Journal of
*Clinical Medicine*

MDPI

*Article*

# Association Between Presenting Bell's Palsy and Subsequent Diabetes Mellitus: A Retrospective Cohort Study

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## Abstract

**Background**: Bell's palsy may be a marker of impaired glucose homeostasis. We aimed to study the time progression to diabetes (TPD) in non-diabetic patients presenting with Bell's palsy. **Methods**: This was a retrospective cohort, and the study matched Bell's palsy cases with controls in a 1:1 manner by age-, sex-, and presentation date. Baseline systolic and diastolic blood pressures (SBP, DBP), body mass index (BMI), fasting blood sugar (FBS), glycated hemoglobin (HbA1c), and the date of diabetes diagnosis were collected. Overall diabetes-free survival time (DFS) was assessed via survival analysis. Independent factors associated with DFS were evaluated using Cox proportional hazard regression ($p < 0.05$). **Results**: Bell's palsy cases exhibited significantly elevated FBS, SBP, DBP, and BMI, as well as shorter overall DFS (log-rank $\chi^2 = 12.6$, $p < 0.001$). The mean DFS was 15.0 years (95% CI 14.2–15.8) in the cases vs. 17.9 years (95% CI 17.4–18.3) in the controls. Bell's palsy patients with high BMI levels showed shorter DFS than did the controls (log-rank $\chi^2 = 18.6$, $p < 0.001$). Bell's palsy patients and controls with elevated FBS showed significantly shorter DFS (log-rank $\chi^2 = 6.2$, $p = 0.046$, and log-rank $\chi^2 = 38.0$, $p < 0.001$, respectively). The Cox proportional hazards model revealed that SBP, DBP, and BMI were significant in the unadjusted analysis, whereas no independent associated factors could be identified. **Conclusions**: Although a causal relationship between Bell's palsy and DM remains inconclusive, screening and longitudinal monitoring of FBS or HbA1c among patients presenting with Bell's palsy are advised.

**Keywords:** Bell's palsy; diabetes mellitus; body mass index; cardiometabolic; prediabetes

Academic Editor: Walter M. Plasencia

Received: 24 September 2026
Revised: 18 November 2026
Accepted: 21 November 2026
Published: 26 November 2026

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## 1. Introduction

Bell's palsy is a well-recognized unilateral facial neuropathy, but its etiology and pathogenesis remain non-definite. Based on the concurrence of herpes labialis and facial palsy, reactivation of latent herpes simplex virus infection in the geniculate ganglion of the facial nerve is usually introduced [1]. Later studies additionally reported that cardiometabolic disorders (CMDs) such as elevated blood pressure (BP), insulin resistance (IR), impaired fasting blood sugar (IFBS), diabetes mellitus (DM), dyslipidemia, and metabolic syndrome (MS) were associated with, or possibly contributed to, Bell's palsy [2–4]. Due to the different study settings and population, the incidence of DM in Bell's palsy was reported from 2.5–11% [5,6]. Two local hospital-based studies done by one of

the authors (PS) showed that DM was associated with Bell's palsy in 4.9 and 6% of cases [7,8]. The specific timeline or causal relationship between CMDs and Bell's palsy has not been comprehensively investigated, as most of the available studies were cross-sectional or enrolled a small sample size. Since the development of overt DM in an individual requires a long time from prediabetes state, i.e., IR and IFBS, to overt DM, whether the patients presenting with Bell's palsy have already acquired a prediabetes condition remains unanswered. Some studies suggested that Bell's palsy patients had a higher than expected incidence of associated prediabetes state [9–11]. While the other mono-cranial neuropathies, i.e., oculomotor, trochlear or abducens nerve involvement commonly presented as a complication of pre-existing DM, most of the patients presenting with Bell's palsy showed unremarkable fasting blood sugar levels [12–15]. It is possible that impaired glucose homeostasis or pre-diabetes state has already emerged in Bell's palsy patients and is progressing to overt DM over a variable length of time. Therefore, Bell's palsy is a possible marker of pre-diabetes state, finally converting to overt DM. The conversion to overt DM depends on an individual's glucose homeostasis tolerance. A population-based cohort study demonstrated an independent association between Bell's palsy and subsequent diabetes, as well as diabetes-related cardiometabolic complications across all age groups, particularly those aged 40 or older. Moreover, diabetic nephropathy and retinopathy were further addressed among this group of patients [16]. These findings support the hypothesis of microvascular thrombosis of the vasa nervorum as an etiopathogenesis of Bell's palsy. Furthermore, three retrospective cohort studies demonstrated a higher incidence of ischemic stroke among the patients previously experiencing Bell's palsy [17–19]. Hence, the "vascular etiology" of Bell's palsy describing facial nerve ischemia deserves more attention in addition to that given to the original theory of herpes virus reactivation.

