Casos Clínicos: Desafíos gastroenterológicos.

Diabetes y Enfermedad Celíaca?

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Caso Clínico

- JS; Sexo Femenino. FN 09/05/96
- Fecha consulta: 25/01/08 (11a 8m)
- Deriva S. de Diabetes para investigar EC.
- Dg DBT Insulinodependiente: 8 años.
- Estudiada por Endocrinologia (2008) Hipotiroidismo subclínico. Test TRH (+)
- Ac APO (-); Ac Tg (-)
- Peso P 50; Talla 50-75.
- Lantus38 U; Correcciones: Ultrarápida (2xdía)
- Actividad física (+). Hipoglucemias.

Caso Clínico.

- Antecedentes: no fliar de DBT ni EC
- Perinatológicos normales.
- LM 2 meses.
- Fla sin lactosa por diarrea y eczemas (2m)
- 9m Leche entera baja lactosa.
- 2 años dieta Cta.
- Papillas 6 meses; gluten 7 meses.
- Menarca 12 años (con Levotiroxina)

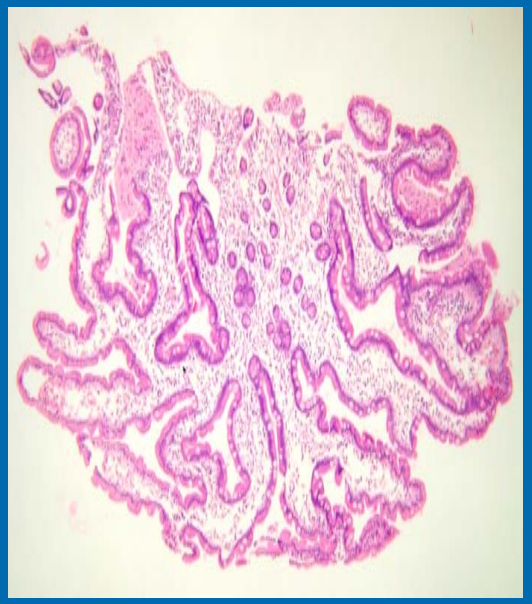
Caso Clínico

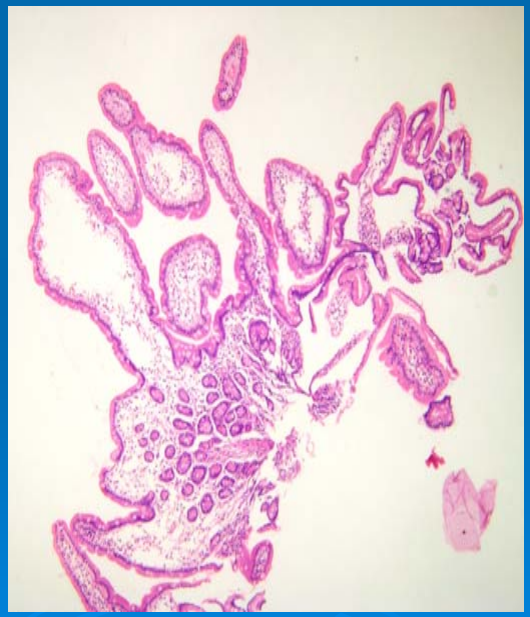
- Deriva S de DBT por Hipoglucemias y Ac AGA (+).
- Ago ’08: IgA 124; IgG 585.
- Ac AGA (A): 120 (+); (G) (-). Reiterados.
- Ac AEM e Ac Atg (AyG) (-).
- Se decide EGC y BID.

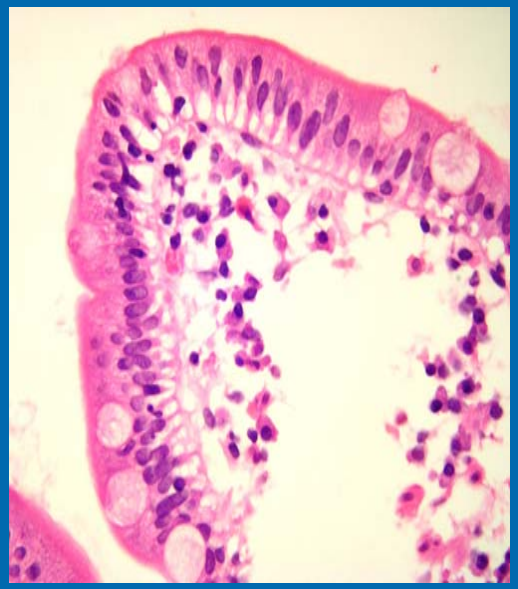
Caso Clínico

- 13/03/09 EGC: Gastropatía antral. Hiperplasia linfoidea duodenal.
- Anatomía Patológica:
- Esofagitis crónica moderada Grado 2
- Estómago sin lesiones a destacar
- Duodenitis con Edema y Eosinófilos en corion. Ensanchamiento focal de Vellosidades por edema. No LIE.

