Conferencias y Simposios

SIMPOSIO 14: ¿Cuándo comienza la diabetes mellitus tipo 1?

Coordinadora: Dra. Liliana Trifone

Preservación de la célula beta al debut

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Hemos aprendido a lo largo de los años que la diabetes mellitus tipo 1 (DM1) es una enfermedad heterogénea en su presentación y evolución, y que su desarrollo depende de la interrelación de factores, genéticos, inmunológicos y ambientales.

En las etapas del desarrollo de la DM1 vemos que, a partir de la predisposición genética con expresión autoinmune en la célula B (etapa 1) luego aparece la dis glucemia (etapa 2) y la expresión clínica florida de diabetes (etapa 3), pero poco se sabe sobre cambios secuenciales en la secreción de insulina y la homeostasis de la glucosa a través de estas etapas. El Trial Net muestra la variación de péptido C seis meses previos al debut y luego del mismo.

Se conoce que la disfunción de las células β en la DM1 puede preceder al diagnóstico de diabetes en más de 5 años. La mayoría de los pacientes con DM1 de larga duración es micro secretor de insulina, tiene célula Beta funcionante que responde a estímulo, la remisión clínica parcial se correlaciona inversamente con cetoacidosis, directamente con BMI y edad al debut.

El control metabólico óptimo temprano dentro de los 10 primeros días de la enfermedad preserva la secreción de péptido C y disminuye complicaciones a largo plazo.

La hiperglucemia temprana deja secuelas permanentes en SNC tan o más deletéreas que las de la hipoglucemia, preserva célula beta, reduce el riesgo hipoglucemias y las complicaciones microvasculares, también previene deterioro de sustancia blanca gris del cerebro, protegiendo un adecuado neurodesarrollo en los pacientes más pequeños.

ISPAD 2018 sugiere la implementación de un tratamiento intensivo una vez compensado el paciente para lo cual la implementación de los análogos insulínico y de la tecnología han facilitado el camino.

Palabras clave: diabetes; célula beta.

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SYMPOSIUM 14: When does type 1 diabetes mellitus begin?

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Preservation of beta cell at diagnosis

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Through the years, we have learnt that type 1 diabetes is a heterogeneous disease in its presentation, evolution and that its development depends on the interrelationship between genetic, immunologic and environmental factors. During the development stages of type 1 diabetes we can observe the genetic predisposition with autoimmune expression in beta-cell (stage 1), the appearance of dysglycemia (stage 2) and it becoming clinically symptomatic (stage 3), but little is known about the sequential changes in insulin secretion and glucose homeostasis through these stages. TrialNEt shows the variation of PEPC six months before the clinical diagnosis and then, after it. It is know that the dysfunction of beta-cells in type 1 diabetes might precede diagnosis for over 5 years. Most patients with long term type 1 diabetes are micro secretors of insulin, have a functional beta-cell that answers to stimuli and the partial clinical remission is inversely correlated to ketoacidosis and directly with BMI and age at the time of diagnosis.

Early and optimum metabolic control within the first 10 days of the disease preserves PEPC secretion and decreases long term complications.

Early hyperglycemia leaves permanent consequents in the CNS, which are as much or more deleterious than hypoglycemia. Preserving the beta-cell reduces the risk of hypoglycemia and microvascular complications. It also prevents the damage of white and gray matter of the brain, protecting an adequate neurodevelopment in younger patients.

ISPAD Guidelines (2018) suggest the implementation of an intensive treatment once the patient is compensated, for which the implementation of insulin analogues and technology have eased the road.

Key words: diabetes; beta cell.

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