CONCLUSION

This case illustrates the profound difficulty in diagnosing dopaminoma when exogenous L-DOPA therapy creates a near-identical biochemical profile. The diagnostic dilemma is further amplified by vague symptomatology, multiple comorbidities, and non-suggestive imaging. However, the finding of isolated dopamine hypersecretion with suppressed metanephrines serves as a critical clinical clue. This pattern points toward an intratumoral biosynthetic defect rather than drug interference. Clinicians must maintain a high index of suspicion and perform meticulous biochemical fractionation to identify these rare, dopamine-isolated secreting tumors.

EP_A009

THE HEMODYNAMIC PARADOX: SYNCHRONOUS ROBOTIC SURGERY FOR NORMOTENSIVE PHEOCHROMOCYTOMA IN VHL

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INTRODUCTION

Normotensive pheochromocytomas in Von Hippel-Lindau (VHL) syndrome present unique perioperative challenges. Standard alpha-blockade may induce intolerable orthostatic hypotension, making calcium channel blockers (CCB) a practical alternative. Furthermore, the primary intraoperative danger in these specific phenotypes may not be a hypertensive crisis, but profound vasoplegia. We report a VHL patient undergoing synchronous robotic surgery exhibiting this paradoxical hemodynamic response.

CASE

A 37-year-old female with VHL syndrome presented with an incidental 3.5-cm left adrenal mass and bilateral renal masses. Biochemistry confirmed a normotensive, noradrenergic pheochromocytoma (24-hour urine normetanephrines 4.2x upper limit of normal). Renal biopsy revealed a clear cell papillary renal cell tumor. Due to prior severe intolerance to Prazosin (hypotension/dizziness with low-dose Prazosin 0.5 mg ON), we utilized amlodipine for preoperative optimization. She was only able to tolerate low-dose 2.5 mg OD alongside oral sodium chloride and ample oral fluid loading. She underwent a synchronous robotic-assisted left adrenalectomy and left midpole renal tumor excision. Strikingly, tumor manipulation did not precipitate a hypertensive crisis. Instead, she developed hypotension requiring an intravenous noradrenaline infusion prior to adrenal vein ligation and tumor removal. Vasopressor support was successfully weaned 12 hours postoperatively, and she was discharged well.

CONCLUSION

Normotensive, noradrenergic pheochromocytomas in VHL are hemodynamically fragile. Chronic catecholamine excess induces homologous desensitization and downregulation of alpha-1 adrenergic receptors. This physiological adaptation explains the normotensive presentation and highlights the intraoperative vasoplegia experienced once sympathetic tone is altered by anesthesia. While CCB monotherapy with volume expansion safely facilitates prolonged, synchronous robotic surgeries, clinicians must anticipate and combat refractory hypotension rather than classical hypertensive spikes.

EP_A010

DISSEMINATED HISTOPLASMOSIS PRESENTING AS ADDISONIAN CRISIS: A DIAGNOSTIC MIMIC OF TUBERCULOSIS WITH BILATERAL ADRENAL MASSES

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INTRODUCTION

Disseminated histoplasmosis is a rare but important cause of adrenal insufficiency (AI), particularly in tuberculosis (TB)-endemic regions, where it may mimic granulomatous diseases. Adrenal involvement occurs in up to 80% of disseminated cases, although overt AI is less common. Reported cases described bilateral adrenal masses mimicking malignancy or TB, even in immunocompetent individuals. Addisonian crisis may be the initial manifestation, especially when the diagnosis is delayed. In TB-endemic settings, fungal infections are often overlooked, leading to delayed diagnosis and inappropriate therapy.

