CONCLUSION

This case illustrates an uncommon pattern of ACC progression characterized by bone-dominant metastases in the absence of visceral disease. It also highlights the importance of reconsidering the initial histopathological diagnosis when clinical behavior is discordant. Vigilance for atypical metastatic patterns is warranted, even years after resection of an adrenal lesion initially classified as benign.

EP_A025

CONUNDRUM IN THE MANAGEMENT OF A RARE CAUSE OF CUSHING SYNDROME

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INTRODUCTION

Primary bilateral macronodular adrenal hyperplasia (PBMAH), characterized by bilateral adrenal macronodules >1 cm, is a rare genetic disease contributing to <2% of Cushing syndrome.

CASE

A 56-year-old male with underlying diabetes mellitus and hypertension presented with worsening proximal muscle weakness. Detailed neurological assessment was unremarkable. Re-assessment revealed more apparent cushingoid features, prompting referral to endocrinology. Basal cortisol was markedly elevated at 957 nmol/L (145.4–619.4 nmol/L). Unsuppressed cortisol after 1 mg overnight dexamethasone and low-dose dexamethasone test confirmed Cushing Syndrome. Suppressed adrenocorticotropic hormone (ACTH <0.33 pmol/L) suggested autonomous cortisol secretion from adrenal glands. Computed tomography adrenal reported massively enlarged hypodense multinodular adrenal glands of varying sizes. Oral ketoconazole, an adrenal steroidogenesis inhibitor, was initiated. Despite careful titration to keep serum cortisol 400–500 nmol/L, he experienced glucocorticoid withdrawal syndrome. Bilateral adrenalectomy was planned but deferred as he continued to lose weight despite persistent hypercortisolism. Extensive investigations were conducted for possible opportunistic infections or malignancy. His chest X-ray revealed a suspicious right lung cavity. Positive serum galactomannan and bronchial alveolar lavage galactomannan suggested pulmonary aspergillosis. Voriconazole was introduced, with drug-drug interaction judiciously addressed. He underwent bilateral adrenalectomy and required lifelong glucocorticoid and mineralocorticoid replacement. Histopathological report confirms bilateral macronodular adrenocortical disease. A referral to a genetic clinic was made to explore the potential hereditary basis.

CONCLUSION

PBMAH is a highly heterogeneous disease with variable presentation and a wide spectrum of hypercortisolism. Definitive management remained controversial and individualized. Multidisciplinary discussion is crucial, and genetic study is highly recommended for long-term prognostication and familial screening.

EP_A026

SYNERGIZING ARTIFICIAL INTELLIGENCE WITH CLINICIANS' EXPERTISE: TOWARDS PERSONALIZED PROACTIVE DIABETES CARE IN MALAYSIAN PRIMARY HEALTH CLINICS

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INTRODUCTION

Diabetes affects 15.6% of Malaysian adults and remains a leading cause of complications and mortality. Although locally developed artificial intelligence (AI) tools have shown strong technical performance, their integration with clinicians' decision-making in primary care remains fragmented. This perspective proposes a synergistic AI–clinician framework to deliver truly personalized proactive diabetes care.

METHODOLOGY

A multidisciplinary team designed a clinical workflow that integrates three validated Malaysian AI components: (1) LightGBM (LGBM) model trained on Malaysian National Diabetes Registry (MNDR) data for complication risk prediction, (2) DR.MATA AI retinopathy screening with automated triage, and (3) AI-enhanced mobile apps for real-time self-management. These layers feed into a single secure clinician dashboard. The framework was modelled using existing datasets (MNDR 2011–2021, NHMS 2023, DR.MATA validation cohort >14,000 images) and aligned with the National AI Roadmap 2021–2025 and the forthcoming National AI Action Plan 2026–2030.

RESULTS

The LightGBM model achieved ROC-AUC values of 0.84 (mortality), 0.71 (retinopathy/nephropathy), 0.66 (ischemic heart disease), and 0.74 (stroke). DR.MATA demonstrated