Prestroke Glycemic Control Is Associated With the Functional Outcome in Acute Ischemic Stroke

The Fukuoka Stroke Registry

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Background and Purpose—Diabetes mellitus is an established risk factor for stroke. However, it is uncertain whether prestroke glycemic control (PSGC) status affects clinical outcomes of acute ischemic stroke. The aim of this study was to elucidate the association between PSGC status and neurological or functional outcomes in patients with acute ischemic stroke.

Methods—From the Fukuoka Stroke Registry (FSR), a multicenter stroke registry in Japan, 3627 patients with first-ever ischemic stroke within 24 hours after onset were included in the present analysis. The patients were categorized into 4 groups based on their PSGC status: excellent (hemoglobin [Hb] A1c on admission <6.2%), good (6.2–6.8%), fair (6.9–8.3%) and poor (≥8.4%). Study outcomes were neurological improvement (≥4 points decrease in the National Institutes of Health Stroke Scale [NIHSS] score during hospitalization or 0 points on NIHSS score at discharge), neurological deterioration (≥1 point increase in NIHSS score) and poor functional outcome (death or dependency at discharge, modified Rankin Scale 2–6).

Results—The age- and sex-adjusted ORs for neurological improvement were lower, and those for neurological deterioration and a poor functional outcome were higher in patients with poorer PSGC status. After adjusting for multiple confounding factors, these trends were unchanged (all probability values for trends were <0.002). These findings were comparable in patients with noncardioembolic and cardioembolic infarctions.

Conclusions—In ischemic stroke patients, HbA1c on admission was an independent significant predictor for neurological and functional outcomes. (Stroke. 2011;42:2788-2794.)

Key Words: diabetes ■ brain infarction ■ prognosis ■ hyperglycemia

Diabetes is an established risk factor for the development of cardiovascular diseases, including stroke. The Hisayama Study revealed that the risk of stroke in diabetic patients was twice as high as in nondiabetic people in a general Japanese population.1,2 Furthermore, if stroke occurs in diabetic patients, their outcomes may be less favorable than in nondiabetic patients. A large number of studies have demonstrated residual neurological deficits and functional outcome to be worse compared with nondiabetics; consequently, hospital and long-term mortality were worse in diabetic patients compared with nondiabetics,3-5 although a few other studies did not confirm these effects.6-8

Very few observational studies have assessed the association between prestroke glycemic control (PSGC) status and clinical outcome in acute stroke patients. A previous study with a small number of subjects suggested that prestroke blood glucose level was not associated with stroke outcome.9 However, the impact of the PSGC status on clinical outcome is still unknown because there have been such few studies conducted.

The aim of the present study was to investigate the association between PSGC status, defined by hemoglobin (Hb) A1c on admission, and neurological and functional outcomes after acute ischemic stroke.

Subjects and Methods

Study Subjects and a Description of the Fukuoka Stroke Registry

The Fukuoka Stroke Registry (FSR) is a multicenter, hospital-based registry in which acute stroke patients were enrolled. Kyushu University, Fukuoka, Japan.

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University Hospital and 6 other stroke centers in Fukuoka, Japan participated in this registry (see Appendix). The study design was approved by the Institutional Review Board of the ethics committees in all hospitals. The Institutional Review Board approved the study protocols and related materials, such as informed consent, and document and study brochures, after careful investigation into the protocols and ethics of the study to protect the rights, safety and welfare of all participants.

The FSR consists of 2 database systems, ie, prospective and retrospective databases. In the prospective database, we have been recruiting stroke patients admitted to the participating hospitals within 7 days after onset since June 2007. A total of 3666 cases of stroke were registered as of August 2010. In a prospective database, the patients participated in the study on a voluntary basis after they were fully informed of the objectives and methods of the study, risk to the patients, and benefit to society. They consented to the collection of data from medical records, inquiry concerning medical condition and activities of daily living after discharge, and blood samples. Written informed consent was obtained from all patients. This database consists of demographic characteristics; medical history; prehospital, emergency, and in-hospital interventions; and patient states including activities of daily living, neurological symptoms, and laboratory data during hospitalization. For the retrospective study, we reviewed medical records of all consecutive patients admitted to participating hospitals within 24 hours of onset between June 1999 and May 2007, and collected similar information as was included in the prospective database. A total of 5497 cases of stroke were registered in the retrospective database during this period.

