Insulin Resistance and ß-Cell Function in Patients with Non-alcoholic Fatty Liver Disease

Ahlam K. Mohammed[1], Salman K. Ajlan [2], Aseel R. Issa [3]

1. Department of Biochemistry, College of Medicine, University of Basrah, Basrah, IRAQ.
2. Professor, Department of Biochemistry, College of Medicine, University of Basrah, Basrah, IRAQ
3. Lecturer, Department of Surgery, College of Medicine, University of Basrah, Basrah, IRAQ

Received: 25.2.2024 **Accepted: 16.4.2024**

Abstract

Background: Nonalcoholic fatty liver disease is a common hepatic disorder marked by the accumulation of lipids in the liver, frequently accompanied by metabolic disturbances, including insulin resistance (IR), a condition where cells become less responsive to insulin's glucose-lowering effects, important in the evolution and progression of NAFLD.
Objective: To evaluate the degree of IR and β-cell function among patients with NAFLD.
Methods: A case-control study was conducted on 75 patients diagnosed with NAFLD and 81 control subjects. Fasting blood glucose, glycated haemoglobin and insulin were determined in all participants. In addition, insulin resistance and pancreatic ß- cell function were estimated by HOMA-IR and HOMA-B equations respectively.
Results: HOMA-IR and HOMA-B were significantly higher among patients with NAFLD (3.59 ± 3.52, 277.57 ± 169.89) than controls (2.56±1.39, 153.46±106.17) respectively, with the comparative $P < 0.05$ and $P < 0.001$. In addition, FBG, HbA1c and insulin were significantly higher among patients with NAFLD than control subjects, $P < 0.01$.
Conclusion: The strong association between HOMA-IR and NAFLD imply the role of IR in the pathogenesis of NAFLD. The significant elevation of HOMA-B among patients with NAFLD reflects the degree of hyperinsulinaemia secondary to IR.

Keywords; NAFLD, insulin resistance, β-cell function

Corresponding author: Ahlam K. Mohammed, College of Medicine, University of Basrah, Basrah, IRAQ

✉ **Email:**ahlamaltaa0@gmail.com

Introduction

Nonalcoholic fatty liver disease(NAFLD) is identified by the buildup of lipids in the liver (hepatic steatosis) without other underlying causes. While a small amount of fat can naturally occur in a healthy adult liver, the condition is considered pathological when at least 5% of hepatocytes have fat deposition. [1,2] Fat accumulation in the liver can be attributed to multiple factors, including increased delivery of free fatty acids (FFAs), elevated hepatic synthesis of fatty acids (FAs), reduced oxidation of FFAs, or diminished synthesis or secretion of very-low-density lipoprotein (VLDL). Various factors may contribute to the development of NAFLD, such as infections like hepatitis C and HIV, medications like tamoxifen, amiodarone, and methotrexate, exposure to toxins, metabolic disorders such as glycogen storage disorders, Wilson's disease, and homocystinuria, as well as nutritional influences like overnutrition, and extreme dieting [3,4]

The prevalence of NAFLD varies significantly across different populations. Estimates demonstrated that it affects around 20% of the US people and ranges from 11.5% to 46% in the general population. In contrast, nonalcoholic steatohepatitis (NASH) is estimated to affect only 2% to 3% of individuals. While NAFLD is more common among white men compared to white women, there are no discernible differences in prevalence between Hispanic and African American individuals. [5] Insulin resistance (IR) refers to a reduced biological response to insulin in target tissues like the muscle, liver, and adipose tissue, leading to impaired glucose disposal and compensatory hyperinsulinemia. [6,7]. This condition often precedes type 2 diabetes (T2D) by 10 to 15 years. As IR develops, the body increases endogenous insulin production, which, being an anabolic hormone, promotes weight gain, exacerbating IR further.[8,9] This cycle persists until pancreatic ß-cell activity fails to meet the demand for insulin, resulting in hyperglycemia and eventually

10.33762/mjbu.2024.147228.1199