This 1:1 age-, sex-, and presentation date-matched case-control study aimed to compare the overall time of progression to DM (TPD) between Bell's palsy cases and their matched controls. The influence of body mass index (BMI) and states of fasting blood sugar (FBS) associated with Bell's palsy on TPD were also evaluated. We proposed that Bell's palsy was possibly an isolated cranial nerve palsy preceding overt DM. The occurrence of Bell's palsy may be a marker of the existence of impaired glucose homeostasis. We expected that the results of this study would alert physicians treating the Bell's palsy patients to the possible development of DM and related CMDs in the future and prompt the performance of diabetes screening and regular monitoring among Bell's palsy patients.

## 2. Material and Methods

### *2.1. Study Setting and Design*

This was a 1:1 age-, sex-, date of Bell's palsy diagnosis (cases) and hospital attendance (controls)-matched case control and retrospective cohort study conducted at Songklanagarind Hospital, which is the medical teaching university hospital affiliated with the Faculty of Medicine, Prince of Songkla University, in southern Thailand. The hospital is a tertiary-care hospital serving the people in lower southern Thailand. The Hospital Information System (HIS), the well-designed computerized medical data record system, was the source for study data retrieval.

### *2.2. Study Population*

The study population comprised patients presenting with Bell's palsy (ICD-10 code: G51.0) at the outpatient department (OPD) of the Department of Medicine (cases), and those presenting for regular check-ups (ICD-10 code: Z00.0) at the OPD of the

Department of Family and Preventive Medicine (controls). The patients, aged ≥ 35 years of both sexes, were identified from HIS using the ICD-10 diagnosis codes mentioned. The diagnosis of Bell's palsy was based on the presentation of acute unilateral lower motor neuron facial palsy, reaching maximal facial paralysis within 3 days from the onset, without attributable causes [3]. The patients who had a diagnosis of diabetes mellitus (DM) 3 months before or after the presentation date were excluded from this study.

### 2.3. Sample-Size Calculation

Schoenfeld's formula for sample-size calculation for proportional hazards regression was used. Assuming relative hazards between 1.5 and 2.0, $\alpha = 0.05$, $\beta = 0.20$, equal group sizes ($q_1 = q_0 = 0.5$), and the required number of events ranging from 65 to 191 were specified. Given a censoring rate of 20% and a follow-up duration of at least 5 years, the estimated sample size was 662 participants (331 patients each for cases and controls).

### 2.4. Data Collection

The cases and controls were identified from the HIS using ICD-10 codes G51.0 and Z00, respectively. Cases and controls were matched by age (±1 year)-, sex- and presentation date (±3 months). The computer-assisted software was applied to randomize the matched controls from the check-up patients pool. We enrolled the eligible cases and matched controls from 1 January 2005 to 31 December 2017 and followed the development of diabetes mellitus (DM) until 31 December 2024. The date of diagnosis with Bell's palsy (cases) and the date of OPD visit (controls) were matched with the predefined time frame (±3 months). Those who did not develop diabetes or who were lost follow-up at any time until the end of follow-ups (31 December 2024) were censored using Kaplan–Meier survival analysis.

The data collected in this study included general demographics, body mass index (BMI), systolic and diastolic blood pressure (SBP, DBP), fasting blood sugar (FBS) and low-density lipoprotein cholesterol (LDL-C), HbA1c, and prescription of prednisolone treatment, among the Bell's palsy cases. The study data were stored in an encrypted electronic database accessible only by the investigators, with personal passwords required.

### 2.5. Terms and Definitions

Diabetes mellitus (DM) was diagnosed according to the American Diabetes Association (ADA) criteria, i.e., fasting plasma glucose (FBS) ≥ 126 mg/dL on at least two occasions, glycated hemoglobin (HbA1c) ≥ 6.5%, or random plasma glucose ≥ 200 mg/dL, with typical symptoms of hyperglycemia.

