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Case Report

Cushing's Syndrome due to a Renal Neuroendocrine Tumor: A Case Report

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Abstract
Introduction: Cushing's syndrome (CS) due to ectopic adrenocorticotrophic hormone (ACTH) is rare and usually due to neuroendocrine neoplasia (NEN). Primary renal NEN is exceptionally rare but may be a cause of rapidly progressive CS. ***Case Presentation:*** A 51-year-old man presented with profound hypokalemia, cellulitis, and new-onset type 2 diabetes and hypertension with 1 month of muscle weakness, labile mood, and insomnia. CS due to ectopic ACTH production was confirmed. Biochemical control was achieved using a "block-and-replace" regimen with dual blockade with ketoconazole and metyrapone and hydrocortisone replacement in addition to mineralocorticoid receptor blockade using spironolactone. CT and ultrasound demonstrated a 24 mm right renal lesion with features concerning for renal cell carcinoma. Right laparoscopic nephrectomy was performed. Histology demonstrated a WHO grade one NEN with ACTH staining. ***Conclusion:*** In CS, where the source of ectopic ACTH production is unable to be identified, a renal source should be considered. Diagnosis may be difficult as there are no reliable radiological characteristics to distinguish renal NENs from renal cell carcinomas, so a high degree of suspicion is required.

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Introduction

Cushing syndrome (CS) may result in significant metabolic, cardiovascular, and infectious complications. Ectopic adrenocorticotropic hormone (ACTH) secretion (EAS) is a rare subset of CS, comprising 5–15% of all CS cases with a 13-fold increase in mortality rate [1]. Due to the high mortality, early diagnosis and management of EAS are paramount [2]. A variety of tumors have been associated with EAS. Most are of neuroendocrine origin, such as small cell lung carcinoma, bronchial carcinoid, pancreatic neuroendocrine tumor, and thymic carcinoid, with rare cases of small cell prostate cancer also reported [3]. Over 4 decades, 166 cases of histologically proven primary renal NEN were identified from the Surveillance, Epidemiology, and End Results (SEER) database [4], making the kidney a rare location for NENs to arise. Primary renal NEN secreting ACTH causing EAS is exceedingly rare, with less than 10 published cases [5–11] (Table 1). Diagnosis of renal NEN may be difficult to distinguish from other renal lesions so awareness of this possibility is important. This is to ensure diagnosis and treatment are not delayed due to the high morbidity and mortality associated with EAS.

Case Report

A 51-year-old European male presented acutely to the emergency department with severe hypokalemia and cellulitis, recent-onset type 2 diabetes and hypertension, and 1 month of proximal myopathy with labile mood and insomnia. He was an ex-smoker with an unclear pack history. Medications included a recent COVID vaccination, metformin, and quinapril. There was no relevant family history. Examination revealed a profound proximal myopathy and right leg cellulitis but no purpura, supraclavicular fat pads, hyperpigmentation, moon facies, or abdominal striae. Laboratory results demonstrated a hypokalemic metabolic alkalosis with severe hypercortisolemia and markedly elevated ACTH levels consistent with ACTH-dependent CS (Table 2). Pituitary MRI was unremarkable. Chest X-ray showed left lung base collapse/consolidation with *Pneumocystis jiroveci* (PJP) isolated from bronchoscopic washings and *Streptococcus* from blood cultures. These were treated with cotrimoxazole and intravenous penicillin, respectively. Significant hypercortisolemia with active PJP and sepsis meant rapid control of CS was paramount. Ketoconazole was initiated at 1.2 g daily in divided doses plus mineralocorticoid blockade with spironolactone (200 mg BD) and potassium supplementation. Once available, metyrapone 6 g daily in divided doses was added with hydrocortisone 40 mg per day in divided doses to prevent hypocortisolism ("block and replace"). Serum cortisol normalized with treatment from 2,173 nmol/L to 213 nmol/L (measured prior to hydrocortisone dose), and his free thyroid hormone levels also normalized.

