Stanazolol Abuse: Diagnostic Dilemma in an Adolescent with Persistent Hypoglycemia

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CONTEXT: Multiple endocrine neoplasia (MEN)1 is a rare autosomal dominant disorder with primary hyperparathyroidism, enteropancreatic neuroendocrine tumors and anterior pituitary adenomas.

PATIENT: A 16-yr-old male adolescent followed-up from another center owing to primary epilepsy was referred to our center due to recurrent seizures. Family history was significant for maternal death owing to metastaic adenocarcinoma of the lung, paternal history of low-grade liposarcomas and nephrolitiasis. Physical examination was non-yielding, with a well-built, muscular adolescent. Biochemical tests revealed venous blood glucose: 24 mg/dl, cortisol: 18 µg/dl, growth hormone: 13 ng/ml, insulin: 8 µU/ml, C-peptide: 1.34 ng/ml; consistent with hyperinsulinism. Anti-insulin antibodies were negative. Serum liver transaminases [Alanine amino transferase (349U/L),Aspartate aminotransferase (158U/L)] and creatine kinase (834 U/l) were high. Metabolic work-up for inborn errors of metabolism, were normal. Hepatic ultrasound for a congenital portal-hepatic shunt was also non-yielding. Thin-slice pancreas BT and panreas MRI were normal. On further questioning, the patient admitted receiving stanazolol (Winstrol) to strengthen his muscles. Liver transaminases and CK levels subsided back to normal ranges within two to three weeks after cessation of stanozolol. Hypoglycemia did not recur on diazoxide (200 mg/day) therapy. Biochemical evaluation also revealed serum Ca: 11.5 mg/dl, P: 2.6 mg/dl, ALP: 520 IU/l, PTH: 320 pg/ml, 25OH-vitamin D:33 ng/ml and 24-h urinary calcium: 4.2 mg/kg/day. Parathyroid scintigraphy revealed an adenoma in the inferior right parathyroid gland (Fig.1).

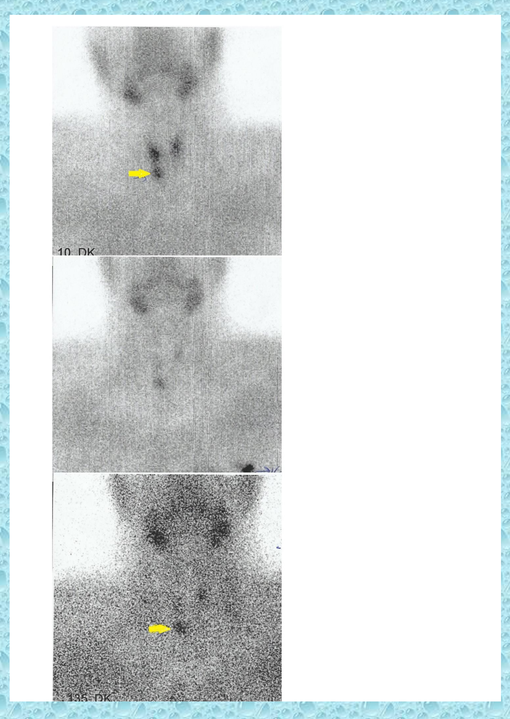

Figure 1. Right inferior parathyroid adenoma (yellow arrow)

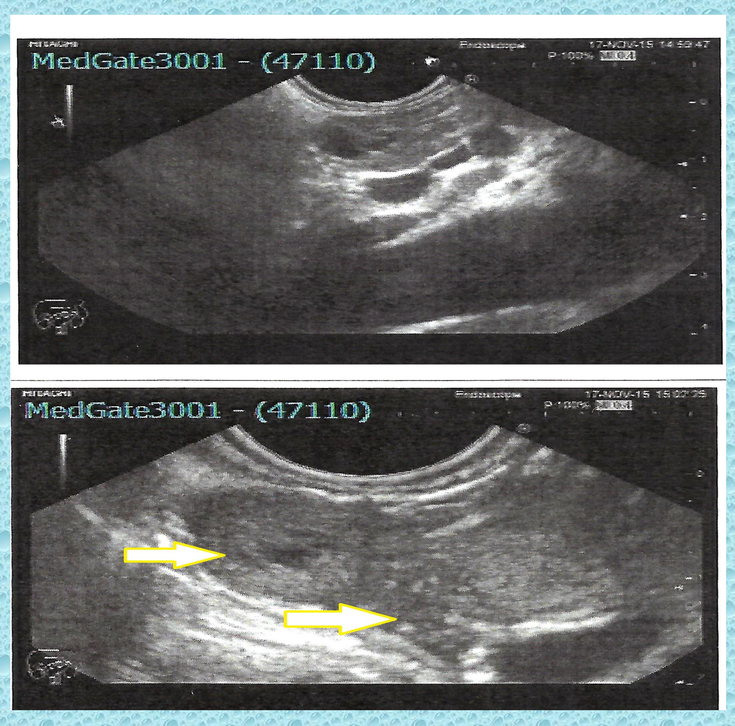

Figure 2.Lesions in the corpus and tail of the pancreas shown by yellow arrow.

Upper gastrointestinal endoscopic ultrasound revealed two lesions in the corpus and tail of the pancreas. (Fig 2). The biopsies of the lesions were consistent with grade 1 neuroendocrine tumor according to WHO classification. Serum prolactin levels and pituitary MRI were normal. DNA sequence analysis of mennin gene revealed a "pW183S" heterozygous deletion. The same mutation was found in his father and his 14-year-old brother, who had asymptomatic primary hyperparathyroidism. Distal pancreatectomy revealed two adenomatous masses (1.1 cm and 1.4 cm); with an insulinoma and a non-secretory adenoma.

CONCLUSION: Our case emphasizes the need to question drug abuse in adolescents presenting to clinics, particularly when the diagnosis is murky. This is the first pediatric case of MEN1 with two synchronous pancreatic adenomas. The genetic confirmation of the clinical diagnosis of MEN1 is mandatory to assess other family members for the presence of the mutation, for regular follow-up for potential endocrine problems and for avoiding unnecessary work-up due to "phenocopy".

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