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The autoimmune hypothesis for acute bilateral cataract in type 1 diabetes

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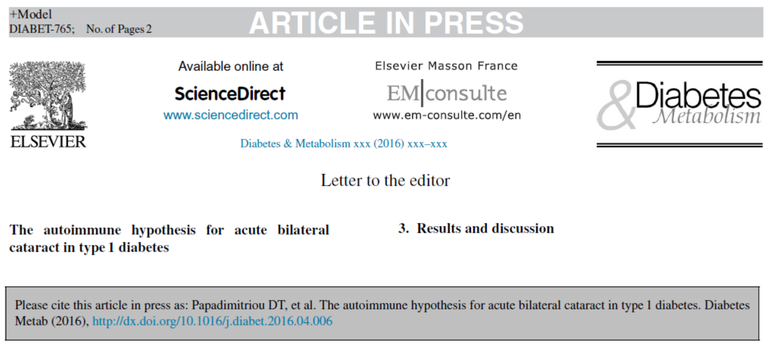

No disclosures

Background:

Cataract as a chronic complication of diabetes in children is well established in the literature, the risk factors being: duration of diabetic symptoms before diagnosis, diabetic ketoacidosis, poor metabolic control, elevated HbA1c, genetic factors and administration of glucocorticoids. However, rare cases of acute bilateral cataract have been reported, all of them happening relatively shortly after diagnosis in T1DM. While the pathophysiology of this phenomenon remains unclear - as a lot of different theories proposed so far fail to explain adequately the acuteness of bilateral cataract formation - there have also been reports of acute bilateral cataract as the first presentation of T1DM .

Objective and hypotheses:

We have recently published a case of acute bilateral cataract in a pediatric patient with T1DM manifested 3 months after presentation with severe diabetic ketoacidosis and initiation of insulin treatment. Although ophthalmologic examination was normal while in the Intensive Care Unit, the 17-year-old boy, 3 months later under optimal metabolic control (HbA1c 6% and 95% of blood glucose measurements falling into the range between 70-150 mg/dl) presented a complete loss of vision within 6 days with a visual acuity of hand motion only. On examination, in psychological shock, he had bilateral milky white cataracts with no view of the fundus in either eye.

Results and Discussion:

The cataract being completely liquefied; no lens tissue could be sent to immunopathology in either of the two operations. The only remarkable change to his biology and autoimmune status were the previously negative at presentation of T1DM, now clearly elevated Insulin autoantibodies (IAA): 1.4 (<1.1).

One of the most accepted theories for cataract formation in the diabetic patient is the osmotic stress induced oxidative damage.

Our "autoimmune hypothesis" however proposes that the timing of the cataract formation – usually few weeks or months after the onset of insulin treatment, as in our case- suggests an autoimmune response. More specifically, the IAA became positive within 3 months after the beginning of insulin treatment and this period coincides with the cataract formation. Unfortunately, we were unable to measure the IRA, which might have strengthened the autoimmune hypothesis, as decreased immunoreactivity for the insulin receptor has been found in the lens when cataract is detected. Insulin receptor antibodies can act either as agonists or antagonists to the insulin receptor. The insulin receptor antibody has been associated with the inhibition of insulin binding to the insulin receptor leading to accelerated receptor degradation and downregulation. Insulin receptor antibodies may also inhibit insulin clearance resulting in elevated levels of plasma insulin. For these reasons it has been suggested that the most important laboratory test in autoimmune hypoglycemia is the direct assay for the presence of antibodies directed against the insulin receptor or insulin. These two types of autoantibodies can interact with each other, although the consequences of this interaction cannot be projected. It is of major importance that while lens epithelium is not dependent on insulin for glucose uptake, still the insulin receptor is present in normal lens, as well as in lens of diabetics, and disappears only after cataract has developed. **Which raises the question: Is the decrease of the insulin receptors a result of the cataract or the decrease of insulin receptors autoimmune in origin – possibly through the elevation of the IAA and/or IRA - causes the cataract? Knowing that IAA can interfere with the pharmacological action of administered insulin, resulting in insulin autoimmune syndrome, one could hypothesize an intriguing autoimmune hypothesis for acute bilateral cataract in diabetes.**

The timing of the occurrence of acute bilateral cataract in patients with good metabolic control, who do not carry the burden of a chronic diabetic decay, clearly implies an autoimmune mechanism on the grounds of a possible genetic predisposition for cataract formation.

Conclusion:

While previously reported theories fail to explain the acuteness of the phenomenon, there are indications to suggest a possible autoimmune mechanism for acute bilateral cataract in diabetes that warrants further investigation. Elucidation of a possibly autoimmune underlying mechanism could lead in early detection and development of prevention strategies to avoid such a stressful complication.

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