CT chest, abdomen, and pelvis performed on day two of his admission demonstrated bilaterally enlarged adrenal glands, upper zone lung changes consistent with his atypical infection, and bibasal atelectasis. An ill-defined 24 mm right renal lesion was identified and a lower pole 15 mm cyst (Fig. 1). On ultrasound 5 days later, the 15 mm lesion was consistent with a simple cyst; the upper pole lesion was possibly an angiomyolipoma. ^{68}Ga-DOTATATE PET/CT was not available locally, and due to the COVID-19 Delta outbreak, travel was not possible.

Given his smoking history and CS severity, small cell lung cancer was considered. ^{18}F-FDG-PET/CT performed on day 10 following admission demonstrated increased FDG uptake in the areas of infection and mildly avid hilar and mediastinal nodes. The renal lesions were not FDG avid. Renal vein sampling was performed by inserting a 5 French sheath into the right common femoral vein and a C2 catheter into the right renal vein. Simultaneous bloods

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Table 1. Ectopic ACTH secretion secondary to renal neuroendocrine neoplasms

Author (reference)	Year	Age/ sex	Imaging	Histology	Size, mm	Chemotherapy	Surgery	Follow-up from CS dx	Outcome at the last follow-up
Hannah et al. [5]	1988	12/F	CT – solid, enhancing	Oncocytic features, well-differentiated	40	No	Left renal mass excised 4 × 3 × 2 cm	2.5 years	Alive/disease free
Arlt et al. [6]	2008	36/M	CT – appearance not described	NEC, well differentiated Ki-67 5%	28	No	Not performed due to acute deterioration	–	Deceased PJP infection ARDS, multiorgan failure
Kocher et al. [7]	2019	11/F	CT – solid/cystic septated mass	High grade, nodal metastases	138	Cisplatin, etoposide; sunitinib; atezolizumab	Left radical nephrectomy	8 months	Deceased fungal pneumonia, widespread metastases
Tamaki et al. [8]	2019	71/M	CT – heterogenous enhancement MIBG – peripherally avid MRI – T2W wall high intensity, low centrally; T1W – early enhancement	'Oncocytic carcinoid'	85	No	Nephrectomy	NS	Clinical and biochemical resolution
Chunharojrith et al. [9]	2021	51/M	CT – 44HU, mild enhancement 10-min delay (79HU) Tc-99m HYNIC-TOC – no avidity	Oncocytic features, extensive necrosis, mitoses 2/10 HPF Ki-67 NS	35	No	Left radical nephrectomy and left adrenalectomy	3 years	Alive/disease free
Xu et al. [10]	2023	26/M	CT – 38HU FDG-PET – intense uptake ^{68}Ga-DOTATATE – moderate uptake, SUVmax 17.6 MRI – T2W – low signal; T1W fat-suppressed – slightly high signal	WHO G1 Ki-67 <1%	NS	No	Left radical nephrectomy	NS	Biochemical remission
Kasajima et al. [11]	2023	32/F	NS	WHO G1 Ki-67 2%	45	NS	Yes	48 months	Alive/disease free
Kasajima et al. [11]	2023	54/F	NS	WHO G3 Ki-67 33%	36	NS	Yes	57 months	Alive/disease free
Current case	2022	51/M	CT – ill-defined hypodense FDG-PET – no uptake	WHO G1 Ki-67 <1%	24	No	Right nephrectomy	2 years	Alive/disease free

NS, not stated; F, female; M, male; NEC, neuroendocrine carcinoma; WHO, World Health Organization; G1, grade 1; G3, grade 3; HPF, high-powered field.

samples for ACTH taken from both the sheath and catheter demonstrated a mildly increased right renal vein ACTH gradient compared to peripheral (1.4:1), suggesting a right renal source for his ectopic ACTH. Twenty-five days after his acute admission (time being allowed for treatment of his PJP), a laparoscopic adrenal-sparing radical nephrectomy was performed.