Diabetes-free survival time (DFS) was the duration (years) from the presentation date of Bell's palsy cases or matched controls to the date of first diagnosis with DM until the end of follow-up (31 December 2024).

Censored patients from the Kaplan–Meier survival analysis were those who did not develop DM or who were lost follow-ups at any time until the end of the follow-up.

Body mass index (BMI, $kg/m^2$) was classified according to the World Health Organization criteria for Asians, i.e., non-obese (<23 $kg/m^2$), overweight (23–24.9 $kg/m^2$), obese class I (25–29.9 $kg/m^2$), and obese class II (≥30 $kg/m^2$).

Fasting blood sugar (FBS) states based on ADA classification comprised normal (<100 mg/dL) and impaired fasting blood sugar (IFBS) (100–125 mg/dL).

Prednisolone treatment was the receiving of oral prednisolone for treatment of Bell's palsy, which was usually 1 mg/kg/d for 1 week, followed by a taper-down dosage until cessation over the next week.

### 2.6. Statistical Analysis

Descriptive statistics such as frequency (percentage), median (IQR), Mann–Whitney U test, and Chi-square test results were used for evaluation of significant difference between groups ($p < 0.05$). Kaplan–Meier analysis was used to estimate DFS. Median survival or restricted mean survival time was reported, where appropriate. Group differences were assessed by log-rank test ($p < 0.05$). Subgroup analyses evaluating the effects of combined BMI and FBS with Bell's palsy on DM occurrence were further conducted. Stratified Cox proportional hazards models were used in univariate analysis to test the significant association of individual variables with DFS. Then, multivariate Cox regression analysis by directly adjusting for the matching covariates (age and sex) within an unstratified multivariable Cox proportional hazards framework was done. Results were reported as hazards ratios (HRs) and adjusted HRs with 95% confidence intervals (Cis) ($p < 0.05$). The analyses in this study were performed in R studio version 4.0 via Jupyter® Notebook using the survival and survminer packages.

### 2.7. Strategies to Handle Missing Data, Sensitivity Analysis, and Collinearity and Proportional Hazards Assumption Assessment (Supplementary Tables S1–S5)

From our preliminary data cleaning, we found a concerning volume of missing data that might interfere with the statistical analysis and the integrity of statistical power. Hence, we applied measures to assess and manage missing data, including events per variables (EPV), complete-case analysis, multiple imputation by chained equation (MICE), and collinearity assessment by variance inflation factor (VIF). The proportional hazards assumption was formally tested using Schoenfeld residuals ( Supplementary Tables S1–S5).

We constructed separate bivariate Cox proportional hazards models, each containing only the primary exposure (Group: Bell's palsy vs. control) and one specific metabolic variable. By evaluating metabolic covariates in separate parsimonious models (group + age + sex + single metabolic factor; total df = 4), our effective EPV remained between 15.75 and 63.0, which was well above the stability threshold of 10.

According to the sensitivity analysis, the hazard ratios between the complete-case analysis and multiple imputation models remain entirely concordant.

All covariates demonstrated a VIF below the strict threshold of 2.5 (approaching 1.0), which confirmed the complete absence of multicollinearity between the exposure group and the respective metabolic variables.

The proportional hazards assumption was formally tested for all revised models using Schoenfeld residuals. The global test for each model yielded a $p$-value greater than 0.05, which confirmed that the hazards remain proportional over time, satisfying the fundamental assumption of the Cox model.

### 2.8. Ethical Approval

The study was conducted in compliance with the updated version of the Declaration of Helsinki and its related guidelines. The study protocol was reviewed and approved for ethical clearance by the Ethics Committee of Prince of Songkla University (protocol code: REC.66-263-14-4; date of approval: 24 July 2023). The personal information of the study participants was completely anonymized. Study analyses were done in aggregation to ensure the confidentiality of the participants.

## 3. Results

### 3.1. General Characteristics

Of 702 participants, 351 were equally distributed in the case and matched control

groups. Bell's palsy patients had significantly elevated FBS, SBP, DBP and BMI. Also, there were significant differences in the proportion of BMI and FBS classifications between cases and controls. From the overall follow-ups, ranging from 0.5 to 19 years, a total of 63 new DM cases were diagnosed, in which 38 and 25 were Bell's palsy cases and controls, respectively. (Table 1).

