The Journal of International Advanced Otology

J Int Adv Otol 2025; 21: 1–8 • DOI: 10.5152/iao.2025.241759

Original Article

The Impact of High Glycated Hemoglobin A1c on Prognosis in Patients with Bell's Palsy: A Propensity Score Matchingatched Analysis

Erhui Yu[1], Binyan Yu[2], Fanyuan Jin[1], Huafeng Cai[1], Jinhua Hu[1], Yingtong Chen[1], Runcheng Wang[1], Xiuzhen Xie[1], Shuhan Yang[1], Lihua Xuan[2]

[1]The First School of Clinical Medicine, Zhejiang Chinese Medical University, Zhejiang, China
[2]The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine), Hangzhou, Zhejiang, China

ORCID iDs of the authors: E.Y. 0009-0005-6420-2933, B.Y. 0000-0001-6680-5133, F.J. 0009-0007-9357-5610, H.C. 0009-0001-2552-7138, J.H. 0009-0003-3249-0235, Y.C. 0009-0002-2841-3782, R.W. 0009-0007-4974-2055, X.X. 0009-0009-1450-4035, S.Y. 0009-0002-0143-6239, L.X. 0000-0002-1257-1244.

Cite this article as: Yu E, Yu B, Jin F, et al. The impact of high glycated hemoglobin A1c on prognosis in patients with Bell's palsy: a propensity score-matched analysis. *J Int Adv Otol.* 2025, 21(2), 1759, doi: 10.5152/iao.2025.241759.

BACKGROUND: Glycated hemoglobin A1c (HbA1c) is an indicator of blood glucose levels, but the impact of hyperglycemia on Bell's palsy (BP) remains unclear. This study aims to assess the influence of high and low HbA1c levels on the prognosis of patients with BP.

METHODS: This monocentric, retrospective study included 712 patients with BP, divided into 103 patients with HbA1c ≥ 6.5% and 609 patients with HbA1c < 6.5%. Receiver operating characteristic curve analysis was used to evaluate the main factors affecting HbA1c levels. Propensity score matching (PSM) was further utilized to avoid selection bias and disproportionate distributions of confounding factors between the 2 groups. The House–Brackmann (H–B) facial nerve grading system was employed to assess the severity of facial motor dysfunction.

RESULTS: Analysis showed that high HbA1c patients were older, had higher body mass index, less frequently suffered from dysgeusia, and more often had hypertension ($P < .05$). According to the area under the curve, age had the greatest impact on HbA1c levels (95% CI = 0.748-0.803, $P < .001$). After PSM 1 : 1 matching, there was no statistical difference in initial H–B grade between the 2 groups, but there was a statistical difference in final H–B grade ($P = .023$), indicating a worse prognosis for patients with BP in the high HbA1c group.

CONCLUSION: The study, after controlling for confounding factors, showed that patients with BP and high HbA1c have a worse prognosis, suggesting that controlling blood glucose levels has a positive significance for the recovery of patients with BP.

KEYWORDS: Bell's palsy, HbA1c, H–B grade, prognosis, propensity score matching

INTRODUCTION

Bell's palsy (BP) is characterized by an idiopathic paralysis of the peripheral facial nerve that primarily innervates the majority of the facial musculature.[1] Bell's palsy is readily discernible in clinical settings, typically portends a favorable outcome, and is considered not to impact the patient's longevity. The etiology of BP is predominantly correlated with herpes simplex virus, ischemia, inflammation, and immunological disorders.[2]

Many studies aimed to focus on various clinical factors related to the recovery of BP, encompassing age, body mass index (BMI) categorization, concurrent underlying pathologies, therapeutic interventions, and duration of hospitalization. Across numerous investigations, age has been identified as a pivotal determinant in the prognosis of BP, with younger patients exhibiting more expedited recovery and a favorable prognosis.[3] Similarly, the convalescence from BP posed a relatively greater therapeutic challenge in patients with obesity.[4,5] Furthermore, the recuperation from facial paralysis in pregnant women was likewise inferior to that in their non-gravid counterparts.[6] Despite extensive research and investigation, there are still no determinable factors.

Corresponding author: Lihua Xuan, e-mail: xlh1083@163.com
Received: October 15, 2024 • **Revision Requested:** November 11, 2024 • **Last Revision Received:** November 12, 2024 • **Accepted:** November 25, 2024 • **Publication Date:** March 24, 2025
Available online at www.advancedotology.org

