JOJ
Urology & Nephrology
ISSN: 2476-0552

Juniper Publishers
key to the Researchers

Opinion
Volume 7 Issue 3 - July 2020
DOI: 10.19080/JOJUN.2020.07.555712

JOJ uro & nephron

Insulin Resistance in Hemodialysis Patients: How to Identify?

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Submission: June 26, 2020; **Published:** July 01, 2020

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Abstract

Insulin resistance is one of the common signs of end-stage renal failure. The onset of hemodialysis is accompanied by an increase in the sensitivity of peripheral tissues to insulin, but it does not eliminate insulin resistance. Clamp test in dialysis patients is difficult to apply. There is currently no consensus on how to replace it. It has been suggested that it is advisable to use a combination of HOMA-IR-1, HOMA2% B, Quantitative insulin sensitivity check index (QUICKI).

Opinion

Insulin resistance is identified as an impaired biologic response to insulin stimulation of target tissues (liver, muscle, and adipose tissue), resulting in a compensatory increase in beta-cell insulin production and hyperinsulinemia [1]. Over the past 20 years, the number of publications dealing with insulin resistance itself has sharply decreased. The emphasis shifted to metabolic syndrome. However, at present, there is no single understanding of metabolic syndrome. Some authors consider insulin resistance to be the trigger, while others consider central obesity. The gold standard for diagnosing insulin resistance is the euglycemic clamp test [2]. However, the technical features of its implementation do not allow its widespread use in hemodialysis patients.

However, the technical features of its implementation do not allow its widespread use in hemodialysis patients. It is proposed to use various indices as surrogate methods for studying insulin resistance: the Caro index [3], Fasting insulin resistance index [4], Fasting glucose-insulin ratio [5], Homeostasis Model Assessment of Insulin Resistance (HOMA-IR-1 and 2) [6,7], Quantitative insulin sensitivity check index (QUICKI) [8].

We conducted a comparative analysis of the use of these methods on a small group of 140 stable dialysis patients without diabetes and a fasting glucose level of not higher than 6 mmol / L. We concluded that for the diagnosis of insulin resistance, it is not enough to use any one formula. In our opinion, the optimal combination is the determination of the HOMA-IR-1 index, pancreatic β-beta cell activity index (HOMA2% B), and peripheral tissue insulin sensitivity index (QUICKI). The HOMA-IR-1 index was higher than 2.7 in 74 patients, the QUICKI index was less than 0.339 in 89 patients, the HOMA2% B index was more than 100% in 91 patients. Thus, in most clinically stable hemodialysis patients there is an increase in the activity of pancreatic β-beta cells and a decrease in the sensitivity of peripheral tissues to insulin. The HOMA-IR-1 index has been shown to be less sensitive. We believe that insulin resistance in dialysis patients develops in several stages: a decrease in the sensitivity of peripheral tissues to insulin - an increase in the activity of pancreatic β-cells in the pancreas - the development of true insulin resistance. The question of whether insulin resistance can be transformed into type 2 diabetes remains open. We believe that the cohort of patients on the kidney transplant waiting list is the most vulnerable in this regard. They are likely the risk group for the formation of steroid diabetes. However, this assumption needs to be clarified in the course of further research.

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DOI: 10.19080/JOJUN.2020.07.555712

How to cite this article: Rumyantsev A S. Insulin Resistance in Hemodialysis Patients: How to Identify?. JOJ Urology & Nephrology, 2020; 7(2): 555712. DOI: 10.19080/JOJUN.2020.07.555712