A challenging case of paraneoplastic Cushing syndrome-case report- ECE 2015

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INTRODUCTION

Paraneoplastic Cushing syndrome represents 5-10% of all Cushing syndrome and has a severe prognosis due to severe metabolic imbalance, denutrition, associated infections and progression of tumor underlying pathology.

CLINICAL CASE

A 67 years old woman presented with mental confusion, progressive weight loss, severe edema and hypokalemia, without typical features of Cushing or hyperpigmentation. Patient’s behaviour altered in the last 5 months. She was nasty with her daughter, blinking, while diabetes and hypertension aggravated in the last 3 months.

The elective imbalance was severe: K 1.65 mmol/l, inspite of multiple attempts to correct it with 150 mmol/day of KCl on peripheral iv line, 40 mmol/day of KCl orally and 200 mg/day of Liproconsolate, treatment was firstly initiated in the National Institute of Endocrinology “C.I. Parhon”. Patient was transferred in the I.C.U. of Central Military Emergency Hospital “Dr. Carol Davila” for the weekend, in order to obtain a better control using a central i.v. catheter

TREATMENT

We initiated treatment with Ketoconazole 400 mg, day 1, then 600 mg, for 2 days, but with inadequate correction of alkalosis and hypokalemia-pH was 7.54-7.59, BE 5.7-9.8 mmol/l, K 3.3-3.16 mmol/l. The third day patient became septic (MRSA Staffilococcus probably) due to central catheter and intestinal pneumonia- fibrinogen 600 mg/dl, AST 87-160 U/l, ALT 95-121 U/l, GGT 348-385 U/l, total bilirubine 2.44 mg/dl, leucocytes 13400/mm3, granulocytes 8500/mm3. Cortisol levels were 28.3-29.2 mcg/dl and Ketoconazole was increased to 1200 mg/day, also associating Tavanic 500 mg intravenously, then Tegicyleine 100 mg/day. The high values of ALT and AST were due to sepsis and did not increase after doubling of ketoconazole dosage. After 1 day of high dose Ketoconazole, K was 4.7 mmol/l, allowing introduction of Miliperinone 200 mg/day. The seventh day after Miliperinone was introduced, cortisol levels were 18.7 mcg/dl(4.3-8.4), allowing surgery. Due to denutrition, pulmonary sepsis, lack of localisation of tumour-kidney/thymus/leiomyoma, recent syncope, severe brain atrophy with cognitive impairment, we decided to perform left adrenal gland resection.

The adrenal gland resection was difficult due to excessive bleeding and lack of tissue elasticity. Hepatic biopsy showed periporal fibrosis, but no necrosis of hepatocytes, probably due to use of toxic substances at work. Left adrenal was 7/3/1.5 cm in diameter, with focal hemorrhage lumenochimistry-CK7, CK20, CEA, TF, ER-negative, MELAN A positive – suggested diffuse hyperplasia of left adrenal gland.