CASE REPORT

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Reninoma: A Rare Cause of Surgically Curable Hypertension and Secondary Hyperaldosteronism

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Abstract

Reninoma is a rare cause of renin-induced hypertension commonly seen in young adults and adolescents. Here, we describe a case of reninoma presenting with hypertension in the young. Further investigation revealed elevated renin and aldosterone levels. Computed tomography of the kidneys showed a right-sided renal mass. A diagnosis of reninoma was made, and the patient underwent nephron-sparing surgery, which rendered her normotensive post-operatively.

Key words: reninoma, renin-induced hypertension

INTRODUCTION

Reninoma is an extremely rare cause of hypertension, with less than 200 cases reported so far.[1] It is a tumour of the juxtaglomerular apparatus which secretes renin, leading to secondary hyperaldosteronism, ultimately causing hypertension. This disease is commonly seen in adolescents and young adults and has a female preponderance. Patients with reninoma mostly present with hypokalaemia although they can also be normokalaemic. Most reninomas are benign, and surgical resection can render the patient normotensive.[2] Here we report a case of reninoma in a young girl who presented with hypertension and palpitations. The patient underwent nephron-sparing surgery, resulting in the cure of her hypertension and palpitations.

CASE

A 23-year-old female was referred to our clinic for hypertension in the young. She has initially presented to a general practitioner with complaints of headache with palpitations for one year. She was found to have a home ambulatory blood pressure reading ranging between 130-180 mmHg (systolic) and 85-120 mmHg (diastolic). She was diagnosed with hypertension and started on doxazocin tablets. On further history, she complained of paroxysms of sweating with palpitations. She did not have symptoms of heat intolerance or diarrhoea. There was no history of weight gain, easy bruising or thinning of skin. She denied consumption of any supplements or over-the-counter medications. She had normal development and growth and had attained menarche at age 12 with regular menses. Her family history was unremarkable.

On examination, she was a moderately-built girl with a body mass index of 20.4 kg/m^2. Her pulse rate was 100 beats per minute and her blood pressure was 153/100 mmHg. She did not appear Cushingoid, there were no features of acromegaly and there was no goitre or exophthalmos. She had a regular pulse with no radio-radial or radio-femoral delay. Her apex beat was on the mid-clavicular line at the 5th intercostal space, with normal heart sounds. Her lungs were clear, and abdomen was soft with no organomegaly. There was no carotid or renal bruit heard. Tanner staging of her breast and pubic hair was appropriate for age.

Her routine blood investigations were normal. Thyroid function and urine metanephrines were within normal limits. Urine test revealed proteinuria of 1+, this was not quantified. Her electrocardiography (ECG) fulfilled voltage criteria for left ventricular hypertrophy (LVH). Transthoracic echocardiogram was normal and 24-hour Holter monitoring showed episodes of sinus tachycardia of up to 120 beats per minute. There were no arrhythmia detected. Her aldosterone and renin levels revealed an elevated renin level at 518 mU/L (4.4- 46.1 mU/L) with an elevated aldosterone of 998 pmol/L (61.2- 997.8 pmol/L).

A contrasted computed tomography (CT) of abdomen and pelvis was performed and this found a well-encapsulated heterogeneously-enhancing mass on the upper pole of the right kidney measuring 2.7 × 3 × 3.3 cm (Figure 1) with no other lesions elsewhere. Both adrenal glands were normal, and there was no evidence of renal artery stenosis. Given the raised renin and aldosterone values, a renin producing tumour was suspected. We proceeded with a biopsy of the renal mass to confirm the diagnosis. The histopathologic

