EP_A021

WHEN TISSUE IS THE ISSUE: PRESUMPTIVE DIAGNOSIS AND TREATMENT OF BILATERAL ADRENAL TUBERCULOSIS

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INTRODUCTION

Adrenal tuberculosis (TB) is a rare but important cause of adrenal insufficiency in TB-endemic regions, usually secondary to pulmonary or extrapulmonary disease, with primary involvement being uncommon. It typically affects both glands via hematogenous or lymphatic spread and presents late with nonspecific features after significant adrenal destruction, making diagnosis challenging. Diagnosis is more straightforward in the presence of extra-adrenal TB, where imaging may obviate the need for biopsy, but remains difficult in isolated adrenal involvement.

CASE

We report a 69-year-old Malay male with diabetes mellitus, hypertension, and hyperlipidemia who presented with a 2-week history of chronic cough and 4 months of constitutional symptoms, including weight loss and anorexia. Initial TB workup in September 2025 was negative. He was subsequently admitted twice in October 2025 for pneumonia, with persistent upper lobe consolidation on chest radiography despite antibiotics.

Contrast-enhanced computed tomography thorax, abdomen, and pelvis revealed bilateral mildly enhancing hypodense adrenal lesions (right 3.7 × 3.0 cm, left 2.5 × 2.6 cm), suggestive of adenoma, hyperplasia, or lymphoma. Bronchoscopy detected Mycobacterium tuberculosis via GeneXpert BAL, with no malignant cells on cytology. Adrenal biopsy was non-diagnostic.

Biochemical evaluation showed low morning cortisol with elevated adrenocorticotropic hormone and findings consistent with adrenal insufficiency. The patient was treated as smear-negative pulmonary TB with adrenal involvement and initiated on anti-TB therapy, currently in the maintenance phase. Hydrocortisone replacement 10 mg twice daily was also initiated.

CONCLUSION

This case highlights the diagnostic challenge of adrenal TB, particularly when tissue sampling is inconclusive. In TB-endemic settings, a presumptive diagnosis based on clinical, radiological, and microbiological evidence is often necessary. Early empiric anti-TB therapy is essential to prevent adrenal crisis and preserve endocrine function.

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BEYOND MITOTANE IN A PATIENT WITH HIGHLY AGGRESSIVE ADRENOCORTICAL CARCINOMA

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INTRODUCTION

Adrenocortical carcinoma (ACC) is an aggressive malignancy with high rates of recurrence even after surgical resection. Surgery remains the mainstay of treatment, while adjuvant options are limited. Mitotane is the only approved systemic therapy. Current guidelines recommend stereotactic body radiotherapy (SBRT) alongside adjuvant mitotane therapy in Rx, R1, R2 resections and in locally advanced disease.

CASE

We present a case of a 40-year-old female who presented with abdominal pain and was found to have a large heterogeneous left adrenal mass measuring 8.2 × 8.5 × 9.5 cm (Hounsfield Unit 63) on computed tomography (CT) imaging. Clinically, she was obese with a body mass index of 33.7 kg/m^2. No discriminatory feature of Cushing's was present. Hormonal evaluation demonstrated autonomous cortisol secretion with failure of suppression on both overnight and low-dose dexamethasone suppression tests at 301 nmol/L and 313.6 nmol/L, respectively. DHEA, testosterone, and urinary metanephrine were within range. Hemoglobin A1c was 6.6%. She underwent open left adrenalectomy. Intra-operatively, a 12 × 10 cm adrenal tumor was identified with multiple areas of tumor rupture and spillage during mobilization. HPE confirmed high-grade ACC with high Weiss score of 8, Ki-67 index 60–80%, and mitotic count 54/50 hpf (pT2Nx). Post-operative CT imaging demonstrated a residual soft tissue lesion in the left adrenal bed (largest diameter 3.8 cm) with fluorodeoxyglucose avidity. We commenced adjuvant mitotane therapy, titrated to 2 g TDS with supraphysiological hydrocortisone replacement. Mitotane level was within therapeutic range (16 mcg/mL). She was deemed unsuitable for repeat surgery due to the proximity of the residual mass to the adjacent vessel and was planned for SBRT therapy after a multi-disciplinary team discussion.

