing to left testicular pain with onset of microscopic hematuria. Urologic evaluation for this constellation of symptoms led to a contrast-enhanced computed tomography (CT) scan of his abdomen/pelvis. The CT scan identified compression of the LRV between the SMA and aorta, with an abundance of venous collaterals in the retroperitoneum (Figure 1). A left renal venography performed at an outside facility confirmed the diagnosis and demonstrated an LRV-inferior vena cava (IVC) pressure gradient of 6 mm Hg (normal value < 1–2 mm Hg).

The patient was counseled regarding the risks, benefits, and alternatives of surgical versus endovascular therapeutic alternatives, and he opted for LRV stenting. After obtaining appropriate informed consent, he was taken to the endovascular suite where the right common femoral vein was accessed under ultrasound guidance and local anesthesia. After sheath insertion and systemic administration of heparin (0.8 mg/kg body weight), a marker pigtail catheter was placed into the IVC and a vena cavagram was obtained. The catheter and sheath were then exchanged for a 7-F Pinnacle Destination sheath (Terumo Interventional Systems, Somerset, NJ), which was advanced to the LRV-IVC junction. The LRV was selected with a Cobra 2 catheter (Cook Medical, Bloomington, IN) and a 0.035-inch hydrophilic Glidewire (Terumo Interventional Systems), and the Cobra 2 catheter was exchanged for a straight flush catheter. A selective left renal venogram was then obtained, which identified abundant large collaterals of the left adrenal and renolumbar veins and an LRV diameter of 12 mm (Figure 2). A 0.035-inch Storq wire (Cordis Corporation, Bridgewater, NJ) was then selectively advanced into the left gonadal vein, and the Destination sheath was advanced into the LRV. Prestent 8-mm balloon angioplasty was performed to facilitate sheath and stent advancement across the stenosis. A 14-mm X 6-cm SMART control stent (Cordis Corporation) was then deployed from the left gonadal vein origin, with 1-cm extension into the IVC. Poststent 10-mm balloon angioplasty was performed to dilate the vein. Completion venography demonstrated a widely patent LRV with decreased collateral filling and disappearance of an LRV-IVC pressure gradient (Figure 2).

Figure 1. CT angiography demonstrating compression of the proximal LRV between the aorta and SMA, with poststenotic dilatation (arrow) and prominent renolumbar collaterals.

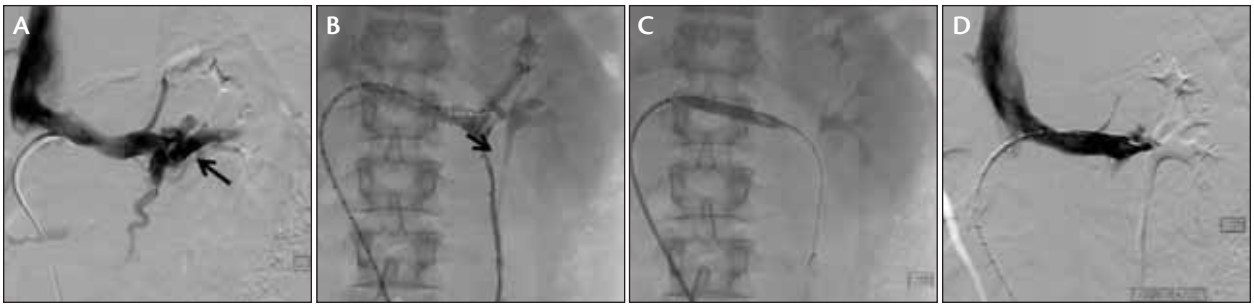

Figure 2. Venogram demonstrating LRV compression with "double-density" and prominent renolumbar collaterals (arrow) (A). The gonadal vein is selected to facilitate flush catheter angiography and for tracking of the sheath across the lesion (B). The lesion is predilated (C), and a 14-mm, self-expanding nitinol (SMART) stent is deployed (D).