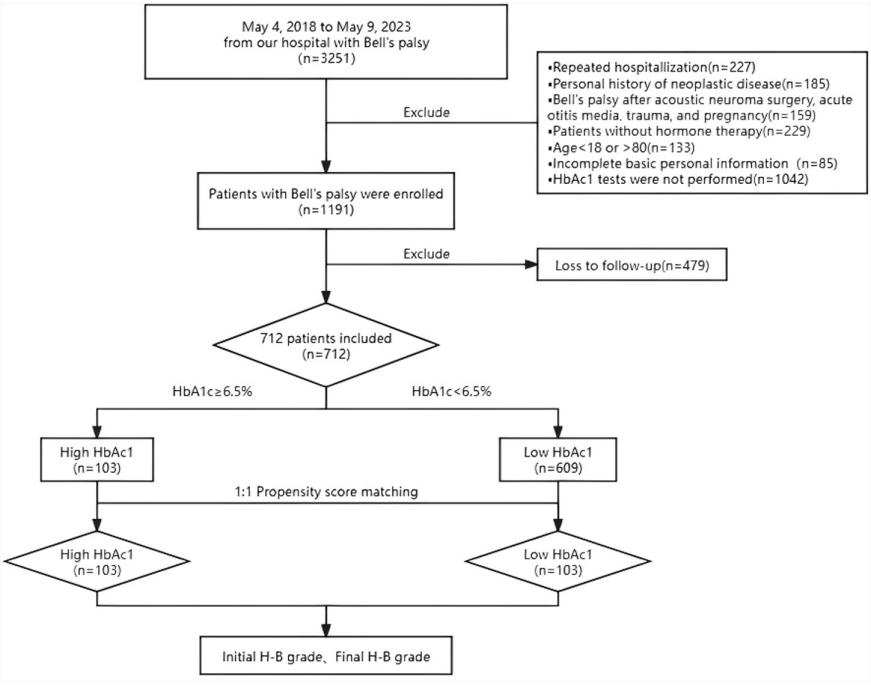

Figure 1. Flow diagram of the study design. HbA1c, glycated hemoglobin A1c; PSM, propensity score matching.

The incidence of BP combined with diabetes was relatively high. A large national cohort study in Korea, including 153 845 patients with BP, reported that the incidence of diabetes was 12.8%.[7] Another study with a smaller sample size (730 cases) reported an even higher prevalence of 43.8%.[8] Diabetes is a high-risk factor leading to poor prognosis in BP. Studies have suggested that the recovery rate in the diabetes group was significantly lower than in the non-diabetes group 6 months after the onset of BP.[9] Psillas's research found that the recovery rate for BP patients with diabetes was only 58.8%, while it was 78.3% for the control group.[10] The actual temporal relationship between the onset of BP and the development of diabetes is still unclear. It remains to be revealed whether neuropathy exacerbates hyperglycemia, diabetes induces neural edema, or both occur simultaneously.

We found that almost all studies on the prognosis of BP had significant limitations due to non-matching subjects, leading to recall bias based on patient descriptions, and failing to eliminate other confounding factors, which affected the accuracy of the study results. Therefore, we conducted this study to analyze variables potentially related to the outcomes of BP through a large sample size. We then used propensity score matching (PSM) to control these confounding variables, thereby further analyzing the impact of high or low HbA1c levels on the prognosis of BP, assessing whether a hyperglycemic state is associated with poor recovery from facial paralysis in patients with BP.

MAIN POINTS

- Bell's palsy patients with high HbA1c have a worse prognosis than those with low HbAc1.
- No significant difference in initial H–B grades between high and low HbA1c groups.
- Age was the most relevant factor associated with HbA1c levels.

METHODS

Study Population and Design

In our retrospective study, patients diagnosed with BP and admitted to The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine), Hangzhou, Zhejiang from May 4, 2018, to May 9, 2023, were included. Of the primary 3251 BP patients, a total of 1191 patients were included in the study. Other patients were excluded based on the following criteria: (1) repeated hospitalization; (2) personal history of neoplastic disease; (3) BP after acoustic neuroma surgery, acute otitis media, trauma, and pregnancy; (4) patients without hormone therapy; (5) age <18 or >80; (6) incomplete basic personal information; and (7) HbA1c tests were not performed. Ultimately, after excluding patients lost to follow-up, the study incorporated a total of 712 BP patients. The selection process is outlined in Figure 1.

All 712 patients with BP were treated with glucocorticoids, neurotrophic drugs, and traditional acupuncture. The hormonal treatment regimen was based on the magnetic resonance imaging and facial electroneuronography (fEnoG).[11] Oral prednisone at a dosage of 30 mg/d (initial dose) for 5 days, followed by a gradual reduction, or intravenous dexamethasone at a dosage of 5-10 mg/d (initial dose), followed by a gradual reduction to oral prednisone. Neurotrophic drugs were administered orally with Mecobalamin 0.5 mg, 3 times daily, continuously until symptoms improved. Acupuncture therapy was administered at specific points including Cuanzhu (BL2), Yangbai (GB14), Taiyang (EX-HN5), Quanliao (SI18), Xiaguan (ST7), Dicang (ST4), and Jiache (ST6) on the affected side, along with bilateral Hegu (LI4). The needles were retained for 30 minutes, with sessions conducted once daily for a total of 10 sessions.

Data Collection and Outcome Evaluation

We utilized a retrospective hospital electronic case system to gather demographic and clinical data from patients. Initial health

information was meticulously collected, including age, gender, height, weight, onset of disease, complications, underlying diseases, and history of alcohol consumption and smoking. Additionally, pretreatment hematological parameters (HbA1c) and the severity of facial motor impairment before and after treatment were recorded. At least 3 professional clinicians assessed facial function using the House–Brackmann (H–B) grading system during admission and follow-up visits post discharge, categorizing it as mild (H–B I-III), moderate (H–B III-IV), or severe (H–B V-VI). All patients underwent a follow-up examination at least 6 months post treatment to determine their final recovery status.

