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Hypokalemia in a Young Patient as Onset Sign of Reninoma, still a Complex Diagnosis

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ABSTRACT

Hypokalaemia is rare in young people without chronic disease and chronic treatment. Rarely it may be associated to secondary hypertension.

We here report a rare case of reninoma with early onset characterized by hypokalemia and chronic kidney dysfunction.

GB a 29 yy black old man was admitted in internal medicine with unexplained hypokalemia associated to onset of severe hypertension and anamnestic kidney dysfunction. During the differential diagnosis evaluation, the presence of a right reninoma was found. Therapeutic management was very difficult because the rare evidence of reninoma and the available options in this clinical setting are not clear.

The complexity clinical management and therapeutic options of similar cases are limitated by the current guidelines and to choose a long term treatment is very difficult.

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Background

Electrolyte alterations may be considered uncommon in young people for which the use of specific diet and\or the use of drugs that may induce electrolytes imbalance is less common than for adults [1]. Among chronic disease that may lead to electrolytes imbalance per se or because the use of specific drugs there are hypertension and progressive kidney dysfunction, in particular secondary hypertension [2-4]. Yet, the occurrence of hypertension in a young subject is related to a complex relationship among genetic and environmental risk factors [5]. Rarely, we may also observe that the onset and clinical management of secondary hypertension may be associated to high rate of complications and reduced pharmacological response to conventional drugs even if they are used in combination [6]. In these clinical conditions, the appearance of hypokalemia is possible [1].

Regarding this clinical setting the occurrence of secondary hypertension in a young patient with hypokalaemia and in presence of chronic kidney dysfunction because the presence of a reninoma (a iuxtaglomerular cell neoplasm) is very rare.

We here report the complexity of clinical management of a secondary hypertension due to a reninoma of right kidney with electrolyte imbalance at the onset.

Case History

GB a 29 yy old black man, was admitted to Medicine department for hypokalemia and anamnestic kidney dysfunction with firs objecvtive determination of severe hypertension. No further symptoms and clinical signs were present as headache, vertigo or nausea\vomiting.

A fast screening to exclude secondary hypertension due endocrynopathies (e.g. hyperthyroidism or hyperaldestorism) or cardiovascular anatomic variability as aortic coartaaction or renal artery stenosis was performed without evidences. Furthermore, also renin angiotensin system was looked for. For this reason, an abdomen magnetic resonance in order to evaluate primary hyperaldosteronism (i.e. Conn syndrome) together to renin values in orthostatism and clinostatism were also evaluated and resulted in serum renin increase. Vanillymandelic acid and metanephrine in the urine were ruled out and tested normal in three different samples.

Blood tests were summarized in table 1 as far as main imaging of abdomen magnetic resonance were reported in Figure 1.

Table 1: Laboratory Tests of Reported Patient

Test	Patient values At admission	Patients values at follow up at 4th day	Normal values
BUN (mg\dL)	81	55	10-50
Creatinine mg\dL	4.14	1.9	0.5-1.2
Hb g\dL	13.3	13.8	12-16
WBC mm cube	7.8	7	4-10
PLT mm cube	201	230	150-400
Serum sodium (meQ)	135	136	132-141
Serum potassium (meQ)	2.6	3.3	3.5-4.5
TSH (mU\L)	2	2	0.8-3.5
C reactive protein	1.21	0.9	0-5
Fibrinogen (mg\dL)	287	296	200-400
Serum aldosterone (clin) (ng\dL)	27	30	1.7-23.1
Serum aldosterone (orto) (ng\dL)	21	29	2.52-39.1
Serum renin (clin) (µU\mL)	119	121	2.1-39.2
Serum renum (orto) (µU\mL)	180	182	4.4-47.1
Serum, albumin (g\dL)	3.8	3.8	2.4-4.2

Legend to Table 1
Abbreviations: BUN (Blood Urea Nitrogen), TSH (Thyroid Stimulating Hormone)

In the meantime, a multidrug approach was attempted to front of severe hypertension and chronic kidney dysfunction with persistent hypokaliaemia: urapidil 9 mg \h iv after first bolus of 18 mg iv for first 24 hours followed by nitroglicerine 125 mg iv for following 2 days with low response to values of systolic blood pressure. For this reason, a withdrawal of this treatment was needed and replaced by nifedipine 60 mg twice daily associated to doxazosine 2 mg daily without a full therapeutic improvements.