Caso Clínico







Caso Clínico

- 26/03/09
- IgE 222,5 UI
- Rast LV (+) grado 2
- Rast B lactoglobulina (+) grado 1
- Rast Caseína Negativo.
- AC APO (-)
- Dieta hipoalergénica. (S/ LV ni alergenos > por un año).
- Evolucion Dic'10: Insulina 20 U. Estable.
- Negativiza AGA (A); Rast LV (+ débil)

Bibliografía

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- Goh C, Banerjee K. Prevalence of celiac disease in children and adolescents with type 1 diabetes mellitus in a clinic based population. Postgrad Med. 2007;83:132-6.
- Sumnik Z, Kolouskova S, Malcova H, Vavrinec J, Venhacova J, Lebl J, et al. High prevalence of celiac disease in siblings of children with type 1 diabetes. Eur J Pediatr. 2005;164:9-12.

Bibliografía

- Outi Vaarala. Intestinal Immunity and Type 1 Diabetes. *University of Linköping,Sweden. Journal of Pediatric Gastroenterology and Nutrition .* **39:**S732–S733 © June 2004

- In humans, **autoreactive T-cells may originate from**
- **the gut immune system**. For example, T-cells derived
- from the pancreas of a patient with T1D adhered to mucosal
- and pancreatic endothelium (6). Autoreactive
- T-cells from patients with T1D expressed gut-associated
- homing receptor α47-integrin, whereas their tetanus
- toxoid reactive T-cells did not (7). The reports of enhanced
- immune responses to dietary cow-milk proteins
- suggest an increased activation of the gut immune responses
- and dysregulation of oral tolerance in T1D (8).

Bibliografía

- When a large panel of cytokines and
- chemokine receptors was studied, we found that mRNA
- levels of **IL-18 were significantly increased** in biopsy
- samples from children with T1D and **without signs of**
- **celiac disease**. In contrast, the levels of **IL-18 mRNA**
- **were decreased** in biopsy samples from patients with
- **celiac disease**, whereas **increased expression of IFN-**
- **and CD25 was found in celiac disease.** In patients with
- T1D and so-called potent celiac disease (i.e., increased
- number of intraepithelial lymphocytes), increased expression
- of CCR9 and TGF- was found in biopsy
- samples. Our results suggest that the **inflammatory activation**
- **of intestinal immunity related with T1D is a separate**
- **entity from that seen in celiac disease.** Our findings
- of intestinal immune activation in T1D were not restricted
- to the patients with DQ2 genotype, which is the
- most common HLA class II genotype in celiac disease.

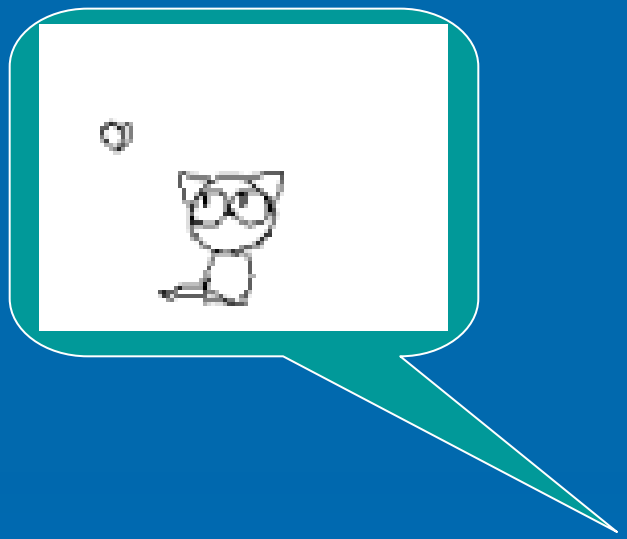
Bibliografía

- Our studies show that infants who have been exposed to cow-milk formulas before the age of three months have higher levels of insulin-binding antibodies and T-cell reactivity to insulin than infants who have been exclusively breast-fed (10,11).

- Both antibody and T-cell response to bovine insulin showed cross-reactive with human insulin. Accordingly, an environmental trigger of insulin-specific immune response in humans is dietary bovine insulin. Bovine insulin differs from human insulin by three amino acids. Immunogenic nature of bovine insulin in humans was recognized when bovine insulin was used for the treatment of diabetic patients and resulted in high levels of insulin-binding antibodies. Since bovine insulin differs from human insulin it can be considered a “modified self-antigen,” which may escape tolerance induced to self-insulin in the thymus.

- **Conclusion: This suggests that the children who develop islet cell autoimmunity may have a failure in tolerance induction to dietary insulin.**

Caso Clínico



Muchas gracias!