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Acromegaly caused by ectopic growth hormone–releasing hormone secretion by a carcinoid bronchial tumor: A rare entity

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Ectopic secretion of growth hormone–releasing hormone (GHRH) is a rare cause of acromegaly.^{1–5} The most common tumors secreting GHRH are bronchial carcinoids and pancreatic islet cell tumors. Differential diagnosis of pituitary adenoma is necessary to avoid unnecessary pituitary surgery and damage to the pituitary gland. Rarely, that syndrome is secondary to a carcinoid bronchial tumor. We present a case of a synchronous GHRH-secreting bronchial carcinoid and a pituitary adenoma in a patient with acromegaly.

Clinical Summary

A 37-year-old man who presented with an acromegaly over the last 8-year period was hospitalized in our department. He had been treated surgically for an acidophil pituitary adenoma 1 year ago, and the histology had revealed an adenoma (1.3 cm × 1.3 cm) producing growth hormone (GH) and prolactin. There was no amelioration of the clinical signs of acromegaly postoperatively, and the GH and insulin-like growth factor levels, although lower, reached 50 ng/mL and 833 ng/mL, respectively. The glucose tolerance test indicated diabetes. Thyroid and parathyroid hormones were within normal levels.

The patient was subjected to magnetic resonance imaging, which showed remaining pathologic pituitary tissue. Sandostatin was administered in subcutaneous doses of 100 µg 3 times because during the test of somatostatin, there had been a tremendous suppression of GH levels. The response to sandostatin was remarkable, but acromegaly was still active, and the patient was submitted to radiation of the pituitary region with Co60 in a total dose of 5000 Gy (200 Gy daily).

Six months later, the patient was re-examined and was found to still have active acromegaly while receiving medication. His chest x-ray film demonstrated a large tumor of the left lung. Computed

Figure 1. Angiography demonstrating a rich vascularized carcinoid tumor.

tomographic scanning revealed a large circumscribed tumor in the left upper lobe without lymph node enlargement. Angiography was undertaken before surgical intervention to exclude angioma or arteriovenous malformation (Figure 1). The patient underwent a left upper lobectomy (Figure 2). Histology demonstrated a carcinoid tumor of 6 × 5.5 × 4.5 cm without cellular atypia or any features of malignancy. The bronchial carcinoid was tested immunohistochemically and was found to be positive for gastrin and somatostatin secretion.

One month later, the basal GH levels decreased to 3.5 ng/mL, the amelioration of the clinical signs was obvious, and the patient recovered completely within 6 months.

Discussion

Acromegaly caused by ectopic GHRH secretion by carcinoids is a rare cause of GH hypersecretion. This diagnosis should be suspected in any case of proved acromegaly in which there is no clear evidence of pituitary adenoma.¹ Of the 39 fully documented cases of acromegaly caused by extracranial tumors synthesizing GHRH recognized by Faglia and colleagues,³ 21 were bronchial carcinoids. Occasionally, carcinoid and pituitary adenomas coexist as parts of a widespread pluriglandular syndrome.⁵

This case differs from the others described in the literature because apart from the bronchial carcinoid, a pituitary adenoma coexisted and not a hyperplasia, as is mentioned in other reports.^{4,5}

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Received for publication Nov 17, 2003; revisions received Jan 30, 2004; accepted for publication Feb 5, 2004.

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J Thorac Cardiovasc Surg 2004;128:631-2

0022-5223/\$30.00

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doi:10.1016/j.jtcvs.2004.02.033

Figure 2. The well-encapsulated large tumor.

The patient's sex also differs. He was a man, and it is clearly stated in the literature that ectopic acromegaly is more often seen in women younger than 40 years.⁴

Unfortunately, the neuroendocrine tests cannot differentiate between ectopic GHRH secretion and pituitary adenoma.¹ High GH plasma levels and excessive production of GHRH can be

suggestive of ectopic secretion, with the exception of very rare cases of excessive GHRH of hypothalamic origin.² The somatostatin receptor scintigraphy could have been an alternative diagnostic tool, but it was not performed in our case.

In inoperable cases or incomplete operations, octreotide seems to be effective.⁵ Surgical excision of the bronchial carcinoid tumor leads to a rapid decrease of GHRH and to a benign clinical course, as is also mentioned in other reports.^{1,2}

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Innominate artery transection in the setting of a bovine arch

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Blunt trauma to the innominate artery is an infrequent but commonly fatal occurrence. We report a case of a patient with a bovine arch who underwent successful graft replacement of the innominate artery and reconstruction of the aortic arch under deep hypothermic circulatory arrest with retrograde cerebral perfusion.

Clinical Summary

A 46-year-old woman was brought to our trauma bay intubated with stable hemodynamics after a head-on motor vehicle collision. A chest x-ray film showed a widened mediastinum. A subsequent computed tomographic scan of the chest and aortogram revealed a contained traumatic pseudoaneurysm of the innominate artery in the setting of bovine arch anatomy (Figure 1). The patient also had a grade III liver laceration, bilateral rib fractures leading to bilateral pneumothoraces, and multiple lower extremity orthopedic injuries. She was taken on an emergency basis to the operating room. An external fixation device was placed to stabilize the right femur and to prevent further blood loss in that compartment. A baseline focused abdominal sonographic exam for trauma was then performed and repeated every 30 minutes to exclude the presence of intraperitoneal hemorrhage from the liver laceration in the setting of heparinization for cardiopulmonary bypass. A median sternotomy was performed, and cardiopulmonary bypass was initiated via ascending aortic and right atrial cannulation. Circulatory arrest with retrograde cerebral perfusion at a flow rate of 250 to 350 mL/min and a central venous pressure of 21 to 23 mm Hg was initiated at a core bladder temperature of 15°C (Figure 2, A). One gram of methylprednisolone sodium succinate (Solu-Medrol) was

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Received for publication Feb 16, 2004; revisions requested March 2, 2004; revisions received March 8, 2004; accepted for publication March 18, 2004.

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J Thorac Cardiovasc Surg 2004;128:632-4

0022-5223/\$30.00

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doi:10.1016/j.jtcvs.2004.03.006