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The effect of insulin resistance on House-Brackmann grade of facial paralysis in patients with Bell's palsy

İnsülin direncinin Bell's palsy hastalarında House-Brackman evrelemesi üzerine etkisi

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Abstract

Aim: Bell's palsy is the most common cause of facial nerve lesion and is frequently observed in diabetic patients and the duration of healing is delayed in diabetic patients. To investigate the effect of insulin resistance on the facial paralysis as graded by the House-Brackmann classification in patients with Bell's palsy and to determine the importance of insulin resistance assessed primarily prior to treatment planning.

Methods: Patients admitting to our emergency department and outpatient neurology clinic with suspected facial paralysis who were not administered steroid therapy within the first 24 hours were studied. Demographic data were collected for patients with Bell's palsy from different age groups with a normal body mass index (BMI) and no chronic endocrine disease. The House-Brackmann (HB) grading scale was used to assess the clinical severity of the facial paralysis. In addition to routine laboratory tests, fasting insulin level was obtained to estimate HOMA-IR (Homeostatic model assessment for insulin resistance) for all patients.

Results: Of 19 patients enrolled, 10 were female (52.6%). Patients had a mean age of 33 years, mean glucose value of 106 mg/dL, mean insulin value of 15.9 µU/mL and mean HOMA-IR value of 4.1. A moderate positive correlation and a statistically significant association were found between mean glucose values and HOMA-IR values (r=0.548; p=0.015) Age and glucose values were not statistically significantly associated with insulin and HOMA-IR values (p=0.858 and p=0.015, respectively).

Conclusion: Higher blood glucose and average insulin levels were found in patients with facial nerve paralysis in comparison to general population. Most of the patients were IR-positive. Therefore, assessment of insulin resistance would be beneficial for both treatment planning and taking proactive measures against future development of diabetes in all patients presenting with Bell's palsy.

Keywords: Insulin resistance, House-Brackmann, Facial paralysis

Öz

Amaç: Bell's palsi fasial sinir lezyonun en sık nedenidir ve diyabetli hastalarda sık izlenir ve iyileşme süresi diyabetli hastalarda gecikmiştir. Bell's palys geçiren hastalarda insulin resistansının House – Brackman evrelemesi üzerine etkisini araştırmak ve bu araştırma sonucunda Bell' palsi tedavisi planlamadan önce öncelikli olarak insulin direncinin bakılmasının önemini araştırmak.

Yöntemler: Çalışmaya fasial paralizi şüphesiyle ilk 24 saat içinde acile veya polikliniğe başvuran ve bu nedenle steroid almamış hastalar dahil edildi. Bell's palys'li hastaların demografik verileri toplanıldı. Endokrin hastalığı olmayan çeşitli yaş gruplarında normal vücut kitle endexi olan hastalar çalışmaya alındı. Fasia paralizinin derecesinin değerlendirilmesi için House-Brackman klinik skalası kullanıldı. Tüm hastaların rutin laboratuar testlerine ek olarak açlık insülin seviyeleri bakıldı ve HOMA-IR değeri hesaplandı.

Sonuçlar: Çalışmaya alınan 19 hastanın 10'u kadın (%52,6) diğeri erkekti. Çalışmaya katılan hastaların ortlama yaşı 33 glukoz değeri ise 106 (mg/dL), ortalama insulin değeri 15,9 (µU/mL) ve ortalama HOMA-IR değeri ise 4,1 idi. Ortalama glucose değeri ile HOMA-IR değeri arasında istatistiksel olarak pozitif yönde orta şiddette anlamlı korelasyon saptanmıştır (r=0,548; p=0,015) Yaş ve glucose değerleri ile insülin ve HOMA-IR değerleri arasında istatistiksel olarak anlamlı bir ilişkiye rastlanmamıştır (sırasıyla p=0,858, p=0,015).

Tartışma: Fasial sinir felci ilen başvuran hastaların çoğunun kan şekeri yüksek ve ortlama insulin değerleri toplumdan daha fazla izlendi. Hastaların büyük çoğunluğunda IR pozitifliğine rastlanıldı. Bu yüzden Bell's palsi ile başvuran tüm hastlarda insulin rezsitansına bakıklmasını ve tedavi planın ve gelecekte hastada gelişebilecek bir diyabet açısından dikkatli olunmasını önermekteyiz.

