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A CASE REPORT OF DIABETES IN A 13-YEAR OLD BOY TREATED FOR NEPHROTIC SYNDROME

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Medication-induced diabetes mellitus (MID) is an iatrogenic glucose metabolism disorder. The prevalence of MID in pediatric population is unclear. By analyzing a few available evidences, we can estimate that the MID occurs less often in children than the diabetes mellitus type 2 (DM2). MID is a heterogenic group of diabetes, which can be subdivided by the main pathway. There are three main pathways (decreased insulin secretion, increased insulin resistance and increased glucose influx). The glucose metabolism disturbances may be normalized or became permanent despite the drug discontinuation. The risk of diabetes persistence is related to factors such as duration of treatment, prescribed medication and body mass index.

A 13-year old boy with a 10-year history of submicroscopic nephrotic syndrome, was diagnosed with diabetes during the check-up visit after treatment modification.

From the beginning he presented with steroid dependent nephrotic syndrome with frequent relapses, what required the introduction of immunosuppression. His medical history also included overweight, hypertension, dyslipidemia, hypothyroidism, and familial history of DM2. On admission he showed cushingoid appearance and complained of dry mouth which was not accompanied by polyuria or polydipsia.

Blood tests showed high fasting (143 mg/dL) and post-prandial (364 mg/dL) glucose levels and an abnormal glycated hemoglobin level (HbA1c = 10.2%) Results of blood gases were in normal ranges. Urinalysis showed glucosuria without ketonuria.

Diabetes was confirmed by fasting glucose re-test next day. Intensive insulin therapy was initially introduced due to HbA1c over the 8,5%. Extended laboratory analyses showed normal concentration of fasting and post-prandial C-peptide, negative islet autoantibodies (anti-islet cell antibody, anti-glutamate decarboxylase antibody and anti-insulinoma associated protein 2 antibody). Those findings prompted us to consider DM2 or MID caused by chronic steroid/tacrolimus treatment.

The patient started the treatment with a low daily dose of insulin per kilogram, which decreased after full steroid withdrawal (from 0,31U/kg to 0,24U/kg). The HbA1c level also decreased from 10,2% at the beginning to 5,5% and remained at similar level. Because he was constantly on diabetogenic tacrolimus therapy, the MID could not be excluded. For this reason, pharmacotherapy of diabetes remained insulin, because there is no high-quality of evidence recommending the treatment of MID other than subcutaneous injections of insulin.

The attempt to change medications to treat diabetes may be possible after the discontinuation of tacrolimus, when the diagnosis of MID could be excluded.

The lack of high-quality evidence on diagnosing and treating the MID in pediatric population, may lead to overprescribing the insulin in cases in which oral antidiabetics could be used, which should lower the risk of hypoglycemia and weight gain.

Further research is needed to develop guidelines that provide the procedures for more accurate differentiation of diabetes in patients treated with diabetogenic medications, and an algorithm of the treatment based on the most possible pathway of hyperglycemia.