This retrospective study received approval from the institutional review board of Zhejiang Provincial Hospital of Chinese Medicine, Hangzhou, Zhejiang (approval no.: 2024-KL-070-01, date: 2024-02-27). Given the nature of the study as retrospective, the decision was made to waive the need for written informed consent.

Statistical Analysis

We used the *t*-test for independent samples to compare continuous variables between high and low HbA1c groups, and the X^2 test for categorical variables. Groups were matched using PSM at a 1 : 1 ratio with a caliper of 0.25, considering age, BMI, comorbidities, and hypertension. Successful matching was indicated by a standardized mean difference (SMD) <0.1. Statistical significance was set at $P < .05$. Analysis was performed using SPSS version 26 (IBM SPSS Corp.; Armonk, NY, USA) and R Studio version 2023.12.1 (Posit Software, PBC Corp.; Boston, Massachusetts, USA).

RESULTS

Demographical and Clinical of Patients' Characteristics Before Propensity Score Matching

Statistical analysis revealed that there were statistically significant differences between the 2 groups in terms of age, degree of BMI, dysgeusia, and hypertension (Table 1). Specifically, the average age of patients in the high HbA1c group was 53.88 ± 12.34 years, which was significantly higher than the average age of 42.51 ± 14.81 years in the low HbA1c group ($P < .01$), indicating an older demographic in the high HbA1c group. In terms of weight, the proportion of overweight patients in the high HbA1c group was 67.96%, compared to 49.92% in the low HbA1c group. This suggested that the BMI index was generally higher in the high HbA1c group, indicating a greater tendency toward obesity ($P < .01$). Regarding taste disorders, the high HbA1c group had a lower proportion at 22.33% compared to the low HbA1c group's 34.32%. Additionally, the percentage of patients with hypertension in the high HbA1c group (37.86%) was significantly higher than that in the low HbA1c group (17.57%).

Identify the Most Relevant Factor Associated with Glycated Hemoglobin A1c Levels

In an effort to identify factors associated with HbA1c levels, we conducted a ROC curve analysis. The findings revealed that the area under the curve (AUC) for age was 0.775 (95% CI = 0.748-0.803, $P < .001$), a value that surpassed the AUC of all other pertinent factors. This suggested that, among the myriad of confounding factors impacting HbA1c levels in patients with BP, age held the strongest correlation and was the most significant influencer.

Table 1. Demographical and Clinical Characteristics Before PSM

	Total (n = 712)	Low HbAc1 (n = 609)	High HbAc1 (n = 103)	*P*
Age, mean ± SD	44.15 ± 15.01	42.51 ± 14.81	53.88 ± 12.34	<.001*
Gender, n (%)				.072
Male	305 (42.84)	265 (43.51)	40 (38.83)	
Female	407 (57.16)	344 (56.49)	63 (61.17)	
Degree of BMI, n (%)				.002*
Skinny	14 (1.97)	14 (2.30)	0 (0.00)	
Normal	324 (45.51)	291 (47.78)	33 (32.04)	
Overweight	374 (52.53)	304 (49.92)	70 (67.96)	
Side, n (%)				.220
Left	357 (50.14)	297 (48.77)	60 (58.25)	
Right	354 (49.72)	311 (51.07)	43 (41.75)	
Bilateral	1 (0.14)	1 (0.16)	0 (0.00)	
The timing of onset, n (%)				.171
<7 day	326 (45.79)	272 (44.66)	54 (52.43)	
7 day-3 months	352 (49.44)	305 (50.08)	47 (45.63)	
>3 months	34 (4.78)	32 (5.25)	2 (1.94)	
Complication, n (%)				
Hearing impairment	164 (23.03)	146 (23.97)	18 (17.48)	.147
Dysgeusia	232 (32.58)	209 (34.32)	23 (22.33)	.016*
Comorbidities, n (%)				
Hypertension	146 (20.51)	107 (17.57)	39 (37.86)	<.001*
Neurasthenia	47 (6.60)	42 (6.90)	5 (4.85)	.440
Social history, n (%)				
Smoke	91 (12.78)	81 (13.30)	10 (9.71)	.313
Drink	37 (5.20)	33 (5.42)	4 (3.88)	.516
Initial H–B grade, n (%)				.412
Mild (I-II)	2 (0.28)	2 (0.33)	0 (0.00)	
Moderate (III-IV)	307 (43.12)	256 (42.04)	51 (49.51)	
Severe (V-VI)	403 (56.60)	351 (57.64)	52 (50.49)	
Final H–B grade, n (%)				.393
Mild (I-II)	402 (56.46)	349 (57.31)	53 (51.46)	
Moderate (III-IV)	306 (42.98)	256 (42.04)	50 (48.54)	
Severe (V-VI)	4 (0.56)	4 (0.66)	0 (0.00)	

BMI, body mass index; H–B grade, House–Brackmann grade; HbA1c, glycated hemoglobin A1c; PSM, propensity score matching.
*$P < .05$

Comparasion of Patients' Characteristics After Propensity Score Matching

After 1 : 1 PSM, there were no statistically significant differences in patient age or other factors between the groups (Table 2). The analysis of standardized mean differences indicated that all covariates had values less than 0.1, suggesting that the matching was satisfactory. Furthermore, the coincidence of propensity score distributions was higher after PSM matching (Figure 2A and B), indicating an improved comparability between the 2 cohorts.

