Normal Glucose Tolerance with a High 1-Hour Postload Plasma Glucose Level Exhibits Decreased β-Cell Function Similar to Impaired Glucose Tolerance (Diabetes Metab J 2015;39:147-53)

Hee Kyung Kim
Department of Internal Medicine, Chonnam National University Hwasun Hospital, Chonnam National University Medical School, Hwasun, Korea

A 75-g oral glucose tolerance test (OGTT) is a sensitive test in most populations for diagnosis of type 2 diabetes mellitus (T2DM) and commonly used to identify high-risk individuals of T2DM [1]. The diagnostic criteria define diabetes as a fasting glucose ≥126 mg/dL or a 2-hour glucose challenge ≥200 mg/dL, but not including 1-hour glucose level. Recently, data obtained in population studies have suggested that 1-hour glucose level after OGTT can provide a better predictor for development of T2DM than fasting or 2-hour glucose [2]. And, subjects with 1-hour glucose levels of ≥155 mg/dL with normal glucose tolerance (NGT 1-hour high) have been considered as high risk of T2DM [3], which were more insulin-resistant and worse β-cell function than subjects with NGT 1 hour-low (<155 mg/dL) [4].

In this article entitled "Normal glucose tolerance with a high 1-hour postload plasma glucose level exhibits decreased β-cell function similar to impaired glucose tolerance," Oh et al. [5] compared β-cell function between subjects with NGT 1 hour-high, 1 hour-low, and impaired glucose tolerance (IGT). The interesting point was that subjects with NGT 1 hour-high group have decreased insulin sensitivity even after adjusting β-cell function, which was similar degree as subjects with IGT in Korean. However, there are several issues that need to be addressed.

First, the sensitivity of indicator of insulin secretion and resistance status should be considered. There are differences of opinion as to which diagnostic test represents the "gold standard" for assessing the insulin sensitivity and resistance. In this study, there are significant difference in NGT 1 hour-high and NGT 1 hour-low groups: Matsuda index, oral disposition index, and insulin secretion-sensitivity index-2. However, insulinogenic index, homeostasis model assessment (HOMA)-β-cell and HOMA-insulin resistance were similar between two groups. It is needed to be clarifying whether insulin resistance is not different between the NGT 1-hour high and low group, because of the conflicting results about insulin resistant marker.

Second, association between NGT 1-hour high group and impaired fasting glucose (IFG) should be considered. Both IGT and IFG conditions are intermediate states of abnormal carbohydrate metabolism between NGT and T2DM, and those are considered as high risk factor for development of T2DM. Lifestyle modification and pharmacologic therapy could prevent the progression of disease in subjects with prediabetic condition (IGT and IFG). In IFG, there is marked hepatic insulin resistance with near-normal muscle insulin sensitivity, whereas this pattern is reversed in IGT [6]. Although both conditions are characterized by reduced early-phase insulin secretion, there is an additional impairment of late-phase insulin secretion in IGT. In this study, insulin sensitivity
and resistance of NGT 1-hour high groups were similar with that of IGT subjects, but this study did not determine the differences between NGT 1-hour high group and IFG subjects.

Lastly, high 1-hour plasma glucose may be an index of metabolic impairment related with nonalcoholic fatty liver disease [7] and dyslipidemia [8]. Several models for prediction of T2DM are based upon established risk factors associated with metabolic disease or insulin resistance in nondiabetic individuals [9]. Therefore large population based epidemiologic studies are necessary to evaluate the association of T2DM and metabolic condition in subjects with NGT 1-hour high group compared with NGT 1-hour low group in Korean subjects. Eventually, treatment guideline should be established for subjects with NGT 1-hour high group such as diet, exercise and pharmacotherapy.

**CONFLICTS OF INTEREST**

No potential conflict of interest relevant to this article was reported.

**REFERENCES**

1. Unwin N, Shaw J, Zimmet P, Alberti KG. Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. Diabet Med 2002;19:708-23.
2. Abdul-Ghani MA, Williams K, DeFronzo RA, Stern M. What is the best predictor of future type 2 diabetes? Diabetes Care 2007;30:1544-8.
3. Abdul-Ghani MA, Abdul-Ghani T, Ali N, DeFronzo RA. One-hour plasma glucose concentration and the metabolic syndrome identify subjects at high risk for future type 2 diabetes. Diabetes Care 2008;31:1650-5.
4. Marini MA, Succurro F, Frontoni S, Mastroianni S, Arturi F, Sciacqua A, Lauro R, Hribal ML, Perticone F, Sesti G. Insulin sensitivity, β-cell function, and incretin effect in individuals with elevated 1-hour postload plasma glucose levels. Diabetes Care 2012;35:686-72.
5. Oh TJ, Min SH, Ahn CH, Kim EK, Kwak SH, Jung HS, Park KS, Cho YM. Normal glucose tolerance with a high 1-hour postload plasma glucose level exhibits decreased β-cell function similar to impaired glucose tolerance. Diabetes Metab J 2015;39:147-53.
6. Abdul-Ghani MA, Tripathy D, DeFronzo RA. Contributions of beta-cell dysfunction and insulin resistance to the pathogenesis of impaired glucose tolerance and impaired fasting glucose. Diabetes Care 2006;29:1130-9.
7. Sesti G, Hribal ML, Fiorentino TV, Sciacqua A, Perticone F. Elevated 1 h postload plasma glucose levels identify adults with normal glucose tolerance but increased risk of non-alcoholic fatty liver disease. BMJ Open Diabetes Res Care 2014;2:e00016.
8. Shimodaira M, Niwa T, Nakajima K, Kobayashi M, Hanyu N, Nakayama T. Correlation between serum lipids and 1-hour postload plasma glucose levels in normoglycemic individuals. J Clin Lipidol 2014;8:217-22.
9. Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM; San Antonio Heart Study. The metabolic syndrome as predictor of type 2 diabetes: the San Antonio heart study. Diabetes Care 2003;26:3153-9.