**Table 1.** Baseline characteristics of the study participants.

| | **Bell's Palsy** | **Control** | ***p*-Value** |
|---|---|---|---|
| | (*n* = 351) | (*n* = 351) | |
| Age, year (median [IQR]) | 52.42 [43.73–61.34] | 52.71 [43.72–61.36] | 0.951 |
| Female sex, *n* (%) | 186 (53) | 186 (53) | |
| Prednisolone treatment, *n* (%) | 297 (84.6) | 0 | |
| Metabolic variables, median (IQR ) | | | |
| HbA1c (%) [a] | 5.65 [5.33–5.98] (*n* = 16) | 5.8 [5.57–6.05] (*n* = 10) | 0.278 |
| FBS (mg/dL) [a] | 96 [90–104.75] (*n* = 124) | 94 [89–100] (*n* = 318) | 0.026 ** |
| LDL (mg/dL) [a] | 148.01 [122–147.33] (*n* = 78) | 139.03 [120.4–160.94] (*n* = 190) | 0.218 |
| SBP (mmHg) [b] | 135 [122–147.33] (*n* = 191) | 123.67 [112.5–137.0] (*n* = 303) | <0.001 ** |
| DBP (mmHg) [b] | 81 [73.7–88.33] (*n* = 191) | 73.5 [67–82.33] (*n* = 303) | <0.001 ** |
| BMI (kg/m$^2$) [b] | 24.88 [22.74–28.15] (*n* = 184) | 23.85 [21.43–26.08] (*n* = 303) | <0.001 ** |
| BMI classifications, *n* (%) | | | <0.001 * |
| Non-obese (<23 kg/m$^2$) | 52 (28.3) | 129 (42.6) | |
| Overweight (23–24.9 kg/m$^2$) | 41 (22.3) | 64 (21.1) | |
| Obese class I (25–29.9 kg/m$^2$) | 67 (36.4) | 92 (30.4) | |
| Obese class II (>30 kg/m$^2$) | 24 (13.0) | 18 (5.9) | |
| N/A | 167 | 48 | |
| FBS classifications, *n* (%) | | | <0.001 * |
| Normal (<100 mg/dL) | 74 (59.7) | 237 (74.5) | |
| IFBS (101–125 mg/dL) | 50 (40.3) | 81 (25.5) | |
| N/A | 227 | 33 | |
| Diabetes mellitus, *n* | 38 | 25 | |

[a] The dates of test performance did not differ by more than 1 year between cases and controls. [b] The dates of the measurements performance did not differ by more than 3 months between cases and controls. N/A, data were not available. * Chi-square test $p < 0.05$; ** Mann–Whitney U test $p < 0.05$.

### 3.2. Comparison of Overall DFS Between Bell's Palsy Cases vs. Controls

The Kaplan–Meier curve (Figure 1) showed that the Bell's palsy patients had a significantly shorter overall DFS than that of their matched controls (log-rank $\chi^2 = 12.6$, $p < 0.001$). The cumulative incidence of diabetes cases diagnosed at 5, 10, and 15 years were 6.4%, 17.2%, and 26.2%, respectively, among the patients with Bell's palsy compared with 2.4%, 6.7%, and 10.5% among the controls. The mean DFS estimated by the Kaplan–Meier method was 15.0 years (95% CI 14.2–15.8) in the Bell's palsy cases and 17.9 years (95% CI 17.4–18.3) in the controls.

