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Surgical treatment of posterior nutcracker syndrome presented with hyperaldosteronism

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Abstract

Posterior nutcracker syndrome is caused by the compression of left renal vein between the abdominal aorta and the vertebral body. Most seen symptoms are haematuria, left flank pain, abdominal pain and varicocele. The nutcracker syndrome may lead to left renal vein thrombosis due to blood congestion within compression of the vessel. Both endovascular and open surgical interventions can relieve symptoms; however, traditional surgical repair is still considered as the gold standard. Here, we present the surgical treatment of a 36-year old female with complaints of hypertension, hyperaldosteronism and diagnosed with posterior nutcracker syndrome.

Keywords: Nutcracker syndrome • Posterior • Surgical treatment • Diagnosis

INTRODUCTION

The nutcracker syndrome/phenomenon is also known as the left renal vein entrapment syndrome and was first described by Grant in 1937. The left renal vein mostly is compressed between the abdominal aorta and the superior mesenteric artery. The nutcracker syndrome may lead to left renal vein thrombosis due to blood congestion within compression of the vessel. Anterior nutcracker syndrome is caused by abnormal branching of the superior mesenteric artery that decreases the aorta-mesenteric angle to below 90°, and posterior nutcracker syndrome is caused by the compression of the left renal vein between the abdominal aorta and the vertebral body [1]. The left renal vein has 1–3% retroaortic course. Rarely, the third portion of the duodenum accompanies left renal vein on the anterior nutcracker syndrome and this phenomenon is also known as the superior mesenteric artery syndrome (Wilkie's syndrome). The exact prevalence of the nutcracker syndrome is unknown, although it is slightly higher in females and is not a hereditary condition. Most commonly seen symptoms are haematuria, left flank pain, abdominal pain, varicocele, abnormal menstruation, pelvic congestion syndrome, gonadal vein syndrome, orthostatic proteinuria and orthostatic abnormalities. Symptoms can be triggered or aggravated by physical activity, pregnancy or multiparity [2].

CASE REPORT

A 36-year old female presented with complaints of hypertension (average blood pressure 140/80 mmHg) for 2 years (Irbesartan 150 mg/day orally), hyperaldosteronism and intermittent left flank pain for 4 years. Plasma cortisol level was 16.45 μg/dl, adrenocorticotropic hormone was 68.29 (0–46 normal limits) pg/ml, creatinine was 0.86 (0.4–1.4 normal limits) mg/dl, potassium was 3.2 (3.5–5.5 normal limits) mEq/l, renin was 0.02 (0.5–1.9 normal limit while lying) ng/ml/h and aldosterone level was 663,17 (8–172 normal limits) pg/ml. Computed tomography scan and venography of left renal vein and inferior vena cava were done (Fig. 1). Left renal vein was compressed between the aorta and vertebral body and the distal part was dilated. She was diagnosed to have posterior nutcracker syndrome. We decided to perform left renal vein transposition surgery. After routine preparation for surgery was made and informed consent was taken, we reached retroperitoneum via partial midline laparotomy above the umbilicus to xyphoid. The left renal vein was found coursing behind the abdominal aorta. We put a clamp on the left renal artery, left renal vein and inferior vena cava after systemic heparin administration (5000 IU), 0.25 mg/kg mannitol, and external cooling was provided by slush ice. The length of the renal vein was not enough to reach the inferior vena cava; therefore, interposition was performed with an 8-mm polytetrafluoroethylene (PTFE) graft between left renal vein and the inferior vena cava (Fig. 2) by anteaortic route. The patient was discharged on postoperative day 3 without any significant event with an asatilsalisilic asit 100 mg per day. Postoperative day 15 and 6 month Doppler ultrasonography, clinical examinations and biochemical blood tests were performed. No complication was detected. Plasma adrenocorticotropic hormone and aldosterone level decreased to 40.06 (0–46 normal limits) and 17.98 (8–172 normal limits) pg/ml, respectively and

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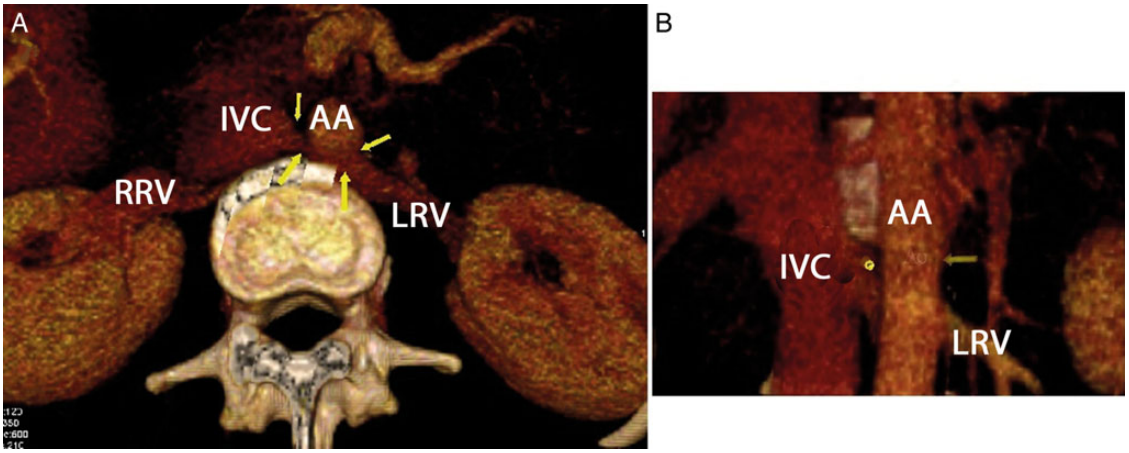


Figure 1: Retroaortic course of the left renal vein (LRV; shown on computed tomography three-dimensional reconstruction). (**A** and **B**) LRV was compressed and dilated behind the abdominal aorta (AA). IVC: inferior vena cava; RRV: right renal vein.

