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Research Article

A YOUNG MALE WITH NUTCRACKER PHENOMENON AND RESISTANT HYPERTENSION

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A B S T R A C T

Nutcracker phenomenon is a vascular compression disorder. This is easily missed by routine diagnostic methods and it's incidence is likely underestimated. We describe a male with resistant hypertension where CT Abdomen revealed posterior nut cracker phenomenon. His BP was controlled partially with three drugs including diuretic without any secondary cause for elevated blood pressure. At present he is on medical management planning for surgical intervention if not controlled.

INTRODUCTION

The nut cracker syndrome or phenomenon implies compression of left renal vein as it passes through the angle between aorta and superior mesentric artery. The terms syndrome and phenomenon have been used quite often interchangeably in literature. Nutcracker syndrome (NCS) must be used only when there is evidence of nutcracker anatomy accompanied with clinical symptoms, otherwise Nutcracker phenomenon (NCP) looks more appropriate. NCS is a rare entity, which can present as hematuria, orthostatic proteinuria, flank pain, left sided varicocele, pelvic congestion, mild anaemia or chronic fatigue. Hypertension is not included in the classical signs of NCS.

Case Repport: A 28 year old male with symptoms of recurrent giddiness and palpitation was evaluated from various hospitals and detected to be hypertensive since the age of 15 years. He was initially started on Metaprolol 50 mg OD 8 years ago. He was started on regular treatment with three drug regimen for hypertension only 3 years ago, which he discontinued after 3 months. He then went abroad where he was treated with another antihypertensive medications, which he continued for 1 year. After returning to India he received multiple drugs for hypertension which included telmisartan 40 mg, amlodipine 5 mg and bisoproplol 5 mg. He was quite irregular in taking medicines.

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He came to us for control of resistant hypertension. At present he is on Telmisartan 40 mg, chlorthalidone 12.5 mg, cilnidipine 10 mg and metaprolol 50 mg. His blood pressure is 150/100 mmHg. He is 157 cm tall with BMI of 26.1 %. His routine clinical and systemic examinations were with in normal limits. No arterial bruit over neck, abdomen or interscapular area. All peripheral pulsations are equally felt. Investigations showed no urine abnormality. Urine total metanephrine levels were normal. His complete blood count, thyroid function, renal function, liver function, blood sugar and lipid profile were with in normal limits. ANA profile and USG abdomen were normal.

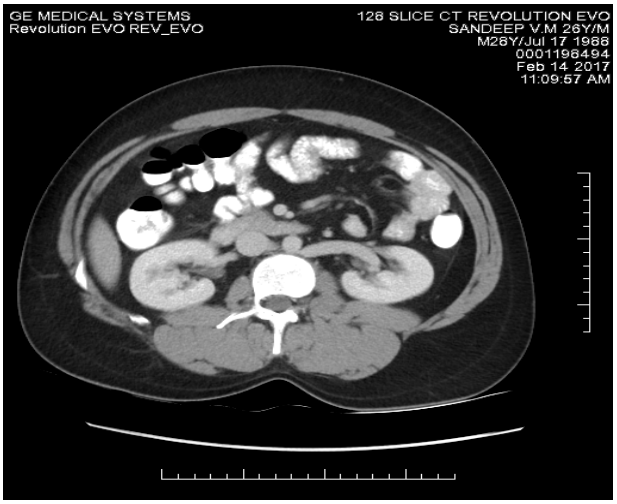

Figure 1 CT abdomen showing retro aortic course of left renal vein, which is compressed between aorta and L2 vertebral body, thereby causing posterior nut cracker phenomenon.

Most of the causes of secondary hypertension like renal artery stenosis and pheochromocytoma were excluded. We proceeded with CT abdomen which revealed retro-aortic course of left renal vein with compression between aorta and L2 vertebral body. (Figure 1)

DISCUSSION

In the nutcracker syndrome anatomical changes generate no specific symptomatology making the condition underdiagnosed[1]. D Archambear *et al* [2] reported 83% incidence of the syndrome in patients with pelvic congestion. Initially it was described by El-Sadr and Mina [3] in 1950 and later named by Schepper as nut cracker syndrome in 1972, which is the left renal vein compression by the aorta and superior mesenteric artery.