Histology demonstrated a well-differentiated (T1a N0 M0 WHO grade one) 30-mm NEN (Ki-67 <1%) with no lymphovascular invasion. Immunohistochemistry markers for neuroendocrine differentiation (CD56, synaptophysin, chromogranin) were positive, and ACTH staining was present (Fig. 2). Immunohistochemistry markers for renal cell carcinoma (CK7, CD10, PAX8) were negative. Postoperatively, his ACTH was undetectable. His hypothalamic-pituitary-adrenal axis rapidly improved (Fig. 3), and glucocorticoid therapy was ceased on postoperative day two. Daily bloods were performed until discharge at postoperative day eight and then twice weekly. Due to the rapidity of axis recovery and concerns for distant disease, a ^{68}Ga-DOTATATE PET/CT was performed 6 weeks postoperatively with no metastatic disease identified. Biochemical testing was reduced to weekly, and as ACTH and cortisol levels remained stable, it was reduced to monthly for the next 6 months. Hypoglycemic and antihypertensive medications were stopped. At 3-year follow-up, he remains well with no evidence of recurrence with ongoing 6-monthly biochemical and clinical assessments.

The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000545734).

Discussion

EAS is an endocrine emergency but often a diagnostic challenge as typical cushingoid features may not be present [12]. This patient presented with classical features of severe hypercortisolism typical of EAS: proximal myopathy, new type 2 diabetes mellitus, hypertension, and hypokalaemic metabolic alkalosis but lacked the weight gain and changes in body habitus typically seen with non-EAS causes of CS. He also had a biochemical pattern suggesting hypopituitarism which was due to the severe hypercortisolism and which rapidly normalized with adrenal steroidogenesis blockade without the need for specific thyroid replacement. The diagnostic workup for EAS is outlined by Young et al. [13]; however, it is important to emphasize that initiation of treatment in severe EAS should not be delayed by the diagnostic workup.

Delay in biochemical control is associated with significant morbidity and mortality [14], patients particularly being at risk of developing life-threatening opportunistic infections such as PJP, as seen in this case and previously reported in EAS due to a renal NEN [6]. As such, prompt treatment is required and should not be delayed while localization investigations are being performed [13]. We aggressively treated the hypercortisolism with dual adrenal steroidogenesis blockade using ketoconazole and metyrapone along with spironolactone for mineralocorticoid receptor blockade, as we have found from experience treating EAS that use of either as a single-agent blockade is insufficient to control severe hypercortisolemia in this setting [15]. In addition, dual blockade in this setting at maximal doses, even in patients with severe hypercortisolism, typically results in the need for glucocorticoid replacement, i.e., a block-and-replace regimen with hydrocortisone dosing titrated based on biochemistry (cortisol, electrolytes) and clinical features and considering the need for higher glucocorticoid dosing to cover illness. In life-threatening settings, etomidate has also been used, traditionally in an ICU setting but has also been reported to be safely used at low doses in medical wards [16]. More recently, osilodrostat has been used off-label in severe hypercortisolism due to EAS

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Table 2. Pertinent laboratory investigations at initial assessment (08:00 h)

Laboratory request	Result	Reference interval
Serum potassium	2.6	3.5–5 mmol/L
Serum bicarbonate	43	22–26 mmol/L
Glucose	23.4	<7.8 mmol/L
HbA1c	63	<41 mmol/mol
Prolactin	237	0–400 mIU/L
LH	0.3	2–9 U/L
FSH	0.4	2–12 U/L
Testosterone	11	9–30 nmol/L
TSH	0.45	0.27–4.2 mU/L
FT4	3	12–22 pmol/L
FT3	1.2	3.1–6.8 pmol/L
Serum morning cortisol	2,173	170–500 nmol/L
24-h urine free cortisol	38,939	<380 nmol/24 h
DHEAS	11	1–10
Plasma ACTH	260	1.2–17 pmol/L

[17] but has a longer time to effect than ketoconazole or metyrapone, and like mifepristone (a glucocorticoid receptor antagonist which has a rapid effect but may exacerbate hypokalaemia and cortisol levels are not able to be used to guide response [18]), is not available in New Zealand. The onset of action of mitotane, an adrenolytic agent, is too slow to be of use in this setting [19]. Bilateral adrenalectomy is also a useful option to rapidly control hypercortisolism if the patient's clinical condition allows [14].