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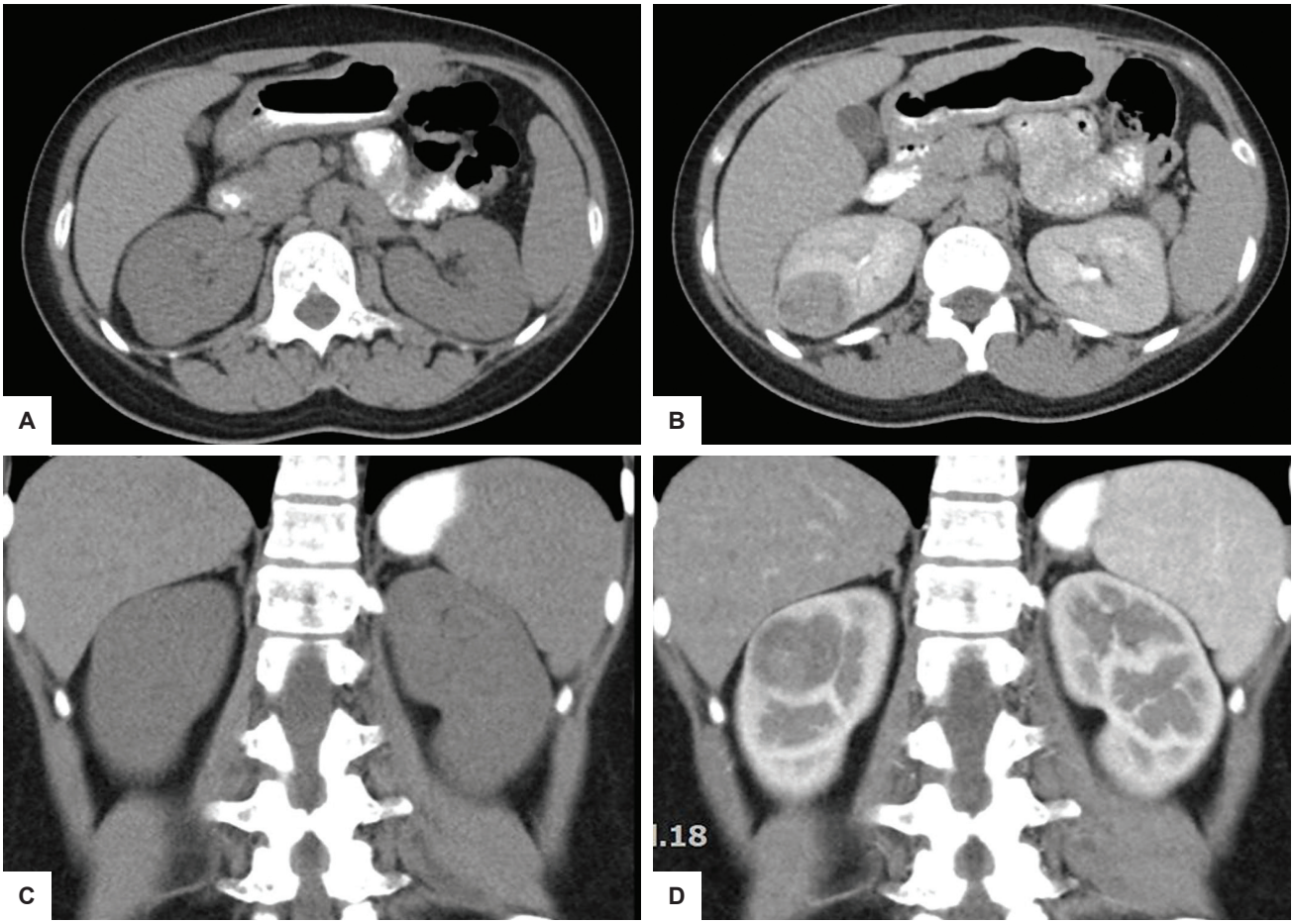

Figure 1. Contrast computed tomography (CT) of abdomen and pelvis demonstrated a well-encapsulated heterogeneously-enhancing mass on the upper pole of the right kidney. **(A)** Pre-contrast, mass appears isodense; **(B)** post-contrast, mass appears hypodense compared to renal cortex; **(C)** pre-contrast, mass appears isodense; **(D)** post-contrast, mass appears hypodense compared to renal cortex.

examination of the biopsy showed neoplastic cells that stained positive for smooth muscle actin, caldesmon and negative for beta-catenin. Examination under electron microscopy was not done due to resource limitations. Overall, these features were suggestive of a reninoma.

She was then switched to an angiotensin-converting enzyme inhibitor (perindopril) and a mineralocorticoid receptor antagonist (spironolactone) for adequate blood pressure control. She reported experiencing fewer episodes of palpitations after the switch of medications were made. She was referred to the urology team and underwent a partial nephrectomy of the right kidney. Her surgery was uneventful, with no fluctuations in blood pressure intraoperatively.

