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Autoimmune Hypoglycemia in a Patient with Slow Type 1 Diabetes: A Rare Cause of Hypoglycemia

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Abstract **Case Report**

Glycemic instability which is common among patients treated with insulin is incapacitating because of the repeated episodes of hypoglycemia that are often related to dietetic errors, insulin overdose or excessive physical effort but rarely to an auto-immune etiology that is due to the presence in the serum of the patient of anti-insulin or anti receiver-insulin antibodies. We report the case report of a 58 years old female patient, Who is a Slow type 1 diabetes carrier, and has been on premixed human insulin for 4 years, the patient presented repeated episodes of hypoglycemia : both nocturnal and late postprandial, The rate of anti-insulin antibodies was positive higher than 50 IU / ml (NR <0.4). The patient was started on corticosteroids containing prednisolone (1mg/kg/j) and insulin analogue. The course was marked by the disappearance of the hypoglycemic episodes.

Keywords: hypoglycemia; anti-insulin antibodies; slow type 1 diabetes.

INTRODUCTION

Hypoglycemic events are common in insulin treated diabetic patients, disabling and sometimes severe life-threatening, most often explained by dietary errors, insulin overdose or excessive physical exertion.

Hypoglycemia is rarely due to an auto-immune etiology which is the presence in the serum of the patient of anti-insulin or anti receiver- insulin antibodies.

The present case is a slow type 1 diabetic patient in whom the autoimmune origin of repeated hypoglycemia was found.

CASE

A 58 years old female patient, Who is a Slow type 1 diabetes carrier, and has been on premixed human insulin for 4 years, the patient presented repeated episodes of hypoglycemia : both nocturnal and late postprandial. she also denied skipping meals or having any drug intake that is likely to cause hypoglycaemia, the search for signs of gastroparesis was negative, and no family history of Autoimmune disease was found, Physical Examination revealed a conscious patient, glucose blood level was 0,6 g/l, No lipodystrophy or lower limb edema were found, Biochemical investigations revealed normal liver and kidney function, Cortisol level at 8 AM was 15 µg / dl; after stimulation (Synacthen test) : 26 µg / dl Anti-transglutaminase IgA antibody detection was negative with absence of IgA deficiency), High levels of anti-insulin antibodies was discovered; it returned higher than 50 IU / ml, The diagnosis of autoimmune hypoglycemia was confirmed by the high level of the Anti-insulin antibody. The indication for corticosteroid therapy was made alongside the use of the insulin analogue.

DISCUSSION

In 1956, Berson *et al*. demonstrated that insulintreated patients possessed insulin-binding immunoglobulins. It was also found that the use of bovine or porcine derived insulin induced IA in more than 95% of diabetic patients receiving insulin treatment [1, 2].

Despite the fact that the advance in insulin manufacturing technology such as preparations with high purity and use of recombinant human insulin had markedly reduced the immunological reaction against insulin injection; there still are patients that develop IA against human insulin preparation [1].

The mechanism of the immunogenicity against human insulin preparations is still unknown. Although

Genetic factors, purity of insulin preparation, or species of insulin may influence and promote antibody production [2, 3].

However, there is less immunogenicity with new insulins, especially analogues, but cross-reactivities can be seen in patients previously treated with animal insulins [4].

The presence of anti-insulin antibody in high titer affects the body's response to insulin by capturing insulin molecules, hence delaying its initial action. This therefore induces post-prandial hyperglycaemia and causes hypoglycaemia in the post-absorptive state or at night by prolonging the release of insulin from the insulin-antibody complexes, making it hard to maintain adequate glycemic control [5-7].

The symptomatology that our patient presented can be explained by the presence of anti-insulin antibodies which capture insulin after the injection and then release it in an uncontrolled way, explaining severe hypoglycaemia in late postprandial time.

The use of insulin analogues may be effective. Corticosteroid therapy based on prednisolone at 1 mg / kg / day can be proposed as a second alternative, divided into several daily doses over a few weeks. The use of plasmapheresis and cyclophosphamides can, however, is reserved for resistant forms [8-10].

CONCLUSION

Autoimmune hypoglycemia is considered a rare etiology of hypoglycemia in the diabetic patient, the role of anti-insulin antibodies is suggested in the presence of postprandial hyperglycaemia and late hypoglycaemia.

The presence of a high level of antibodies confirms this rare etiology of hypoglycemia. Human insulin remains as an immunogenic product that induces the secretion of specific antibodies, especially among patients with autoimmune diseases. Steroid therapy might be useful for the treatment of brittle diabetes, if the patient had high titer of IA with high binding capacity to insulin.

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