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Original Research Paper

The relationship between lipid levels and leptin concentration in obese adolescents compare with control group

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Abstract: This study investigated the relationship between lipid metabolism and leptin concentration in obese adolescents. A total of 70 obese and 20 normal-weight adolescents [aged 14–19 years] were recruited from Iraqi schools. Serum lipid profile parameters [HDL, LDL, TG, VLDL, and total cholesterol] and leptin concentrations were measured. The results revealed decreased HDL, non-significant changes in LDL and cholesterol, and significant increases in TG and VLDL in obese adolescents. Unexpectedly, leptin levels were lower in obese participants compared to controls. These findings suggest that altered lipid metabolism may influence leptin regulation in obese adolescents.

Keywords: Lipid Profile , Leptin , Obese

1.Introduction

The process of putting on too much fat in an unhealthy way that puts a person's health and life at risk is called obesity [1]. One of the most important problems of the 21st century is obesity, which leads to many long-term illnesses like diabetes, cancer, and others [2]. When someone is between the ages of 14 and 19, they are said to be adolescents [3]. People of all ages are getting fat, which has made it a big threat around the world. This issue affects more than just wealthy nations; it also affects some emerging nations, like Iraq. Baghdad, the city of Iraq, is a good example of how the number of overweight teens is rising[5]. The World Health Organization says that obesity is when a person's fat levels rise too quickly and build up in places like the waist and buttocks[6]. Lack of exercise, living in cities, and easy access to fast food and soft drinks are some of the things that are spreading it. Leptin is a hormone that the body makes from fat cells. The amount of body fat in babies, teens, and adults is closely linked to the amount of leptin in the blood. Leptin is the first chemical in the body that tells us about energy balance, controlling body weight, and the metabolism and control of fats and glucose [8]. High levels of the hormone leptin are always linked to being overweight. Leptin can get into the brain from the blood and connect to leptin receptors there. This makes the brain eat less and burn more energy. Through the leptin receptor [OBR], leptin does its job [9].

The OBR gene is on chromosome 1 and makes a protein with 1,162 amino acids. A part of the OBR gene called OB-Rb is highly expressed in the brain and cerebellum. Through a feedback loop, leptin controls body weight and energy balance by telling regulatory centers in the brain to stop people from eating, keep weight in check, and keep energy balance [10]. High-density lipoprotein [HDL] is a mixed particle made up of lipids, enzymes, and apolipoproteins. HDL not only moves cholesterol to the liver, but it also protects against oxidative stress, inflammation, and cell death. Another important job it has is to keep atherosclerosis at bay. This means that having more HDL may help guard against heart disease and atherosclerosis. Most of the time, HDL values are lower in fat [11]. LDL is a small lipoprotein that is very important for controlling how fats are used in the body. The tiny structure of LDL gives it special physical and

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chemical qualities, such as the ability to carry more payload, stay in the bloodstream for a longer time, be compatible with living things, be smaller, and naturally target other cells. [12]. Lipid profile imbalance is often present in people who are overweight, a situation that could be considered a sickness. In this way, being overweight is linked to higher levels of LDL-C and sdLDL-C/LDL-C, as well as higher levels of TG and TG/HDL-C. sdLDL-C is a substitute biomarker that is strongly linked to CVD. sdLDL-C affects more than just cardiovascular health. It also plays a role in the development of several diseases and is harmful in the cases of liver disease, metabolic syndrome, and obesity [13]. Triglycerides are nonpolar lipids that don't dissolve in water. They need to be moved around in the plasma with different lipoprotein particles. [8]. It is possible for triglycerides to help put together lipoprotein molecules and store and transfer energy. Adipocerides can make fatty acids, which the body's muscles can use to get energy for tasks or keep as energy in the form of fat or adipose tissue [14]. Apolipoprotein B is only found in very-low-density lipoprotein [VLDL], which is released from the liver. VLDL is made up of apolipoproteins, cholesterol, and lipids. VLDL's main job is to bring cholesterol and other fats to cells and organs so they can be used. VLDL plays a part in regular biological processes, but it is also known to help cause inflammatory cardiovascular disease. [15]. Total cholesterol, on the other hand, is the sum of all the cholesterol in the body. The high rate of cardiovascular disease is closely linked to being overweight. When there is more LDL in the blood than HDL, the body's metabolism and heart function will be slowed down. Some people call HDL "the good fat" because it cleans LDL cholesterol off of blood vessel walls by taking it back to the liver. [16]. The aim of this study was to determine the relationship between lipid levels and leptin hormone concentration in obese individuals.

