

Secondary hypertension associated with nutcracker syndrome: a case report

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ABSTRACT

Nutcracker syndrome (NCS) is defined as the compression of the left renal vein (LRV) between the aorta and the superior mesenteric artery, accompanied by clinical findings. Here, we present an adolescent patient diagnosed with NCS during the investigation of secondary causes of arterial hypertension. A 13-year-old female patient presented to our outpatient clinic with complaints of headache and a home-measured blood pressure of 160/100 mmHg. In the clinic, her blood pressure was measured at 150/100 mmHg in the right arm and 160/100 mmHg in the left arm. CT Imaging revealed compression of the LRV between the superior mesenteric artery and the aorta. The angle between the superior mesenteric artery and the abdominal aorta was measured as 18.3 degrees. In conclusion, blood pressure should be routinely monitored in patients with NCS, as hypertension may develop during follow-up.

Keywords: Nutcracker syndrome, child, hypertension

INTRODUCTION

Nutcracker syndrome (NCS) refers to a condition in which the left renal vein (LRV) is compressed between the aorta and the superior mesenteric artery (SMA), leading to various clinical symptoms. Individuals with NCS may experience symptoms such as hematuria, proteinuria, pain in the left flank or abdomen, varicocele on the left side in males, and pelvic congestion syndrome in females.^{1,2} There are only a handful of reported pediatric cases in the literature describing the coexistence of NCS and arterial hypertension.^{3,4} Here, we present an adolescent patient diagnosed with NCS during the investigation of secondary causes of arterial hypertension. This case aims to demonstrate the association between NCS and secondary hypertension.

CASE

A 13-year-old female patient presented to our outpatient clinic with complaints of headache and a home-measured blood pressure of 160/100 mmHg. In the clinic, her blood pressure was measured at 150/100 mmHg in the right arm and 160/100 mmHg in the left arm. Although not obese, the patient was

considered overweight (BMI=28.5 kg/m²). Blood chemistry results were as follows: blood urea 30 mg/dl, creatinine 0.8 mg/dl, triglyceride 124mg/dl, cholesterol 132mg/dl. Free T4 and TSH were in the normal range. Urinalysis revealed 3–4 red blood cells per microliter, consistent with borderline microscopic hematuria. Plasma renin and aldosterone levels could not be assessed due to the initiation of pharmacological treatment. She was admitted for further evaluation. Despite 12 hours of monotherapy with an antihypertensive agent (Amlodipine), her blood pressure remained between 140/90 and 150/100 mmHg. Therefore, a thoracoabdominal CT angiography was performed. Imaging revealed compression of the LRV between the SMA and the aorta. The angle between the SMA and the abdominal aorta was measured as 18.3 degrees (**Figure**) which is smaller than the normal range (38–65°).⁵ Based on these findings, the patient was diagnosed with NCS. A second antihypertensive agent (Enalapril) was added to her treatment regimen. Following this adjustment, her blood pressure stabilized between 110/70 and 120/80 mmHg. The patient was consulted for cardiovascular surgery, and surgical intervention was deemed unnecessary.



Figure. Compression of the left renal vein between the superior mesenteric artery and the aorta. The angle between the SMA and the abdominal aorta was measured as 18.3 degrees
SMA: Superior mesenteric artery

DISCUSSION

There are very few studies demonstrating an association between NCS and arterial hypertension. In one report, a 17-year-old girl presented with intermittent hypertension, loin pain, and hematuria, and imaging revealed compression of the LRV consistent with NCS.³ In another adolescent male case, transient high blood pressure was incidentally associated with a nutcracker phenomenon, with normal renin/aldosterone levels and resolution of hypertension after weight gain and conservative management.⁴ In our case, which was evaluated for secondary hypertension, no other pathology was identified to explain the elevated blood pressure. Hypertension in NCS is thought to result from venous stasis within the left kidney, leading to renal venous hypertension and consequent activation of the renin-angiotensin-aldosterone system (RAAS). Compression of the LRV impairs venous outflow, increasing intrarenal venous pressure and reducing effective renal perfusion. This decrease in perfusion stimulates renin release from juxtaglomerular cells, which promotes systemic vasoconstriction and sodium retention through RAAS activation. These processes ultimately lead to elevated arterial blood pressure.⁶ Among the surgical treatment options for NCS are open surgery, open or laparoscopic extravascular stent placement, and endovascular stenting.⁷

CONCLUSION

As a result, blood pressure should be routinely monitored in patients with NCS, as hypertension may develop during follow-up.

ETHICAL DECLARATIONS

Informed Consent

The patient's family signed the free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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