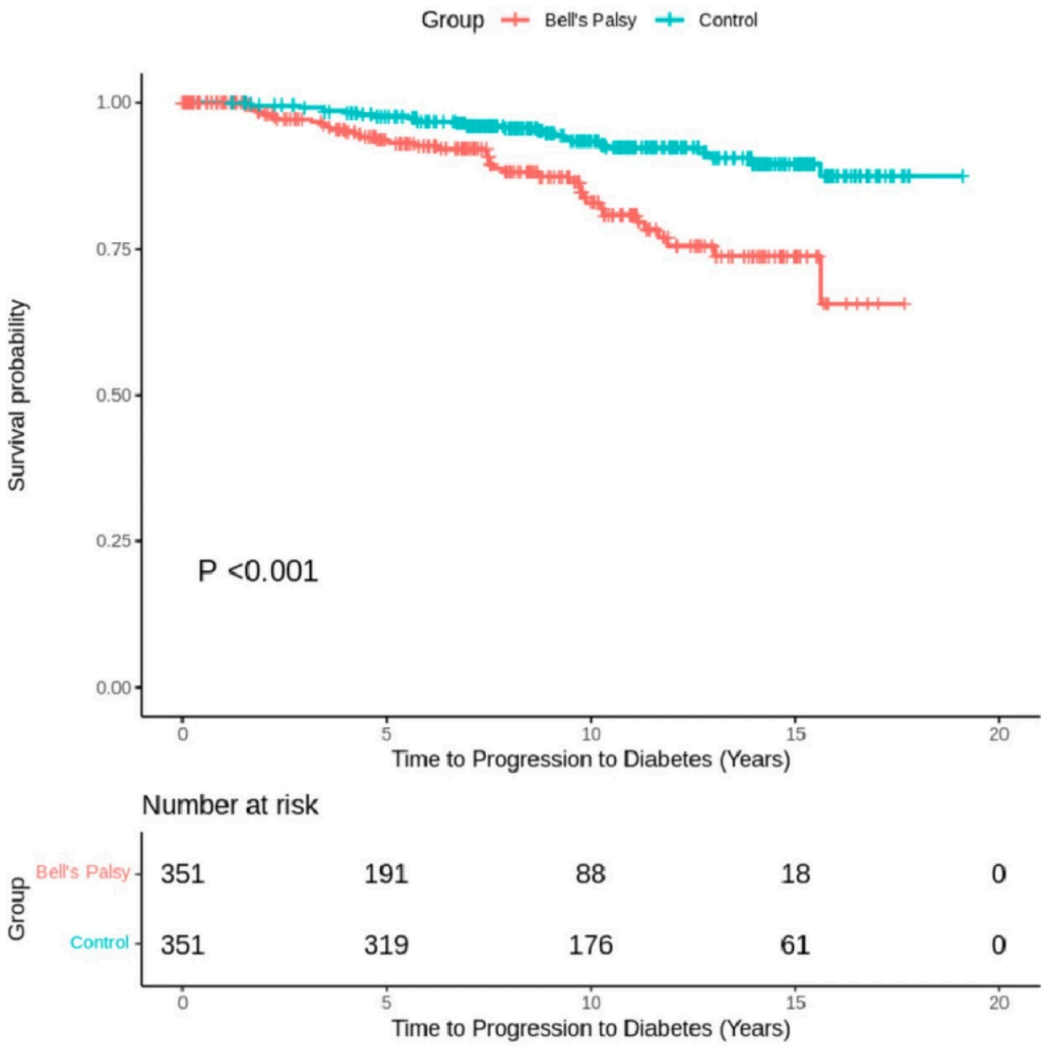



**Figure 1.** Kaplan–Meier curves compare overall DFS of Bell's palsy cases vs. controls.

### *3.3. Comparison of DFS Between Bell's Palsy vs. Controls Considering BMI Classifications*

With an increasing BMI, Bell's palsy patients showed significantly shorter DFS (log-rank $\chi^2 = 18.6$, $p < 0.001$), while only a borderline effect was found in the controls (log-rank $\chi^2 = 9.5$, $p = 0.050$) (Supplementary Figures S1 and S2). In Bell's palsy patients, obese class I was associated with significantly shorter DFS compared with that for the controls (log-rank $p = 0.014$) (Table 2).

**Table 2.** Mean DFS between Bell's palsy cases vs. controls considering BMI classifications.

| BMI Classification [a] | Bell's Palsy Mean Survival, Years (95% CI) | Controls Mean Survival, Years (95% CI) | Difference, Years | Log Rank (Mantel–Cox) |
|---|---|---|---|---|
| N/A | 14.46 (13.0–15.9) | 17.80 (16.6–19.0) | −3.3 | 0.094 |
| Non-obese | 16.75 (15.8–17.7) | 16.81 (16.4–17.3) | ≈0 | 0.457 |
| Overweight | 15.37 (14.6–16.1) | 16.93 (16.3–17.6) | –1.6 | 0.511 |
| Obese class I | 13.09 (11.7–14.5) | 15.75 (14.8–16.7) | –2.7 | 0.014 * |
| Obese class II | 10.59 (8.6–12.6) | 15.07 (13.3–16.9) | –4.5 | 0.106 |

− (minus), time earlier than controls. N/A, data was not available. * Chi-square test, $p < 0.05$. [a] World Health Organization (WHO) criteria for Asians, i.e., non-obese (<23 kg/m$^2$), overweight (23–24.9 kg/m$^2$), obese class I (25–29.9 kg/m$^2$), obese class II (≥30 kg/m$^2$).

