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Expression of miR-24-3p and miR-198 in newly diagnosed type 2 diabetes mellitus patients

Prasenjit Mitra[1], Abhilasha .[1], Smriti Suri[1], Indu Saxena[3], Ravinder KG Shukla[2], Praveen Sharma[1]
[1]Department of Biochemistry, All India Institute of Medical Sciences, Jodhpur, India
[2]Department of Endocrinology, All India Institute of Medical Sciences Jodhpur, India
[3]Department of Biochemistry, All India Institute of Medical Sciences Gorakhpur, India

Objectives: T2DM is a metabolic condition caused by a combination of genetic, lifestyle, and environmental factors. Hyperglycemia is a symptom, which is caused by insulin resistance. MicroRNAs have been implicated in the regulation of post-transcriptional gene expression, mostly via repressing protein synthesis, according to recent research. In type 2 diabetes, dysregulated miRNA disrupts the insulin signalling cascade as well as multiple physiological processes, leading to disease progression. Because of their stable nature, miRNAs are now known as a novel class of biomarkers that are discharged from cells in circulation. Although miR-24-3p and miR-198 are linked to a variety of disorders, their role in type 2 diabetes is unknown. The aim of the study was to examine the expression levels of miR-24-3p and miR-198 in newly diagnosed T2DM patients with non-T2DM controls.

Methods: After obtaining informed consent, thirty-five newly diagnosed type 2 diabetes cases and thirty-five non-T2DM controls were recruited. Blood was drawn from a vein under aseptic conditions. Biochemical parameters were analyzed using the autoanalyzer. TaqMan Advanced miRNA assays were used to measure the expression levels of miR-24-3p and miR-198 using Real-time PCR. As an internal control, miR-16-5p was used.

Results: In terms of glycaemic status, T2DM patients had significantly higher levels of FBG, HbA1c compared with Non-T2DM controls ($p<0.0001$). There were no significant differences between the study groups for HDL Whereas TC, TG, and LDL differed significantly between subject groups and was higher in T2DM subjects ($p<0.0001$). The difference in miR-24-3p and miR-198 circulating levels in whole blood was statistically significant among the study participants ($p<0.01$). miR-24-3p showed a fold change of 0.312 and miR-198 showed a fold change of 0.203.

Conclusions: Our findings reveal that in newly diagnosed Type 2 Diabetes Mellitus, expression of miR-24-3p and miR-198 is downregulated.