Few days after, because the persistence of severe hypertension although the multidrug approach a local sample of renin and aldosterone was performed with venous catheterism and from both renal veins and inferior vena cava after 30 minutes were pick up venous samples to be analysed. Persistent high levels of renin from right renal vein and in the vena cava system after 30 minutes, so demonstrating the local primary hyperproduction of renin typical of reninoma; on the other hand aldosterone levels followed pathological trend because the primary reninoma.

After the acquisition of these data a prompt change in treatment was performed adding Ramipril 10 mg daily although the kidney dysfunction and confirming oral nifedipine 60 mg twice daily with improvements on potassium levels and blood pressure after 48 hours from the onset. No additional support of oral or iv potassium was planned and serum levels of potassium restored at fourth day from the onset of treatment. Follow up blood tests were also reported in Table 1.

Table 2: Values of Iuxta Renal Renin and Aldosterone During Vascular Catheterization

Tests	Renal renin (right)	Renal Aldosterone (right)	Renal renin (left)	Renal aldosterone (left)
T0	224 µU\mL	25.1 ng\dL	83 µU\mL	100 ng\dL
T1	224 µU\mL	25.1 ng\dL	114 µU\mL	100 ng\dL
Normal values	< 40 µU\mL	< 50 ng\dL	<40 µU\mL	< 50 ng\dL

A following possible step to consider intervention for renal denervation or local embolism of renal artery was take into account but discharged because in off label.

The prolongation of treatment in following weeks confirmed a positive trend in blood pressure levels and in electrolytes (including potassium levels), BUN and creatinine levels after discharge and the patients was addressed to outpatient clinic to look for damages of secondary hypertension in order to evaluate further therapeutic steps.

Discussion
Juxtaglomerular neoplasm are uncommon in clinical practice and they are frequently associated to onset of vascular complications [7]. Reninoma, is one of juxtaglomerular neoplasm and its presence is associated to increased production of renin inducing severe hypertension and hypokaliemia. Yet the differential diagnosis of severe hypertension and hypokaliaemia should be quickly performed with Conn's disease or other adrenal neoplasm as pheocromocytoma as we reported [8].

On the other hand, reninoma is very rare (few of 500 cases described in Literature) and to understand its distribution in general population is very hard although men seem to be more affected than women.

The case that we reported describes the occurrence of severe hypertension in a black male that may offer a confounding epidemiological aspect and that could influence our daily clinical practice because severe hypertension when not associated to malignancies is typical of black males.

Yet, the onset with hypokalemia with anamnestic kidney dysfunction is atypical too and for this reason a thorough differential diagnosis was performed with primary hyperaldosteronism as occurring in Conn's syndrome. In this way, samples made in renal vein and in a second time in vena cava helped us to identify a right reninoma.

On the other hand, the uncertain response to first multidrug approach with alternate trend to develop asymptomatic spikes of hypertension mainly during treatment was atypical too as far as the low therapeutic response to intravenous treatment compared to oral treatment with ACE-inhibitors; this ex adiuvantibus clinical aspect helped us to go on with a differential diagnosis among Conn's disease and reninoma.

Furthermore, in order to explore other chances different from pharmacological administration of ACE-inhibitors, other off label treatments were ruled out as the evaluation for renal artery embolism that was more appropriated for kidney transplant as far as the renal denervation that is more appropriated to patients without any type of clinical response to oral treatment [9-11]. Therefore, the off label use of ACE-inhibitors in chronic kidney dysfunction was evaluated to be more appropriated in a first phase of treatment [12].

Unfortunately, few laboratory data are useful to suspect a reninoma as in our case. Blood samples of our patient, as reported in table 1, did not reveal dysfunction of thyroid metabolism nor of suprarenal glands. Only data on potassium metabolism were useful to suspect secondary hypertension. For these reason, radiological imaging were needful to improve our clinical approach to the patient.

Abdomen CT scan with particular interest toward both vascular renal districts was performed in order to evaluate the chance of malignant hypertension due to renal artery stenosis. Abdomen CT scan was associated to abdomen magnetic resonance in order to identify possible causes of secondary renal hypertension (i.e. adrenal glands neoplasm) without clinical evidence. Being radiological imaging lacking of any type of vascular abnormalities or neoplasm that may explain the clinical trend of malignant hypertension, a selective catheterism of renal veins was performed in order to have a quantification of secreted renin in local vein and in a second time in inferior vena cava to evidence the systemic distribution of renin too.

With this clinical acquisitions associated to the improvements of potassium levels we have not a clear indication to interventional treatments as renal artery embolization or renal denervation.

