marked by the appearance of a new pulmonary nodule. Notably, however, the adrenal mass remained stable in size and morphology.

CONCLUSION

The absence of mixed androgen/glucocorticoid hypersecretion, combined with the radiographically static nature of the mass, suggests a lower probability of adrenocortical carcinoma. This case highlights that while size is a major risk factor for adrenocortical carcinoma, it must be interpreted in conjunction with hormonal activity, HU, and growth patterns. Identifying these "mimics" helps avoid over-staging lung cancer and ensures patients receive targeted therapy instead of unnecessary adrenalectomies.

EP_A018

A HIDDEN DIAGNOSIS: DISSEMINATED HISTOPLASMOSIS MIMICKING TUBERCULOSIS IN THE ELDERLY

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INTRODUCTION

Histoplasmosis is a rare opportunistic, inhalation-acquired systemic mycosis caused by *Histoplasma capsulatum*, endemic to Southeast Asia, including Malaysia. Although classically seen in immunocompromised hosts, it is increasingly reported in immunocompetent individuals. Infection is associated with environmental exposures such as bat or bird droppings and soil disruption. Histoplasmosis is a progressive granulomatous disease that can closely mimic tuberculosis and malignancy, making diagnosis challenging.

CASE

An 87-year-old Malay male with a history of treated pulmonary tuberculosis presented with a 6-month history of intermittent fever, anorexia, weight loss, and hypotension. Initial computed tomography imaging demonstrated bilateral heterogeneous adrenal masses (right: 2.2 × 3.7 × 5.0 cm; left: 2.0 × 3.7 × 5.2 cm) with indeterminate washout characteristics. Biochemical adrenal evaluation, including a short Synacthen test, confirmed primary adrenal insufficiency. Extensive microbiological and malignancy workup, including bronchoscopy, cultures, and tumor markers, was non-diagnostic. In view of clinical deterioration and epidemiological risk, empirical antituberculous therapy was initiated; however, no clinical improvement was observed after 2 months. PET-FDG revealed intensely hypermetabolic bilateral adrenal masses (SUVmax right 16.7, left 13.2) with no other abnormal foci. Non-invasive fungal investigations were negative. Definitive diagnosis was established via computed tomography-guided adrenal biopsy, which demonstrated necrotizing granulomatous inflammation with intracellular yeasts, subsequently identified as *H. capsulatum*. The patient was treated with oral itraconazole and corticosteroid replacement, resulting in significant clinical improvement and planned interval radiological reassessment.

CONCLUSION

Disseminated histoplasmosis is a diagnostic challenge, particularly in frail elderly patients, where invasive procedures may be delayed. It can mimic tuberculosis and malignancy and may lack an identifiable exposure history. Non-invasive tests may be inconclusive, making tissue biopsy essential. Adrenal involvement may result in primary adrenal insufficiency, further complicating the clinical picture. Early consideration and timely confirmation are crucial for appropriate management.

EP_A019

OVERWHELMING OPPORTUNISTIC INFECTIONS AS THE INITIAL PRESENTATION OF SEVERE CUSHING SYNDROME

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INTRODUCTION

Severe hypercortisolism is associated with profound impairment of both innate and adaptive immune responses, predisposing affected individuals to opportunistic infections. Excess glucocorticoids alter leukocyte trafficking, suppress pro-inflammatory cytokine production, and impair cellular immunity, increasing susceptibility to bacterial, viral, and fungal pathogens. In some cases, severe infections may precede the diagnosis of Cushing syndrome and represent the initial clinical manifestation. Early recognition is important as untreated hypercortisolism can lead to substantial morbidity and mortality.

CASE

A young adolescent male presented with progressive facial fullness and facial hyperpigmentation for 4 months, followed by 1 month of intermittent fever, cough, lower limb weakness, and hallucinations. On examination, he was tachypneic with cushingoid features including moon facies, pigmented acne over the face and chest, and nail bed hyperpigmentation. He was hypertensive and had severe hypokalemia with lymphopenia.

Radiological imaging demonstrated multiple cavitary lung lesions and intracranial tuberculomas. Bronchoalveolar lavage identified multiple opportunistic pathogens, including *Pneumocystis jirovecii, Aspergillus fumigatus*, and *Haemophilus influenzae*, while cerebrospinal fluid testing was positive for cytomegalovirus.

Given the unusual combination of infections, an underlying immunocompromised state was suspected. Endocrine evaluation revealed markedly elevated serum cortisol, with loss of diurnal rhythm and elevated adrenocorticotropic hormone (ACTH). Twenty-four-hour urinary cortisol was significantly increased, confirming severe ACTH-dependent Cushing syndrome. Magnetic resonance imaging of the pituitary gland and computed tomography imaging of the thorax, abdomen, and pelvis did not identify the source of ACTH secretion.

CONCLUSION

This case highlights that overwhelming opportunistic infections may be the first manifestation of severe Cushing syndrome in children. Excess cortisol disrupts host defenses by impairing neutrophil chemotaxis and macrophage phagocytosis, suppressing T-cell-mediated immunity, and reducing cytokine signaling necessary for pathogen clearance. These mechanisms contribute to susceptibility to simultaneous bacterial, fungal, and viral infections. Clinicians should therefore consider underlying hypercortisolism in patients presenting with multiple or unusual opportunistic infections to enable earlier diagnosis and appropriate multidisciplinary management.

EP_A020

BEYOND HYPONATREMIA: UNMASKING ADDISON'S DISEASE

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INTRODUCTION

Primary adrenal insufficiency is rare and potentially life-threatening, with an estimated prevalence of five cases per million in Southeast Asia. Local data remain limited, and diagnosis is frequently delayed due to non-specific clinical manifestations. Widespread use of traditional medication in Malaysia may further undermine recognition, particularly when steroid exposure is concealed. We report a female on prolonged use of traditional remedies presented with classic features of Addison's disease rather than cushingoid features, confirmed by biochemical results.

CASE

A 65-year-old female with underlying dyslipidemia and osteoarthritis presented with 4 days of giddiness, poor intake, nausea, and diarrhea. Further history revealed prolonged use of multiple traditional Chinese medicines, discontinued months prior, raising suspicion of prior steroid exposure. She claimed her skin has become darker over the past 2 months. She denied any infectious symptoms, contact with PTB patients, or exposure to birds. There was no family history of autoimmune disease. Clinically, she was dehydrated and hypotensive. Her blood pressure improved after fluid resuscitation. There was hyperpigmentation involving the face, extremities, tongue, and buccal mucosa.

Investigation results showed severe hyponatremia (119 mmol/L), hyperkalemia (4.93 mmol/L), with normal creatinine and negative infective markers. Hyponatremia persisted despite adequate hydration. Thyroid function test was normal (Free T4 12.28 pmol/L and thyroid-stimulating hormone 4.16 μIU/mL). Morning cortisol was suppressed (37 nmol/L) with markedly elevated adrenocorticotropic hormone levels (1,134 pg/mL), confirming the diagnosis of primary adrenal insufficiency. Hence, hydrocortisone was commenced, and serum sodium was normalized 2 days later. The underlying etiology remains under evaluation, although autoimmune adrenalitis is the most likely cause.

CONCLUSION

Primary adrenal insufficiency should be considered in patients presenting with unexplained hyponatremia and hypotension. In a setting where traditional medication use is prevalent, unrecognized steroid exposure may further complicate diagnosis. A thorough clinical and appropriate biochemical assessment is crucial to differentiating primary from secondary adrenal insufficiency.