Impaired Crosstalk Between Insulin and Glucagon Secretion in Patients With Type 2 Diabetes

Hidekatsu Yanai

In this issue (P6), Morimitsu and Hamasaki report that once-weekly glucagon-like peptide-1 (GLP-1) receptor agonist, dulaglutide ameliorated glycemic control by suppressing fasting and postprandial glucagon secretion in a patient with type 2 diabetes [1]. Their report reminded me of the significance of impaired crosstalk between insulin and glucagon secretion in patients with type 2 diabetes.

In normal individuals, serum glucagon falls after carbohydrate ingestion. In diabetic patients, serum glucagon is not suppressed by carbohydrate ingestion despite hyperglycemia [2]. An inverse relationship between insulin and glucagon secretion observed in normal individuals has been lost in patients with type 2 diabetes [3]. Such an impaired crosstalk between insulin and glucagon secretion was also observed in prediabetic individuals [4]. Postprandial hyperglucagonemia in type 2 diabetes is likely due to loss of intra-islet postprandial suppression of glucagon secretion by insulin [5]. Therefore, altered insulin-to-glucagon ratio which was used in the report by Morimitsu and Hamasaki should play an important role in the pathophysiology of type 2 diabetes [6].

GLP-1 receptor agonist improved glycemic control by amelioration of insulin-to-glucagon ratio. Effects of other anti-diabetic drugs on insulin-to-glucagon ratio should be studied in the future.

Conflict of Interest

The author declares that he has no conflict of interest concerning this article.

References