### 3.4. Comparison of DFS Between Bell's Palsy vs. Controls Considering FBS Classifications

Overall, Bell's palsy patients and controls demonstrated differences in DFS (log-rank $\chi^2$ = 6.2, $p$ = 0.046, and log-rank $\chi^2$ = 38.0, $p$ < 0.001, respectively) ( Supplementary Figures S3 and S4). When stratified FBS was considered, Bell's palsy patients with normal fasting glucose exhibited significantly shorter DFS compared with that of the controls (log-rank $p$ = 0.041), but those with impaired fasting glucose showed no significant differences (Table 3).

**Table 3.** Mean DFS between Bell's palsy cases vs. controls considering FBS classifications.

| FBS Classifications | Cases Mean Survival, Years (95% CI) | Controls Mean Survival, Years (95% CI) | Difference, Years | Log Rank (Mantel–Cox) |
|---|---|---|---|---|
| N/A | 13.99 (13.0–15.0) | 15.91 (14.6–17.3) | –1.9 | 0.094 |
| Normal (<100 mg/dL) | 14.99 (14.3–15.7) | 18.73 (18.4–19.0) | –3.7 | 0.041 * |
| IFBS (100–125 mg/dL) | 14.75 (12.8–16.7) | 14.38 (13.1–15.6) | ≈0 | 0.86 |

– (minus), time earlier than controls; Abbreviations: N/A, data were not available; IFBS, impaired fasting blood sugar. * Chi-square test, $p$ < 0.05.

### 3.5. Cox Proportional Hazards Analysis

Univariate analysis showed that BMI, SBP, and DBP were significantly associated factors of diabetes development. However, no significant metabolic variables were identifiable by multivariate analysis (Table 4).

**Table 4.** Univariate and multivariate analysis of association of metabolic factors with development of diabetes in Bell's palsy cases via Cox proportional hazards models.

| Metabolic Variables | HR (95% CI) | *p*-Value | Adj HR (95% CI) | *p*-Value |
|---|---|---|---|---|
| FBS (mg/dL) | 1.05 (0.98–1.13) | 0.200 | 0.14 (0.02–1.11) | 0.063 |
| LDL (mg/dL) | 0.97 (0.93–1.01) | 0.174 | 0.15 (0.00–5.36) | 0.297 |
| SBP (mmHg) | 1.06 (1.01–1.10) | 0.011 * | 0.42 (0.12–1.53) | 0.191 |
| DBP (mmHg) | 1.06 (1.01–1.12) | 0.017 * | 0.32 (0.10–1.01) | 0.051 |
| BMI (kg/m$^2$) | 1.35 (1.07–1.72) | 0.013 * | 0.34 (0.11–1.06) | 0.064 |

* $p$ < 0.05. HR, hazards ratio; Adj HR, adjusted hazards ratio for metabolic variables.

### 3.6. The Effect of Prednisolone Use in Bell's Palsy on the Development of DM

The use of prednisolone among Bell's palsy patients was not significantly associated with the subsequent development of DM (HR 1.51, 95% (CI 0.59–3.87), $p$ = 0.39).

## 4. Discussion

The baseline characteristics showed significantly elevated FBS, SBP, DBP, and BMI among Bell's palsy cases. Also, BMI and FBS classifications by standard definitions were significantly different between Bell's palsy cases and controls (Table 1). During the follow-ups, Bell's palsy patients had a significantly shorter overall DFS than did their matched controls (*log-rank* $\chi^2$ = 12.6, $p$ < 0.001). The mean time to develop overt DM was 15.0 (95% CI 14.2–15.8) years in Bell's palsy compared with 17.9 (95% CI 17.4–18.3) years in controls (Figure 1). Bell's palsy was possibly a neurological presentation during the course of the diabetes clinical spectrum, from IR, to IFBS, and to overt DM. Therefore, Bell's palsy may be a marker of abnormal glucose metabolism [9,10,20]. Diabetes-related

