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Research Article:

Role of Melatonin in the Onset of Metabolic Syndrome in Men

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Abstract

Background: This prospective hospital-based study conducted in Tikrit City (Iraq) aimed to investigate the role of melatonin and the level of haemoglobin A1c and lipid profile concentrations in metabolic syndrome patients. The present study sought to identify the role of serum melatonin in MetS male patients. **Methods**: Participants were all males aged 18-50 years. A total of 60 metabolic syndrome male patients versus 30 healthy control male participants were enrolled in the present study. Blood samples were collected at a specific time (10 P.M. to 1 A.M.) and analyzed for measurement of haemoglobin A1c, lipid profile and melatonin. **Results**: Melatonin is significantly (p>0.001) lower in patients with metabolic syndrome (206.55 ±105 pg/ml) compared with the control group (298.82±110.4 pg/ml). The metabolic syndrome has significantly (p >0.001) elevated HbA1c % and lipid profile compared with the control group. **Conclusion**: In metabolic syndrome patients, serum melatonin concentration is reduced alongside elevated lipid profile glycemic control compared to normal people, these characteristics could participate in further enhancing the underlying pathology of metabolic syndrome diseases.

1. Introduction

Metabolic syndrome (MetS) has mostly centred on whether high abdominal obesity should be a required component (1). Insulin resistance syndrome and Syndrome X are other names for MetS and dysmetabolic syndrome. The updated definition comprises five criteria, three of which are required for a diagnosis (2). The characteristics include high waist circumference, triglycerides, blood pressure, fasting hyperglycemia, and low HDL cholesterol. Differences in baseline waist circumference across sexes and ethnicities have also raised concerns, prompting the development of sex- and ethnicity-specific recommendations (3).

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The hormone that the pineal gland produces, melatonin, is an effective antioxidant. Furthermore, it has been demonstrated to play a function in metabolic control. There is convincing evidence linking circadian system disruption (chronodisruption), lack of sleep, and the reduction of melatonin in obesity and MetS (4).

Melatonin effects on obesity-associated metabolic changes are emphasized on body weight/adiposity and resistance to the two essential hormones involved in the control of metabolism, insulin, and leptin (5). The function of melatonin as a multipurpose chemical as well as any potential consequences on the heart. Special consideration has to be given to the fact that the pleiotropic regulatory effects of melatonin in combination, as opposed to its antioxidant capabilities alone, are more likely to have an overall impact on obesity-related illnesses (6). It is directly pertinent to examine its potential function in the genesis of MetS to guide the therapeutic use of melatonergic agonists in the treatment of MS as well as to offer fresh insights into the illness (7).

Melatonin was stated to decrease systolic blood pressure (SBP) along with aortic pulse wave velocity, it is thought to be a significant indication of diastolic blood pressure and overall cardiovascular risk assessment in individuals with nocturnal hypertension. Regulation and modulation of blood pressure are a complex mechanism involving many components. Several reports have indicated a direct effect of melatonin on blood pressure (6,7).

The study aims to evaluate the role of serum melatonin in MetS male patients. The Correlation among melatonin levels, Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and haemoglobin (Hb) A1c in MetS patients.

2. Materials and Methods

2.1. Study design

The case-controlled study included sixty male patients with MetS, aged 18 to 50, 30 control and 60 MetS patients. The samples were taken at the Tikrit Teaching Hospital in Tikrit City (Iraq) between October 2023 and the end of November 2023 at a set time (10 A.M. to 1 P.M.). The investigator prepared a questionnaire form that was used for the interviews with these individuals. It had questions on their age, weight, length, and illness history.

2.2. Study population

2.2.1. Inclusion criteria

Males with MetS 18-50 years old were included in the study.

2.2.2. Exclusion criteria

-All patients with chronic liver and renal disorders, or Patients with Myocardial Infarction.
-Women were excluded from this study.
- Patients who take aspirin, ibuprofen, or melatonin

2.3. Study groups

MetS group: Included sixty male patients who meet the criteria for MetS. Control group: Included thirty samples from a healthy, typical control group.

