Introduction: Cushing’s syndrome can present with a variable constellation of symptoms, and is most commonly caused by iatrogenic steroids, followed by pituitary Cushing’s disease. Rarely is fulminant Cushing’s syndrome encountered, and still rarer is it due to occult ACTH-producing neuroendocrine tumors. This is the case of a previously healthy, uninsured never-smoker who presents with classic findings of Cushing's syndrome caused by an ACTH-secreting bronchial tumor.

Case Description: A 43-year-old male presents to an outside ER for new, non-traumatic back pain in the setting of 30-pound weight loss over six months, progressive lower extremity weakness, loss of height, and recently diagnosed hypertension and diabetes. CT chest notable for T7/T9 compression and pathologic rib fractures; no intrathoracic "masses" are seen, though a 7 millimeter well-circumscribed round nodule is appreciated in the left lower lung, suggestive of a "granuloma". Labs return with elevated ACTH and cortisol levels, consistent with ACTH-dependent hypercortisolism. Subsequent MRI brain is unremarkable. DEXA confirms new osteoporosis (T score -4.2). He is referred to outpatient endocrinology, but prior to evaluation, he re-presents to our ER for worsening back pain and has the following findings:

- PMH: hypertension, diabetes, and hyperlipidemia; each diagnosed within past year.
- Medications: Lisinopril, atenolol, furosemide glimepridime, and hydrocodone for back pain.
- Social hx: immigrated from Guadalajara > 25 years ago. Works as mechanic and preacher. Married, has young children. Rare alcohol intake; never used tobacco or illicit drugs.
- No known allergies. No previous surgeries.
- Family hx: brother with lupus

P.E.: middle-aged male with hypertensive urgency (180/110), moon facies, kyphosis, central obesity with extremity wasting, fragile, diffuse ecchymoses, lower extremity edema and symmetric, proximal LE weakness with intact reflexes.

Initial imaging: CT spine which re-demonstrates thoracic spine and rib fractures with newly involved thoracic and lumbar regions.

Initial Presentation:

- High-dose (8mg) dexamethasone suppression test shows non-suppressible ACTH and cortisol (38.8 mcg/dL), suggestive of ectopic origins of ACTH
- Inferior petrosal sinus sampling confirms non-pituitary ACTH production by lack of central-to-peripheral gradient
- CT chest/abdomen/pelvis, obtained to evaluate for possible ectopic sources, is without additional masses aside from the previously seen 9 mm, well-circumscribed left lower lung nodule
- Octreotide scan reveals mild signal uptake in lung nodule, indicating neuroendocrine behavior
- Serum chromogranin A is elevated (122 ng/ml), supportive of neuroendocrine tumor diagnosis

Histopathology reported a well-differentiated, low grade neuroendocrine tumor positive for synaptophysin, chromogranin, ACTH and TTF-1, the latter representing pulmonary primary.

Treatment:
- Video-assisted thoracoscopic surgery with wedge resection
- Postoperative ACTH and cortisol concentrations declined dramatically (ACTH 96->8 pg/ml)
- Histopathology reported a well-differentiated, low grade neuroendocrine tumor positive for synaptophysin, chromogranin, ACTH and TTF-1, the latter representing pulmonary primary
- Initiated on hydrocortisone for anticipated secondary adrenal insufficiency following resection
- Successfully discharged home

Discussion: This case demonstrates a seldom considered etiology for fulminant Cushing’s syndrome, bronchial carcinoid tumor. Carcinoid tumors of the lung are exceedingly rare, representing less than 2% of all lung malignancies. Within bronchial carcinoids, only 1-2% are associated with Cushing’s syndrome. Furthermore, presenting symptoms of bronchial carcinoids are classically either asymptomatic, or associated with respiratory symptoms typical of other primary lung malignancies (cough, hemoptysis, recurrent pneumonia). Our never-smoker lacked any respiratory symptoms to invoke lung cancer; indeed, initial radiology interpretation of the nodule was benign granuloma. However, the rapid onset to Cushing’s syndrome along with weight loss (latter atypical for hypercortisolism) alluded to a more malignant cause, leading to prompt diagnostic investigation enabling definitive cure for a potentially fatal case of fulminant Cushing’s syndrome.