**Definition of Stroke**

Stroke was defined as sudden onset of nonconvulsive and focal neurological deficit persisting for more than 24 hours, and was classified into ischemic stroke, brain hemorrhage, subarachnoid hemorrhage, or other type of stroke by means of brain imaging (computed tomography and/or magnetic resonance imaging). Ischemic stroke was further classified into 4 subtypes, ie, lacunar infarction, atherothrombotic infarction, cardioembolic infarction, and unclassified infarction; it was classified on the basis of the Classification of Cerebrovascular Disease III proposed by the National Institute of Neurological Disorders and Stroke, as well as on the basis of the diagnosis criteria of the Trial of Org 10172 in Acute Stroke Treatment (TOAST) study and the Cerebral Embolism Task Force for ischemic stroke subtypes. Noncardioembolic infarction was defined either as lacunar, atherothrombotic or unclassified infarction.

**Patient Selection**

Among a total of 9163 cases in the FSR prospective and retrospective databases, 8637 cases were classified as ischemic strokes. After excluding cases with recurrent strokes, impairment of daily living before onset, hospitalization more than 24 hours from onset, and no HbA1c data on admission, 3627 cases were included in the present study (Figure 1).

**Clinical Assessment**

HbA1c was measured according to the Japan Diabetes Society (JDS)/Japan Society of Clinical Chemistry (JSCC) standardization scheme. The values were standardized with national calibrators, the Japanese standard reference materials. The HbA1c values used in the present analysis were obtained by the addition of 0.4% to the JDS/JSCC-based HbA1c values, because the JDS/JSCC-based HbA1c is 0.4% lower than that measured by the National Glycohemoglobin Standardization Program system. The patients were then classified into 4 groups according to their PSGC status, based on the criteria suggested by the JDS as follows: excellent (HbA1c <6.2%), good (6.2% ≤ HbA1c <6.9%), fair (6.9% ≤ HbA1c <8.4%) and poor (HbA1c ≥8.4%). Poststroke casual blood glucose was also measured on admission. A diagnosis of diabetes mellitus was determined by the diagnostic criteria of the JDS in the chronic stage or based on a medical history of diabetes. In patients with known diabetes, the use of oral hypoglycemic agents and insulin were investigated.

Hypertension was defined as systolic blood pressure ≥140 mm Hg or diastolic pressure ≥90 mm Hg in the chronic stage, or as current treatment with antihypertensive drugs. Dyslipidemia was defined as either a low-density lipoprotein-cholesterol level ≥3.62 mmol/L, high-density lipoprotein-cholesterol level <1.03 mmol/L, triglycerides ≥1.69 mmol/L, or current treatment with a cholesterol-lowering drug. Atrial fibrillation was diagnosed based on electrocardiographic findings on admission or during hospitalization. Smoking was defined as current cigarette smoking and alcohol intake as habitual consumption of alcohol beverages before onset of stroke.

Blood pressure and body mass index were measured on admission. Thrombolytic therapy was defined as intravenous or intra-arterial administration of thrombolytic agents such as recombinant tissue-type plasminogen activator and urokinase in the acute phase of stroke. Infectious complications were defined as any infectious diseases such as pneumonia, urinary tract infection, and sepsis during hospitalization.

