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Adres : Kahramanmaraş Sütçü İmam Üniversitesi Tıp Fakültesi Dahili Tıp Bilimleri
Telefon : (344)280-3344
e-Posta:kgul@ksu.edu.tr

Bilgi için: Kamile GÜL
Unvan: Prof.Dr.
Telefon : (344)280-3344

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Relationship Between Serum Betatrophin Levels and Non-alcoholic Fatty Liver Disease in Hypogonadal Males

ABSTRACT

Objective: Betatrophin is a hepatokine that modulates hepatic glucose and lipid metabolism and contributes to non-alcoholic fatty liver disease (NAFLD) pathogenesis. Therefore, this study aimed to investigate the relationship between NAFLD and betatrophin levels in hypogonadal males.

Methods: The study included 56 newly diagnosed hypogonadal males aged 18-60 and 60 eugonadal males of similar age and body mass index. All participants were assessed for anthropometric and metabolic parameters, liver function tests, and betatrophin levels. Transient elastography was used to evaluate liver steatosis [controlled attenuation parameter (CAP)] and fibrosis [liver stiffness measurement (LSM)]. Accordingly, hypogonadal and control groups were divided into NAFLD (n = 64) and non-NAFLD (n = 52).

Results: Controlled attenuation parameter, LSM, waist circumference (WC), triglycerides (TG), IR index homeostasis model assessment (HOMA-IR), and betatrophin were significantly higher in the hypogonadal group than controls. Hepatic steatosis and fibrosis (67.9%-43.3%) were higher in hypogonadal males. Triglycerides, HOMA-IR, and betatrophin were higher, and total testosterone was significantly lower in the NAFLD group. Serum betatrophin was also significantly higher in patients with fibrosis than without. There was a significant positive correlation between WC, TG, HOMA-IR, betatrophin, and LSM and CAP. The predictive factors were TG ($\beta = 0.329$, $P < .001$), betatrophin ($\beta = 0.221$, $P = .029$), HOMA-IR ($\beta = 0.213$, $P = .019$) for CAP, and betatrophin for LSM ($\beta = 0.466$, $P < .001$).

Conclusion: Non-alcoholic fatty liver disease is more common in hypogonadal males than in eugonadal males. Betatrophin is an independent risk factor for developing and progressing NAFLD. However, more research is needed to explain the causal relationship between betatrophin and NAFLD.

Keywords: Hypogonadism, non-alcoholic fatty liver disease, betatrophin

Introduction

Non-alcoholic fatty liver disease (NAFLD) covers a wide clinical spectrum. It ranges from hepatic steatosis to fibrosis and cirrhosis.[1] It is an integral part of metabolic diseases that develop in the center of insulin resistance (IR), such as obesity and type 2 diabetes (T2DM). It is becoming an increasingly important problem worldwide.[2] Hypogonadism in males is a clinical situation that occurs as a result of insufficient testicular sperm production or testosterone (TT) or both.[3] It also causes an increased risk of cardio-metabolic diseases.[4,5] Low sex hormone levels have a negative effect on glucose and lipid metabolism. Several studies revealed that low TT levels are associated with abdominal obesity, metabolic syndrome (MS), IR, and NAFLD.[5-9]

Recently, betatrophin has been shown to contribute to the pathogenesis of MS, NAFLD, and T2DM.[10] It has even been suggested that it may be a possible biochemical marker of NAFLD progression.[11,12] Many mechanisms have been speculated to explain this relationship. Betatrophin is a hepatokine and has been shown to modulate liver glucose and lipid metabolism.[13] Insulin markedly increases betatrophin in adipose tissue and the liver.[14] It has been shown in animal studies that mice with IR have significantly increased betatrophin levels as a compensatory response.[15] While insulin directly activates lipoprotein lipase (LPL), it also indirectly regulates LPL by modulating betatrophin levels. It has been reported that betatrophin, which increases in the presence of IR, increases triglyceride (TG) synthesis by inhibiting LPL.[14,16]

Feyza Sarışık[1]
Ayten Oğuz[2]
Murat Şahin[2]
Murat İspiroğlu[3]
Ergül B. Kurutaş[4]
Özlem Nakış Bozkuş[4]
Ümit Nur Özbay[2]
Kamile Gül[2]

[1]*Department of Internal Medicine, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Türkiye*
[2]*Department of Endocrinology and Metabolism, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Türkiye*
[3]*Department of Gastroenterology, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Türkiye*
[4]*Department of Biochemistry, Kahramanmaraş Sütçü Imam University Faculty of Medicine, Kahramanmaraş, Türkiye*

Corresponding author:
Ayten Oğuz
✉ aytenoguz@windowslive.com

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