Research Article (Araştırma Makalesi)

Hematological and Inflammatory Parameters Effective on Inflammation and Insulin Resistance in Obesity

Obezitede İnflamasyon ve İnsülin Direncine Etkili Hematolojik ve İnflamatuar Parametreler

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Özet

Amaç: Obezite; insülin direnci, tip 2 diyabet ve pek çok metabolik sendromun altında yatan baskın risk faktörlerinden biridir. Bu çalışmada obez hastalarda inflamasyon parametrelerini, bu parametrelerin insülin direnci (İD) ve kan grupları ile ilişkisi araştırmayı amaçladık.

Gereç ve Yöntemler: Hasta ve kontrol grubunun demografik özellikleri, vücut kitle indeksleri (VKİ), biyokimya, hemogram, C-reaktif proteinin (CRP) ve kan grupları kayıt edilerek gruplar arasında karşılaştırıldı.

Bulgular: Obez hastaların yaş ortalaması 37.37±11.43 yıldı ve %41.8'inde (n=77) insülin direnci vardı. Obez ve İD olan grubun nötrofil ve monosit düzeyleri İD olmayan kontrol ve obez gruplardan anlamlı derecede yüksekti (p<0.001). İD olan obezlerde nötrofil/lenfosit oranı (NLO), monosit/HDL oranı (MHO) ve sistemik immün-inflamatuar indeks (Sİİ) düzeyleri İD olamayan obezlerden anlamlı derecede yüksekti. B kan grubunda olan obezlerde İD ve nötrofil oranları O kan grubundakilere göre anlamlı derecede yüksek bulundu (p=0.023).

Sonuç: Obez ve İD olan grupta hematolojik inflamatuar parametreleri ve NLO, MHO ve Sİİ gibi sistemik kronik inflamasyonu gösteren oranları yüksek bulduk. Çalışmamızın artmış yağ dokusunun neden olduğu inflamasyon ve obezlerde İD gelişimi arasındaki ilişkiyi gösterdiğini, gelecekte bu konuda yapılacak çalışmaların erken teşhis ve tedavilerin tasarlanmasında yardımcı olacağını düşünüyoruz

Anahtar kelimeler: İnsülin resistansı, İnflamasyon, Kan grubu, Obezite

Abstract

Objective: Obesity is one of the predominant risk factors associated with insulin resistance (IR), type 2 diabetes, and many metabolic syndromes. In this study, we aimed to investigate inflammatory parameters and their relationship with IR and blood groups in obese individuals.

Materials and Methods: The demographic characteristics, body mass index (BMI), biochemical parameters, hemogram values, and blood group types of individuals in the obesity and control groups were recorded and compared.

Results: The mean age was 37.37±11.43 years in obese individuals and 41.8% (n=77) of them had IR. Neutrophil and monocyte counts of the obese individuals with IR were significantly higher than those of the individuals without IR in the control and the obesity groups (p<0.001). The neutrophil/lymphocyte ratio (NLR), the monocyte/high-density lipoprotein cholesterol ratio (MHR), and the systemic immune-inflammation index (SII) were significantly higher in obese individuals with IR compared to obese individuals without IR. IR was more common and neutrophil ratios were significantly higher in obese individuals with B blood type compared to those with O blood type (p=0.023).

Conclusion: We found that the values of hematological inflammatory parameters and the levels of NLR, MHR, and SII as indicators of systemic chronic inflammation were increased in obese individuals with IR. We think that our study shows the relationship between inflammation due to excess adipose tissue and the development of IR in obese individuals. We are of the opinion that future studies investigating this subject will help to develop processes for early diagnosis and treatment.

Keywords: Blood groups, Inflammation, Insulin resistance, Obesity

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INTRODUCTION

The prevalence of obesity and its metabolic complications have significantly increased in recent years globally (1). Obesity is one of the predominant risk factors associated with metabolic syndrome. It increases the risk of developing various pathological conditions such as insulin resistance (IR), type 2 diabetes, dyslipidemia, and hypertension (2). Obesity is the most common cause of IR in humans. In nondiabetic individuals, hyperinsulinemia usually maintains normal or near-normal glucose homeostasis by compensating for the underlying insulin resistance (IR) (3).

