Abstract.—A patient with suspicion of a neuroendocrine tumor of the pancreas underwent a somatostatin receptor scintigraphy using $^{111}$In-Pentetreotide. $^{111}$In-pentetreotide scintigraphy showed discrete uptake of the radiotracer in the head of the pancreas and focal uptake in the right upper thyroid lobe. Tracer uptake in the 24h planar image was higher compared to the 4h image, and decreased after 48 hours. Normal thyroid tissue and thyroid disorders, such as cancers, Hashimoto's thyroiditis, and adenomas often show increased uptake of $^{111}$In-pentetreotide resulting in a possible false positive interpretation in patients with neuroendocrine tumor. Adding a 48h planar image might contribute to the differential diagnosis between benign or malignant lesions, as in the present case where the uptake decreased in an adenoma after 48 hours.

KEY WORDS: $^{111}$In-Pentetreotide, thyroid adenoma.

INTRODUCTION

$^{111}$In-pentetreotide is used for the evaluation and therapy planning of somatostatin receptor positive neuroendocrine tumors and their metastases. In thyroid benign disorders, such as Grave’s disease and ophthalmopathy, Hashimoto and De Quervain thyroiditis, nodular goiter, toxic adenoma as well as malignant tumors, such as papillary, follicular, anaplastic, and medullary thyroid carcinoma, and non-functional metastases of differentiated thyroid carcinoma uptake of $^{111}$In-pentetreotide was observed 1-3. Recently, $^{111}$In-pentetreotide accumulation in a thyroid gland mimicking a metastasis of a previously operated, renal-cell carcinoma in a patient with multiple endocrinological neoplasms was published 4. $^{111}$In-pentetreotide uptake was observed in normal functioning colloidal thyroid nodules, multinodular, nodular, colloidal nodule with chronic thyroiditis, cellular colloid nodule, and in endemic goiter 1,2,5-7. Accumulation of $^{111}$In-pentetreotide in various tissues and organs such as pituitary gland, spleen, liver, kidney and urinary bladder, in colon, sarcoidosis, tuberculosis, ventral hernia, parapelvic renal cyst, granuloma etc. was also reported 1,3.

CASE REPORT

A 54 year-old men with the suspicion of a neuroendocrine tumor of the pancreas was referred to the Department of Nuclear Medicine. The patient underwent a somatostatin receptor scintigraphy after injection of 200MBq of $^{111}$In-Pentetreotide. Four and 24 hour post injection thoracic and abdominal planar images and a Single Photon Emission Computed Tomography (SPECT) study were acquired. $^{111}$In-pentetreotide scintigraphy showed discrete uptake of the radiotracer in the head of the pancreas (fig. 1) and focal uptake in...
the right upper thyroid lobe (fig. 2). $^{111}$In-pentetreotide uptake in the 24h planar image was higher compared to the 4h image, and decreased after 48 hours. Thyroid ultrasonography revealed a $27 \times 14 \times 18$ mm sized iso-echoic homogenous thyroid lesion of the right thyroid lobe with good vascularization in the Duplex scan, and a second, echopenic nodule with a diameter of 5 mm located below this lesion (fig. 3). On $^{99m}$Tc-pertechnetate scintigraphy (fig. 4), the lesion was a cold lesion suggesting a possible malignant tumor suggestive for a metastasis of the neuroendocrine tumor. The patients thyroid hormone tests were normal. The tumor was removed surgically. The histopathological diagnosis was a thyroglobulin-positive follicular thyroid adenoma. The lesion was negative for serotonin or chromogranin A.

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FIG. 1.—24 h. abdominal Single Photon Emission Computed Tomography revealed a positive $^{111}$In-pentetreotide uptake in the region of pancreas-head (arrow).

FIG. 2.—24 h. (A) and 48 h. (B) planar $^{111}$In-Pentetreotide static images show the tracer accumulation in follicular adenoma in the upper thyroid pole (arrows).

FIG. 3.—Patients thyroid ultrasonography revealed a upper side located follicular adenoma (arrow) and the second hypo-echoic nodule (arrow head).

FIG. 4.—$^{99m}$Tc-Pertechnetate thyroid scan shows the cold adenoma located on the upper pole of the right thyroid (arrow head).
DISCUSSION

According to the uptake of octreotide in various thyroid lesions such as C-cell (medullary thyroid) carcinoma, activated lymphocytic infiltration (Hashimoto’s thyroiditis, Grave’s disease)12, it is concluded that the presence of somatostatin receptors in these cells is responsible for octreotide uptake. This is supported by in vitro and in vivo studies on the effect of somatostatin on the thyroid gland. Ahren et al10 found a blocking effect of somatostatin after systemic administration of thyroid hormones induced by injection of TSH in humans. An inhibiting effect (being more pronounced in neoplastic thyroid tissue) of somatostatin on basal and TSH-stimulated adenylate cyclase activity in normal and neoplastic thyroid tissue was reported by Sipersstein et al10. In experimental studies it was shown that somatostatin inhibits the growth of thyroid cells11, DNA synthesis in thyroid cells12, and the proliferation of thyroid cell lines13. However, Hoelting et al14 reported that octreotide has a stimulatory effect at low concentrations and an inhibitory effect at high concentrations regarding the growth and invasion of follicular thyroid cell lines. This was not observed in animals.

Recently, high expression of mRNA for the somatostatin receptor subtype 3 (SSTR3) and SSTR5 and weak expression of mRNA for SSTR1 and SSTR2 was reported in normal thyroid tissue15. Although the expression of mRNA of SSTR does not always accurately reflect the level or the presence of the SSTR in thyroid cells, the positive uptake of octreotide in benign and malignant thyroid tissues indicates the presence of SSTRs in thyroid cells. Additionally, it may be possible that octreotide uptake in activated lymphocytic infiltration does contribute to the octreotide uptake in differentiated thyroid carcinoma, autoimmune thyroiditis and Grave’s disease. In Hurthle cell carcinoma, mainly the SSTR2 expression, and in follicular adenoma, papillary and follicular thyroid carcinoma SSTR1, SSTR3, SSTR4 and SSTR5 expression was found14. In normal parafollicular C-cells and medullary thyroid carcinoma, all subtypes of somatostatin receptor subtypes are responsible for the uptake of 111In-pentetreotide as in the present adenoma the uptake decreased after 48 hours.

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REFERENCES