Table 2. Clinical Characteristics Before and After PSM

Variable	Before PSM				After PSM			
	Low HbAc1 (n = 609)	High HbAc1 (n = 103)	*P*	SMD	Low HbAc1 (n = 103)	High HbAc1 (n = 103)	*P*	SMD
Age, mean ± SD	42.51 ± 14.81	53.88 ± 12.34	<.001*	0.922	53.57 ± 12.35	53.88 ± 12.34	.857	0.025
Degree of BMI, n (%)			.002*				.650	
Skinny	14 (2.30)	0 (0.00)		−0.166	0 (0.00)	0 (0.00)		
Normal	291 (47.78)	33 (32.04)		−0.337	30 (29.13)	33 (32.04)		0.062
Overweight	304 (49.92)	70 (67.96)		0.387	73 (70.87)	70 (67.96)		−0.062
Complication, n (%)								
Dysgeusia	209 (34.32)	23 (22.33)	.016*	−0.288	21 (20.39)	23 (22.33)	.734	0.047
Comorbidities, n (%)								
Hypertension	107 (17.57)	39 (37.86)	<.001*	0.418	34 (33.01)	39 (37.86)	.466	0.100

BMI, body mass index; H–B grade, House–Brackmann grade; HbA1c, glycated hemoglobin A1c; PSM, propensity score matching.
*$P < .05$.

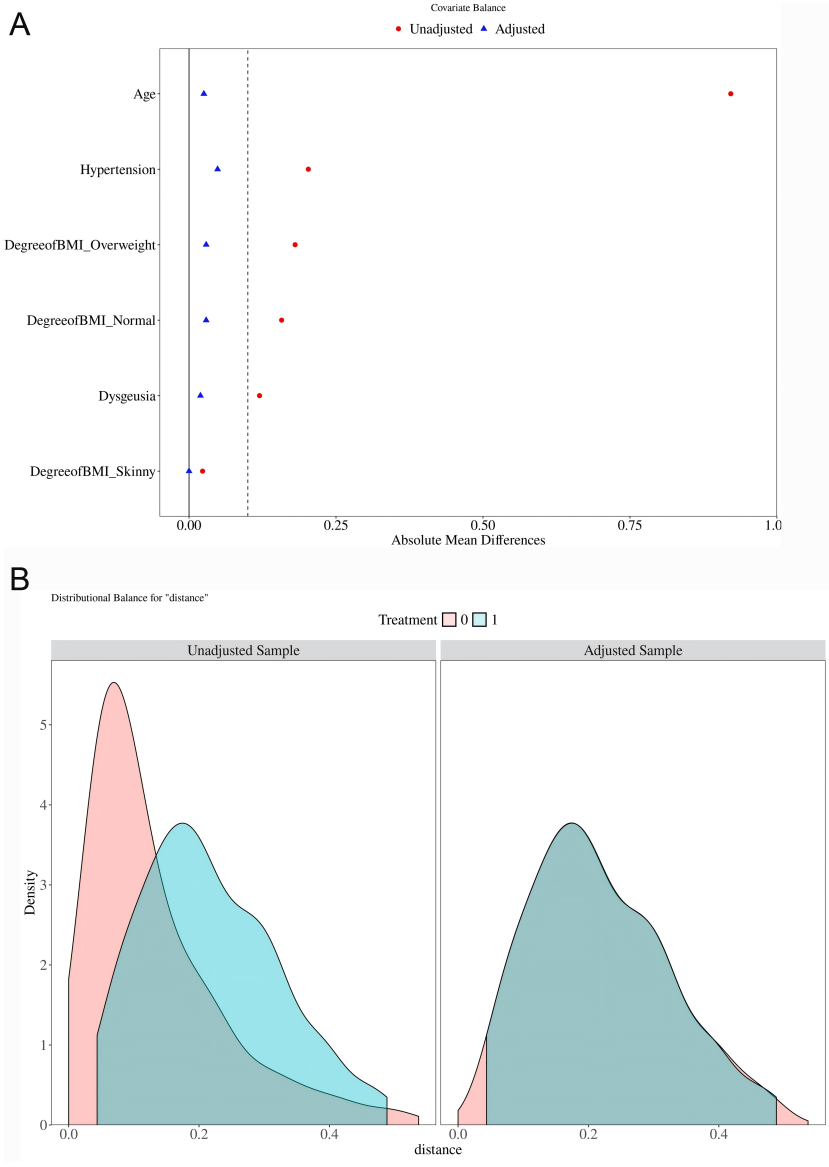

Figure 2. A. The assessment of covariate balance for standardized mean difference. B. The probability density image. H–B grade, House–Brackmann grade; HbA1c, glycated hemoglobin A1c; PSM, propensity score matching.