Nut cracker syndrome and nut cracker phenomenon are two different entities, though these two are used interchangeably in the literature. Nut cracker phenomenon refers to the compression of left renal vein most commonly between the aorta and superior mesenteric artery with lateral dilatation and medial narrowing leading to impacted outflow from left renal vein into the inferior venacava, but this phenomenon may represent a normal variant or be accounted for any other conditions. Shin and Leeemphasize that the nutcracker anatomy is not always associated with clinical symptoms and that some of the anatomic findings suggestive of nutcracker may represent a normal variant or be accounted for by other conditions. Therefore, the term *nutcracker syndrome* should be reserved for patients with characteristic clinical symptoms associated with demonstrable nutcracker morphologic features. No consensus exists on what symptoms are severe enough to warrant the designation of a clinical syndrome or to what extent various findings may simply reflect different evolutionary stages of the process [4].

There is increased pressure in the left renal vein and may result in severe symptoms and signs such as pain in flanks and haematuria, left varicocele, orthostatic proteinuria and pelvic congestion. It is due to venous reflux with formation of collaterals. This may lead to chronic pelvic pain associated with dysmenorrhea, dyspareunia and dysuria and chronic fatigue syndrome [5,6]. Most typical nut cracker features imply compression of left renal vein (LRV) between the aorta and the superior mesenteric artery (SMA), known as anterior nutcracker syndrome. Sometimes a third part of duodenum crosses in front of the LRV between the aorta and the SMA. Thus anterior nut cracker may occur along with compression of the duodenum by the SMA known as the superior mesenteric artery syndrome (WILKIE syndrome) [7,8]. The retro-aortic or circum-aortic renal vein may be compressed between the aorta and the vertebral body and known as posterior nut cracker syndrome[9].

In our patient the retro-aortic segment appears flattened between aorta and L2 vertebral body. The segments on either side of the retro aortic part appears ectatic suggesting the possibility of posterior nut cracker phenomenon. Dilated lumbar veins are seen draining into left renal vein. It is assumed that nut cracker phenomenon is caused by nephroptosis above or decreased retro peritoneal adipose tissue leading to the elongation of the left renal vein and reduce the angle between the SMA and the aorta[10]. He had no symptoms described in NCS. His body mass index was 26.1% with height of 167 cms. Lower body mass correlates positively with NCP [11]. Hosotoni *et al* have reported a case of NCP with hypertension with increased plasma renin activity and aldosterone levels in the peripheral blood of a young Japanese woman. Patient became normotensive and plasma renin activity levels normalised after endovascular stent placement of the affected renal vein [12].

He is on four anti hypertensive medications including a diuretic and his BP only partially controlled. We were not able to identify any secondary causes for hypertension, like renal artery stenosis, pheochromocytoma or endocrinopathy except for CT abdomen finding suggestive of posterior nut cracker phenomenon. NCP/NCS has been reported concurrently with various clinical entities such as IgA nephropathy, membranous nephropathy, idiopathic hypercalcemia, henochschonleinpurpura, but our patient has no evidence of glomerular damage[13]. After discussing with urologist we came to conclusion that these finding might represent a normal variant and do not require interventional management at this moment.

In patients with severe symptoms like haematuria or flank pain, interventions aiming to decrease left renal vein hypertension were undertaken[14-15]. Various surgical techniques like endovascular therapy or renal auto transplantation were performed with rather acceptable results. In our case we were not able to establish any correlation of hypertension with the described nut cracker anatomy.

CONCLUSION

Current literature so far reviewed revealed that only one case was reported earlier where renal vein stent replacement normalized the blood pressure. As there was no established pathological links, hypertension could not be included in clinical findings that may commonly accompany Nutcracker phenomenon and may be considered as coincidence until more such cases studies are reported in future.

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