Inflammation is a physiological response of the organism to physical, chemical, or biological harmful stimuli. The precise triggers of obesity-associated inflammation are poorly understood and may be different across several tissues (4). The inflammatory state in obesity is characterized by a low-grade chronic inflammation with no accompanying signs of infection or autoimmune reaction and no major tissue damage. The inflammatory response in obesity is induced by impaired metabolic homeostasis (5). Adipose tissue, which contains immune cells, as well as several other types of cells including preadipocytes, adipocytes, fibroblasts, and endothelial cells, is the origin of immune-mediated inflammation in obesity (6).

Studies have shown that obesity-associated inflammation may be an important cause of IR with the involvement of many cellular and molecular factors in this process (7). Studies in the literature suggest that ABO blood group antigens may play a role in the pathogenesis of cancers and cardiovascular, endocrine, and metabolic disorders (8,9). The relationship between ABO blood groups and metabolic disorders suggests the likelihood of the availability of similar links between obesity and IR. In this study, we aimed to investigate inflammatory parameters and relationships of these parameters with IR and blood groups in obese individuals.

MATERIALS AND METHODS

This retrospective study was approved by the Ethics Committee of Health Sciences University Diskapi Yildirim Beyazit Training and Research Hospital (2021-05/111/07). All procedures were applied in accordance with the principles of the Declaration of Helsinki. The study included 184 obese individuals, who presented to the internal diseases outpatient clinic in the period between January 2021 and January 2022, and who had a body mass index (BMI) value of ≥30 kg/m^2. Patients with a history of chronic diseases (diabetes and chronic liver, kidney, heart, lung, thyroid, or hematologic disorders), immune deficiency, acute or chronic inflammatory disorders, oncologic diseases, thalassemia; patients with a history of infection and regular use of medications over the last month, and pregnant women were excluded from the study. The control group included 90 healthy individuals, who had BMI values of 20-25 kg/m2, who presented to the outpatient clinic for regular check-ups, and who were free from diseases. Obese individuals were further categorized into the obese with IR (OWIR) and obese with no IR (OWNIR) subgroups. The control group was categorized into the controls with IR (CWIR) and controls with no IR (CWNIR) subgroups.

The demographic characteristics, biochemical parameters including C-reactive protein (CRP) levels, hemogram values, and blood group types of the obesity and control groups were recorded. The neutrophil/lymphocyte ratio (NLR) was calculated by dividing the neutrophil count by the lymphocyte count; the platelet/lymphocyte ratio (PLR) was calculated by dividing the platelet count by the lymphocyte count, and the monocyte/high-density lipoprotein cholesterol ratio (MHR) was calculated by dividing the monocyte count by the HDL level. The systemic immune-inflammation index (SII) was calculated using the following equation: SII = neutrophil count X platelet count/lymphocyte count (10). The calculated values were compared between the obesity and control groups. Blood tests for HDL, cholesterol, and fasting plasma glucose were performed by enzymatic colorimetric assay after 12 hours of fasting. BMI was calculated by dividing weight in kilograms by the square of height in meters. Individuals with BMI values of ≥30 kg/m2 were considered obese. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the following equation: HOMA-IR=Fasting Glucose (mg/dL) x Fasting Insulin (uIU/mL)/405. Individuals with HOMA scores of ≥2.7 were considered to have IR (11).

Statistical Analysis

The study data were collected from 274 individuals, comprising 90 individuals with normal BMI and 184 with obesity. Analyzes were conducted using the IBM SPSS Statistics 26 package software. Study data were summarized using frequencies (number, percentage) for categorical variables and descriptive statistics (mean, standard deviation, median, minimum, and maximum) for numerical variables.

The normality assumptions of numerical variables were examined by the Kolmogorov Smirnov test of normality, which revealed that the variables were not normally distributed. Therefore, non-parametric statistical methods were used. Differences between two independent groups were evaluated using the Mann-Whitney U Test. Differences between more than two independent groups were evaluated by the Kruskal-Wallis

test. Relationships between two independent categorical variables were tested by the Chi-Square analysis. Statistical significance was interpreted at the 0.05 level in analyses.