Bell's Palsy Patients' Initial and Final House–Brackmann Grade After Propensity Score Matching

The results indicated that there was no statistical difference in the initial H–B grade between the high HbA1c group and the matched low HbA1c group (P = .208). However, after treatment, the H–B grade of the 2 groups showed a statistically significant difference (P = .023), with fewer patients in the high HbA1c group recovering to mild facial paralysis compared to the low HbA1c group (Table 3), and a higher proportion of patients remaining with severe paralysis post treatment (Figure 3A and B).

DISCUSSION

In this study, by employing a PSM 1 : 1 matching method, we effectively controlled for potential confounding factors associated with elevated HbA1c levels. Based on this, we found that patients with higher HbA1c levels had a significantly poorer prognosis compared to those with lower HbA1c levels. This outcome suggested that among BP patients with comorbid hyperglycemia, a greater number failed to fully recover. Additionally, our study confirmed that age was the most significant factor influencing the level of HbA1c.

HbA1c is broadly recognized as an index of average blood glucose concentrations, indicative of the average blood glucose level over the preceding 2-3 months.[12] In 2010, the American Diabetes Association underscored the importance of HbA1c levels, mandating a threshold of 6.5% or higher as a principal diagnostic standard for diabetes.[13] China also agreed with this diagnostic threshold.[14] Thus, we conducted the study utilizing the HbA1c standardized at 6.5%. Our investigation revealed a significant correlation between elevated HbA1c levels (≥6.5%) and suboptimal facial functional recovery (HB > II). A large-scale Korean study indicated that the risk of acute facial nerve paralysis was heightened in diabetic patients compared to non-diabetic individuals.[15] Kanazawa et al[9] discovered that the recovery process in diabetic patients with BP was slower and their facial paralysis scores were higher compared to non-diabetic individuals with the condition. In addition, the association between diabetes and different types of peripheral facial paralysis varied. One study found that among patients with persistent hyperglycemia, those with Ramsay Hunt syndrome showed significantly better recovery rates than those with BP.[16] However, some studies believed that the presence of hyperglycemia had no significant impact on the prognosis of BP.[17] Şevik Eliçora and Erdem,[18] after following BP patients for 1-3 years, found that hyperglycemia did not substantially influence the severity, recovery rate, or rate of recuperation in BP. Hence, there was still some controversy about the relationship between hyperglycemia and BP. This diversity could stem from the varying case selection criteria across studies, as well as the influence of age, weight, and other variables. Our study, after minimizing the possibility of bias, bolstered this theory that hyperglycemia could contribute to adverse outcomes in individuals with BP. Additionally, Mantsopoulos reported that a critical factor affecting the prognosis of BP was the initial severity of facial weakness.[19] However, in our study, the initial severity of facial weakness exhibited no substantial disparity between the high HbA1c and low HbA1c groups, indicating that elevated HbA1c or hyperglycemia primarily affected the long-term prognosis of patients' facial paralysis rather than the incidence of BP.

Table 3. Facial Function Grading for Patients After PSM

Variables	Total (n = 206)	Low HbAc1 after PSM (n = 103)	High HbAc1 (n = 103)	Statistic	P
Initial H–B grade				X^2 = 1.59	.208
Mild (I-II)	0 (.00)	0 (.00)	0 (.00)		
Moderate (III-IV)	93 (45.15)	42 (40.78)	51 (49.51)		
Severe (V-VI)	113 (54.85)	61 (59.22)	52 (50.49)		
Final H–B grade				X^2 = 5.15	.023*
Mild (I-II)	122 (59.22)	69 (66.99)	53 (51.46)		
Moderate (III-IV)	84 (40.78)	34 (33.01)	50 (48.54)		
Severe (V-VI)	0 (.00)	0 (.00)	0 (.00)		

H–B grade, House–Brackmann grade; HbA1c, glycated hemoglobin A1c; PSM, propensity score matching; X^2, chi-square test.
*P < .05.

Hyperglycemia was considered a factor contributing to an unrecoverable prognosis in patients with BP. The etiology of BP was complex and believed to primarily encompass 5 mechanisms: ischemia, inflammation, anatomical variations, viral infections, and cold exposure.[20] Within these mechanisms, hyperglycemia may exacerbate local ischemia, augment inflammatory responses, and compromise the body's resistance to viruses, thereby causing damage to the facial nerve. Initially, it was posited that hyperglycemia could lead to microvascular changes. Persistent elevation of blood glucose levels may harm vascular endothelial cells, resulting in vascular dysfunction and causing ischemia of the facial nerve. Upon reperfusion, the damaged microvasculature might be unable to regulate blood flow properly, leading to an unstable blood supply to the facial nerve, which further exacerbated neural degeneration and triggered Bell's palsy.[21,22] Secondly, hyperglycemia affected the immune system and inflammatory responses, posing a risk factor for metabolic disorders and being closely associated with neurological diseases. Research indicated that under hyperglycemic conditions, increased oxidative and nitrative stresses, activation of the polyol pathway and protein kinase C pathway, as well as gene activation related to neuronal damage, all potentially contributed to facial nerve injury.[23-25] Ultimately, impaired glucose regulation diminished the immune system's capacity to combat infections, leading to increased susceptibility to viral infections. Insulin itself was considered a molecule capable of directly modulating immune cells, particularly T-cell function. However, in patients with hyperglycemia, there was a reduction in the expression of insulin receptors on T cells, which hindered the proliferation of antiviral T cells and the production of cytokines, making individuals more prone to infections.[26] Moreover, in the era of the novel coronavirus (COVID-19) pandemic, diabetes was one of the most common comorbidities among patients with severe acute respiratory syndrome, further underscoring the susceptibility of individuals with hyperglycemia to viral infections.[27,28]

