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CASE REPORT

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Unravelling a mystery of hypokalemic hypertension- a rare case report of a reninoma

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Abstract

Background Reninoma is a rare cause of secondary hypertension, which can be cured with surgery if identified early before any target organ damage occurs. It leads to hypokalaemia and hypertension and typically responds well to treatment with renin–angiotensin–aldosterone system blockers. However, confirmation of the diagnosis and the localisation of this rare culprit lesion can be challenging.

Case presentation We describe a case of young-onset hypertension in a 19-year-old girl due to a reninoma. She had resistant hypertension with marked hypokalaemia, which required exceedingly high doses of potassium supplements. Biochemical Investigations revealed secondary hyperaldosteronism. Thus, she underwent a renal angiogram to exclude a renovascular cause for her hypertension. While the renal artery anatomy was normal, there was an exophytic renal lesion in the lower pole of the left kidney. Hence, the diagnosis of a reninoma was suspected. She underwent renal vein sampling to confirm the functionality of the detected tumour, but the results were inconclusive. After a multidisciplinary discussion, based on the clinical evidence, the renal lesion was thought to be a reninoma and a partial nephrectomy was done, removing the lesion. Immediately following resection, her blood pressure and potassium normalised without further drug treatment, and the resected lesion was later confirmed to be a reninoma by histopathological examination.

Conclusion In young people with hypokalemic hypertension, reninoma should be considered when the more common causes are excluded since prompt treatment with excision of the culprit lesion can cure hypertension and prevent associated morbidity and mortality.

Keywords Reninoma, Hypokalaemic hypertension

Introduction

Global hypertension prevalence studies in 2010 suggested that it affects 1 in 4 adults, amounting to a daunting total of 1.4 billion hypertensives worldwide [1]. Hypertension is the number one risk factor for deaths globally [2]. In addition to the contribution to morbidity and mortality of the population, there is a significant financial burden related to hypertension-related complications. To further complicate the issue, hypertension typically remains asymptomatic until signs of end-organ damage become apparent.

Depending on the population studied, 5%–10% of hypertensive patients have underlying secondary causes for hypertension, but the exact prevalence is unknown as there is selection bias in most studies. These secondary causes can be successfully treated when identified, curing hypertension. Primary aldosteronism, or renin-independent autonomous aldosterone production, is the most common and curable form of secondary hypertension

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[3]. Although relatively uncommon, secondary aldosteronism, also known as renin-dependent aldosteronism, does occur. The main reason for secondary aldosteronism is the renovascular causes like renal artery stenosis secondary to atherosclerosis or rare forms like fibromuscular dysplasia or polyarteritis nodosa affecting the renal arteries. Renin-secreting tumours: reninomas/juxtaglomerular cell tumours are the rarest form of secondary aldosteronism [4].

Reninomas are more frequently reported in females (female to male ratio of 2:1), particularly those of childbearing age, with an average age of 27 years [5]. The most common clinical features are hypertension and hypokalaemia, present in about 80% of cases, and are challenging to manage without renin–angiotensin–aldosterone system (RAAS) blockers [5].

Biochemical tests typically show elevated plasma renin concentration and activity with elevated aldosterone levels. Further imaging studies are needed to find the cause of excess renin. Renal artery doppler and a CT angiogram are essential to rule out renal artery stenosis. If they are normal, the possibility of a reninoma should be entertained.

Reninomas usually appear as small cortical or corticomedullary lesions on imaging. On CT, they are isodense with slight contrast enhancement [4]. On MRI T1 images, the lesion will be isointense, but T2 images can be iso, hypo or hyperintense [6]. Functional imaging techniques like FDG PET or DOTATATE PET have not been effective in diagnosing reninomas [4, 6].

Renal vein sampling (RVS), with measurement of plasma renin activity or direct renin concentration, is the preferred test to confirm the secretory nature of the lesion, although its sensitivity is limited to around 56% [5]. Different protocols for RVS exist, but a recent study by Hayes et al. proposed a preparation protocol to improve RVS results by focusing on the underlying physiology of renin secretion [4].