RESULTS

The mean age was 37.37±11.43 years and 42.30±11.57 years in the obesity and control groups, respectively. Women constituted 73.3% of the control group, while this rate was 81.5% in the obesity group. The percentage of individuals with the AB blood type in the obesity group was significantly lower than that in the control group (p=0.038). The rate of those with IR in the obesity group was significantly higher compared to the control group (p<0.001) **Table 1**.

Blood glucose levels were significantly lower in the CWNIR group compared to the OWIR and CWIR groups. Blood glucose levels of the OWIR group were significantly higher compared to the OWNIR group (p=0.001). Insulin levels in the CWNIR group were significantly lower compared to the OWIR, OWNIR, and CWIR groups. Insulin levels in the OWIR and CWIR groups were significantly higher compared to the OWNIR group (p<0.001). Neutrophil counts in the OWIR group were significantly higher than those in the OWNIR and CWNIR groups (p=0.01). Lymphocyte and platelet counts in the OWIR group were significantly higher compared to the CWNIR group. Monocyte counts in the OWIR group were significantly higher than those in the CWNIR and OWNIR groups. HDL levels were significantly higher in the CWNIR group compared to the OWIR group. CRP levels were significantly higher in the OWIR and OWNIR groups compared to the CWIR and CWNIR groups (p<0.001). NLR was significantly higher in the OWIR group compared to the OWNIR group (p=0.046). MHR was significantly higher in the OWIR group compared to the OWNIR and CWNIR groups (p<0.001). SII was significantly higher in the OWIR group compared to the OWNIR and CWNIR groups (p=0.01) **Table 2**.

In the obesity group, neutrophil counts of individuals with blood type B were significantly higher than those with blood type O (p=0.023). The percentage of individuals with IR was significantly higher among those with blood type B compared to those with blood type O (p=0.024) (**Table 3**).

DISCUSSION

It is well recognized that obesity, prediabetes, and IR are closely related (12). In prediabetes, the metabolism is considered insulin resistant with increased circulating insulin levels to achieve a glucose-lowering response (13). In our study, 41.8% of obese individuals had IR and this rate was significantly higher compared to the control group. In obese individuals with IR, blood glucose levels were significantly higher compared to those without IR. The blood glucose levels of the non-IR individuals in the control group were significantly lower

Table 1. Demographic characteristics of patients

	Obese (n=184)	Control (n=90)	Z	p
	Mean±SD	**Mean±SD**		
Age	37.37±11.43	36.00±12.44	-1.212	0.225
BMI	34.59±4.38	21.85±2.35	-13.487	**0.000***
	n(%)	**n(%)**	**Chi-Square**	**p**
Gender				
Female	150(81.5)	66(73.3)	2.428	0.119
Male	34(18.5)	24(26.7)		
Blood Group				
0	53(28.8)	17(18.9)	8.402	**0.038***
A	90(48.9)	40(44.4)		
AB	**15(8.2)**	**16(17.8)**		
B	26(14.1)	17(18.9)		
Insulin Resistance				
No	107(58.2)	74(82.2)	15.617	**0.000***
Yes	77(41.8)	16(17.8)		