The second challenge is identifying the cause. The most common are small cell lung cancer and NEN [3]. Most NENs are found in the gastrointestinal (67%) and respiratory (25%) tracts [20]. Neuroendocrine cells are not found in the renal parenchyma; hence, primary renal NENs are rare and may develop from renal stem cells that undergo neuroendocrine differentiation [21] or intestinal metaplasia of renal cells [22]. Nguyen and colleagues identified 166 histologically proven primary renal NENs from the SEER database from 1973 to 2014 [4]. Of these, 51% were male with a median age of 59 years, and 42% had distant disease at diagnosis. Most (93%) were in the renal parenchyma, with 7% in the renal pelvis. Overall 5-year survival was 50% with surgery associated with a decreased mortality. Older age, male sex, regional and distant disease and higher grade were associated with poorer survival. While primary renal NENs are rare, those resulting in EAS appear to be exceptionally rare (Table 2). Of those identified in a literature search and including our case, the median age was 36 years but ranged from 11 to 71 years with no obvious gender predilection. Tumor grade ranged from WHO grade 1 to high grade, and 2 patients died due to opportunistic infections [6, 7], again supporting the need for early aggressive control of the hypercortisolism.

Here the differential diagnosis of the renal lesion included renal cell carcinoma (RCC). Renal NEN cannot be differentiated from RCC or benign lesions by radiological features [23]. In a review of 85 renal NENs, 29% were hyperdense, 55% hypodense, and 15% unspecified [23]. Calcifications were present in one-third [23]. Current recommendations for EAS are to use a combination of anatomical (CT +/− MRI) and functional (^{68}Ga-DOTATATE) imaging in tumor localization [24]. ^{68}Ga-DOTATATE PET/CT has one of the highest detection rates, particularly for non-pulmonary NENs [24]. However, RCC may also demonstrate ^{68}Ga-DOTATATE uptake [25]. Little is known about the somatostatin receptor status of renal NENs, and hypercortisolism may suppress tumor somatostatin type 2 receptor concentrations

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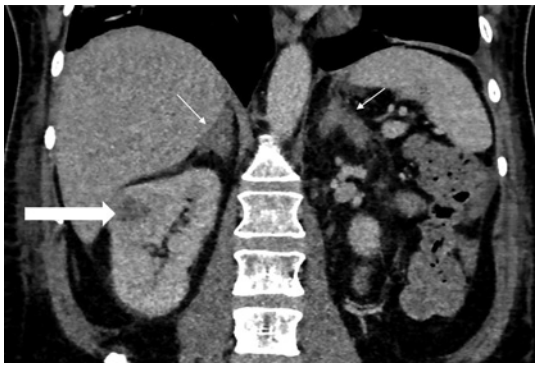

Fig. 1. Coronal CT scan demonstrating right renal lesion (solid white arrow) and bilateral adrenal hyperplasia (thin white arrows).

