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ABSTRACTS OF SHORT PAPERS

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Effects of somatostatin on calcitonin and ectopic ACTH release in a patient with medullary thyroid carcinoma *

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The association of a calcitonin-producing tumor with ectopic ACTH production is rare.— Although the importance of somatostatin (SRIF) in modulating the secretion of a variety of polypeptide hormones has been amply demonstrated, there is so far no information about the influence of SRIF on calcitonin (CT) and ectopic ACTH release.

Therefore we studied a 38-year-old man who had two weeks prior to our investigations undergone total resection of his goiter because of medullary thyroid carcinoma (MCT). He complained about rapid weight loss, diffuse abdominal pain, dyspnea, profuse diarrhea and muscular cramps. Blood pressure was elevated and a nonregional spread of the MTC in the liver and in a variety of lymph nodes diagnosed. Although immediate combined chemotherapy was started, the patient died two months later due to massive visceral dissemination of the tumor. A postmortem examination was refused.

The main *laboratory findings* postoperatively were the following: The mean serum CT-levels were extremely elevated (about 40 ng/ml). Serum calcium was low normal (4.5 mEq/l); serum phosphorus was normal. Serum K^+: 2.4 mEq/l and Na^+: 148 mEq/l; urinary excretion in 24 h for K^+ was 90 mEq and for Na^+ – 60 mEq. The arterial blood gases (mm Hg) were: pO_2 66, pCO_2 53; pH 7.57; base excess +22. Basal serum cortisol was 86 µg/100 ml, after exogenous ACTH 67 µg/100 ml and after dexamethasone (2 mg) 64 µg/100 ml. Plasma renin activity was completely suppressed due to a tenfold increase of serum corticosterone (13.4 µg/100 ml) with increased urinary excretion of tetrahydro-DOC glucuronide (Dr. *Vecsei*, Heidelberg). Urinary aldosterone excretion, however, was very low. The basal plasma ACTH levels were markedly elevated (initially around 1 ng/ml, increasing within 8 weeks up to 20 ng/ml), both with radioimmunoassay and with bioassay. There was no decrease of plasma ACTH under 8 mg dexamethasone for two days. The common association of MCT with pheochromocytoma could be excluded in our patient by the normal values of metanephrine and vanillylmandelic acid. Low potassium and glucocorticoid excess led to an overt diabetes mellitus with hypoinsulinemia. An additional overproduction of vaso-inhibitory peptide (VIP) could be excluded by normal serum VIP values (Dr. *Bloom*, London), although profuse diarrhea, low potassium, low gastric acid output after pentagastrin stimulation and elevated basal gastrin values (175 pg/ml) were initially thought to be due to a Verner-Morrison syndrome. Increased serotonin production as cause for the gastrointestinal symptoms could also not be shown.

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Time min	Calcitonin ng/ml	ACTH ng/ml	Insulin µU/ml	Prolactin µU/ml	
– 15	41	—	18	163	before SRIF
0	48	20.48	19	129	
15	13	17.25	7	1202	during SRIF
30	17	4.93	9	1297	
60	35	2.12	12	915	
90	80	2.60	9	729	
120	96	1.53	14	557	
150	81	2.54	26	556	after SRIF
180	33	1.23	31	486	

The *influence* of *SRIF* on calcitonin and ACTH release was tested using 250 μg SRIF (i.v.) as a bolus followed by a two-hour infusion with 500 μg. The effectiveness of the inhibitory action of SRIF could be demonstrated by the insulin release pattern as shown in the Table. CT decreased initially, followed by a marked increase during SRIF infusion. ACTH could be markedly suppressed, followed by a slight rebound after removal of SRIF. There was a so far unexplainable paradoxical rise of prolactin under SRIF. Growth hormone and TSH were already completely suppressed by the glucocorticoid excess and thyroid hormone therapy, respectively.

Conclusion: The inhibitory effect of SRIF is only briefly or not observed on CT secretion, as shown in this patient and further confirmed by the study of a second patient with MCT [1]. In contrast, ectopic ACTH was diminished markedly during the infusion of SRIF. The same phenomenon could be demonstrated in patients with pituitary ACTH excess [2]. Therefore SRIF cannot be regarded as a tool for CT reduction, whereas disorders with ACTH excess might be therapeutically influenced by SRIF.

References

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2. Tyrrell, J. B., Lorenzi, M., Gerich, J. E., and Forsham, P. H.: J. Clin. Endocrinol. Metab. 40 (1975) 1125