Z: Mann Whitney U, *:p<0,05, SD: Standard deviation, BMI: Body mass index

Table 2. Biochemical Test Results and Differences by Groups

	1.OWIR (n=77)	2.OWOIR (n=107)	3.CWIR (n=16)	4.CWOIR (n=74)	KW	p	Dif.
	Mean±SD	Mean±SD	Mean±SD	Mean±SD			
Glucose(mg/dl)	104.51±19.14	94.78±9.45	102.19±16.1	90.58±7.72	46.575	**0.000***	**4-1.3 1-2**
Insulin(uIU/ml)	21.19±12.53	5.85±2.84	21.05±8.77	4.13±2.66	189.942	**0.000***	**4-1.2.3 2-1.3**
Neu(×10³ µl)	5.01±1.27	3.89±1.52	4.85±1.94	3.92±1.19	43.735	**0.000***	**1-2.4**
Lym(×10³ µl)	2.79±0.88	2.84±3.45	3.65±4.84	2.82±3.34	8.267	**0.041***	**1-4**
Plt(×10³ µl)	295.35±59.6	272.88±63.4	263.81±67.08	264.34±60.49	10.004	**0.019***	**1-4**
Mon(×10³ µl)	0.74±0.76	0.52±0.61	0.71±1.17	0.46±0.89	40.562	**0.000***	**4-1.2 1-2**
HDL(mg/dl)	49.16±11.09	52.7±10.77	54.81±15.09	56.39±12.1	14.220	**0.003***	**1-4**
CRP(mg/l)	1.15±1.62	0.99±2.03	0.19±0.17	0.37±1.27	76.725	**0.000***	**4-1.2 3-1.2**
Ferritin(ng/ml)	52.3±60.98	42.38±50.78	46.14±47.4	33.23±42.33	5.968	0.113	-
NLR	2.03±1.26	2.58±9.83	1.97±1.06	1.72±0.75	7.986	**0.046***	**1-2**
PLR	118.39±64.28	209.46±985.56	106.78±45.61	117.27±47.14	0.476	0.924	-
MHR	0.02±0.04	0.01±0.01	0.01±0.02	0.01±0.02	49.487	**0.000***	**4-1.2 1-2**
SII	591.77±376.01	739.46±3043.98	543.54±369.41	463.59±269.19	18.057	**0.000***	**1-2.4**

Z:Mann Whitney U Analizi *:p<0,05, KW: Kruskal Wallis, Dif.(Difference): Tukey's multiple comparison test, OWIR: Obese with insülin resistance, OWOIR:Obese without insulin resistance, CWIR: Control insulin resistance, CWOIR: control without insulin resistance. Neu: Neutrophils, Lym: Lymphocytes, PLT: Platelets, Mon: Monocytes HDL: High-density lipoprotein,CRP: C-reactive protein, NLR: Neutrophil/lymphocyte ratio, , PLR: Platelet/lymphocyte ratio, MHR: Monocyte/HDL ratio, SII: Systemic immune-inflammation index

Table 3. Biochemical Results According to Blood Groups in Obese

	1.O group	2.A group	3.AB group	4.B group	KW	p
	Mean±SD	Mean±SD	Mean±SD	Mean±SD		
Neu(×10³ µl)	4.2±1.71	4.31±1.38	3.93±1.58	5.07±1.41	9.558	**0.023* Dif: 0-B**
Lym(×10³ µl)	2.66±0.79	2.99±3.77	2.66±0.69	2.67±0.82	1.377	0.711
Plt(×10³ µl)	288.96±66.22	274.14±57.75	294.13±78.28	290±61.97	2.622	0.454
Mon(×10³ µl)	0.53±0.22	0.61±0.76	0.55±0.42	0.81±1.06	2.936	0.402
HDL(mg/dl)	50.55±10.81	52.15±11.62	51.73±12.44	49.08±8.31	3.029	0.387
CRP(mg/l)	1.01±2.21	1.18±1.86	0.4±0.35	1.09±1.62	5.489	0.139
Ferritin(ng/ml)	45.02±52.06	45.47±53.09	46.45±41.35	53.35±75.69	0.534	0.911
NLR	3.52±13.94	1.88±1.25	1.55±0.68	2.08±0.9	6.240	0.101
PLR	305.51±1399.72	117.32±63.13	114.05±30.79	117.97±48.67	0.145	0.986
MHR	0.01±0.001	0.02±0.04	0.01±0.01	0.02±0.02	4.618	0.202
SII	1059.78±4312.84	516.7±381.38	444.75±204.45	590.23±286.78	5.813	0.121
	n(%)	n(%)	n(%)	n(%)	CS	p
Insulin Resistance					9.408	**0.024***
No	37(**69.8**)	51(56.7)	10(66.7)	9(**34.6**)		
Yes	16(**30.2**)	39(43.3)	5(33.3)	17(**65.4**)		

KW: Kruskal Wallis, *: p<0,05, Dif.(Difference): Tukey's multiple comparison test CS: Chi-Square Neu: Neutrophils, Lym: Lymphocytes, PLT: Platelets, , Mon: Monocytes HDL: High-density lipoprotein, CRP: C-reactive protein, NLR: Neutrophil/lymphocyte ratio, , PLR: Platelet/lymphocyte ratio, MHR: Monocyte/HDL ratio, SII: Systemic immune-inflammation index

than those of the individuals with IR in the obesity and control groups. In association with these findings, insulin levels of the individuals with IR in the obesity and control groups were significantly higher than those of the individuals without IR in the obesity group.

