NEPHROTIC SYNDROME AND TYPE 1 DIABETES: A THERAPEUTIC APPROACH

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INTRODUCTION

Paediatric nephrotic syndrome has an estimated incidence of 2 per 100,000 children per year and type 1 diabetes had a reported incidence of 9.5 per 100,000 habitants in Portugal (2018 data). To the best of our knowledge, the simultaneous occurrence of idiopathic nephrotic syndrome and type 1 diabetes is rare – we found 19 published cases in paediatric age worldwide.

Diabetes management under glucocorticoid treatment is a challenge and new technologies can be an ally.

The aim of this case is not only to report a new case of nephrotic syndrome and type 1 diabetes mellitus, but also to make known a different approach concerning patients with diabetes mellitus insulin-treated under glucocorticoid treatment.

CLINICAL CASE

5-year-old boy, Caucasian

Past Medical History: 2-year history of **idiopathic nephrotic syndrome** (2 relapses)

Family History: irrelevant

Emergency Department: admitted with **palpebral oedema and** home detected **proteinuria** (300 mg/dL) in urine dipstick test since febrile rhinopharyngitis started, 4 days before. Other symptoms were denied, such as polyuria, polydipsia, weight loss and polyphagia. Unremarkable physical examination, besides the palpebral oedema

Urinalysis: **protein** (300 mg/dL) and **glycose** (2000 mg/dL) **by dipstick**
Blood evaluation: glucose 267 mg/dL, **cetonemia 1.2 mmol/L**, **HbA1c 8.6%**, insulin 2.0 pmol/L, **positive Anti-GAD2 antibodies** and blood gases without alterations

Diagnosis:

1. **Relapsing nephrotic syndrome (3rd)**
2. **Inaugural type 1 diabetes without ketoacidosis**

Treatment:
Oral **prednisolone** 60 mg/m^2/day and **multiple insulin injection** therapy

Clinical course:

- During hospital stay **glycaemia control was difficult** and he was discharged with total daily dose of insulin (TDDI) of 1.90 IU/kg
- When he started to take **prednisolone every other day**, our option was to **diminish 10% of the basal insulin** in the **days without corticoid**, with satisfactory results (HbA1c 7.6%)
- Eight months after the diagnosis, **continuous subcutaneous insulin infusion (CSII)** was initiated, achieving a **HbA1c** of **6.5%** with a **TDDI** of **0.68 IU/kg**, without glucocorticoid treatment
- Since CSII, is reported a **better glycaemic control in relapses**, achieved using temporary basal rates (HbA1c 7.1-7.3%). To note that **TDDI increases** to a maximum of 1.17 IU/kg under glucocorticoid treatment
- He is now on a **slow glucocorticoid tapering** (7th relapse) and he will be maintained on prednisolone on alternate days at least during 12 months
- Additional study revealed human leucocyte antigen (**HLA**) genotyping **DQA1*0201, *0501** and **DQB1*02,*02**, both associated with DQ2 heterodimer

CONCLUSIONS

It is challenging to control hyperglycaemia during glucocorticoid treatment. Previous work have been limited to discuss treatment approaches for better glycaemic control. Goldman et al. report hyperglycaemia control by maintaining a regular glucocorticoid dose, rather than alternate days tapering. We have obtained better glycaemia control with CSII compared to multiple insulin injections in our patient, besides stating our approach during corticosteroid progressive weaning when in multiple insulin injection therapy. As our patient is now on a long course of glucocorticoid treatment, we are working to program and adjust two basal rates (one for prednisolone and other for non-prednisolone day) on CSII, alternatively to the use of temporary rates.

Type 1 Diabetes has a genetic background and HLA characterization can help set up a prognosis when the result is associated with other autoimmune diseases. HLA result of the presented patient has been associated with a genetic predisposition to type 1 diabetes but was not described in other cases of patients with both nephrotic syndrome and type 1 diabetes.

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