If localised, reninomas are easily cured by surgery, with typical blood pressure and potassium response evident within days after surgery. Surgery is nephron-sparing partial nephrectomy. It can be done laparoscopically using a retroperitoneal approach if anatomy is favourable [7, 8]. There have been cases of reninomas treated successfully with radiofrequency ablation when the anatomical location is favourable [9, 10].

Medical management of reninomas includes RAAS blocking agents if the lesion is not localised. However, the blood pressure response to these drugs can wane over time with resultant end-organ damage due to hypertension. Agents which can be used include angiotensin-converting enzyme blockers (enalapril, captopril), angiotensin receptor blockers (losartan, irbesartan), mineralocorticoid receptor blockers (spironolactone, eplerenone), epithelium sodium channel blockers (amiloride) and lastly direct renin inhibitor (aliskiren). Considering that reninomas primarily affect women of childbearing age, if a patient with reninoma becomes pregnant after medical treatment, managing high blood pressure becomes challenging since all these antihypertensive medications are contraindicated during pregnancy [4].

Histopathology can confirm the diagnosis of reninoma. Reninomas exhibit similarities with other renal cell tumours by having round, oval, or polygonal cells, eosinophilic cytoplasm, and oval or round nuclei, which show minimal atypia [11, 12]. Immunohistochemistry stains help differentiate reninomas from more common tumours: Reninomas typically show positive staining for CD34, renin, and vimentin [12–14]. Rhomboid renin crystals are observable through electron microscopy [14].

Even though it is rare, once diagnosed early and treated surgically, reninomas have excellent prognoses and good clinical outcomes. So, it is essential to bear in mind the possibility of reninoma while assessing young individuals with hypokalemic hypertension.

Case description

We report a case of a 19-year-old unmarried female who was incidentally detected to have high blood pressure during hospital admission for a febrile illness one year ago. At the time of presentation to our institution, she had hypertension for six months duration and was on losartan 50 mg BID. She denied any symptoms suggestive of proximal muscle weakness, polyuria or nocturia. There was no history of recent rapid weight gain, easy bruising, acne, or hirsutism. She did not have any phaeochromocytoma-suggestive spells. History of any joint pains, intermittent abdominal pains, or any other symptoms to suggest an underlying rheumatological illness was absent. Other system inquiries were completely normal. Her family history was negative for young-onset hypertension or intracerebral haemorrhages. At the time of presentation or prior to that, she has not used any long-term medications or supplements, including liquorice or any illicit substances. Throughout the illness, she did not suffer from any hypertensive emergencies despite having difficulty to control blood pressure.

Examination revealed a female with a body mass index of 22 kg/m^2. Cardiovascular system examination was normal without any radio-radial or radio-femoral delays. Precordial examination revealed a non-displaced apex. Dual rhythm was heard without any murmurs. However, blood pressure was 200/120 mmHg without discrepancies in both upper limbs. Her pulse rate was 88 beats per minute and was regular. The abdominal examination did

not reveal any ballotable masses, or renal bruits. There were no carotid bruits as well. Other system examinations were unremarkable except for the evidence of silver wiring on fundoscopy.

Investigations revealed persistent hypokalaemia despite replacement and a normal magnesium level. She underwent aldosterone renin ratio (ARR)while being on prazosin 5 mg TDS diltiazem 60 mg TDS, and potassium replacement with oral potassium chloride 3 tablets QDS. Blood investigation results are listed in Table 1.

Her 2D echo showed a normal ejection fraction with preserved biventricular function, and there was no evidence to suggest coarctation of the aorta. Her ultrasound revealed no evidence of chronic renal parenchymal changes, renal size discrepancy or suprarenal masses. The renal artery doppler was also found to be normal. Lastly, her renal angiogram was also found to be normal, excluding a renal artery stenosis. However, it showed a small, well-defined exophytic lesion (1.2*1 cm) with delayed enhancement and minimal washout in the 5-min phase involving the lower pole of the left kidney (Fig. 1).

