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.

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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.