2.4. Sampling

Every subject included in this research should be fast for at least 14 hours before that. patient's blood samples were taken between 10:00 p.m. and 1:00 a.m. All subjects had their antecubital veins punctured with a disposable syringe to collect around five milliliters of blood. The drawn blood was split into two parts. Three milliliters of the first part were placed in a separation gel tube, which helps separate the serum by centrifugation at 3000 rpm for ten to fifteen minutes. The transparent serum was transferred into dry, clear Eppendorf tubes and kept cold at -20 °C until the ELISA test was performed to determine melatonin and the U.V. Spectrophotometer's for lipid profile. The second portion, 2 mL of blood, was placed in a blood collection tube with ethylene diamine tetra acetic acid (EDTA) as an anticoagulant so that the Cobas C 111 analyzer could detect glycated haemoglobin (HbA1c) right away.

2.5. Physical examination

Every participant involved in the research had his height, weight, and waist circumference (WC) measured. The formula for calculating BMI was weight in kilograms divided by height in square meters. It measured obesity using an international obesity task force and the WHO's BMI categorization.

2.6. Ethical consideration

According to the research consent form, both sick and healthy individuals gave their approval to participate in this study, where a personal interview was conducted for each person through which a questionnaire was filled out that included the sequence, name, age, height, weight, residence, sex, duration of injury, and manner of treatment.

2.7. Statistical analysis

The statistical program (SPSS) version (23) was used to examine the data from the current study. The mean and standard deviation were tested, and the Duncan test was used to ascertain the difference between the groups at the probability level of 0.05.

3. Results

The serum level of TG, TC, VLDL, and LDL in the patients group was significantly higher in comparison to the control group while the serum level of HDL was significantly higher in the control group in comparison to the patients group. HbA1c (%) has shown significantly higher blood levels in patients compared to the control group (**Table 1**). The blood pressure was measured for all patients and reported to be more than 140/90 mm Hg for all patients compared to equal or less than 120/80 mmHg in the control group .

Table 1. Lipid profile in patients and control group

Lipid profile (mean±SD)	Control group (n=30)	MetS group (n=60)
TG (mg/dl)	189.75± 9.1	221.87±18.2*
TC (mg/dl)	164.60± 9.8	225.91±31.4*
VLDL(mg/dl)	37.95± 1.8	44.37±3.6*
HDL(mg/dl)	48.68±7*	31.57±7.0
LDL(mg/dl)	77.97± 13	149.97±32.4*
Hb_{A1c} (%)	4.8±0.47	7.05±1.41*
*significantly higher at p<0.001		

The melatonin plasma concentration was significantly higher in the control group compared to the patient group, moreover, the concentration of melatonin in hypertensive patients was significantly higher than in diabetic patients (**Table 2**).

Table 2. Melatonin levels in patients and control group

Parameter	Control group (n=30)	Hypertensive group (n=24)	Diabetes mellitus group (n=36)
Melatonin (pg/ml)	215.71±108.9*	194.58±100.6^	102.07± 81.2
*^significantly higher at p<0.001, * significantly higher compared to other groups, ^ is significantly higher compared to the diabetic group			

The results have confirmed that the correlation between serum melatonin and BMI in patients with metabolic syndrome is negative (**Figure 1**).

