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

Tze Liang Lee[1], Shaleni Nagappen[1], Deviga Latchumanan[1]
[1]Department of Internal Medicine, Hospital Port Dickson

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

Mohd Fyzal Bahrudin[1], Jia Miao Tan[1], Chin Voon Tong[1]
[1]Endocrine Unit, Department of Medicine, Hospital Putrajaya

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.

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

Wee Mee Cheng[1], Lit Sin Yong[1], Poh Shean Wong[1], Nor Afidah binti Karim[1], Noor Lita Adam[1]
[1]Endocrinology Unit, Department of Internal Medicine, Hospital Tuanku Ja'afar Seremban

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

Sindeh Woweham[1], Mohd Nazri Mohd Daud[1], George George Mathew[1], Hazwani Hanum Hashim[1], Khairun'naim Khairuddin[1]
[1]Faculty of Medicine and Health Sciences, Universiti Malaysia Sabah

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