microvasculopathy involving the vasa nervorum, which supplies the facial nerve, as well as oculomotor nerves or peripheral nerves, can be attributed. This hypothesis was supported by a study reporting significantly lower median nerve amplitudes in diabetes patients presenting with Bell's palsy. Some studies suggested that Bell's palsy could be a focal form of peripheral neuropathy complication in diabetes patients [21,22]. Because of the retrospective study design, the causal relationship between Bell's palsy and impaired glucose metabolism cannot be concluded from the previous and current studies.

When BMI was considered in combination with the presence of Bell's palsy, Bell's palsy patients exhibiting higher BMI levels had significantly shorter DFS (log-rank $\chi^2$ = 18.6, $p < 0.001$) compared with those of the controls (log-rank $\chi^2$ = 9.5, $p$ = 0.050). Also, Bell's palsy patients with obese class I (BMI = 25–29.9 kg/m$^2$) had a 2.7-year shorter DFS ($p$ = 0.014) (Table 2). This subgroup analysis should be carefully interpreted because of the lower events rate and large missing data of the current study. Individuals with high BMI levels usually exhibit obesity, particularly central obesity. BMI was a universal and standard tool used for public assessment of obesity [23]. High body adiposity induces systemic cellular inflammation, including vascular endothelium. This results in atherosclerosis and vascular thrombosis of both the macro- and microvasculature of the human body. High BMI was reported as a significant factor increasing the risk of diabetes, as well as stroke, among the patients with a previous diabetes diagnosis [24–29]. Furthermore, high BMI increased the global health burden via the significant rise in global death and disability-adjusted life year (DALY) in a global survey conducted in 1990–2021. The leading causes of BMI-attributable DALYs reported in this survey were diabetes mellitus, ischemic heart disease, hypertensive heart disease, chronic kidney disease, low back pain and stroke [30]. Therefore, high BMI patients should be under surveillance and long-term monitoring for the occurrence of diabetes and related cardiovascular complications [8,31,32]. Based on the available evidence, although diabetes-related microvascular thrombosis causing Bell's palsy may be possible, the exact causal relationship between high BMI, Bell's palsy, and overt DM remains unproven. In clinical practice, Bell's palsy can present prior to, together with, or after the diagnosis of DM.

Considering FBS, Bell's palsy patients and controls showed significantly shorter DFS (log-rank $\chi^2$ = 6.2, $p$ = 0.046, and log-rank $\chi^2$ = 38.0, $p < 0.001$, respectively). Until now, there has been no prospective cohort study to demonstrate timeline association and the percentage of patients transforming from normal blood sugar levels, or IFBS, to overt DM. Studies reported that the patients presenting with Bell's palsy could possibly already have a prediabetes condition [9,10]. Hence, the rapidity of turning from prediabetes to overt DM varied individually, confounded by various co-existing cardiometabolic and genetic factors. In this study, we did not adjust the events using confounding cardiometabolic factors such as lifestyle, dietary intake, physical exercise, etc. While stratified FBS results showed significantly shorter DFS in Bell's palsy patients with normal baseline FBS (Table 3), interpretation of the result should be concerned about possible bias caused by missing study data like those reported in Table 2.

In this study, there were no identifiable independent factors associated with overt DM occurrence; only BMI, SBP and DBP showed significant association in the unadjusted Cox regression model (Table 4). The absence of independent factors from multivariate analysis could be attributed to the large missing baseline data. From a clinical point of view, the three cardiometabolic risk factors possibly co-contributed to Bell's palsy following the proposed theory of its vascular etiology. Lastly, prednisolone treatment of Bell's palsy had no significant effect on the later development of overt DM. This ensured the natural progression in this study of pre-diabetes state to overt DM among the patients presenting with Bell's palsy, without corticosteroid-induced hyperglycemia.