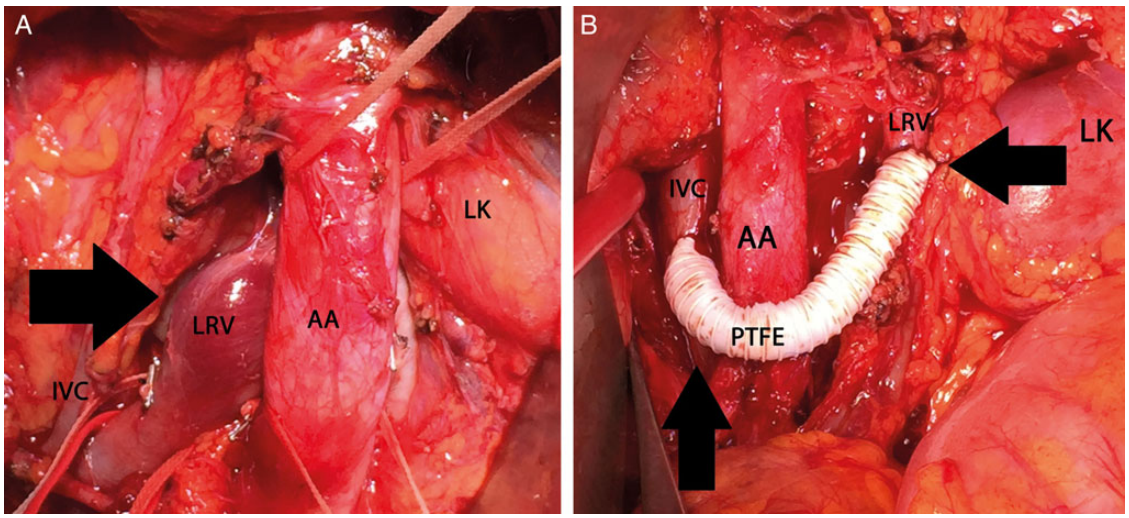


Figure 2: Intraoperative images. (**A**) The retroaortic course of the left renal vein (LRV, arrow) and the courses of the abdominal aorta (AA) and left kidney (LK). (**B**) Polytetrafluoroethylene (PTFE) graft bypass between LRV and inferior vena cava (IVC).

plasma renin level increased to 0.96 (0.5–1.9 normal limits while lying) ng/ml/h. She underwent Doppler ultrasound with the patency conformation of the bypass graft. Her blood pressure was within normal limits. The patient had no more complaints.

DISCUSSION

Renal venography, computed tomography angiography, digital subtraction angiography, standard magnetic resonance angiography and Doppler ultrasound are used for diagnosing the nutcracker syndrome [3]. Only 13 cases have been reported in the literature; among them, 4 patients were treated surgically and 4 were treated conservatively (Supplementary Table 1). Severity and stage of symptoms and patient's age are essential for treatment modality in order to reduce the left renal vein hypertension; however, management is controversial. For patients who are younger than 18 years old or those presenting with mild haematuria, the best treatment option is the conservative therapy with a follow-up of at least 2 years. Hence, 75% of patients have complete resolution of haematuria (fat tissue increases in time between the superior mesenteric artery and the left renal vein). A variety of surgical approaches have been used including left renal vein bypass, left renal vein transposition with PTFE, Dacron or saphenous vein graft, medial nephropexy with excision of renal varicosities, superior mesenteric artery transposition, renal autotransplant, gonadocaval bypass and nephrectomy, wrapping of the left renal vein with ringed PTFE. Endovascular stenting can also be used; however, it may lead to stent migration, thrombosis, restenosis, deformities and erosions [4]. In this case, we chose surgical therapy on account of the lack of improvement after 4 years with medical therapy. We chose left renal vein bypass rather than transposition on account of shortness of left renal vein and subsequent possibility of high pressure on left renal vein. And also, we chose PTFE graft rather than saphenous vein graft to avoid diameter mismatch. Long-term results of left renal vein transposition demonstrate a reasonably high rate resolution of symptoms of flank pain, hypertension and haematuria [5]. Endovascular stent placement for retroaortic left renal vein may cause aorto-venous fistula due to stent erosion of the posterior aortic wall [3]. Traditional surgical repair is still considered as the gold standard. Based on our

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experience, open surgical approach with left renal vein bypass or transposition provides a safe method with satisfying long-term results and should be considered as the first therapeutic option.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *ICVTS* online.

Conflict of interest: none declared.

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