CASE

We reported a case of a 68-year-old male with underlying diabetes mellitus who presented with fever, cough, dysphagia, and weight loss for 1 month. He was empirically treated as smear-negative disseminated TB. On day 2 of therapy, he developed hypotension (80/50 mmHg), hypoglycemia (3.9 mmol/L), hyponatremia (Na 129 mmol/L), and hyperkalemia (K 5.1 mmol/L), suggestive of adrenal crisis, and was started on intravenous hydrocortisone. Serum cortisol prior to treatment was 61

nmol/L. Computed tomography (CT) imaging revealed bilateral lipid-poor adrenal lesions (right: 3.6 × 2.2 × 4.9 cm, Hounsfield Unit (HU) 36 and absolute washout 33%; left: 3.5 × 2.4 × 5.4 cm; HU 35 and absolute washout 17%), raising suspicion of infectious or malignant etiologies. Endoscopic ultrasound-guided biopsy demonstrated necrotizing granulomatous inflammation with budding fungal yeasts on Pituitary Apoplexy Score and GMS staining, consistent with *Histoplasma capsulatum*. TB and malignancy were excluded. He received amphotericin B for 14 days, followed by oral itraconazole for 1 year, and oral hydrocortisone replacement. At 1-year follow-up, adrenal lesions remained stable on CT images, and he continued to require hydrocortisone replacement.

CONCLUSION

This case highlights the importance of considering disseminated histoplasmosis as a differential diagnosis of bilateral adrenal masses with AI, especially with poor response to anti-TB therapy. Early tissue diagnosis is essential, as imaging findings are non-specific. Prompt recognition is critical to prevent life-threatening adrenal crisis and improve clinical outcomes.

EP_A011

LARGE "GROWING" ADRENAL MASS WITH AN UNEXPECTED HISTOLOGY

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INTRODUCTION

Large, rapidly enlarging adrenal masses typically mandate surgical intervention given the high probability of adrenocortical carcinoma (ACC). However, benign processes can mimic these aggressive growth kinetics, creating a diagnostic challenge for clinicians.

CASE

A 74-year-old male with hypertension, atrial fibrillation on rivaroxaban, and prostate cancer was referred for evaluation of a right adrenal incidentaloma detected on computed tomography imaging performed for prostate cancer staging. The well-defined mass measured 8.9 × 7.8 × 7.5 cm (AP × W × CC) with areas of calcification. Mean attenuation was +40 Hounsfield Unit, with no significant washout. Biochemical evaluation confirmed a non-functioning lesion (24-hour urine metanephrines 756 mcg/24 hours [N 246–753 mcg/24 hours]; serum cortisol 62 nmol/L post overnight dexamethasone suppression test). The patient was otherwise asymptomatic and declined surgical intervention. Repeat imaging 4 months later demonstrated interval enlargement of the mass to 9.8 × 8.8 × 10.7 cm. Due to this rapid growth, which was highly concerning for ACC, the patient underwent laparoscopic right adrenalectomy. Histopathological examination unexpectedly revealed an adrenal hematoma without evidence of an underlying tumor or malignancy.

This case illustrates the difficulty in differentiating aggressive cortical tumors from atypical hemorrhagic events. Larger series on adrenal hemorrhage reported trauma and procedural complications as the leading causes, with a median size of 3–4 cm. Idiopathic large adrenal hematomas presenting as rapidly expanding "pseudotumors" are exceptionally rare, with fewer than 20 cases reported in which surgical resection was performed due to high preoperative suspicion of malignancy. Additionally, adrenal hemorrhage attributed to anticoagulant therapy often occurs bilaterally. Unilateral lesion, especially when large, can pose a diagnostic challenge as highlighted in this case.

CONCLUSION

In patients on anticoagulation, rapid interval growth of an adrenal mass does not always equate to malignancy. Recognizing this mimicry in the era of widespread Direct Oral Anticoagulant use is essential for refining treatment decision regarding surgical intervention, as most cases exhibited a self-limiting process with spontaneous resolution.

EP_A012

SILENT ADRENAL MASS WITH DIAGNOSTIC CHALLENGE: A CASE OF HUGE NON-FUNCTIONING ADRENAL LESION MIMICKING MALIGNANCY

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INTRODUCTION

Adrenal incidentalomas are increasingly detected with the widespread use of imaging, whereby the large or heterogeneous lesions often raise concern for adrenocortical carcinoma (ACC). However, certain rare benign