## Adult E-Poster

**CASE**

A 48-year-old female, with no known medical illness presented with right neck swelling for 5 months duration. The painless neck swelling progressively increased in size, with no obstructive symptoms. She did not exhibit any symptoms related to catecholamine excess. No medications were given as well. There was no other significant personal or family medical history, including familial cancer syndromes such as multiple endocrine neoplasia type 2 (MEN 2), Von-Hippel Lindau (VHL) and neurofibromatosis (NF1). She was normotensive (133/64 mm Hg), with normal heart rate (90 beats per minute). Neck examination revealed right neck swelling measuring 2.5 cm x 3 cm, well demarcated, firm and immobile. Biochemistry results showed normal metanephrine (0.43 umol/24H), normal normetanephrine (0.66 umol/24H) but elevated 24 hour urine 3-Methoxytyramine (6.66 umol/24H). Computed tomography scan and MRI of the neck demonstrated a right carotid space enhancing mass measuring 3.2 x 3.0 x 4.1 cm. Subsequently, CT scan of the thorax, abdomen and pelvis were carried out, but no adrenal nodule or mass was noted. After a week of alpha-blockade as preoperative management, she successfully underwent pre-embolization and tumor excision via transcervical approach. Intra-operatively, neither hypotension nor hypertension was noted After the operation, she required voice rehabilitation and recovered well. Histopathology report confirmed the diagnosis of exclusively dopamine secreting carotid body paraganglioma with no extension to the lymph nodes. Post-operatively, PET scan and 24 hour urine metanephrine/ normetanephrine/ 3-methoxytyramine were conducted and no biochemical or imaging evidence of recurrence or metastasis was observed.

**CONCLUSION**

Dopamine secreting paragangliomas are rare and difficult to diagnose. Hence as clinicians, one needs to have a high index of suspicion to enable early diagnosis and management.

# EP_A015

## EUGLYCEMIC DIABETIC KETOACIDOSIS: ELUSIVE, YET A DIAGNOSIS NOT TO BE OVERLOOKED IN CASES OF UNEXPLAINED METABOLIC ACIDOSIS

https://doi.org/10.15605/jafes.040.S1.023

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**INTRODUCTION**

Euglycemic diabetic ketoacidosis is a rare but serious condition. The absence of hyperglycemia frequently causes a delay in diagnosis and treatment initiation. We present a case of acute coronary syndrome in cardiogenic shock in which the euglycemic DKA diagnosis was missed.

**CASE**

A 65-year-old female with underlying diabetes mellitus, hypertension, chronic kidney disease and ischemic heart disease presented with typical chest pain and heart failure symptoms. Patient was tachypneic with Grd 2 edema, BP 107/58 mm Hg, HR 98 beats/min, $SpO_2$ 89% at room air and blood glucose 6.3 mmol/L. Electrocardiogram had dynamic changes. Initial blood investigations showed urea 15.3 mmol/L, sodium 133 mmol/L, K 4.4 mmol/L, Cl 105 mmo/L, creatinine 376 umol/L, pH 7.236, lactate 6.7 mmol/L, bicarbonate 12.2 mmo/L and anion gap 16.6 mmol/L. Bedside ultrasound revealed ejection fraction of 40-50%, RWMA, plethoric IVC measuring 2.3 cm. As the patient's blood pressure dropped, noradrenaline was administered with the furosemide infusion. The patient was assessed to have acute decompensated heart failure in cardiogenic shock secondary to acute coronary syndrome and acute on chronic kidney disease. Despite optimal doses of diuretics, there was no urine output. Dialysis was initiated due to refractory fluid overload. Venous blood gas post dialysis showed pH 7.184, $HCO_3$ 11.5 mmol/L, glucose 7 mmol/L, lactate 1.6 mmol/L and anion gap 17 mmol/L. Despite dialysis and improved serum lactate levels, the metabolic acidosis worsened. Capillary ketone was taken for unexplained acidosis showing an alarming value of 4.3 mmol/L, confirming the diagnosis of euglycemic DKA. Insulin infusion with dextrose was initiated. Follow-up VBG indicates an improvement of pH to 7.273 and $HCO_3$ to 13.6 mmol/L.

## 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

*https://doi.org/10.15605/jafes.040.S1.024*

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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). Adrenocorticotrophic 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

*https://doi.org/10.15605/jafes.040.S1.025*

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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