2.Methodology

This study included 70 overweight and 20 normal-weight adolescents, aged 14 to 19 years. A venous blood sample [5 ml] was collected. Serum was extracted and stored at -20°C until analysis. Leptin levels were measured using commercially available ELISA kits. Lipid profiles were analyzed using an automated chemist [Biolis 30i] to measure total cholesterol, HDL, LDL, and VLDL. Body mass index [BMI] was calculated based on height and weight, using World Health Organization guidelines. Statistical analysis was performed using the t-test in GraphPad Prism version 8. [17, 18, 19].

3. Results and discussion

The study results showed that leptin levels in obese adolescents were significantly [$p < 0.01$] lower than in the control group as show [figure 1].also show were significantly [$p < 0.01$] lower High-density lipoprotein [HDL] cholesterol levels in obese adolescents compared to the control group as show [figure 2]. But The study results showed that Cholesterol levels, Low-density lipoprotein, Triglyceride levels, and Very low-density lipoprotein levels were significantly higher [$p < 0.01$] in obese adolescents compared to the control group, as shown [Figure 3,4,5,6].

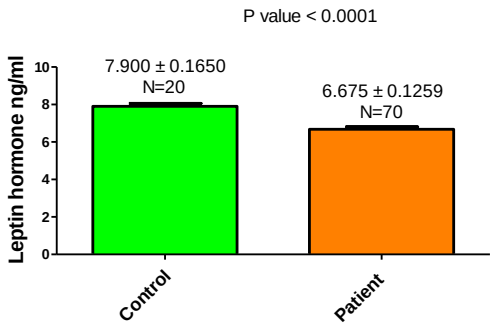

Fig 1: Leptin hormone levels in obese adolescents compared to the control group

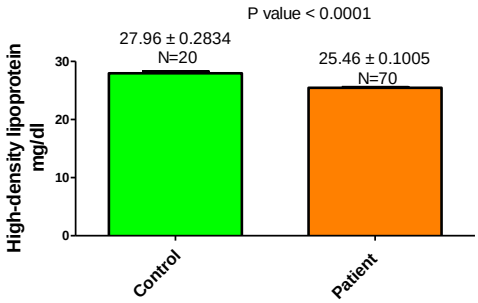

Fig 2: High-density lipoprotein [HDL] cholesterol levels in obese adolescents compared to the control group

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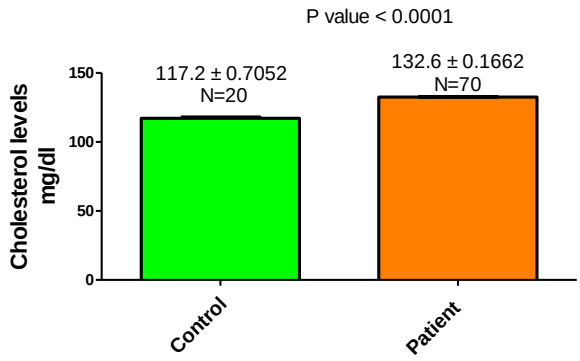

Fig 3: Cholesterol levels in obese adolescents compared to the control group

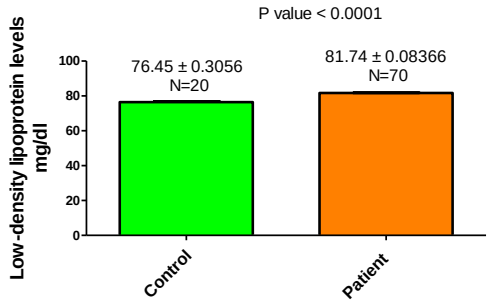

Fig 4: Low-density lipoprotein [LDL] cholesterol levels in obese adolescents compared to the control group

