J Diabetes Metab 2016, 7:5(Suppl)
http://dx.doi.org/10.4172/2155-6156.C1.043

9th Diabetologists Conference

June 06-08, 2016 Dallas, Texas, USA

Shared genetic etiology underlying type 2 diabetes and Alzheimer's disease

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Epidemiological evidence supports the observation that subjects with type-2 diabetes (T2D) is at higher risk to develop Alzheimer's disease (AD). However, how these two conditions are causally linked is unknown. Possible mechanisms include shared genetic risk factors, which we investigated in a recent study based on recent genome wide association study (GWAS) findings. We retrieved single nucleotide polymorphisms (SNPs) associated with T2D and AD from large-scale GWAS meta-analysis consortia and tested for overlap among the T2D and AD associated SNPs. We found 927 SNPs associated with both AD and T2D with p-value≤0.01, an overlap significantly larger than random chance (overlapping p-value of 6.93E-28). Among these, 395 of the shared GWAS SNPs have the same risk allele for AD and T2D, suggesting common pathogenic mechanisms underlying the development of both AD and T2D. We found that gene annotations from these shared SNPs are significantly enriched for specific KEGG pathways pertaining to immune responses, cell signaling and neuronal plasticity and cellular processes in which abnormalities are known to contribute to both T2D and AD pathogenesis. Our observation suggests that among T2D subjects with common genetic predispositions, dysregulation of these pathogenic pathways could have contributed to the onset of T2D, while simultaneously contributing to the increased risks of these subjects to eventually develop AD. Collectively, our GWAS studies will bring up to date one of the most important issues related to the influences of genetics on why some individuals with T2D are at high risk for developing AD.

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When basal insulin fails in type 2 diabetes: What next

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It is well known that type 2 diabetes is associated with a progression with an inevitable progression of beta cell dysfunction, in the absence of established methods to permanently protect the beta cells; the only clinical option available to physician is to intensify therapeutic measures at each stage of diabetes. Therefore, when one or more oral hypoglycemic agents are not available to maintain A1c targets, basal insulin is often added, as this is an option endorsed by the diabetes association across the world. However, given the progressive nature of type 2 diabetes, the A1c continue to rise and this requires the addition of prandial insulins. The choice that physician often face at this point may be divided into 5. Is there a possibility of adding a single prandial insulin injection, i.e a basal-plus regimen or a single injection co-formulation based regimen? A second option is to switch to a premix or a co-formulation based regime, third option is the clinician could consider the optimization of the existing oral drug regime. A fourth option is the institution of basal regimen. A fifth option is to add an incretin based injectable regimen. This article will discuss the pros and cons of the various options in the management of type 2 diabetes.

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