Post operatively, her blood pressure ranged from 100-117 mmHg (systolic) and 75-90 mmHg (diastolic) and both anti-hypertensive medications were withheld. She was reviewed for 2 months post-surgery in our clinic. She no longer complained of palpitations and her heart rate was between 75-86 beats per minute. Her home blood pressure monitoring was within normal limits. ECG revealed resolution of left ventricular hypertrophy, urine dipstick was negative for protein and renal profile done showed normokalaemia. Histopathological examination of the resected tumour revealed an encapsulated lesion that stained positive for CD 34 (Figure 2A), CD 117 (Figure 2B), vimentin (Figure 2C) with absence of mitotic figures, confirming the diagnosis of reninoma with benign features.

DISCUSSION

In general, all patients with reninomas were found to have a high renin and aldosterone levels, while some cases have reported high renin levels with a normal aldosterone level,[2] albeit methods of sampling renin and aldosterone where not specified in most case reports. Typically, reninomas are small in size and have a cortical or subcortical location.[3] There is also evidence that these tumours grow in size with time, suggesting that repeated interval scanning maybe useful in cases of uncertainty or when initial scan fails to identify a lesion.[4] In the presence of renin-mediated hypertension, a reninoma should be suspected in the absence of renovascular disease. In our patient, in addition to high blood pressure, she also had palpitations and was found to have sinus tachycardia. Activation of sympathetic nervous system by the renin-angiotensin aldosterone

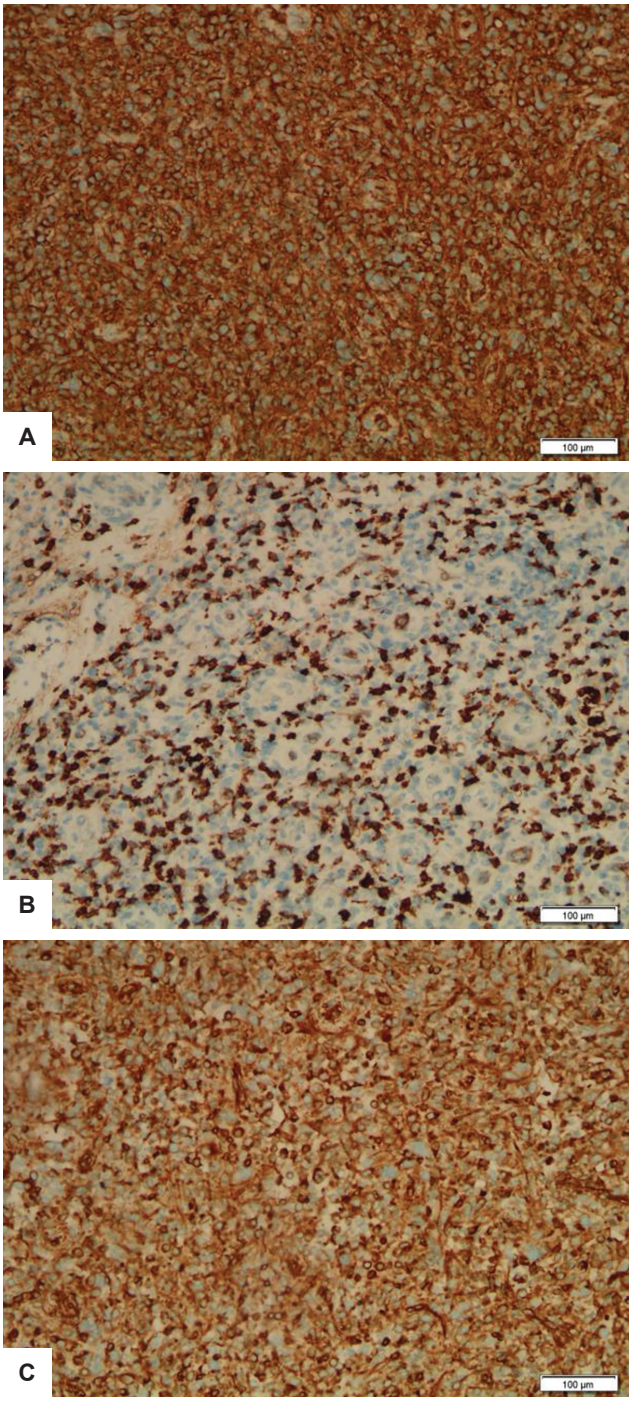

Figure 2. Histopathologic staining of the resected tumour (Immunohistochemistry, 200×): **(A)** CD34; **(B)** CD117; **(C)** vimentin.

