Use of Somatostatin Analog for Localization and Treatment of ACTH Secreting Bronchial Carcinoid Tumor*

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A 29-year-old woman presenting with an ectopic adrenocorticotropic hormone syndrome and a nodule of the upper lobe of the left lung was explored by intralobar sequestration. Hemothorax in intralobar sequestration is exceedingly rare and in extralobar sequestration has not been previously described.

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REFERENCES

Table 1—Biological Evaluation Prior to Treatment, on Day 24 of SMS Treatment, and 1 Week and 2 Months Postsurgery*

<table>
<thead>
<tr>
<th>Values</th>
<th>Initial Value</th>
<th>Treatment Day 24</th>
<th>1 Week Postsurg</th>
<th>2 Months Postsurg</th>
</tr>
</thead>
<tbody>
<tr>
<td>UFC, nmol/24 h</td>
<td>806 (260)</td>
<td>13</td>
<td>24</td>
<td>33</td>
</tr>
<tr>
<td>Plasma cortisol, nmol/L</td>
<td>1,493 (220-610)</td>
<td>116</td>
<td>123</td>
<td>151</td>
</tr>
<tr>
<td>16 h</td>
<td>1,103 (100-290)</td>
<td>211</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone test</td>
<td>1,166</td>
<td>101</td>
<td>18</td>
<td>32</td>
</tr>
<tr>
<td>Plasma ACTH, pg/mL, basal</td>
<td>214 (9-52)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRF test</td>
<td>246</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma LPH, pg/mL</td>
<td>1,674 (46-124)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

* Surgery took place on treatment day 60. Normal value ranges are shown in parentheses. UFC = urinary free cortisol; LPH = lipotropin hormone; pg = picogram.
1. 2-day high-dose dexamethasone suppression test (8 mg dexamethasone/d).

Key words: bronchial carcinoid; Cushing’s syndrome: ectopic ACTH secretion; somatostatin receptor scintigraphy

Carcinoid tumors are responsible for 40% of cases of adrenocorticotropic hormone (ACTH) dependent Cushing’s syndrome. Bronchial carcinoid tumors usually are small (0.3 to 1.3 cm) and are difficult to localize. In addition, hypercortisolism must be controlled prior to surgery. In this report, we describe, in a patient with ectopic ACTH syndrome, the rapid normalization of hypercortisolism by somatostatin analog in the rapid control of hypercortisolism prior to surgery. (CHEST 1996; 109:45-46)

A 29-year-old woman was admitted to the hospital for the exploration of a rapidly evolving hypercortisolism, discovered as the patient complained of generalized edema, anorexia, and hypokalemia (2.8 mmol/L). She weighed 80 kg and was 163-cm tall. She had a typical appearance of Cushing’s syndrome. Biological evaluation confirmed the ACTH-dependent hypercortisolism. Results of a high-dose dexamethasone test were negative, and the plasma ACTH level did not increase after cortisol releasing factor administration, suggesting an ectopic ACTH secretion (Table 1).
Pituitary imaging with nuclear magnetic resonance was normal, and an abdominal CT scan was also normal. The plasma immunoreactive calcitonin level was below 10 ng/mL (normal, <10 ng/mL), plasma serotonin was below 10 ng/mL (normal, <300 ng/mL), and urinary 5 HIAA was 12.7 mg/g of creatinine (normal, <18). Chest x-ray film and thoracic CT scan showed a 1-cm nodule of the upper lobe of the left lung. Bronchoscopy was normal. SMS receptor scintigraphy was performed using a 111-megabequerel IV injection of 111In diethylene triamine pentaacetae (dTPA) phenyl pentetrotide (Mallinkrodt; Petten, Netherlands) and revealed a significant pathologic uptake in the upper part of the left lung (Fig 1). The patient was then treated with octreotide (Sandostatin; Sandoz; Bäle, Switzerland), 200 μg subcutaneously 3 times daily. She complained of diarrhea and mild joint pain during the first week of treatment. Urinary free cortisol returned to normal on day 4 (16 nmol/24 h). On day 50, the patient underwent surgery, and octreotide therapy was discontinued. During thoracotomy, a small rounded tumor was found in the upper left lobe. Pathologic examination characterized the mass as a carcinoid tumor. The staining was positive for neuron-specific enolase, chromogranin A, and synaptophysin. Mediastinal lymph nodes were free of metastases. On day 10 after surgery, urinary free cortisol, plasma cortisol, and ACTH levels all were normal.

**DISCUSSION**

This observation suggests the interest of SMS receptor imaging technique to allow the diagnosis and localization of ectopic ACTH-secreting tumors. This technique also allows the assessment of a somatostatin treatment sensitivity.

Biologic and clinical normalization was rapidly obtained in this patient treated with octreotide only. Alternatively, the use of anticoicorticoid drugs provides a slow normalization of hypercorticism, generally associated with marked side effects. In addition, persistent adrenal insufficiency can be observed. In our patient, side effects were mild and rapidly reversible, and adrenal function was normal after octreotide treatment.

Octreotide inhibits the adenylate cyclase system via type 2 somatostatin receptors, and this is associated with a decrease in tumoral ACTH secretion. However, complete normalization of ACTH level was not achieved, and this might be due to receptor heterogeneity in tumor cells or to dysfunction of the adenylate cyclase. We cannot rule out the possibility that this tumor may secrete other peptides that may increase cortisol production, such as a bombesin-like peptide, in addition to ACTH.

A correlation between the presence of SMS receptors in binding studies and the response to octreotide treatment has been described. Octreotide scintigraphy may represent an easy technique to assess the presence of these receptors in vivo.

In conclusion, we believe that octreotide is an efficient and well tolerated treatment for preoperative preparation of patients with ectopic ACTH syndrome due to a carcinoid tumor, at least when the tumor appears to be positive on SMS receptor scintigraphy.

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**REFERENCES**


**Pulmonary Vein Thrombosis and Peripheral Embolization**

Mario J. Garcia, MD; Leonardo Rodriguez, MD; and Peter Vandervoort, MD

A 78-year-old woman was admitted to the hospital with bilateral femoral arterial occlusion. Her medical

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