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Riaz, B., Muraleedharan, V., Fernando, D. et al. (2 more authors) (2024) 7898 A case of dual secreting adrenal lesion with multiple complications resulting from hormonal abnormalities. In: Journal of the Endocrine Society. ENDO 2024 Abstracts Annual Meeting of the Endocrine Society, 01-04 Jun 2024, Boston, USA. The Endocrine Society

<https://doi.org/10.1210/jendso/bvae163.060>

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Abstract citation ID: bvae163.060

**Adrenal (Excluding Mineralocorticoids)
7898*****A Case of Dual Secreting Adrenal Lesion With
Multiple Complications Resulting From Hormonal
Abnormalities***Babar Riaz, MBBS, MRCP¹,Vakkat Muraleedharan, MBBS, MD, FRCP, PhD²,Devaka Fernando, MD, FRCP, MSc¹,Sabapathy Balasubramanian, MBBS MS DMI FRCSEd PhD³,and John Newell-Price, MD, PhD, FRCP⁴¹Sherwood Forest Hospitals NHS Foundation Trust, Mansfield, United Kingdom; ²Sherwood Forest Hospitals NHS Foundation Trust, Mansfield, United Kingdom, Nottingham, United Kingdom; ³School of Medicine and Population Health, University of Sheffield, Sheffield Teaching Hospitals NHS Foundation Trust., Sheffield, United Kingdom; ⁴University of Sheffield, Sheffield, United Kingdom**Disclosure:** **B. Riaz:** None. **V. Muraleedharan:** None. **D. Fernando:** None. **S. Balasubramanian:** None. **J. Newell-Price:** None.

Introduction: Dual secreting adrenal tumours with catecholamine and ACTH secretion are rare. This dual secretion may alter the presentation of a Pheochromocytoma, which makes the diagnosis challenging [1]. **Case report:** A 47-year-old woman presented to the local emergency department with intermittent palpitations, abdominal pain and high blood pressure 220/96 mmHg. Investigation showed hypokalemia 3.0 mmol/L (3.5 - 5.3), and metabolic alkalosis (pH 7.50, HCO₃ 32 mmol/L). A CT abdomen showed a 36 mm right adrenal lesion, which was initially reported as a myelolipoma by the local hospital. Plasma normetadrenaline 1.18 nmol/L (0 - 1.07), metadrenaline 0.88 nmol/L (0 - 0.33), and cortisol 2251 nmol/L were raised. She was started on spironolactone and phenoxybenzamine. Subsequent MRI showed a 3.4 cm right adrenal adenoma with cystic areas. ACTH was 156 ng/L (7.2-63.3) and 1mg overnight Dexamethasone suppression test failed to suppress serum cortisol. MRI pituitary was normal. Whilst awaiting outpatient review, the patient was re-admitted with DKA (new onset diabetes mellitus with high ketones and anion gap acidosis), and severe hypokalemia 1.4 mmol/L, requiring critical care admission. She also required intubation and a broncho-alveolar lavage and was treated for pneumocystis jiroveci pneumonia (PJP). A discussion at the regional specialised endocrine MDT made a diagnosis of right pheochromocytoma with ectopic ACTH secretion with hyperplasia of the left adrenal gland, with no other clear source of ACTH on axial imaging, and high dose metyrapone initiated resulting in rapid control of the hypercortisolaemia. Autoantibodies for Type 1 diabetes mellitus were negative. On review at the regional endocrine centre, she was clearly Cushingoid, but with normal

electrolytes, and maintained on medical therapy for 30 weeks to improve her clinical state prior to a right retroperitoneal adrenalectomy. Following this there was complete resolution of her clinical state and normalisation of plasma metanephrines, blood glucose, blood pressure, and plasma ACTH levels (30 ng/L) on no therapy. Histology showed a pheochromocytoma and adrenal cortical hyperplasia, although ACTH immunoreactivity was negative.

Conclusion: This case highlights an unusual presentation of Cushing's syndrome secondary to an ACTH-secreting pheochromocytoma (the challenges were absence of initial clinical signs of Cushing's and borderline plasma metanephrine levels); complicated by new onset Diabetes Mellitus, DKA (in the absence of absolute insulin deficiency), and development of PJP [2]. Clinical resolution following surgery points towards the pheochromocytoma being the source of ACTH, as ACTH immunoreactivity may be negative. Normal pituitary morphology argues against ectopic CRH secretion. **References:** 1. Doi:10.1210/clinem/dgaa4882. Doi.org/10.1016/j.ando.2022.09.02

Presentation: 6/2/2024