

Published by Al-Nahrain College of Medicine ISSN 1681-6579 Email: iraqijms@colmed-alnahrain.edu.iq http://www.colmed-alnahrain.edu.iq

Evaluation of Ghrelin, Insulin and Leptin Levels in Obese Type 2 Diabetic Patients on Metformin or Glimepiride Therapy in Basra, Iraq

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Abstract

Background Recent evidence has demonstrated the complex function of adipose tissue and gastric cells as an

endocrine organ through release of hormones into the blood stream and involved in physiological activities of the body; of them is ghrelin. The consequences of insulin resistance manifest at many levels and in many metabolic processes, producing a cluster of homeostatic abnormalities referred to

what is called metabolic syndrome.

Objectives To evaluate and compare the possible effects of using metformin or glimepiride on serum

concentrations of ghrelin, leptin and insulin resistance in obese type 2 diabetic patients in Basra, Iraq.

Methods Forty type 2 diabetic obese patients and twenty healthy subjects were studied. The patients were divided into 2 groups (each of 20 patients); group 1 on glimepiride therapy while group 2 on metformin

treatment. Blood samples were taken after at least 8 hours fasting for measurement of serum glucose,

leptin, ghrelin and insulin.

Results Ghrelin levels were significantly lower in the two patient groups with greater significant reduction in

metformin group. The highest serum insulin concentration and insulin resistance levels were clearly reported in glimepiride treated group as compared to control and metformin treated group. Leptin

levels show no significant differences in all studied groups.

Conclusion Metformin treatment associated significantly with improved insulin sensitivity; insulin resistance

associated significantly with decreases ghrelin concentration. Ghrelin is negatively correlated with leptin and obesity while positively correlate with insulin resistance. Our data support the role of body

weight as the major determinant of circulating leptin levels.

Keywords Diabetes mellitus, obesity, ghrelin, metformin, leptin.

List of abbreviations: DM = diabetes mellitus, T2DM = type 2 diabetes mellitus, IR= insulin resistance, AMPK = adenosine monophosphate-activated protein kinase, ELIZA = enzyme-linked immune sorbent assay, HOMA-IR = homeostasis model assessment-insulin resistance, CNS = central nervous system.

Introduction

public health problem with an estimated prevalence of 171 million people worldwide in the year 2000, and this number will

almost double by the year of 2030 ⁽¹⁾. Type 2 DM (T2DM) is a more complex metabolic disorder characterized by obesity, impaired β-cell function, increased endogenous hepatic glucose output and insulin resistance (IR) in target tissues ⁽²⁾. DM and obesity have a complex relationship where obesity may be a precursor for T2DM following IR ⁽³⁾. Obesity is associated with decreased responsiveness to insulin in muscle, liver and fat. On the other hand, weight