*Strengths and Limitations*

Due to the retrospective study design, a significant number of patients dropped out, which could contribute to attrition bias in this study. Moreover, a single-center study and inadequate events (63 cases, where 65–119 cases were required) would limit its generalizability. Confounders such as current use of anti-hypertensive and lipid-lowering drugs, lifestyle, physical activity, and dietary status should be considered when interpret the results of this study. Nevertheless, considering the strict inclusion criteria of the study, explicit definitions of terms and clinical condition used, the strategies for management of missing data, assessment of collinearity and hazards model assumption, and long follow-up time, the results of the study showed that BMI, SBP and DBP were dependently associated with overt DM development in this cohort, whereas no independent associated factors were identifiable due to the lower event rate. The findings suggested that Bell's palsy was possibly a marker of subclinical diabetes. Because of the benign course and good recovery rate associated with Bell's palsy, attending physicians usually pay less attention to the possible co-occurrence of diabetes disorder among Bell's palsy patients. With stronger research methodology, we expect that the time sequence or causal relationship between the presentation of Bell's palsy and progression along the diabetes clinical spectrum, or vice versa, could be elucidated in the future studies.

## 5. Conclusions

Bell's palsy is possibly a focal neuropathy complication occurring at some time point along the diabetes clinical spectrum. Its occurrence may mark the existence of impaired glucose homeostasis. The causal relationship and chronological sequence connecting Bell's palsy and diabetes cannot be demonstrated from this study. A complex interrelationship among central obesity, microvascular thrombosis of the facial nerve, and diabetes is unclear pending for further investigation in prospective studies. However, shorter DFS among Bell's palsy patients found in this study suggests diabetes screening at the presentation of Bell's palsy, and longitudinal glycemic monitoring should be performed, since early treatment of diabetes and prevention of diabetes-related cardiovascular complications are essential.

**Supplementary Materials:** The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/jcm15239304/s1, Figure S1: Kaplan–Meier curves compare DFS of Bell's palsy cases according to BMI; Figure S2: Kaplan–Meier curves compare DFS of controls according to BMI; Figure S3: Kaplan–Meier curves compare DFS of Bell's palsy cases according to FBS; Figure S4: Kaplan–Meier curves compare DFS of controls according to FBS; Table S1: Events per variables (EPV); Table S2: Cross tabulation of events per variables; Table S3: Collinearity testing per models based on variance inflation factor (VIF); Table S4: Test for proportional hazard assumption; Table S5: Complete case analysis and multiple imputation (MICE).

**Author Contributions:** Conceptualization, S.S. (Suppanut Siriseth), T.I., and P.S.; methodology, S.S. (Suppanut Siriseth), T.I., and P.S.; software, S.S.(Suppanut Siriseth) and T.I.; validation, S.S. (Suppanut Siriseth), T.I., and P.S.; formal analysis, S.S.(Suppanut Siriseth) and T.I.; investigation, S.S.(Suppanut Siriseth) and P.S.; resources, P.S.; data curation, S.S.(Suppanut Siriseth) and T.I.; writing—original draft preparation, S.S.(Suppanut Siriseth), T.I., and P.S.; writing—review and editing, S.S.(Suppanut Siriseth), T.I., P.S., T.A., P.K. (Pat Korathanakhun), P.K. (Prut Koonalintip), and S.S. (Suwanna Setthawatcharawanich); visualization, T.I., P.S., T.A., P.K. (Pat Korathanakhun), P.K. (Prut Koonalintip), and S.S. (Suwanna Setthawatcharawanich); supervision, T.I. and P.S.; project administration, T.I., P.S., T.A., P.K. (Pat Korathanakhun), P.K. (Prut Koonalintip), and S.S (Suwanna Setthawatcharawanich). All authors have read and agreed to the published version of the manuscript.

**Funding:** This research received no external funding.

**Institutional Review Board Statement:** The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Faculty of Medicine, Prince of Songkla University (REC.66-263-14-4; date of approval: 24 July 2023).

**Informed Consent Statement:** Patient consent was waived due to the retrospective study design involving only medical records review.

**Data Availability Statement:** The study methods, statistical analyses and results are available in the published manuscript.

**Acknowledgments:** The authors thank Trevor Pearson of the Foreign Affairs Unit, Faculty of Medicine, Prince of Songkla University, for the English editing of the manuscript.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## Abbreviations

Diabetes mellitus (DM); diabetes-free survival time (DFS); body mass index (BMI); fasting blood sugar (FBS); impaired fasting blood sugar (IFBS); systolic blood pressure (SBP); diastolic blood pressure (DBP).

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