Anahtar kelimeler: İnsülin resistansı, House-Brackman, Fasial paralizi

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Introduction

Bell's palsy (BP) is the most common cause of facial nerve paralysis. In this condition, the muscles on one side of the face suddenly become weak or paralyzed due to malfunction of the 7th cranial nerve. This causes drooping of the affected side of the face and inability to keep one eye closed, and the face is drawn across to the opposite side on smiling. For most people, Bell's palsy is temporary. Symptoms usually start to improve within a few weeks, with complete recovery in about six months. A small number of people continue to have some Bell's palsy symptoms for life. Rarely, Bell's palsy can recur. Globally, approximately 11–40/100,000 people are affected by BP every year [1,2]. It has been found that there are no sex-related differences in its prevalence. Viral infections, vascular ischemia, disorder of the autonomic regulation and inflammation have been implicated in BP [3]. Controversies still exist about the incidence and exact cause of BP. Some authors reported increased incidence of BP among people who are aged between 30 and 39 years and a tendency for BP to occur more frequently during the colder seasons (autumn and winter) [4, 5]. However, other researchers showed an increased rate of BP during warmer spring months [6,7].

Insulin resistance (IR) is a condition in which the body's cells become resistant to the effects of insulin in the blood circulation. As a result, the normal response to a given amount of insulin is reduced. IR may develop in obese, non-diabetic individuals as well as patients with type 2 diabetes mellitus [8]. A transient IR state occurs physiologically during puberty as part of normal development and pregnancy to maintain adaptation and homeostasis. IR may be present without comorbidities such as obesity or hypertension. It was shown that IR may also occur in non-obese individuals with normal glucose tolerance. Abnormalities in the concentration or affinity of the insulin receptors or both result in impaired insulin effectiveness [9].

In the present study, clinical grading of BP patients was performed using the House-Brackmann (HB) Facial Nerve Grading System (Table 1) and IR was estimated by HOMA-IR (Homeostatic model assessment for IR) to assess whether IR affected HB grading of the patients [10].

Materials and methods

Non-diabetic patients with Bell's palsy and a normal BMI who admitted to Gaziantep Sanko University Medical Faculty in 2017 were included in the study. A retrospective chart review was conducted. Patients admitting to our emergency department and outpatient neurology clinic with suspected facial paralysis who were not administered steroid therapy within the first 24 hours were studied. Patients were mostly enrolled in the fall season.

Patients with paralysis, secondary to trauma, zoster oticus, hypertensive cerebral hemorrhage, diabetes and cerebellopontine angle tumors were excluded. Data including gender, age, month of disease onset and clinical features were recorded. The House-Brackmann (HB) grading scale was used to assess the clinical severity of the facial palsy (Table 1).

Table 1: House-Brackmann scale ranges between I (normal) and VI (no movement)

Grade	Explanation
I	Normal symmetrical function
II	Slight weakness noticeable only on close inspection
	Complete eye closure with minimal effort
	Slight asymmetry of smile with maximal effort
	Synkinesis barely noticeable, contracture, or spasm absent
III	Obvious weakness, but not disfigurment
	May not be able to lift eyebrows
	Complete eye closure and strong but asymmetrical mouth movement
	Obvious, but not disfiguring synkinesis, mass movement, or spasm
IV	Obvious disfiguring weakness
	Inability to lift eyebrows
	Incomplete eye closure and asymmetry of mouth with maximal effort
	Severe synkinesis, mass movement, or spasm
V	Motion barely perceptible
	Incomplete eye closure, slight movement at mouth corner
	Synkinesis, contracture, and spasm usually absent
VI	No movement, loss of tone, no synkinesis, contracture, or spasm

Medical history, age, and gender data were recorded by an expert neurologist. Height and body weight were measured to obtain body mass index (BMI; kg/m2) and age- and sex-specific BMI percentiles. Associations between average doses of study medications and blood glucose, insulin and HOMA-IR values were examined in relation to age and gender. IR was calculated using the HOMA-IR formula of fasting insulin (μU/mL) x fasting glucose (mmol/L) / 405 using a cut-off value of 2.5. The homeostasis model assessment of IR (HOMA-IR) is a non-invasive and effective alternative method to evaluate insulin sensitivity based on the glucose level and the level of serum insulin measured in fasting conditions. HOMA-IR is considered a standard method of measuring IR in epidemiological studies [11].

Statistical Analysis

Normality of numerical data was tested by Shapiro-Wilk test. For normally distributed numerical data, one-way analysis of variance (ANOVA) and LSD (least significant difference) multiple comparison tests were used to compare 2 independent groups. Kruskal-Wallis test and all pairwise multiple comparison tests were used for non-normally distributed data. Relationships between independent categorical variables were tested by Chi-square test and relationships between numerical variables were tested by Spearman's rank correlation coefficient. For descriptive statistics, mean ±standard deviation and median (Inter quartile range 25%-75%) values were presented for numerical variables and number and percentage (%) for categorical variables. All analyses were performed using SPSS for Windows, version 24.0 and a p value equal and smaller than 0.05 was considered significant.

