Commentary
Volume 7:6, 2022

Journal of Diabetic Complications and Medicine

ISSN: 2475-3211 Open Access

Type 2 Diabetes with Early Onset and Younger Age

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Description

Given the high risk of complications and early death associated with this aggressive subtype of diabetes, the rising incidence and prevalence of young-onset type 2 diabetes 1-3 is concerning. New research from the RISE consortium suggests that young-onset type 2 diabetes has a different physiology than older-onset type 2 diabetes. The existence of underlying physiological differences in young-onset type 2 diabetes that might increase susceptibility to a wider range of diabetes complications is currently a crucial question. The excess morbidity that is associated with young-onset type 2 diabetes is thought to be caused by a number of factors, including a lower glycaemic control, a higher rate of pharmacotherapy failure, and a longer duration of diabetes exposure (8-10). It is unclear, across the range of complications, whether there is a residual long-term risk for young people after taking into account these and other factors. We investigated the possibility of an inherent susceptibility to complications over time given access to a large prospective dataset collected over 30 years. We compared the likelihood of microvascular and macrovascular complications after prolonged disease exposure in young-onset and older-onset type 2 diabetes. Our dataset's increased granularity made it possible to make meaningful adjustments to important confounders and to look for a separate connection between age at diabetes onset and long-term complications. The mean (standard deviation) or median (interquartile range) values of the data from descriptive analyses are presented. For continuous variables and Pearson's chi-squared tests for categorical variables, comparisons between groups were made [1,2].

For each duration of diabetes exposure band, logistic regression analyses were used to determine differences in the log-odds of having a micro- or macrovascular complication of interest between distinct age groups at the time of diagnosis. Three retinopathy models were considered for this study. From minimal non-proliferative to proliferative diabetic retinopathy, Retinopathy Model 1 considered all retinopathy grades. Only moderate non-proliferative, severe non-proliferative, and proliferative diabetic retinopathy were included in Retinopathy. Retinopathy includes maculopathy, proliferative diabetic retinopathy, and severe non-proliferative diabetic retinopathy. The primary independent variables in our models were the age-at-diagnosis group and the duration-of-diabetes-exposure group, with the binary outcome variable related to the complication of interest serving as the dependent variable. An interaction term between the age-at-diagnosis group and the duration-of-diabetes-exposure group was included in the study to further investigate the specific odds in relation to particular disease durations. Age at diagnosis and age at present were not included as independent variables in our models because of collinearity. Understanding the links between diabetes complications and age at diagnosis was our primary focus; Our modeling did not include the current age variable.As previously mentioned, some study participants provided data for multiple duration bands. Within the framework of generalized estimating equations, logistic regression analyses were carried out in order to address the issue of within-participant correlation. Within-participant correlations can be taken into account with this framework. The 95% confidence intervals (CIs) for the adjusted odds ratios were calculated. The reference group was the group with an onset age of 60 to 70 years for age-at-diagnosis group comparisons. Our primary model specifically excluded individuals who had undergone a complications assessment prior to ten years of diabetes exposure to allow for sufficient time for the development of complications; Our data may have been more susceptible to selection bias due to this exclusion. As a result, an expanded retinopathy model was used to conduct a sensitivity analysis on complication assessment data from individuals who had diabetes for 0 to 10 years and 10 to 25 years, respectively. In addition, expanded retinopathy model 1 included adjustments for the use of renin angiotensin system blockers and 3-hydroxy-3-methylglutaryl-CoA (HMG CoA) reductase inhibitors (in place of blood pressure and cholesterol), which may have affected the prevalence of retinopathy. These adjustments were made in order to rule out a potential effect of the cohort [3-5].

In conclusion, this study contributes to the expanding body of knowledge regarding the likelihood of long-term complications in young-onset type 2 diabetes. The persistent increase in adjusted odds of diabetic retinopathy suggests a particular susceptibility for retinopathy in youth that is not seen for the other vascular complications, despite the fact that duration and glycaemia contribute to the poorer outcomes seen in young-onset type 2 diabetes. A prospective cohort study would be the most effective way to verify our findings, but due to the time lag between diagnosis and complications, confirmatory data will take a long time to arrive. Until then, we believe that our findings support the need for aggressive management of known risk factors and more frequent surveillance of diabetic retinopathy in young-onset type 2 diabetes, which may be reflected in clinical care guidelines.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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Received: 02 November, 2022, Manuscript No. jdcm-23-86191; **Editor Assigned:** 04 November, 2022, PreQC No. P-86191; **Reviewed:** 16 November, 2022, QC No. Q-86191; **Revised:** 22 November, 2022, Manuscript No. R-86191; **Published:** 28 November, 2022, DOI: 10.37421/2475-3211.2022.7.193

How to cite this article: Jatana, Kris. "Type 2 Diabetes with Early Onset and Younger Age." J Diabetic Complications Med 7 (2022): 193.