BRIEF COMMUNICATIONS

LOCALIZATION OF ADRENOCORTICOTROPIC HORMONE-PRODUCING PULMONARY CARCINOID BY SOMATOSTATIN RECEPTOR SCINTIGRAPHY

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We report the case of a 67-year-old woman with Cushing's syndrome in whom somatostatin receptor scintigraphy was used to localize a corticotropin-producing pulmonary carcinoid tumor. Over several years, the patient had worsening depression, lower extremity edema, glucose intolerance, and severe muscle weakness. Eventually, she was taken comatose to another hospital. Initial evaluation revealed a serum potassium concentration of 1.9 mmol/L and a serum adrenocorticotrophic hormone level of 132 pmol/L (normal range 9 to 52 pmol/L). Computed tomograms and magnetic resonance images of the abdomen and brain did not reveal any abnormalities. Thoracic computed tomography revealed a solitary 8 mm nodule in the peripheral right upper lobe without mediastinal adenopathy (Fig. 1). The patient was transferred to the Thoracic Surgical Service of Memorial Sloan-Kettering Cancer Center. Serum renin, aldosterone, calcitonin, and gastrin levels were within normal limits. Low- and high-dose dexamethasone suppression tests were performed without suppression of either serum or urinary cortisol levels. Somatostatin receptor scintigraphy demonstrated increased uptake in the right upper lobe of the lung that corresponded to the nodule seen on the computed tomographic scan; no additional abnormal areas of uptake were identified (Fig. 2). Through a limited right anterior thoracotomy, a 9 mm well-circumscribed nodule was excised from the anterior segment of the right upper lobe. Histologic examination revealed a well-differentiated pulmonary carcinoid tumor with positive staining for corticotropin hormone. Within several days, glucose and electrolyte levels returned to normal values, her depression lifted, and, with rehabilitation, muscle strength improved.

A corticotropin-secreting bronchial carcinoid may be an occult cause of Cushing's syndrome. These well-differentiated neoplasms originate from the neuroendocrine cells of the bronchial mucosa and may remain clinically silent until the stigmata of hypercortisolism appear. Computed

tomograms and magnetic resonance images of the brain, together with bilateral petrosal sinus sampling, will reliably distinguish pituitary from ectopic corticotropin-producing tumors. In two thirds of patients with Cushing's syndrome, the source of corticotropin production is small-cell carcinoma of the lung.¹ A high percentage of patients with small-cell lung cancer will have asymptomatic elevations of serum corticotropin, and clinically apparent Cushing's syndrome will be present in 2%.² Small-cell lung cancer is usually radiographically apparent; if not present, localizing an ectopic site of corticotropin production within the thorax can be difficult, because the next most common cause, pulmonary carcinoid tumors, are rare, often small, and often not readily distinguishable from other types of indeterminate lung nodules. If an indeterminate nodule is seen, establishing a diagnosis is problematic: transthoracic needle aspiration is associated with a significant false negative rate and exploration of the thorax by either thoracotomy or video-assisted techniques in the severely myopathic patient carries increased risks.

Recently, somatostatin receptors have been found to be expressed in the majority of carcinoid tumors. In vitro autoradiographic labeling of somatostatin analog binding sites was demonstrated in 54 of 62 carcinoid tumors³ and correlates well with anatomic localization by somatostatin receptor scintigraphy.⁴

Activated leukocytes, as accumulate in pulmonary granulomas or hilar lymph nodes with some autoimmune disorders, can be a source of false positive somatostatin

Fig. 1. Thoracic computed tomogram demonstrates a solitary 8 mm indeterminate right upper lobe nodule.

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Fig. 2. Somatostatin receptor scintigraphy demonstrates increased uptake in the right upper lobe (*arrow*) corresponding to the nodule seen on computed tomographic scan.

receptor scans.⁵ False positive lung scans have also been noted to result from increased hilar uptake after an upper respiratory tract infection, external beam

irradiation, bleomycin administration, or surgical treatment.⁵ However, if interpreted in an appropriate clinical setting, somatostatin receptor scintigraphy may provide an accurate, noninvasive technique for the characterization of indeterminate pulmonary nodules as hormonally active and should be considered in the diagnostic evaluation of the patient with Cushing's syndrome.

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PULSATING MASS AT THE STERNUM: A PRIMARY CARCINOMA OF ECTOPIC MEDIASTINAL THYROID

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During embryogenesis the descent of the thymus, thyroid, and parathyroid glands may be disturbed, leading to various possibilities for anomalous locations of each gland. One of the aberrant locations of the ectopic thyroid is the mediastinum. Rarely, a mass in the anterior upper mediastinum

may be a primary mediastinal goiter.¹ Even more rare is primary carcinoma in the ectopic mediastinal thyroid.²

Case report. A 62-year-old woman was referred to Tampere University Hospital in 1993 because of a palpable pulsating tumor in the sternum. The patient was free of symptoms and in good general condition. Eight years earlier she had been operated on for a benign follicular goiter and 6 years earlier she had had a benign gastric ulcer.

Clinical examination revealed a mass in the middle portion of the sternum with a clear pulsation. Palpation of the neck demonstrated a pretracheal thyroid of normal size without abnormal nodules. Doppler examination demonstrated a strong whizzing sound in the tumor during systole. The chest x-ray film showed a weak soft tissue expansion behind the sternum (Fig. 1). A strongly vascularized tumor or arteriovenous fistula was suspected and angiography was performed. The angiogram revealed a vascular tumor behind the sternum with vascular supply from both internal thoracic arteries (Fig. 2). The vascular

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