The FDA approved the clinical indication to renal denervation only for patients that are not able to control blood pressure but multiple randomized clinical studies using rigorous trial designs showed the efficacy to reduce blood pressure values but in a limitated number of patients [13, 14]. Therefore, the clinical indication for off label denervation of renal system in presence of reninoma is still matter of discussion.

Therefore, any type of off label interventional approach in case of reninoma shows actually several limitations. First, regarding long term outcomes, few data are available regarding major adverse cardiovascular events such as stroke as myocardial infarction or rate off kidney transplantation rate. Furthermore, studies regarding the adherence and to pharmacological treatment and individual lifestyle improvements are still lacking as in the case we reported.

Furthermore, because the evidence of a reninoma is very rare also in clinical trials and complications rate as seizures and syncope are not rare, we suggest a thorough clinical evaluation of risk\ benefit of any therapeutic approach in particular if the onset is associated to hypokalemia, because the chronic kidney dysfunction with hypertension is most frequently associated to normal value or increased values of potassium. Clinical trials should be needed although epidemiological data on reninoma make them very hard.

Statements and Contributions

Author Contributions

PDM wrote the manuscript, CC, MD, RS, GS managed the case, VB, AL, MG revised the manuscript, FFB, CdG performed English check and revision of ethnicity on human studies.

Conflict of Interest

The authors declare that they have no competing interests and all authors confirm accuracy.

Ethics Approval and Consent to Participate

Not Applicable.

Informed Consent

Not applicable. Patient consent was released from patient to corresponding author for publication.

Availability of data and materials

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References

1. Muthaluraj GP, Sathyanarayanan A, Larsen N, Fernandez C (2024) Optimising patient care: comprehensive evaluation of inpatient hypokalaemia. Br J Hosp Med (Lond) 85: 1-7.
2. Alicic RZ, Neumiller JJ, Tuttle KR (2025) Combination therapy: an upcoming paradigm to improve kidney and cardiovascular outcomes in chronic kidney disease. Nephrol Dial Transplant 40: i3-i17.
3. Puckett L (2023) Renal and electrolyte complications in eating disorders: a comprehensive review. J Eat Disord 11: 26.
4. Lauder L, Mahfoud F, Azizi M, Bhatt DL, Ewen S, et al. (2023) Hypertension management in patients with cardiovascular comorbidities. Eur Heart J 44: 2066-2077.
5. Tandan N, Lavie CJ, Stewart MH, Milani RV, Ventura HO (2023) Secondary hypertension: evaluation and management. Curr Opin Cardiol 38: 318-325.
6. Sudano I, Suter P, Beuschlein F (2023) Secondary hypertension as a cause of treatment resistance. Blood Press 32: 2224898.
7. Arturo C, D Alessandro L, Poggiano MR, Orefice G, Longobardo A, et al. (2023) A rare cause of recurrent syncope: chemodectoma of left carotid. Italian Journal of Medicine 17: 1585.

8. Araujo Castro M, Parra Ramírez P (2022) Diagnosis of primary hyperaldosteronism. Med Clin (Barc) 158: 424-430.
9. Li D, Pua BB, Madoff DC (2014) Role of embolization in the treatment of renal masses. Semin Intervent Radiol 31: 70-81.
10. Del Tatto B, Gogeneata I, Ohana M, Thaveau F, Caillard S, et al. (2022) Arterial Embolization of Polycystic Kidneys for Heterotopic Transplantation. J Endovasc Ther 29: 885-892.
11. Pisano A, Iannone LF, Leo A, Russo E, Coppolino G, et al. (2021) Renal denervation for resistant hypertension. Cochrane Database Syst Rev 11: CD011499.
12. Ku E, Inker LA, Tighiouart H, McCulloch CE, Adingwupu OM, et al. (2024) Angiotensin-Converting Enzyme Inhibitors or Angiotensin-Receptor Blockers for Advanced Chronic Kidney Disease: A Systematic Review and Retrospective Individual Participant-Level Meta-analysis of Clinical Trials. Ann Intern Med 177: 953-963.
13. Cluett JL, Blazek O, Brown AL, East C, Ferdinand KC, et al. (2024) American Heart Association Council on Hypertension; Council on Cardiovascular and Stroke Nursing; Council on the Kidney in Cardiovascular Disease; and Council on Peripheral Vascular Disease. Renal Denervation for the Treatment of Hypertension: A Scientific Statement from the American Heart Association. Hypertension 81: e135-e148.
14. Sharp ASP, Fisher NDL (2025) Renal Denervation to Treat Hypertension: A Clinical State-of-the-Art Review. Am J Hypertens 23: 134.