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CLINICAL CASE REPORTS

New-onset iatrogenic diabetes in a teenager with ulcerative colitis – an additional challenge

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ABSTRACT

Introduction: Glucocorticoids (GC) are long-established agents for the induction of remission in inflammatory bowel disease (IBD). Hyperglycemia is a known complication of GC, and this risk increases when combined with immunosuppressive treatments, particularly with mycophenolate mofetil (MMF). In patients with IBD, there is insufficient information regarding the adverse outcomes of using both therapies concerning hyperglycemia and new-onset diabetes.

Case Report: A 13-year-old girl with ulcerative colitis (UC), two years after diagnosis, started MMF along with GC. She developed polydipsia and polyuria one week earlier. Follow-up laboratory workup revealed hyperglycemia (696 mg/dL), without ketosis or acidosis, a baseline HbA1c of 7.4%. Insulin treatment was initiated, MMF was suspended, and GC weaning was attempted again. New-onset diabetes complicated the management of a previously diagnosed depressive and anxiety disorder. On follow-up, insulin needs progressively decreased, and insulin was discontinued after five months.

Conclusion: The concomitant use of GC and MMF might have posed a cumulative risk. This case highlights the importance of a multidisciplinary team when establishing a healthcare plan for an adolescent with multifactorial chronic disease burden.

Keywords: diabetes; glucocorticoids; immunosuppression; inflammatory bowel disease; mycophenolate mofetil

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INTRODUCTION

Comorbid diseases in ulcerative colitis (UC) include several immune-mediated conditions, among which type 1 diabetes mellitus (T1DM) is one of the most frequently associated. [1] A large case-control study involving 488 children with UC showed that it is associated with a higher prevalence of T1DM compared to controls (OR = 2.7, 95% CI: 1.1-6.6), with an overall prevalence of 2049 cases per 100,000 children.[2] To confirm the association between diabetes and steroid therapy, it is necessary to rule out an autoimmune background. The presence of autoantibodies to GAD and anti-α tyrosine phosphatase (IA-2) confirms the diagnosis of T1DM.[10]

Glucocorticoids (GC) are long-established and widely used agents for the induction of remission in inflammatory bowel disease (IBD). They are the first-line agents for induction in moderate to severe UC and are also used in IBD patients admitted to the hospital with severe disease relapses.[3,4] Steroids are well known for causing drug-induced diabetes. While the effect of steroids on blood sugar regulation is well-documented, it is essential to consider other medications in these cases as well.

In organ transplant recipients, immunosuppressive therapies have been associated with a markedly increased risk of hyperglycemia and new-onset diabetes. [6] This risk is heightened when GC are combined with other immunosuppressants, particularly mycophenolate mofetil (MMF).[7] This combination has the highest hyperglycemia rate (81.3%) with a significant reporting odds ratio value of 8.38 (95% CI 2.33-30.16).[8] The combined use of GC and other immunosuppressive therapies may contribute to the high metabolic and hyperglycemic adverse effects observed in these patients.[7,8] In patients with IBD, there is insufficient information regarding the adverse outcomes of using GC and MMF in relation to hyperglycemia and new-onset diabetes.

CASE REPORT

A 13-year-old girl was evaluated at the Pediatric Gastroenterology outpatient clinic with UC and sclerosing cholangitis for two years (diagnosed at the age of 11), both evolving with a pattern of severe and relapsing disease. Disease control had been particularly challenging, with little improvement achieved using prednisolone (PDN), infliximab, or azathioprine. At diagnosis, an autoimmunity workup was performed, including tests for anti-pancreatic antibodies and C-peptide levels. Attempts to taper corticosteroids led to multiple relapses, and the introduction of corticosteroid-sparing drugs (namely infliximab and azathioprine) was unsuccessful, as the patient continued to require high doses of PDN. Hyperglycemia screening was conducted approximately every 3 months, since diagnosis.

Two years after diagnosis, MMF (500 mg twice daily) was initiated in combination with PDN (0.5 mg/kg/day). One month after starting MMF, follow-up laboratory workup revealed hyponatremia (126 mmol/L), marked hyperglycemia (696 mg/dL), with corrected sodium level for hyperglycemia within normal range (137.9 mmol/L). Capillary ketonemia was negative (0.1 mmol/L) and there was no metabolic acidosis (pH 7.39, HCO3 24.3 mEq/L). Glucosuria was high, above 1000 mg/dL. When questioned, the patient reported mild symptoms of polyuria and polydipsia, which had previously gone unnoticed.