In retrospective studies, PSM is becoming an increasingly popular method to control factors. Our study used PSM to control for the influence of confounding variables in order to determine the

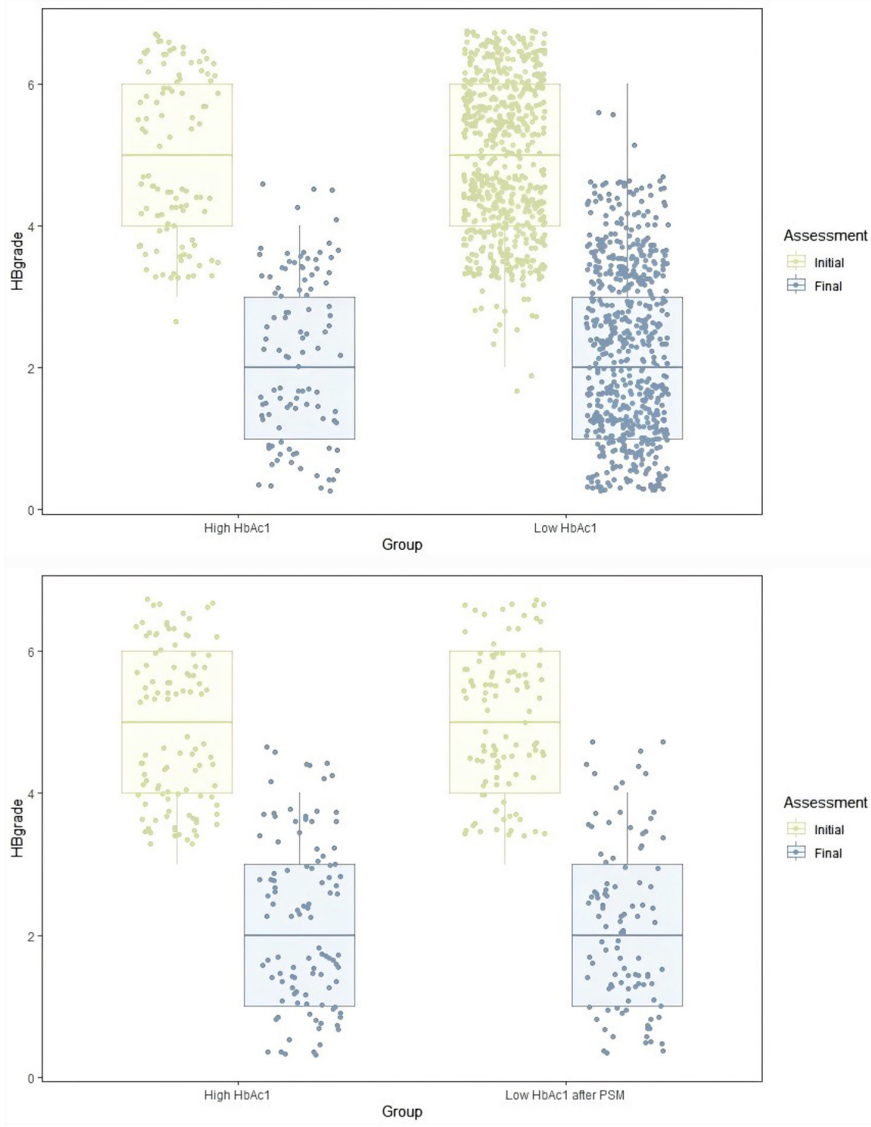

Figure 3. A. Comparison of H–B grade between high and low HbA1c group before PSM. B. Comparison of H–B grade between high and low HbA1c group after PSM. H–B grade, House–Brackmann grade; HbA1c, glycated hemoglobin A1c; PSM, propensity score matching.

relationship between HbA1c and BP prognosis. Characteristics included age, degree of BMI, dysgeusia, and hypertension. Age was regarded as a significant determinant influencing the initial severity and prognosis of BP.[29] Amalanathan reported that individuals over the age of 40 may experienced incomplete recovery from BP.[30] Additional research had also found that individuals with obesity, indicated by a high BMI, had a lower recovery rate from BP compared to people of normal weight.[31] However, age and BMI were also closely related to diabetes. Consequently, it became challenging for some studies to conclusively demonstrate that diabetes was the causative factor of poor prognosis in BP. It was plausible that individuals with hyperglycemia were generally older and have a higher BMI, which could directly impact the unfavorable outcome in patients with BP. Hypertension was also a significant factor related to the prognosis of BP. It may disrupt the intricate equilibrium of the pressure system within the facial canal, leading to impaired canal circulation and consequently causing neural damage.[32,33] Patients with BP and comorbid high HbA1c exhibit markedly diminished gustatory impairment compared to patients with low HbA1c, a result that further corroborated the vascular pathogenesis theory in BP. The vasculature irrigating the distal facial nerve segments may be more implicated in hyperglycemic patients, who were prone to activate supplementary compensatory mechanisms to preserve gustatory function.[34,35]