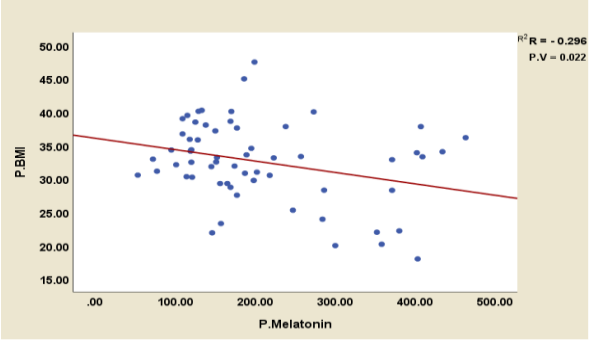

Figure 1. Correlation between serum Melatonin Level and BMI with metabolic syndromes men

4. Discussion

The present study has included a group of patients with MetS diagnosed by laboratory and clinical methods, the biochemical parameters have shown that lipid profile (TG, TC, LDL, and VLDL) have been elevated in MetS patients compared to the healthy control group alongside elevated Hb_{A1c} compared to the control group, with concomitant decreased HDL in MetS patient compared to healthy control group. The melatonin measured at night in MetS patients has been reduced significantly compared to the control group.

According to previous studies (8-11), the metabolic syndrome has been confirmed by laboratory findings, including elevated blood pressure, lipid profile, and glycemic parameters. The findings of these studies were in line with the present study.

The present study found that serum concentration of melatonin has been reported to be lower in MetS patients compared to the control group. In line with this study, Dolores et al. (2012) and Demirtas et al. (2015), have reported a significantly higher serum melatonin concentration in patients diagnosed with metabolic syndrome (8,9). Surprisingly, Dolores et al. (2012) measured melatonin concentration in the morning and night in both the control and MetS patients group and the outcome has been confirmed no differences between the control and MetS patients group in the morning measured melatonin (8). However, evening-measured melatonin was remarkably higher in MetS patients compared to control, henceforth, the present study was designed to measure melatonin at night.

Concerning animal studies, rats fed from weaning with a high-fat diet, melatonin decreased body weight gain, feed efficiency and plasma glucose, leptin and triglyceride levels (12). In middle-aged rats receiving a high-calorie liquid diet, melatonin reduced weight gain and

plasma insulin and leptin levels (13). In high-fat diet-fed mice, melatonin improved insulin sensitivity and glucose tolerance (14). In ovariectomized rats, melatonin was effective in reducing obesity (15-17). In high-fat-fed rats, melatonin attenuated body weight increase, the increase in plasma glucose, insulin, adiponectin, leptin, triglycerides and cholesterol levels, and counteracted disrupted 24-hour patterns (18). Melatonin reduced body weight gain, visceral adiposity, blood triglyceride and insulin levels, and thiobarbituric acid reactive substances under a high-calorie diet in rats (19). Melatonin attenuated high-fat diet-induced fatty liver disease in rats (20). The study by Pai and Majumdar shows the Protective effects of melatonin against metabolic and reproductive disturbances in polycystic ovary syndrome in rats (21).

In observational studies, low plasma melatonin levels in type 2 diabetic patients were reported (22), also an increased number of melatonin receptors in type 2 diabetic patients (23). Recent studies have shown that the melatonin receptor gene polymorphism is associated with an increased risk of type 2 diabetes (24), Furthermore, research has also found a genetic link between the MT2 receptor and impaired fasting glucose in youth with obesity (25), Additionally, studies have also identified a potential connection between melatonin receptor gene polymorphisms and polycystic ovary syndrome (PCOS). PCOS is a hormonal disorder that affects women and has been linked to insulin resistance and an increased

risk of diabetes (26). Furthermore, low melatonin production is associated with myocardial infarction (27,28). Moreover, low melatonin production has also been observed in elderly individuals with hypertension (29). Melatonin treatment ameliorated MS in obese patients (30,31). Melatonin treatment ameliorated the MS caused by second-generation antipsychotics in bipolar and schizophrenic patients (32-34). Melatonin alone or in combination with metformin improved glycemic control in type 2 diabetic patients (35).

Melatonin works by modulating the expression of Glucose transporter type 4 (GLUT4), inducing the phosphorylation of the insulin receptor through its G-protein-coupled membrane receptors, and activating the insulin signalling pathway through its intracellular substrates. Strong chronobiotic melatonin is partly responsible for the daily distribution of metabolic processes, coordinating the insulin-resistant metabolic phase of the day with rest and fasting and the high insulin sensitivity associated with the activity/feeding period of the day (36). Furthermore, By controlling energy flow to and from storage, as well as directly controlling energy expenditure by activating brown adipose tissue and aiding in the browning of white adipose tissue, melatonin plays a major role in maintaining a sufficient energy balance (37).