Results

Of 19 patients enrolled, 10 were female (52.6%). Patients had a mean age of 33 years, mean glucose value of 106 mg/dL, mean insulin value of 15.9 μU/mL and mean HOMA-IR value of 4.1 (Table 2). HOMA-IR was greater than 2.5 in 14 (73.7%) patients, all of whom were found to have insulin resistance. The remaining five patients did not have insulin resistance. Based on HB grading, moderate dysfunction of the facial nerve was found in 9 (47.4) patients and 4 (%21.1) patients had moderately severe dysfunction (Table 3). A moderate positive correlation and a statistically significant association were found between mean glucose value and HOMA-IR value ($r=0.548$; $p=0.015$). Age and glucose values were not statistically significantly associated with insulin and HOMA-IR values

(p=0.858 and p=0.015, respectively, Table 4). Comparison of HBS classes in terms of age, glucose, insulin and HOMA-IR values did not yield any statistically significantly differences between these variables (p=0.791, p=0.715, p=0.405 and p=0.693, respectively; Table 5). HB grade was not significantly associated with sex (p=0.454). Similarly, HB grade was not associated with IR either (p=0.710; Table 6).

Table 2: General identifier for numeric variables

Variable (n=19)	M [Q_1 Q_3]	Mean±SD
Age	33.00 [30.00 50.00]	37.000±18.00
Glucose (mg/dL)	106.00 [95.00 118.00]	112.74±33.52
Insulin (µU/mL)	15.90 [10.00 51.00]	30.25±30.33
HOMA-IR	4.10 [2.32 11.00]	6.90±6.38

Q_1: First quartile (25%), Q_3: Third quertile (75%), M: Median, SD: Standard deviation, HOMA-IR: Homeostatic model assessment for insulin resistance

Table 3: Demographic characteristics of patients

Variable		n	%
Gender	Male	9	47.4
	Female	10	52.6
HOMA-IR	IR+	14	73.7
	IR-	5	26.3
HBS	Slight Dysfunction	3	15.8
	Moderate Dysfunction	9	47.4
	Moderately Severe Dysfunction	4	21.1
	Severe Dysfunction	3	15.8

HOMA-IR: Homeostatic model assessment for insulin resistance, HBS: House-Brackmann Scale

Table 4: Relationship between age, glucose insulin and HOMA-IR

		Insulin	HOMA-IR
Age (years)	r	0.018	-0.044
	p	0.941	0.858
	n	19	19
Glucose (mg/dL)	r	0.301	0.548*
	p	0.211	0.015
	n	19	19

*(p<0.05), HOMA-IR: Homeostatic model assessment for insulin resistance

Table 5: Comparison of HBS classes in terms of age, glucose, insulin and HOMA-IR

Variable		n	Mean±SD	Test Statistics	p
Age	Slight Dysfunction	3	32.33±2.52	F=0.348	0.791
	Moderate Dysfunction	9	41.67±17.18		
	Moderately Severe Dysfunction	4	33.25±15.19		
	Severe Dysfunction	3	33.00±33.05		
	Total	19	37.05±17.67		
Glucose (mg/dL)	Slight Dysfunction	3	100.00±6.25	F=0.458	0.715
	Moderate Dysfunction	9	122.33±37.98		
	Moderately Severe Dysfunction	4	103.50±10.38		
	Severe Dysfunction	3	109.00±57.38		
	Total	19	112.74±33.52		
Insulin (µU/mL)	Slight Dysfunction	3	16.66±1.60	x^2= 2.917	0.405
	Moderate Dysfunction	9	42.39±36.51		
	Moderately Severe Dysfunction	4	27.03±31.23		
	Severe Dysfunction	3	11.70±2.07		
	Total	19	30.25±30.33		
HOMA-IR	Slight Dysfunction	3	4.88±1.65	x^2=1.454	0.693
	Moderate Dysfunction	9	8.80±7.28		
	Moderately Severe Dysfunction	4	7.08±8.35		
	Severe Dysfunction	3	2.93±1.04		
	Total	19	6.89±6.38		

F: ANOVA test, x^2: Kruskal-Wallis Test, HOMA-IR: Homeostatic model assessment for insulin resistance

