Unmasking the Hidden Culprit: ACTH-Secreting Bronchial Tumor in Resistant Hypertension

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Introduction

Resistant hypertension, characterized by persistently elevated blood pressure despite optimal treatment, necessitates a thorough exploration of secondary causes such as obstructive sleep apnea (OSA), primary aldosteronism, renal artery stenosis, Cushing syndrome, and thyroid disorders. This case highlights the imperative of a comprehensive diagnostic strategy in resistant hypertension.

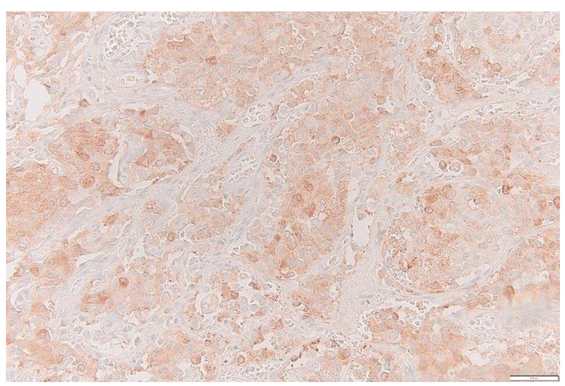

Figure 1: Neoplastic cells positive for ACTH

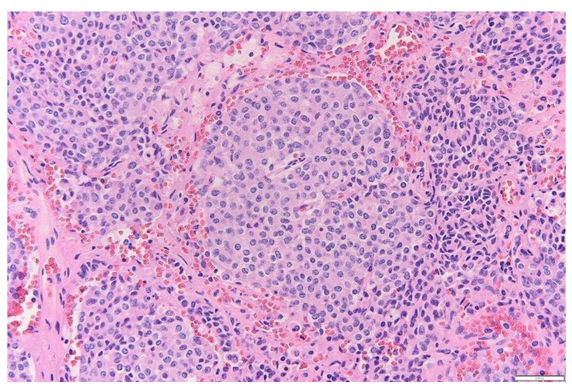

Figure 2: Nests of neoplastic cells with polygonal to round nuclei with salt-and-pepper chromatin. There is minimal cytologic atypia and rare mitotic figures.

Case Presentation

A 33-year-old male with a history of obesity, hyperlipidemia and MAFLD initially presented to a primary care clinic with elevated blood pressure, lower extremity edema, and difficulty losing weight. Despite treatment adjustments from amlodipine-valsartan to olmesartan, nifedipine, eplerenone, and doxazosin, his hypertension remained uncontrolled. Diagnosis of OSA led to CPAP therapy initiation, yet blood pressure control remained elusive. Episodic hypokalemia prompted further investigation, revealing normal TSH, low aldosterone (<1 ng/dL), and normal renin levels. A negative renal ultrasound did not show evidence of renal artery stenosis. Clinical examination subsequently unveiled features consistent with Cushing's syndrome: moon facies, facial plethora, violaceous striae, lower extremity and facial swelling, erectile dysfunction, and proximal muscle weakness. Laboratory investigations revealed markedly elevated morning cortisol levels (55.0 mcg/dL) and a urine potassium creatinine ratio (>40), indicative of hypercortisolism. Nephrology and endocrinology evaluations revealed high ACTH levels (196 pg/mL), positive dexamethasone suppression test (cortisol 43.8 mcg/dL), and elevated midnight salivary cortisol levels (1.60, 1.39 mcg/dL), leading to a diagnosis of ACTH-dependent Cushing Syndrome. Adrenal CT showed no adrenal nodules and pituitary MRI was unremarkable. CT chest revealed a 1.9 cm non-calcified peribronchiolar nodule in the left lower lobe, suggesting an ectopic ACTH-secreting lesion. The patient commenced osilodrostat for hypercortisolism management.

He was referred to oncology and thoracic surgery and underwent left video-assisted thoracoscopic surgery (VATS) with left lower lobectomy and mediastinal lymph node sampling. Histopathology confirmed an ACTH-producing bronchial carcinoid tumor. Post-operatively, he transitioned from intravenous to oral hydrocortisone for adrenal insufficiency prophylaxis, which was tapered off over several months. Ongoing care includes regular serum cortisol and ACTH monitoring, with biannual CT scans for recurrence surveillance. Blood pressure is now well controlled, and all antihypertensive medications have been discontinued.

Discussion

This case underscores the importance of thoroughly investigating secondary causes in patients with resistant hypertension. Early identification and management of rare etiologies such as ectopic ACTH-dependent Cushing Syndrome are crucial for improving patient outcomes and diminishing the potential for cardiovascular complications and organ damage. A comprehensive, interdisciplinary approach involving primary care providers, nephrologists, endocrinologists, oncologists, and thoracic surgeons ensured timely diagnosis and effective treatment, emphasizing the need for personalized care strategies tailored to complex secondary hypertension cases.

Conclusion

This case underscores the critical importance of a systematic approach to uncovering and addressing secondary causes of resistant hypertension, emphasizing the transformative impact of early diagnosis and targeted treatment on patient health outcomes.

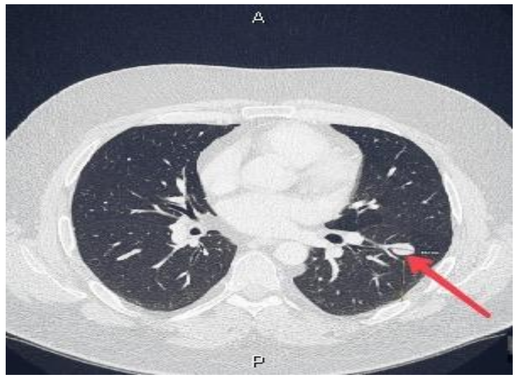

Figure 3

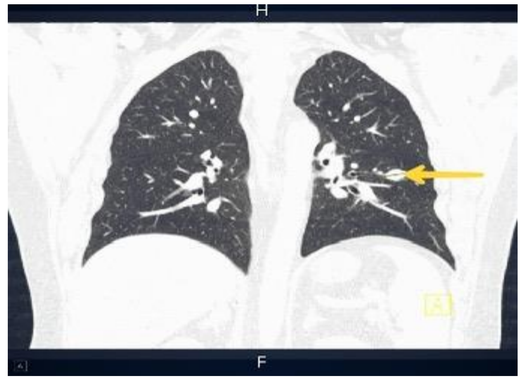

Figure 4

Figure 3,4: CT chest showing 1.9 cm non-calcified peribronchiolar nodule in the left lower lobe (red and yellow arrow)

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