Several changes occur in obesity, including ectopic lipid accumulation in non-adipose tissue and alterations in the distribution of leukocyte populations, the activity of lymphocytes, and the general immune defense (14). Cruz-Pineda et al. presented evidence that IR might impair the metabolism and functioning of leukocytes and that leukocytes might significantly contribute to the development of systemic IR (15). In our study, neutrophil and monocyte counts of obese individuals with IR were significantly higher than those of the non-IR individuals in the control and obesity groups. Hernández Vera et al. showed in their study that obesity is associated with increased platelet counts and that obesity is associated with significantly increased thrombosis in the presence of IR (16). Similarly, in our study, we found significantly higher platelet counts in obese individuals with IR compared to the non-IR individuals in the control group. Previous studies have shown the association of CRP levels with metabolic disorders such as IR, obesity, type 2 diabetes, and reported increased CRP levels in obese individuals (17,18). We found that CRP levels were significantly higher in the obesity groups compared to healthy controls. Increased levels of hematological inflammatory parameters in obese individuals with IR, who are free from chronic diseases, suggest the existence of a robust relationship between IR and inflammation.

NLR, PLR, MHR, and SII are inexpensive and easily calculated indices that have been introduced recently for the diagnosis and the prediction of prognosis in systemic disorders including chronic inflammatory diseases, malignancies, coronary artery disease, and diabetes mellitus (19-21). In our study, NLR was significantly higher in obese individuals with IR compared to non-IR obese counterparts. Similarly, Karakaya et al. found significantly higher NLR levels in obese individuals with IR compared to the non-IR obese group (22). While some studies have shown significantly increased PLR levels in obese individuals, others have reported no correlations or a low level of correlation of PLR with the amount of adipose tissue (23-25). In our study, we did not find a significant difference in PLR between the groups. Battaglia et al. showed that MHR and NLR were significantly increased in patients with metabolic syndrome, and reported that BMI and hyperglycemia were important variables affecting MHR (26). In another study, a positive correlation was found between the BMI and SII levels of patients (24). In our study, we found that MHR and SII levels in obese individuals with IR were significantly higher than non-IR individuals in the obesity and control groups. These results show that IR in obesity is associated with increases in the levels of indicators of chronic inflammation including NLR, MHR, and SII.

The establishment of the hereditary nature of blood group antibodies and antigens has led researchers to investigate potential relationships and susceptibility between ABO blood groups and increased BMI and obesity. In our study, we found that individuals with the AB blood type in the obesity group constituted a significantly lower percentage compared to the percentage of their counterparts in the control group. Similarly, Parveen et al. compared BMI values and blood group types in their study and found the lowest BMI values among individuals with the AB blood type (27). In a study evaluating the relationship between ABO blood types and the type 2 diabetes risk, it was shown that A and B blood types were at a higher risk of developing type 2 diabetes compared to individuals with the blood type O (28). Another study has shown that those with the B blood type were more susceptible to develop diabetes and obesity (29). In our study, in the obesity group, the rates of IR and the neutrophil counts were significantly higher in individuals with the B blood type compared to those with the O blood type. The similarities between our study results and those reported by previous studies show that blood type B may be involved in obesity and IR susceptibility.

In conclusion, we found high levels of hematological inflammatory parameters and indicators of systemic chronic inflammation including NLR, MHR, and SII in the group of obese individuals with IR. Furthermore, we have shown that the B blood type is associated with a higher risk for the development of IR and inflammation compared to the O blood type among obese individuals. We have found that the AB blood type occurs at a significantly lower percentage among obese individuals compared to the control group. We think that our results show a robust relationship between inflammation associated with excess adipose tissue and the development of IR in obese individuals. We think that our study will pave the way for the elucidation of the pathophysiological mechanisms involved in obesity and IR and that further studies on this subject matter will contribute to early diagnosis and the development of innovative modes of treatment.

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