Table 6: Comparison of categorical variables

		HBE			
		Slight Dysfunction	Moderate Dysfunction	Moderately Severe Dysfunction	Severe Dysfunction
		Count	Count	Count	Count
Gender	Male	2	3	3	1
	Female	1	6	1	2
		x^2= 2.621 P=0.454			
HOMA-IR	Yes	3	6	3	2
	None	0	3	1	1
		x^2= 1.380 P=0.710			

HOMA-IR: Homeostatic model assessment for insulin resistance, HBS: House-Brackmann Scale

Discussion

IR is a pathogenic factor for type 2 diabetes mellitus (DM) [12]. Increased insulin secretion and chronic hyperinsulinemia can develop when pancreatic beta cells can no longer compensate and maintain glucose homeostasis, leading to the development of type 2 DM [13]. In adults, a cut-off value of 2.5 is generally used for HOMA-IR. Another study in 691 apparently healthy Indian adolescents (aged 10–17 years) established a HOMA-IR cut-off of 2.5 [14]. In a prevalence study, IR was positive in 28.9% of females and 25.1% of males [15]. In one study, on average, 25% of overweight individuals had insulin resistance; however, in the present study, 52.5% of the total study sample tested positive for IR although our patients had normal body weight [16]. In contrast to previous studies, 73.7% of our patients tested positive for insulin resistance. IR coexists with neurodegenerative and infectious diseases, and metabolic abnormalities such as obesity and type 2 diabetes are frequently associated with underlying immune disorders. A higher prevalence of neurodegenerative diseases was shown in individuals with IR [17]. Vascular endothelium maintains the balance between vasodilation and vasoconstriction and IR may cause hypertension by disrupting the balance of active endocrine functions of the vascular endothelium [18]. Animal and human studies demonstrated inhibition of insulin signaling after exposure to oxidative stress at the cellular level [19] and established the link between IR and oxidative stress. In insulin resistance, plasminogen activator inhibitor-1 increases the risk for macrovascular disease by elevating Factor VII, Factor VIII, von-Willebrand factor and fibrinogen levels [20]. A study found a greater incidence of infection among patients with IR [21]. Previous studies have reported that HSV (herpes simplex virus), as an opportunistic infection agent, may cause BP in IR-positive patients and it seems likely that BP might be a manifestation of prediabetes. In one study, recovery from Bell's palsy in a diabetic group was found to be delayed in comparison to a nondiabetic group and the authors stated that more aggressive treatments might be considered in diabetic patients with severe Bell's palsy [22]. In another study, the recovery rate of BP was significantly lower in the group with metabolic syndrome (MS) than in the non-MS group and was particularly affected by diabetes mellitus obesity and high triglycerides [23]. Consistently, a separate study showed a tendency for incomplete recovery from BP among diabetic patients [24]. Aforementioned three studies also a statistically non-significant effect of high blood glucose on BP and HB grade in the diabetic group compared to the non-diabetic group, supporting our finding. In a review study involving 372 cases; it was found that patients with facial palsy most commonly presented with HB grades III and IV [25]. Similarly, in the present study, our patients presented mostly with HB grades III and IV. In the review study mentioned above, the highest incidence of BP was identified in patients between 39 and 50 years of age.

Consistently, the mean age of our BP patients was 33 years. Limitations of our study include lack of follow-up of patients during the treatment process and the failure to investigate the effect of IR on the duration of treatment. Other limitation of this study is that the number of patients is low, patients are not followed for a long time and therefore the recovery period of patients with IR is not monitored. In this sense there is a need for further study.

IR is a metabolic disorder which is closely related to obesity and abdominal adipose tissue mass. Further studies are needed to clarify the pathogenesis of insulin resistance. Polygenic etiology of IR should be investigated by genetic research. Prevention of IR might be possible in the future if its

mechanisms are fully described through molecular studies. Increasing evidence demonstrate the strong relation between insulin signaling pathways and complications of insulin resistance. It is crucial to prevent and reduce obesity in order to be able to overcome insulin resistance. IR may be present in apparently healthy individuals. Screening targeted to individuals at risk for IR may allow early detection of the condition. IR may be easily diagnosed using HOMA-IR formula based on fasting insulin and fasting glucose levels. Potentially severe complications of IR might be prevented by early diagnosis and lifestyle changes including dietary modification, regular exercise, and weight loss. Longstanding IR accompanied by obesity will inevitably lead to single or multiple manifestations of type 2 diabetes mellitus, cardiovascular diseases, hypertension and cancer. Estimation of pretreatment HOMA-IR value would be beneficial for both treatments planning and taking proactive measures against future development of diabetes in patients presenting with facial paralysis.

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