system (RAAS) via angiotensin 2 has been well studied and is likely the reason for tachycardia in our patient, although other cases have not reported the same.[5] Her tachycardia had significantly reduced with the ACE inhibitors and MR antagonists and had resolved completely post-operatively. Recommended medical management of reninomas are medications directly inhibiting the RAAS pathway such as ACE inhibitors and MRAs.[2] In one case report, aliskiren, a direct renin inhibitor at doses of 300 mg daily successfully controlled the blood pressure in a patient awaiting surgery.[6] However, resistance to aliskiren after 5-6 months of usage was reported.[6] Hypertensive organ damage has been commonly associated with reninomas. In a case series of 9 patients, 3 had severe hypertensive damage resulting in fetal demise in one pregnant patient, intracranial bleed in the second patient and severe retinopathy in the third patient.[7] There is insufficient evidence to conclude if hypertensive complications are reversible post-surgery. In our patient, however, there was resolution of LVH and proteinuria seen post-operatively. The best diagnostic imaging is a contrasted computed tomography (CT) scan, which has shown 100% sensitivity.[8] Reninomas generally are isodense or hypodense pre-contrast, with delayed enhancement seen post-contrast. Renal vein sampling (RVS) has been used as an adjunct to aid with diagnosis of reninomas. It is done by canulating both renal veins and comparing renin values on both sides. A review of previous renal vein sampling found a sensitivity and specificity of a lateralizing ratio of 1.5 to be 56% and 94% respectively.[2] In a small case series of 3 patients with reninoma, RVS has successfully lateralized the lesion in all 3 patients. These patients were meticulously prepared pre-procedure; interfering medications were withheld; patients were put on a low sodium diet of less than 40 mmol/day and were maintained in a recumbent position overnight and during the procedure. Patients were also given captopril, and renin values were sampled 15 minutes after, which increased the sensitivity of the test.[8] Overall, this suggests that if renal vein sampling were to be performed, good patient preparation and administration of captopril may yield a better sensitivity of RVS. In our case, due to the lack of resources and the rarity of this disease, a biopsy of the lesion was undertaken to ascertain the diagnosis prior to subjecting the patient to surgery. While most cases of reninomas are benign, there have been very few cases of malignant reninomas in the literature. In one case report, there was metastatic disease in a paraaortic lymph node with a recurrent mass seen 1 year post initial resection of a reninoma, and histopathological examination revealed a high mitotic index with perivascular invasion.[3] Histologically, these tumours are made of closely packed polygonal cells with oval to round nuclei. Although moderate nuclear atypia is seen, mitotic figures are usually absent. The diagnosis is made with the demonstration of renin positivity in the cytoplasm, although this feature may also be observed in other disease such as renal cell carcinoma and Wilms tumour. Most reninomas stained diffusely for vimentin and CD 34, while CD 117 positivity has been reported in most but not all cases.[9] Presence of rhomboid-shaped renin protogranules via an electron microscopy is diagnostic.[1] Ideally, a nephron-sparing surgery is the mainstay of treatment for reninomas. However, in very large tumours or centrally located tumours, a total nephrectomy may be required. Generally, blood pressure fluctuations are not seen intraoperatively, but there has been hypotension reported intraoperatively during a resection of a large reninoma.[3] Radiofrequency ablation use has also been reported in 2 patients by Ru et al, however one patient developed hypertension post ablation requiring treatment.[10]

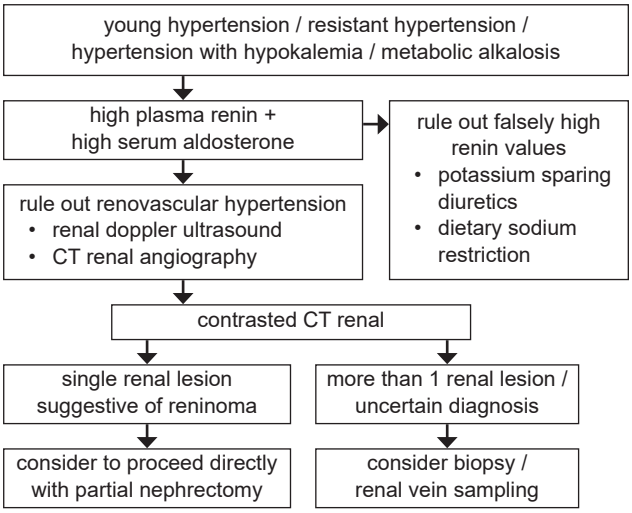

Figure 3. Approach to Reninoma.