CONCLUSION
High-risk ACC with suspected residual disease remains a therapeutic challenge. While mitotane remains the cornerstone of adjuvant therapy, SBRT may represent a promising adjunctive local treatment modality in carefully selected patients. Further studies are required to define its role in improving local control and outcomes in ACC.

EP_A023

WHEN CORTISOL OVERWHELMS THE HEART: A FATAL CASE OF METASTATIC ADRENOCORTICAL CARCINOMA PRESENTING AS ACUTE HEART FAILURE

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INTRODUCTION
Cushing's syndrome is a multisystem disorder with significant cardiovascular morbidity, yet presentation as acute heart failure is uncommon. When driven by adrenocortical carcinoma (ACC), the clinical course is often aggressive and rapidly fatal, with particularly poor out-comes in resource-limited settings where access to therapy is constrained.

CASE
A 39-year-old previously well female presented with acute decompensated heart failure, newly diagnosed hypertension, type 2 diabetes mellitus, and obesity, preceded by a 3-year history of secondary amenorrhea and progressive weight gain. On admission, she exhibited florid Cushingoid features. Biochemical evaluation confirmed severe adrenocorticotropic hormone (ACTH)-independent hypercortisolism: morning serum cortisol 1,950 nmol/L, failure of suppression on overnight dexamethasone suppression test (post-ODST cortisol 2022.9 nmol/L), and elevated 24-hour urinary free cortisol (2,069 nmol/24 hours, 2.56 × upper limit of normal). Androgen excess was evident, with elevated dehydroepiandrosterone sulfate (DHEAS more than 27 µmol/L) and testosterone (9.91 nmol/L). ACTH was suppressed, supporting an adrenal source, while aldosterone was normal. Contrast-enhanced computed tomography demonstrated a large left adrenal mass (12 cm) with tumor thrombus extending into the inferior vena cava and renal veins, with extensive hepatic and pulmonary metastases, consistent with advanced ACC. Management was limited by disease severity and resource constraints. Ketoconazole was contraindicated due to transaminitis, and alternative steroidogenesis inhibitors were unavailable, leaving metyrapone as the only feasible option. Oncological therapy was deferred due to sepsis and clinical instability. Her course was fulminant, complicated by recurrent heart failure, sepsis, and metabolic derangements, culminating in refractory cardiopulmonary failure. She died within 1 month of diagnosis, prior to definitive oncological intervention.

CONCLUSION
Fulminant cortisol-secreting ACC may present catastrophically as acute heart failure and progress rapidly. Early recognition and timely access to multimodal cortisol-lowering therapy are critical, particularly in resource-limited settings. In fulminant hypercortisolism, the challenge is not diagnosis—but timing.

EP_A024

SOLITARY PROGRESSION TO BONE: A RARE MANIFESTATION OF ADRENOCORTICAL CARCINOMA

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INTRODUCTION
Adrenocortical carcinoma (ACC) is a rare and aggressive malignancy with a predilection for metastasis to the liver, lungs, and lymph nodes. Bone involvement is less common and typically occurs alongside widespread disease. Isolated skeletal progression without visceral involvement is unusual and not well characterized.

CASE
A 60-year-old female underwent left adrenalectomy in 2019 for an incidentally detected adrenal mass, which was reported as a benign adrenal cortical adenoma (Ki-67 <3%). In 2022, she presented with persistent low back pain. Imaging demonstrated fluorodeoxyglucose-avid lesions involving the T12 vertebra and right ilium, without evidence of local recurrence or visceral metastases. Histopathological evaluation of a bone biopsy initially suggested a neuroendocrine neoplasm based on synaptophysin positivity. Following multiple expert reviews and integration of clinical, radiological, and immunohistochemical findings, a consensus diagnosis of metastatic ACC was established.

She received palliative radiotherapy to symptomatic skeletal sites and subsequently completed six cycles of etoposide, doxorubicin, and cisplatin chemotherapy in 2023, achieving disease stabilization. Surveillance imaging in May 2025 demonstrated progression confined to the axial and appendicular skeleton, with no involvement of the adrenal bed or visceral organs. Mitotane therapy was initiated in December 2025. Ongoing management focuses on systemic disease control, symptom palliation, and multidisciplinary supportive care.