After the procedure, the patient was started on aspirin (81 mg/day) and low-molecular-weight heparin (1 mg/kg subcutaneously twice/day). He had no postoperative complications but did report new onset back and left flank pain, which was relieved with analgesics and anti-inflammatory medication. The patient underwent LRV duplex imaging, which demonstrated a patent stent, and he was discharged to home on postoperative day 1. At 1-month follow-up, the patient had resolution of his symptoms, and repeat CT demonstrated a widely patent LRV (Figure 3). Anticoagulation was discontinued, and the patient remained symptom-free at 6 months. He is scheduled to undergo annual duplex examination for surveillance.

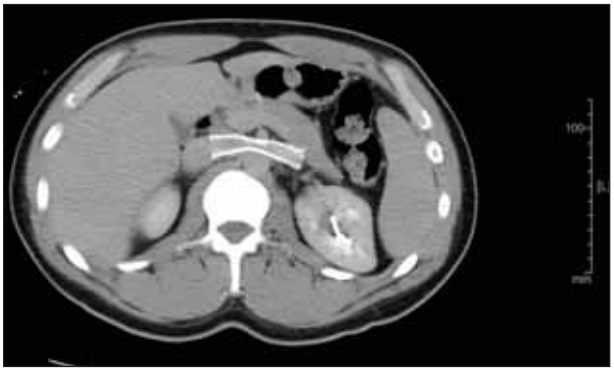

Figure 3. Follow-up CT demonstrates a widely patent LRV stent with decrease in the retroperitoneal venous collaterals.

DISCUSSION

Nutcracker syndrome describes an array of incapacitating symptoms caused by LRV hypertension secondary to vein compression between the SMA and the aorta. Diagnosis of this syndrome requires a high index of suspicion and can be accomplished with duplex sonography, CT, magnetic resonance imaging, or venography, and it is confirmed by measured pressure gradient across the lesion. Treatment of nutcracker syndrome is controversial, with a wide array of therapeutic options. Most reports consist of small case series, and long-term results are limited. Surgical approaches reported include venolysis and anterior nephropexy, renal vein bypass or interposition grafting, renocaval reimplantation, and autotransplantation.[3-7]

More recently, endovascular approaches have been reported for this uncommon problem. The first case report of endovascular stent placement for renal vein hypertension was in 1996 by Neste et al who described a 58-year-old man successfully treated with a Wallstent (Boston Scientific Corporation, Natick, MA).[8] Additional early reports described successful stenting for nutcracker syndrome,[9-12] and the largest series of five patients was reported by Hartung et al in 2005, in which five patients were treated with Wallstents for pelvic vein congestion.[13] All were asymptomatic at 1 month, and two patients had recurrence at 3 to 4 months, with repeat imaging showing stent migration. One patient had recurrent pain that later demonstrated to be related to endometriosis, and the remaining two were symptom free at 4 months and 2 years, respectively. Interestingly, Hartung et al reported success with 60-mm-long stents, whereas both patients with recurrence due to migration had placement of shorter 40-mm-long devices.

To prevent potential stent migration, either during deployment or afterward, we also chose to use a longer (ie, 60 mm) stent, with extension from the gonadal vein to 1 cm into the IVC. We used a 14-mm, self-expanding nitinol SMART stent, which allowed for approximately 15% vessel oversizing. Because this is the largest diameter SMART stent available in the United States, larger-diameter renal veins, although unusual, would require a Wallstent or other device. Another technical point is the importance of using a stiff guidewire advanced distally into the gonadal vein to facilitate stent tracking from the groin. Once across the lesion, the wire can be retracted and carefully advanced distally into the renal vein before stent deployment.

Most venous interventions call for a 3- to 6-month period of systemic anticoagulation, although there are limited data on the necessity of this algorithm in the absence of an acute thrombotic event. We arbitrarily chose to treat this patient with 1 month of low-molecular-weight heparin followed by long-term antiplatelet therapy with low-dose aspirin to reduce the risk of stent thrombosis. Clearly, long-term data are needed to help guide future treatment protocols.

CONCLUSION

Although it is in its infancy, endovascular therapy for nutcracker syndrome is a viable option that may circumvent more invasive procedures. Percutaneous angioplasty and stenting for symptomatic LRV compression and the nutcracker syndrome is an excellent alternative to more invasive approaches. However, further follow-up and investigation will be required to determine if the durability of this approach compares favorably to current open options for this rare condition. ■

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