A notable strength of our study lay in its successful neutralization of potential confounding variables, such as age and BMI, which could have skewed the outcomes. Leveraging a substantial dataset from within the institute over a 5-year span, our research conducted a methodical self-comparative analysis. To our knowledge, this constituted the inaugural report to exclusively appraise the impact of elevated HbA1c on the prognosis of BP after nullifying factors like age and BMI. The findings underscored the potential significance of hyperglycemia and its associated complications within the realm of neurology. Consequently, these revelations underlined the importance of glucose level management to enhance patient prognosis during the treatment of BP.

The investigation conducted by our team does indeed have limitations. First, due to the retrospective cohort design, there may be a risk of recall bias. Secondly, the investigation was confined to a solitary center, potentially restricting its broader applicability.

Based on our study findings, clinicians should be vigilant regarding the risk of BP in the diabetic population, as it may portend a less favorable prognosis and augment the probability of ensuing sequelae. Judicious regulation and administration of hyperglycemia could potentially mitigate the linked prognostic dangers, thereby underscoring the imperative of prompt diagnosis and intervention for patients with hyperglycemia concurrently presenting with BP.

CONCLUSION

Individuals afflicted with BP and elevated HbA1c levels tended to have a relatively worse prognosis, suggesting that hyperglycemia may impact the outcome of BP, with age being the most significant factor affecting the level of HbA1c.

Availability of Data and Materials: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: This study was approved by the Ethics Committee of Zhejiang Provincial Hospital of Chinese Medicine (approval no.: 2024-KL-070-01; date: February 27, 2024).

Informed Consent: Given the nature of the study as retrospective, it was decided to waive the requirement for written informed consent.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – E.Y., L.X.; Design – E.Y., L.X.; Supervision – L.X.; Resources – L.X.; Materials – L.X.; Data Collection and/or Processing – E.Y., F.J., H.C., J.H., Y.C., R.W., X.X., S.Y.; Analysis and/or Interpretation – E.Y.; Literature Search – E.Y.; Writing – E.Y.; Critical Review – B.Y.

Declaration of Interests: The authors assert that the research was carried out devoid of any commercial associations, which could potentially lead to a perceived conflict of interest.

Funding: The authors declared that this study received no financial support.