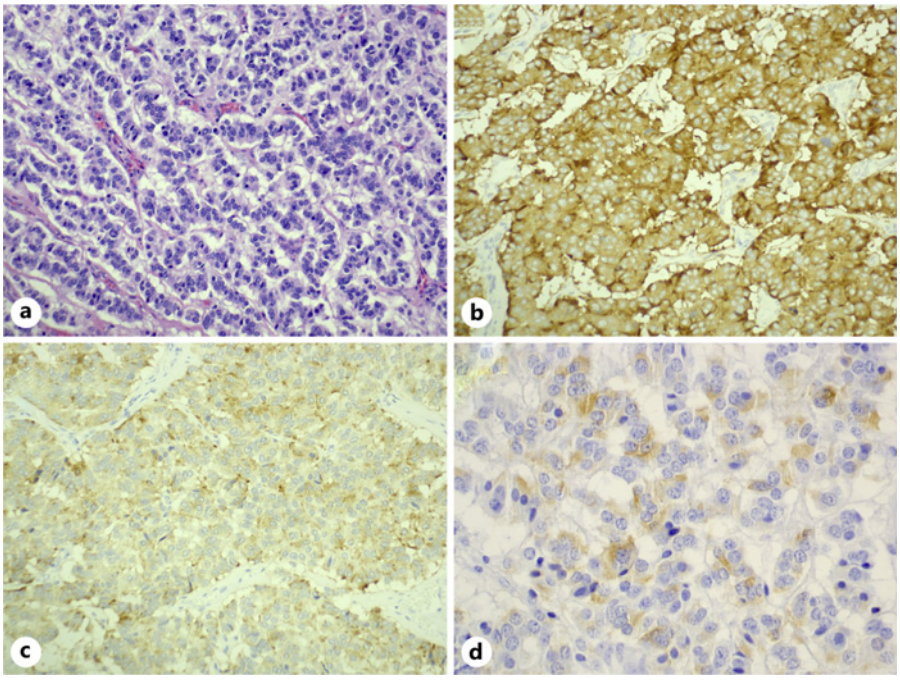

Fig. 2. Histology and immunohistochemistry of the renal lesion. **a**. H&E ×20. **b** Synaptophysin ×20. **c** Chromogranin ×20. **d** ACTH ×40.

[26], potentially leading to false-negative results. Chunharojrith et al. [9] did not identify the tumor on somatostatin receptor scintigraphy (Octreotide scan); however, there was extensive necrosis within the tumor. In comparison, Xu et al. [10] reported moderate uptake of a WHO grade 1 renal NEN on ^{68}Ga-DOTATATE PET/CT, although notably there was intense uptake on ^{18}F-FDG. Use of ^{18}F-FDG-PET/CT in renal tumors may be limited due to physical renal excretion of ^{18}F-FDG, meaning that there is less difference in intensity between the lesion and the normal renal tissue so metabolically active renal lesions may not be easily distinguished [27]. In the current case, ^{18}F-FDG-PET/CT was performed to look for evidence of small cell lung cancer, which at the time was thought to be a much more likely cause of his EAS, given his smoking history and that this is a more common cause of EAS than primary renal NEN [3]. Multidisciplinary input is important, and there should be a high degree of suspicion for all lesions identified, even if initially thought unrelated. Venous sampling may prove helpful for identification of the ACTH source, as was used here. Venous sampling is commonly used in CS