Because of this finding in the background of her hypokalemic hypertension with secondary aldosteronism, the possibility of reninoma was entertained, and she underwent RVS with renin testing while on a low-salt diet, prazosin 5 mg TDS, and diltiazem 60 mg TDS for blood pressure control. At the time of testing, her potassium was 3.8 mmol/L with potassium chloride 3 tablets QDS. RVS was done by entering the femoral vein, and renal vein cannulation was done sequentially. Cannulation was confirmed through visualisation of the renal vein by dye injection. Renin sample analysis was done without a delay. However, the results did not confirm the secretory nature of the left renal lesion as both renal veins and the inferior vena cava renin levels did not differ significantly (refer to Table 2).

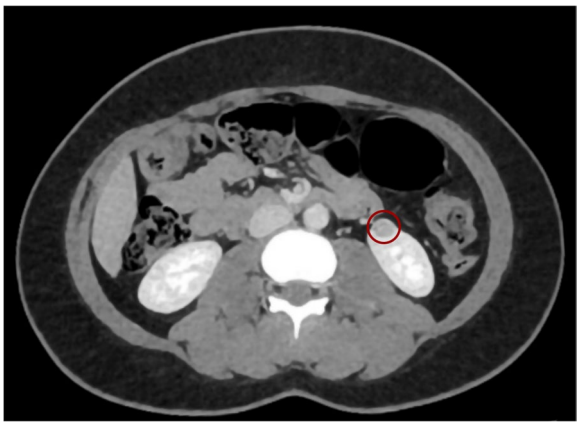

Fig. 1 CT image showing an exophytic iso-dense lesion in the lower pole of the left kidney

Soon after RVS, she required three antihypertensives: losartan 50 mg BID, prazosin 5 mg TDS, and diltiazem 60 mg TDS, but she still had suboptimal blood pressure control, indicating resistant hypertension.

Then, her antihypertensive medication was changed to spironolactone 50 mg BID and losartan 50 mg BID. Despite being on these potassium-sparing diuretics, she still needed regular potassium chloride tablets BID to maintain her potassium level above 3.5 mmol/L. However, after changing her antihypertensives, her blood pressure was well controlled.

Multidisciplinary team (MDT) discussion was held with the participation of the endocrinology team, genitourinary surgical team and the radiology team. Considering the biochemical investigation results and the excellent blood pressure response to RAAS blockade, the possibility of reninoma was remarkably high despite inconclusive RVS. However, the CT characteristics of the lesion also had significant similarities to the reported reninomas in the literature. The possibility of an ectopic source for renin in this otherwise healthy patient was also

Table 1 Biochemical test results

Potassium	**2.8 mmol/L (3.5–5.2 mmol/L)**
Sodium	136 mmol/L (135–145 mmol/L)
Bicarbonate	24.8 mEq/L (22- 26 mEq/L)
ODST	< 11 nmol/L (Normal < 50 nmol/L)
Serum aldosterone	49.7 ng/dl (1.76–39.2 ng/dl)
Plasma Renin concentration-	1275μIU/mL (2.8–32.9μIU/mL)
Plasma renin activity	106.3 ng/ml/hr
ARR	0.5 > 30 suggestive of primary aldosteronism < 10 suggestive of secondary aldosteronism
TSH	1.35μIU/L (0.5–4 μIU/L)
24-h urinary metanephrines	0.35 mg/24 h (< 1 mg/24 h)
Serum creatinine	0.67 mg/dl (0.7–1.3 mg/dL)
Urine full report	No RBCs or proteinuria
ESR	4 mm/1sthr (< 10 mm/1st hr)

ODST Overnight Dexamethasone Suppression test, *ARR* Aldosterone -Renin-Ratio, *TSH* Thyroid Stimulating Hormone, *RBC* Red Blood Cells, *ESR* Erythrocyte Sedimentation Rate

Table 2 Renal Vein Sampling with renin measurement

Site	**Plasma Renin Concentration (μIU/mL/ml)**
Right renal vein	1445
Left renal vein	1100
Inferior Vena cava	1315

very low. Thus, the decision was made to continue with the surgical removal of the lesion.