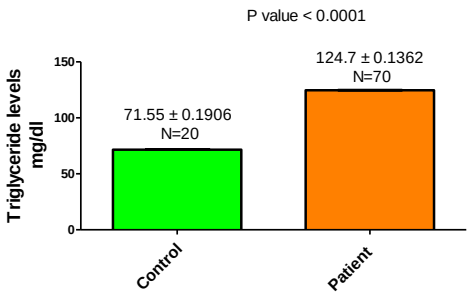

Fig 5: Triglyceride levels in obese adolescents compared to the control group

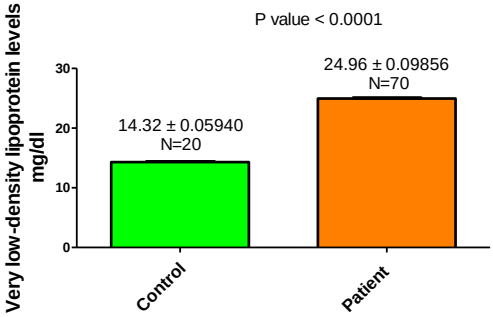

Fig 6: Very low-density lipoprotein [VLDL] cholesterol levels in obese adolescents compared to the control group**.**

The present study examined the association between serum lipid profile parameters and leptin concentration in obese adolescents compared to normal-weight controls. Unexpectedly, our findings demonstrated significantly lower leptin levels in obese adolescents, despite the well-established role of leptin as an adiposity signal that typically increases in proportion to body fat mass [11,12]. This paradoxical observation requires careful interpretation within the context of metabolic, genetic, and environmental influences.

Previous studies consistently reported hyperleptinemia in obesity, which is often accompanied by leptin resistance, particularly in adolescents [13,14]. In contrast, our results indicated decreased leptin concentrations in obese adolescents relative to controls. Several explanations may account for this discrepancy. First, fasting and caloric restriction, such as during the month of Ramadan, are known to suppress circulating leptin irrespective of adiposity [15,16]. Since part of the sample collection coincided with Ramadan, this factor likely influenced the hormonal outcomes. Second, genetic polymorphisms in the leptin gene [LEP] or its receptor [LEPR], such as the Met94 allele, have been shown to affect leptin secretion and signaling, leading to altered leptin levels in obese individuals [20] . Finally, chronic obesity in adolescents may trigger adaptive downregulation of leptin synthesis or secretion, reflecting a state of metabolic inflexibility.

Consistent with previous reports, obese adolescents exhibited significantly higher triglyceride [TG] and very-low-density lipoprotein [VLDL] concentrations[21,22]. Hypertriglyceridemia in obesity is largely attributed to insulin resistance, which impairs the antilipolytic action of insulin, thereby increasing free fatty acid flux to the liver and promoting hepatic TG synthesis and VLDL secretion[23,24]. The elevated TG and VLDL observed in our study suggest an early manifestation of metabolic syndrome in obese adolescents.

High-density lipoprotein [HDL] levels were lower in obese participants, although not statistically significant. This trend aligns with evidence that obesity is strongly associated with reduced HDL-C, often due to increased catabolism of HDL particles and impaired reverse cholesterol transport [25]. Low HDL is a hallmark of atherogenic dyslipidemia and is particularly concerning in adolescents, as it predisposes them to early cardiovascular disease [5,22].

Total cholesterol and low-density lipoprotein [LDL] concentrations did not differ significantly between groups. However, the literature indicates that obesity frequently increases small dense LDL particles [sdLDL],

which are more atherogenic despite normal total LDL levels [4,5]. Therefore, the absence of significant changes in LDL in our study does not preclude underlying lipoprotein abnormalities that may elevate cardiovascular risk.

The interaction between leptin and lipid metabolism is complex. Leptin regulates hepatic lipid oxidation and influences plasma triglyceride clearance via stimulation of lipoprotein lipase activity [25,26]. In theory, higher adiposity should raise leptin and promote lipid utilization; however, our data suggest that obese adolescents may develop an impaired leptin–lipid regulatory axis, causing cholesterol and lower amounts of leptin at the same time. This imbalance could be an early sign of metabolic failure that happens before problems like type 2 diabetes and heart disease become obvious.

Conclusion

Lipid metabolism significantly influences leptin concentration in obese adolescents. The observed reductions in leptin, along with dyslipidemia, highlight complex metabolic disturbances. These findings underscore the importance of monitoring lipid–leptin interactions in obesity prevention and management among adolescents.

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Author's Contributions

Authors had equal contribution in this manuscript.

Ethics

This study was conducted under approval by the medical ethics committee at the University of Kufa [2025]. Verbal and written consent was provided by parents and agreement for publication was obtained from both participants and researchers.

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