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Session OR11: Clinical Dilemmas and Outcomes in Diabetes.
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This session was chaired by Fahmy Amara, MD and Mary Ann Banerji, MD.

The first speaker, Jens Øllgaard, was the primary researcher from his team based out of Denmark and presented on their research entitled: Increased Survival and Reduced Macrovascular Disease with 7.8 years of Intensified, Multifactorial Intervention in Patients with Type 2 Diabetes and Microalbuminuria in the Steno-2 Study.

Their research encompassed 21 years of follow up in a cohort of 160 patients with type 2 diabetes and microalbuminuria. The patients were assigned to conventional or intensified, multi-factorial therapy targeting multiple risk factors. Mean treatment duration was 7.8 years, at which time the study continued as an observational follow up with all patients treated as the original intensive-therapy group.

The primary endpoint was survival time after randomization and survival time before the first cardiovascular disease (CVD) event. The secondary endpoint was the relative risk reduction in a 3-point MACE defined as: death from CVD, coronary artery disease, and cerebrovascular disease (defined as non-fatal and fatal ischemic or hemorrhagic stroke).

Their results showed both a reduction in CVD death by 60% and an increased survival rate of 7.9 years in the intensified treatment group. There was substantial and stable risk reduction in all endpoints. In response to a question following his presentation, Dr. Øllgaard further theorized that "the reduction in cardiovascular disease and risk of stroke could possibly minimize associated dementia" as well.

In summary, he felt that their work strongly supported the importance of early intervention in persons with type 2 diabetes.

As part of this session, Andrea Kelly, MD, MSCE, from the University of Pennsylvania, presented on research entitled: New Criteria for Indeterminate Glucose Tolerance in Pancreatic Insufficient Cystic Fibrosis Based on Defects in β - Cell Secretory Capacity.

Dr. Kelly reported that estimates point out that 40-50% of adults will develop type 2 diabetes; this is clinically relevant to the cystic fibrosis (CF) population as diabetes would constitute a second chronic disease on top of the already-debilitating disease of CF. The combination of CF and diabetes is associated with not only microvascular disease, but also worsening of nutritional status, deterioration of pulmonary function, and increase in mortality. Early recognition and treatment of the diabetes risk can help mitigate the increased mortality risk.

Insulin insufficiency largely accounts for the development of CF diabetes but the mechanisms have not been fully understood. According to Dr. Kelly, the decrease in β - cell mass does not always identify which patients will develop diabetes. To better identify those patients with early defects in insulin secretion, she and her colleagues sought evidence of impaired β - cell secretory capacity (a measure of functional β - cell mass) by using a more stringent parameter for characterizing indeterminate glucose tolerance. They speculated that this might be the earliest clinical sign of insulin secretion abnormalities. Their parameter was a 1-hour plasma glucose of ≥ 155mg/dL as this had been shown to detect early signs of cardiovascular disease associated with type 2 diabetes risk. Metabolic tests were conducted across groups of patients with pancreatic insufficient cystic fibrosis (PI) categorized by an oral glucose tolerance test (OGTT) as normal, indeterminate, impaired, and diabetic.

Dr. Kelly and her colleagues concluded that PI patients with a 1-hour OGTT glucose as low as 155mg/Dl can already be identified with early impairments in β -cell secretory capacity and that this capacity is further taxed by increased insulin secretory demand. She suggested that further studies could address whether dietary and/or pharmacological interventions to reduce pancreatic β -cell stress in PI patients may delay the progression to the development of CF diabetes.