She underwent partial nephrectomy, and during the immediate postoperative period, blood pressure normalised without any requirement for antihypertensives, and the hypokalaemia also fully resolved without supplements.

The resected lesion's macroscopy showed a black tumour measuring 17×12x10mm confined to the renal capsule. As shown in Fig. 2, microscopy revealed a subcapsular, well-circumscribed, unencapsulated tumour composed of sheets of round to polygonal cells having central round vesicular nuclei and eosinophilic cytoplasm with indistinct cell borders. Immunohistochemistry further confirmed the diagnosis of reninoma as the membranes were positive for CD34, CD117 and vimentin.

Currently, almost two years after surgery, she is normotensive and normokalaemic without any medication.

Discussion

In our patient with young onset hypokalemic hypertension without any specific clinical features, multiple differential diagnoses can be considered. The possible list of differential diagnoses for her is mentioned in Table 3.

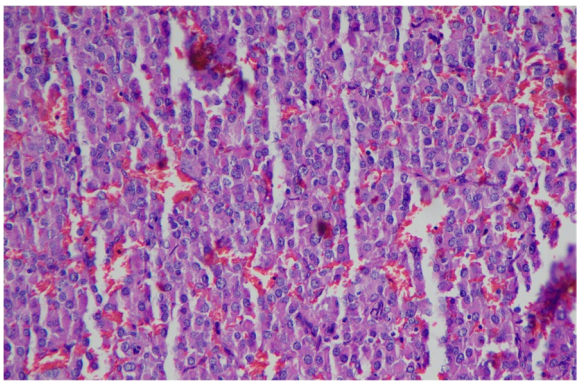

Fig. 2 The cells show mildly pleomorphic round nuclei with moderate eosinophilic cytoplsm

Her ARR helped us narrow the list to three possibilities, giving rise to secondary hyperaldosteronism. Of these three, she did not have a high STOP-BANG score, indicating a low possibility of obstructive sleep apnoea [15]. However, considering the prevalence, renovascular hypertension was the next most likely diagnosis.

The renal angiogram was normal, but an iso-dense lesion was detected in the lower pole of the left kidney. Thus, the diagnosis of reninoma was considered at this point.

Since there are no pathognomonic radiological features for a reninoma and considering the rare possibility of ectopic renin-secreting neoplasms as a part of paraneoplastic syndrome, proving the functional nature of this renal lesion was important. There have been cases reported with paraneoplastic renin secretion involving an ovarian steroid cell tumour and an abdominal desmoplastic small round cell tumour [16, 17]. Additionally, benign renal cysts, which may resemble reninomas on radiological images, is another reason for the functional testing in reninomas. Other renal lesions like Wilm's tumour (nephroblastoma), renal cell carcinoma and renal oncocytoma are also known to secrete renin but are found to be less potent clinically [18, 19].

Throughout the case reports on reninomas, the level of renin elevation differs, and it is impossible to decide on a renin cutoff that should give rise to the suspicion of reninoma. Instead, the clinical picture of hypokalemic hypertension with an excellent clinical response to RAAS blocking agents should alert the clinician to entertain the diagnosis of reninoma.

Preparation for RVS in our patient followed a protocol similar to the one described by Hayes et al., with non-interfering antihypertensive dosing, potassium supplementation, and a low-salt diet starting three days before testing [4]. We could not follow the step of giving IV enalaprilat to augment the renin secretion from the lesion since IV enalaprilat is not available in Sri Lanka. However, IV furosemide can also be used as a substitute for enalaprilat [4]. But, in this case, it was not done because

Table 3 Differential diagnoses for hypokalemic hypertension

Low renin and high aldosterone- primary aldosteronism	High renin and high aldosterone- secondary aldosteronism	Low renin and low aldosterone- Apparent aldosterone excess
I. Aldosterone-producing adenoma	I. Renovascular hypertension	I. Cushing syndrome
II. Bilateral adrenal forms of primary aldosteronism	II. Reninoma	II. Apparent mineralocorticoid excess syndrome
III. Familial forms of primary aldosteronism (including glucocorticoid-remediable hypertension)	III. Obstructive sleep apnoea	III. Liddle syndrome
		IV. Excess consumption of grapefruit or liquorice
		V. Congenital adrenal hyperplasia17α-hydroxylase and 11β-hydroxylase deficiencies

repeat cannulation of the renal vein was not considered possible with the limited facilities at that time.