Table 1. Imaging modalities and treatment options for Reninomas

	Medical management	Imaging modality	Treatment options
Preferred	Mineralocorticoid receptor antagonist (MRA)	Computed tomography of the kidneys	Partial nephrectomy or total if large reninoma or centrally located
Alternative/ Additional	Angiotensin converting enzyme inhibitors (ACE)	Renal MRI Renal vein sampling	Radiofrequency ablation

CONCLUSION

Reninomas are mostly benign neoplasms that can lead to hypertension and severe end organ damage. Despite primary aldosteronism being the more common cause of young hypertension, when faced with elevated renin alongside an elevated aldosterone, a reninoma should be considered a possibility and renal imaging should be performed. A high clinical suspicion is required to diagnose this disease and to avoid unwarranted imaging and procedures. Nephron-sparing surgery can render the patient normotensive.

Ethical Consideration
Patient consent forms were obtained before manuscript submission.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement
GK: Writing – original draft, Writing – review and editing; **LJL:** Writing – original draft, Writing – review and editing; **THC:** Supervision; **SK:** Supervision.

Data Availability Statement
Datasets generated and analyzed are included in the published article.

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References
1. Wang B, Ding L, Xu S, et al. A case of atypical reninoma with mild hypertension and normal plasma renin activity but elevated plasma renin concentration. BMC Endocr Disord. 2022;22(1):71. PMID: 35303838 PMCID: PMC8932068 DOI: 10.1186/s12902-022-00977-w
2. Wong L, Hsu TH, Perlroth MG, Hofmann LV, Haynes CM, Katznelson L. Reninoma: Case report and literature review. J Hypertens. 2008;26(2):368-73. PMID: 18192852 DOI: 10.1097/HJH.0b013e3282f283f3
3. Shera AH, Baba AA, Bakshi IH, Lone IA. Recurrent malignant juxtaglomerular cell tumor: A rare cause of malignant hypertension in a child. J Indian Assoc Pediatr Surg. 2011;16(4):152-4. PMID: 22121315 PMCID: PMC3221159 DOI: 10.4103/0971-9261.86876
4. Diker-Cohen T, Abraham SB, Rauschecker M, et al. Reninoma presenting in pregnancy. J Clin Endocrinol Metab. 2014;99(8):2625-6. PMID: 24848711 PMCID: PMC4121021 DOI: 10.1210/jc.2014-1730
5. Bealer SL. Systemic angiotensin II alters intrinsic heart rate through central mechanisms. Brain Res Bull. 2002;58(1):61-5. PMID: 12121814 DOI: 10.1016/s0361-9230(02)00756-6
6. Rosei CA, Giacomelli L, Salvetti M, et al. Advantages of renin inhibition in a patient with reninoma. Int J Cardiol. 2015;187:240-2. PMID: 25838223 DOI: 10.1016/j.ijcard.2015.03.280
7. Liu K, Wang B, Ma X, et al. Minimally invasive surgery-based multidisciplinary clinical management of reninoma: A single-center study. Med Sci Monit. 2019;25:1600-10. PMID: 30822300 PMCID: PMC6407327 DOI: 10.12659/MSM.913826
8. Wolley M, Gordon RD, Stowasser M. Reninoma: The importance of renal vein renin ratios for lateralisation and diagnosis. Am J Nephrol. 2014;39(1):16-9. PMID: 24401674 DOI: 10.1159/000357410
9. Vidal-Petiot E, Bens M, Choudat L, et al. A case report of reninoma: Radiological and pathological features of the tumour and characterization of tumour-derived juxtaglomerular cells in culture. J Hypertens. 2015;33(8):1709-15. PMID: 26132761 DOI: 10.1097/HJH.0000000000000592.
10. Zhang R, Xu M, Xie XY. The role of real-time contrast-enhanced ultrasound in guiding radiofrequency ablation of reninoma: Case report and literature review. Front Oncol. 2021;11:585257. PMID: 33686368 PMCID: PMC7935754 DOI: 10.3389/fonc.2021.585257