

## Adult E-Poster

### CONCLUSION

Failure to recognize euglycemic DKA may lead to catastrophic outcomes. Clinicians must maintain a high index of suspicion in high-risk populations and advocate for ketone testing in unexplained metabolic acidosis, regardless of glucose levels. Early recognition and targeted therapy can rapidly reverse acidosis and prevent morbidity.

## EP\_A016

### CUSHING'S DISEASE AND THE COST OF DELAY: FROM METABOLIC TO SKELETAL FRAGILITY

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#### INTRODUCTION/BACKGROUND

Cushing's disease, caused by an ACTH-secreting pituitary adenoma, can lead to profound metabolic disturbances, including insulin resistance, hypertension, osteoporosis, and an increased risk of fractures. Timely intervention is crucial to prevent long-term complications. Surgical removal of the pituitary adenoma via transsphenoidal surgery remains the gold standard treatment, offering potential for disease remission and metabolic recovery.

#### CASE

A 39-year-old female was initially investigated at age 34 for young-onset hypertension, recurrent hypokalaemia, and diabetes mellitus. Despite the absence of classical Cushingoid features, biochemical evaluation revealed persistent hypercortisolism, with an unsuppressed overnight dexamethasone suppression test (ONDST 565 nmol/L), low-dose dexamethasone suppression test (607 nmol/L), and markedly elevated 24-hour urinary cortisol (1401 nmol/L). Adrenocorticotropic hormone (ACTH) levels were elevated (5.4 pmol/L), and a cortisol day curve confirmed the loss of cortisol and ACTH diurnal rhythm. Magnetic resonance imaging identified a left pituitary microadenoma (5.9 × 6.5 mm). However, the patient was lost to follow-up and was only reinvestigated after sustaining a T10 compression fracture from a trivial fall. Repeat biochemical testing reaffirmed hypercortisolism (ODST: 750 nmol/L, 24-hour urinary cortisol: 1544 nmol/L, ACTH: 9.7 pmol/L). Magnetic resonance imaging showed a stable pituitary lesion (6.2 × 4.0 mm), and inferior petrosal sinus sampling confirmed a pituitary source of ACTH hypersecretion, with post-DDAVP central-to-peripheral ACTH ratios >3. Ketoconazole was initiated (titrated to 400 mg BD) for biochemical control. She successfully underwent

endoscopic transsphenoidal surgery with adenomectomy and hypophysectomy in October 2024. Postoperatively, she achieved remission but developed panhypopituitarism, necessitating hormone replacement with hydrocortisone, L-thyroxine, and estradiol (Progyluton). Remarkably, she no longer required diabetes treatment, and her hypertension improved, requiring only a single antihypertensive agent.

### CONCLUSION

This case highlights the challenges of diagnosing Cushing's disease in the absence of overt clinical features, the devastating skeletal consequences of delayed treatment, and the transformative impact of successful surgical intervention. Early recognition, multidisciplinary management, and timely surgical intervention remain paramount in optimizing patient outcomes.

## EP\_A017

### AN UNUSUAL SITE OF ADRENOCORTICAL CARCINOMA

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#### INTRODUCTION/BACKGROUND

Adrenocortical carcinoma (ACC) is a rare malignancy with an incidence of 0.5–2 cases per million per year. Typically, ACC originates in the adrenal glands. Although exceedingly rare, ectopic presentations can occur due to developmental anomalies and rarely may arise from an adrenal rest. These adrenal rests are usually clinically silent, but on rare occasions, may undergo malignant transformation and hormonal secretion.

#### CASE

We report the case of a 33-year-old female with underlying hypertension and diabetes who had an incidentally discovered right adnexal mass which was asymptomatic during a routine medical checkup. She underwent complete laparoscopic tumour resection without complications. Comprehensive histopathologic evaluation revealed a low-grade ectopic ACC arising from an adrenal rest. Postoperative imaging demonstrated no residual tumor and normal adrenal glands. She remains under active surveillance.

The case highlights the diagnostic challenge posed by an ectopic ACC masquerading as an adnexal mass. Detailed histopathologic and immunohistochemical analyses are