During RVS, renin release in the renal vein sinus has been effectively induced through intravenous (IV) administration of furosemide or enalaprilat. Samples are obtained from renal veins and the periphery before and 20 min after renin stimulation. A lateralisation ratio exceeding 1.5, preferably with contralateral suppression, has proven valuable for finding autonomous renin secretion [4].

However, the sensitivity of the RVS was as low as 56% throughout the limited literature [5]. This was attributed to the possible variations in venous drainage of subcapsular reninomas, where a lesion might not drain into the renal vein. However, a recent study which retested three patients with previous inconclusive results on RVS by careful pre-procedural preparation, giving priority to optimising renin secretion from the culprit lesions, was able to identify and cure a reninoma in a patient which was previously undetected on cross-sectional imaging [7].

Our patient's renal veins and peripheral blood demonstrated the same renin concentration without the lateralisation ratio. Possible venous drainage into subcapsular vessels rather than the renal vein or displaced catheter during sample collection were considered reasons for these results. Giving IV enalaprilat /furosemide to stimulate renin secretion from the culprit lesion could have enhanced our patient's RVS results.

However, the presence of the renal lesion, with the radiological characteristics suggestive of reninoma, the excellent blood pressure and potassium response to RAAS blockade and considering the rarity of the ectopic renin-secreting lesion in this young female without any other clinical features suggesting an underlying malignancy, MDT decision was made to remove it surgically, as radiofrequency ablation was not deemed possible with the available resources considering the size and the location of the renal lesion.

Immediately following surgery, her blood pressure normalised, and her potassium supplements were withheld, which confirmed the diagnosis of reninoma clinically. A histological examination of the specimen further confirmed the diagnosis.

Limitations

Repeating RVS after 20 min following administration of renin secretion augmenting agents such as enalaprilat / furosemide would have enhanced the yield of RVS. We were unable to follow this step due to logistic reasons. Also, due to the limited availability of ARR testing capabilities, we did not perform post-operative ARR testing on this patient, who had immediate cure of hypertension and hypokalemia following surgery, to confirm a biochemical cure.

Conclusion

Reninoma is a rare but curable cause of hypokalaemic hypertension with secondary hyperaldosteronism. Diagnosing reninoma prior to surgery can be challenging and requires collective evidence through clinical features, imaging evidence, and dynamic testing with RVS for renin. However, taking extra measures to localise the lesion in suspected reninoma is essential, as surgical removal of the lesion can cure the hypertension.

Abbreviations

RAAS	Renin Angiotensin Aldosterone System
CT	Computed Tomography
MRI	Magnetic Resonance Imaging
FDG PET	Fluorodeoxyglucose Positron Emission Tomography
DOTATATE PET	Positron Emission Tomography scan with a radiopharmaceutical tracer DOTATATE
RVS	Renal Vein Sampling
BID	Two times a day
TDS	Three times a day
QDS	Four times a day
ODST	Overnight Dexamethasone Suppression test
ARR	Aldosterone -Renin-Ratio
TSH	Thyroid Stimulating Hormone
RBC	Red Blood Cells
ESR	Erythrocyte Sedimentation Rate
MDT	Multi-Disciplinary Team

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12902-024-01804-0.

Supplementary Material 1.

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Authors' contributions
PNSW, DP, PS, SMR, and EMDU contributed to patient management, manuscript writing, and editing. HDA and AADP contributed by attending to the radiological aspect of the case and by editing the manuscript. STM was involved with the surgical management of the patient and manuscript editing.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent was obtained from the patient for publication of their clinical details and clinical images.

Competing interests
The authors declare no competing interests.

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