She was hemodynamically stable, without clinically evident dehydration. She had lost 2 kg in one month (6% of her body weight). Anthropometric data were: height 134.5 cm (z-score -2.6), weight 32 kg (z-score -1.7), body mass index (BMI) 17.7 kg/m2 (z-score -0.4). Her Tanner stage was II.

Initial HbA1c was 7.4 %, and both C-peptide (2.0 ng/mL, normal range: 0.9-7.1) and insulin levels (4.2 mUI/L, normal range: 1.9-23) were normal. Anti-protein tyrosine phosphatase islet antigen-2, anti-insulin and anti-GAD autoantibodies were negative. Antibodies to Langerhans cells and zinc transporter were not tested.

She was admitted to the ward to achieve glycemic control and intensive diabetes education. Intravenous fluid therapy was started with isotonic saline solution 10 mL/ kg over one hour, followed by subcutaneous insulin in a basal-bolus regimen. The total daily dose was titrated to 0.9 units/Kg/day (40% glargine, with insulin aspart according to glycemia and carbohydrate counting), with adequate glycemic profile. On the second day of admission, MMF was suspended and PDN weaning was initiated to the lowest dose of 2.5 mg daily (0.1 mg/Kg/day). She was discharged after five days. Three weeks later, she was started on oral vancomycin 500 mg three times a day, with progressively significant improvement in gastrointestinal symptoms. The stool culture, Yersinia test, Clostridium difficile, stool virus test (astrovirus, adenovirus, norovirus, rotavirus and enterovirus), stool parasitology test and Giardia test were all negative.

Soon after the diabetes diagnosis, the teenager developed a reactive depressive and anxiety disorder, and she was followed in Pediatric Psychiatry consultations and undergoing psychotherapy. In this setting, the diabetes education process, guidance, and lifestyle counseling were very challenging, and she had difficulty in coping with the disease, especially with insulin administration. Anxiety symptoms, including trichotillomania with severe eyebrow picking, had a significant impact on her daily life. The pharmacological approach began with a benzodiazepine (lorazepam 1 mg/day) and four months after diagnosis, a mood stabilizer (oxcarbazepine 300 mg/day) was added. The patient always complied with the treatment for UC, but her adherence to psychiatric medication was poor, and she only took the medication for a few months. The entire process involved a multidisciplinary team approach, with a pediatric psychiatrist, gastroenterologist, and endocrinologist, as well as specialized nursing educators and a nutritionist.

On follow-up, insulin needs progressively decreased, and basal insulin was stopped after two months. Five months after

diabetes onset, continuous glucose monitoring (CGM) showed 90-100% time in range (70-180 mg/dL), without hypoglycemia or other acute complications, with a total daily insulin dose of 0.2 units/Kg/day. Bolus insulin was then also suspended, and after two months without insulin therapy, HbA1c was 5.3%. Lowering the burden of capillary blood tests, CGM use, and insulin shots was helpful in improving depressive and anxiety symptoms, allowing for progressive weaning of oxcarbazepine.

DISCUSSION

The differential diagnosis of new-onset diabetes in an adolescent with UC is challenging. Indeed, there were overlapping risk factors related to the underlying condition and potential iatrogenic effects.

The hypothesis of T1DM associated with UC as a coexisting autoimmune disease became less likely because pancreatic autoimmunity was negative and baseline insulin and C-peptide levels were normal. The fact that insulin requirements decreased after MMF withdrawal, simultaneously with the weaning of the GC strongly suggests that this was a case of iatrogenic diabetes. In this case, the prognosis is good, although the likelihood of developing T1DM is higher when compared to the general population.

CONCLUSIONS

The concomitant use of corticosteroid therapy (PDN) along with an immunosuppressive agent (MMF) may have had a additive risk effect. Priority should be given, when feasible, to corticosteroid-sparing therapies. In the setting of combined immunosuppressive therapeutics, it is essential to accurately monitor blood glucose levels and investigate signs and symptoms of hyperglycemia as part of routine medical practice.[5]

This case highlights the importance of a multidisciplinary team approach when establishing a healthcare plan for adolescents with a complex, multifactorial chronic disease burden.

AUTHORSHIP

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