These aforementioned impacts of melatonin could be explained in the context of that melatonin regulates energy metabolism in the pancreatic islets, which have an impact on the production and release of insulin and glucagon (38). In isolated rat pancreatic islets, melatonin activity via MT1 or MT2 reduces glucose-stimulated insulin production. The Activation of these receptors results in the suppression of insulin secretion produced by glucose and forskolin, suggesting that melatonin works by blocking the adenylate cyclase/cAMP pathway and lowering protein kinase A (PKA) content while maintaining Protein Kinase C Alpha (PKCa)-subunit content at the same time that cGMP is reduced (39). Concerning the physiological and pathophysiological importance of melatonin's modulatory effect on pancreatic islet function, genome-wide association studies have revealed that common noncoding variants in melatonin receptor 1B (MTNR1B), the gene that codes for melatonin receptor 1B, or MT2, increase the risk of type 2 diabetes (40). It should be noted that insulin can control the synthesis of pineal melatonin by increasing the amount of melatonin produced in response to norepinephrine during two critical times of the night: right before lights out and right after lights on (41).

5. Conclusion

This study concluded that individuals with metabolic syndrome have lower levels of serum melatonin, as well as elevated levels of lipids and impaired glycemic control, in comparison to those without the syndrome. This combination of factors may contribute to the progression of metabolic syndrome and its associated diseases. Therefore, further research should be conducted to explore the potential role of melatonin in the pathogenesis of metabolic syndrome and its potential as a therapeutic target.

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دور الميلاتونين في بداية متلازمة التمثيل الغذائي لدى الرجال

الخلاصة

الخلفية: تهدف هذه الدراسة المستقبلية التي أجريت في المستشفى في مدينة تكريت (العراق) إلى التحقيق في دور الميلاتونين ومستوى الهيموغلوبين السكريوتركيزات الدهون في مرضى متلازمة التمثيل الغذائي. سعت الدراسة الحالية إلى تحديد دور الميلاتونين في مصل الدم في مرضى متلازمة التمثيل الغذائي الذكور. **الطرق:** كان جميع المشاركين من الذكور الذين تتراوح أعمارهم بين 18-50 سنة. تم تسجيل ما مجموعه 60 مريضا من الذكور المصابين بمتلازمة التمثيل الغذائي مقابل 30 مشاركا من الذكور الأصحاء في الدراسة الحالية. تم جمع عينات الدم في وقت محدد (من 10 مساء إلى 1 صباحا) وتحليلها لقياس الهيموغلوبين السكري وملف الدهون والميلاتونين. **النتائج:** الميلاتونين أقل بشكل ملحوظ ($p<0.001$) في المرضى الذين يعانون من متلازمة التمثيل الغذائي (206.55 ±105 بيكوغرام / مل) مقارنة مع المجموعة الضابطة (298.82±110.4 بيكوغرام / مل). متلازمة التمثيل الغذائي لديها بشكل ملحوظ ($p <0.001$) ارتفاع نسبة الهيموغلوبين السكري وملف الدهون مقارنة مع المجموعة الضابطة. **الخلاصة:** في مرضى متلازمة التمثيل الغذائي ، انخفض تركيز الميلاتونين في الدم إلى جانب التحكم في نسبة السكر في الدم في ملف الدهون المرتفع مقارنة بالأشخاص العاديين ، يمكن أن يشارك هذا التوصيف في زيادة تعزيز الأمراض الأساسية لأمراض متلازمة التمثيل الغذائي.

الكلمات المفتاحية: الميلاتونين، متلازمة التمثيل الغذائي، اليحمور السكري، الملف الشخصي للدهون