REFERENCES

1. Yoo MC, Soh Y, Chon J, et al. Evaluation of factors associated with favorable outcomes in adults with Bell palsy. *JAMA Otolaryngol Head Neck Surg*. 2020;146(3):256-263. [CrossRef]
2. Monini S, Lazzarino AI, Iacolucci C, Buffoni A, Barbara M. Epidemiology of Bell's palsy in an Italian Health District: incidence and case-control study. *Acta Otorhinolaryngol Ital*. 2010;30(4):198.
3. Lovin BD, Sweeney AD, Chapel AC, Alfonso K, Govil N, Liu YCC. Effects of age on delayed facial palsy after otologic surgery: a systematic review and meta-analysis. *Ann Otol Rhinol Laryngol*. 2022;131(10):1092-1101. [CrossRef]
4. Breitling V, Leha A, Schiller S, Kruizenga M, Gärtner J, Rosewich H. Association of overweight and obesity with Bell palsy in children. *Pediatr Neurol*. 2023;139:43-48. [CrossRef]
5. Ragaban A, Alsharif L, Alshaikh NA, et al. Prevalence, etiology, risk factors, and complications of facial nerve palsy at King Abdulaziz Medical City: a multicenter study. *Cureus*. 2024;16(2):e53403. [CrossRef]
6. Evangelista V, Gooding MS, Pereira L. Bell's palsy in pregnancy. *Obstet Gynecol Surv*. 2019;74(11):674-678. [CrossRef]
7. Lee JS, Kim YH. Epidemiological trends of Bell's palsy treated with steroids in Korea between 2008 and 2018. *Muscle Nerve*. 2021;63(6):845-851. [CrossRef]
8. Kim JY, Kim MS, Kim MH, Kim DK, Yu MS. Bell palsy and the risk of cardiocerebrovascular disease: a population-based follow-up study. *Laryngoscope*. 2019;129(10):2371-2377. [CrossRef]
9. Kanazawa A, Haginomori SI, Takamaki A, Nonaka R, Araki M, Takenaka H. Prognosis for Bell's palsy: a comparison of diabetic and nondiabetic patients. *Acta Otolaryngol*. 2007;127(8):888-891. [CrossRef]
10. Psillas G, Dimas GG, Sarafidou A, et al. Evaluation of effects of diabetes mellitus, hypercholesterolemia and hypertension on Bell's palsy. *J Clin Med*. 2021;10(11):2357. [CrossRef]
11. Jin F, Yu E, Chen J, et al. Monocyte to high-density lipoprotein ratio as a novel-potential biomarker for predicting prognosis of Bell's palsy. *Eur Arch Otorhinolaryngol*. 2024;281(5):2293-2301. [CrossRef]
12. Ding L, Xu Y, Liu S, Bi Y, Xu Y. Hemoglobin A1c and diagnosis of diabetes. *J Diabetes*. 2018;10(5):365-372. [CrossRef]
13. American Diabetes Association. Standards of medical care in diabetes--2010 [published correction appears in *Diabetes Care*. 2010;33(3):692]. *Diabetes Care*. 2010;33(suppl 1):S11-S61. [CrossRef]
14. Jia W, Weng J, Zhu D, et al. Standards of medical care for type 2 diabetes in China 2019. *Diabetes Metab Res Rev*. 2019;35(6):e3158. [CrossRef]
15. Seo HW, Ryu S, Lee SH, Chung JH. Diabetes mellitus and acute facial palsy: a nationwide population-based study. *Neuroepidemiology*. 2024;58(1):37-46. [CrossRef]
16. Kim SH, Jung J, Jung SY, et al. Comparative prognosis in patients with Ramsay-Hunt syndrome and Bell's palsy. *Eur Arch Otorhinolaryngol*. 2019;276(4):1011-1016. [CrossRef]
17. Riga M, Kefalidis G, Danielides V. The role of diabetes mellitus in the clinical presentation and prognosis of Bell palsy. *J Am Board Fam Med*. 2012;25(6):819-826. [CrossRef]
18. Şevik Eliçora S, Erdem D. Does type 2 diabetes mellitus affect the healing of Bell's palsy in adults? *Can J Diabetes*. 2018;42(4):433-436. [CrossRef]
19. Mantsopoulos K, Psillas G, Psychogios G, Brase C, Iro H, Constantinidis J. Predicting the long-term outcome after idiopathic facial nerve paralysis. *Otol Neurotol*. 2011;32(5):848-851. [CrossRef]
20. Zhang W, Xu L, Luo T, Wu F, Zhao B, Li X. The etiology of Bell's palsy: a review. *J Neurol*. 2020;267(7):1896-1905. [CrossRef]
21. Nukada H. Ischemia and diabetic neuropathy. *Handb Clin Neurol*. 2014;126:469-487. [CrossRef]
22. Cameron NE, Eaton SE, Cotter MA, Tesfaye S. Vascular factors and metabolic interactions in the pathogenesis of diabetic neuropathy. *Diabetologia*. 2001;44(11):1973-1988. [CrossRef]
23. Mendonca HR, Carpi-Santos R, da Costa Calaza K, Blanco Martinez AM. Neuroinflammation and oxidative stress act in concert to promote neurodegeneration in the diabetic retina and optic nerve: galectin-3 participation. *Neural Regen Res*. 2020;15(4):625-635. [CrossRef]
24. Pop-Busui R, Ang L, Holmes C, Gallagher K, Feldman EL. Inflammation as a therapeutic target for diabetic neuropathies. *Curr Diab Rep*. 2016;16(3):29. [CrossRef]
25. Liston SL, Kleid MS. Histopathology of Bell's palsy. *Laryngoscope*. 1989;99(1):23-26. [CrossRef]
26. Turk Wensveen T, Gašparini D, Rahelić D, Wensveen FM. Type 2 diabetes and viral infection; cause and effect of disease. *Diabetes Res Clin Pract*. 2021;172:108637. [CrossRef]
27. Rizvi AA, Kathuria A, Al Mahmeed W, et al. Post-COVID syndrome, inflammation, and diabetes. *J Diabetes Complications*. 2022;36(11):108336. [CrossRef]
28. Sun B, Huang S, Zhou J. Perspectives of antidiabetic drugs in diabetes with coronavirus infections. *Front Pharmacol*. 2021;11:592439. [CrossRef]

29. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol Suppl*. 2002;(549):4-30.
30. Amalanathan S, Colbert KR, Kumar CS, Mathyalagen P. Clinical prognostic indicators in predicting the outcome in patients with Bell's palsy: a descriptive, longitudinal study from Puducherry, South India. *Indian J Otolaryngol Head Neck Surg*. 2022;74(suppl 3):4270-4275. [CrossRef]
31. Choi SA, Shim HS, Jung JY, et al. Association between recovery from Bell's palsy and body mass index. *Clin Otolaryngol*. 2017;42(3):687-692. [CrossRef]
32. MacArthur AM, Minson S. Facial nerve paralysis in hypertension: answers. *Pediatr Nephrol*. 2021;36(2):305-306. [CrossRef]
33. Liu YN, Yang LY, Xue ZW, Zhou S. Association between hypertensive disorders and Bell's palsy in pregnancy: protocol for a systematic review and meta-analysis. *BMJ Open*. 2024;14(5):e080322. [CrossRef]
34. Stamatiou I, Papachristou S, Papanas N. Diabetes mellitus and Bell's palsy. *Curr Diabetes Rev*. 2023;19(1):e080322201913. [CrossRef]
35. İnan S, Jafarov S. Prognostic role of neutrophil lymphocyte ratio and mean platelet volume in Bell's palsy: comparison of diabetic and non-diabetic patients. *Braz J Otorhinolaryngol*. 2023;89(1):98-103. [CrossRef]