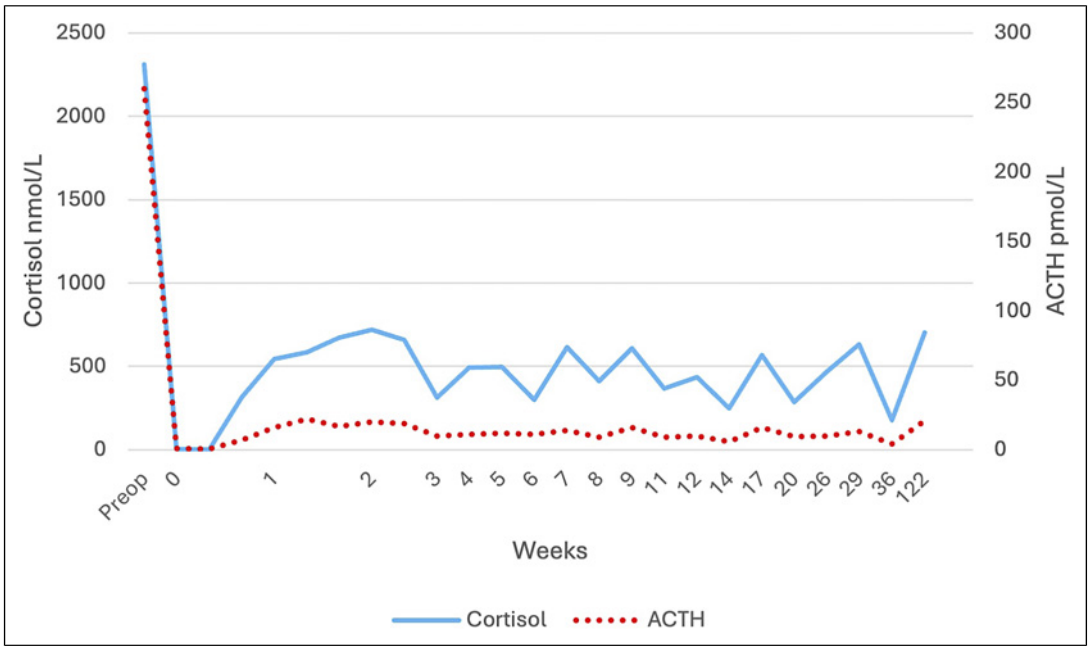

Fig. 3. ACTH and cortisol levels preoperatively and following surgical resection.

(bilateral inferior petrosal sinus sampling) to look for a central-to-peripheral gradient where an unstimulated 2:1 gradient can be accepted as a central source with prolactin measured to ensure appropriate catheter placement. Venous sampling is also routinely undertaken in patients with primary hyperaldosteronism looking for lateralization, as adrenal incidentalomas are common and may be misdiagnosed as an aldosterone-producing lesion which can be small. In adrenal vein sampling, either cortisol or plasma metanephrines are used to correct for blood flow. In our case, we did not have an analyte to correct for flow, and the gradient was 1.4:1, thought likely due to the larger vessel sizes and dilution of the ACTH.

The median time for HPA axis recovery with EAS after successful surgery is 0.6 years with an 82% 5-year recovery rate [28]. Our patient's recovery was rapid – initially this led to concerns for persistent disease, but longer-term follow-up has not demonstrated this. Rather, the rapid recovery of his HPA axis, we now think, was due to the short prodrome and early aggressive management of his hypercortisolism. Due to the rarity, there remains uncertainty about his prognosis, but given the low-grade tumor histologically and the lack of recurrence after 3 years of follow-up, his prognosis is looking promising.

Strengths of this report are the use of various diagnostic imaging modality when ^{68}Ga-DOTATATE was unavailable to help confirm that the kidney was the source of the excess ACTH production and the involvement of a multidisciplinary team in the workup and management. Limitations include the inability to obtain a ^{68}Ga-DOTATATE.

Conclusions

EAS is a rare endocrine emergency. Early diagnosis, prompt control of hypercortisolism, and early surgical intervention are essential and offer the potential for a cure. Primary renal neuroendocrine tumor is a rare cause of EAS with radiological features potentially indistinguishable from other renal lesions and needs to be considered when investigating a patient with EAS.

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Statement of Ethics

The Local Institutional Review Board (Te Whatu Ora Waikato) granted permission for the study (reference number not applicable). Written informed consent was obtained from the patient for publication of the details of this case report and accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

V.P. – data acquisition, analysis, writing the initial draft of the manuscript, and final approval of the version to be published. M.S.E. and V.B. – conception, design, data acquisition, analysis, interpretation, writing the manuscript, and final approval of the version to be published. S.N. and M.J.S. – data acquisition and analysis and final approval of the version to be published. A.D. – data acquisition and final approval of the version to be published.

Data Availability Statement

The data that support the findings of this study are not publicly available due to privacy reasons but are available from corresponding author, V.B., upon reasonable request.

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