**Study Outcomes**

The neurological severity was scaled by the National Institutes of Health Stroke Scale (NIHSS) score on admission and at discharge. Neurological improvement was defined as ≥4 point decrease in NIHSS during hospitalization or a 0 point status on NIHSS at discharge. Neurological deterioration was defined as ≥1 point increase in NIHSS during hospitalization. In this study, to evaluate the neurological changes during hospitalization, we modified the definition originally reported by Weimar et al, in which neurological worsening was defined as an increase of ≥1 point on NIHSS from hospital admission until 48–72 hours later. Death was defined as all-cause mortality during hospitalization. To evaluate the short-term functional outcome, poststroke functional impairment at discharge was graded using a modified Rankin Scale (mRS). A poor functional outcome was defined as death (mRS 6) or dependency (mRS 2–5).
The mean age of the study subjects was 69 years, and the mean duration of hospitalization was 22 days.

Statistical Analysis

Statistical analyses were performed using the JMP version 7 software program (SAS Institute Inc). The clinical characteristics among PSGC groups were compared by logistic regression analysis, analysis of variance, or the Wilcoxon rank sum test. Age- and sex-adjusted or multivariate-adjusted ORs and 95% CIs for each study outcome were estimated by logistic regression analysis. The heterogeneity of the PSGC’s effects on each outcome between noncardioembolic and cardioembolic subtypes was tested by adding an interaction term to the relevant multivariate logistic model. Probability values <0.05 were considered to be statistically significant.

Table 1 shows the clinical characteristics of patients according to their PSGC status. The frequencies of hypertension, dyslipidemia, and smoking increased with a poorer PSGC status. The body mass index and blood pressure and casual blood glucose on admission were significantly higher in patients with poor PSGC status. Concerning stroke subtypes, the proportion of noncardioembolic infarctions was higher in subjects with poorer PSGC status. The median (interquartile range) of NIHSS on admission was 4 (2–8), 4 (2–8), 4 (2–8), and 3.5 (2–6) in patients with excellent, good, fair, and poor PSGC, respectively. There was no statistically significant difference in NIHSS at admission among PSGC groups (P=0.20; Wilcoxon rank sum test).

Results

Demographics of the Patients

The mean age of the study subjects was 69±12 years old, and 1366 patients (37.7%) were women. The mean value of HbA1c was 6.4±1.5%, and 1233 patients (34.0%) had been diagnosed during hospitalization. A total of 588 patients (16.2%) were administered antidiabetic treatment before stroke, including oral hypoglycemic agents and insulin. The mean duration of hospitalization was 27±22 days.

Table 1 shows the clinical characteristics of patients according to their PSGC status. The frequencies of hypertension, dyslipidemia, and smoking increased with a poorer PSGC status, although atrial fibrillation was less frequent in those with a poorer PSGC status. The body mass index and

Association Between PSGC Status and Neurological Outcomes

Table 2 shows the association between PSGC status and each study outcome. The age- and sex-adjusted ORs for neurological improvement decreased substantially as PSGC status became poorer. In contrast, signs of neurological deterioration increased with poorer PSGC status. These findings were still observed after adjustment for possible confounding factors such as age, sex, baseline NIHSS, stroke subtype, systolic blood pressure, hypertension, dyslipidemia, atrial fibrillation, body mass index, thrombolytic therapy, and infectious complications (model 1; P<0.001). In this model, the probability of achieving neurological improvement was significantly lower, and the risk of a neurological deteriora-
tion was significantly higher in both the fair and poor PSGC groups than in the excellent PSGC group. Similar trends were observed when admission blood glucose levels were included as well (model 2; P < 0.002). The frequency of death during hospitalization was not different among the PSGC groups. We further analyzed the association between the PSGC status and each outcome separately for noncardioembolic and cardioembolic infarctions. As shown in Figure 2, multivariate-adjusted ORs for neurological improvement significantly decreased with poorer PSGC status in patients with noncardioembolic infarctions.

Table 2. Odds Ratios for Clinical Outcome According to PSGC Status

<table>
<thead>
<tr>
<th>Clinical Outcome</th>
<th>No. Events/Total</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological improvement</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent PSGC</td>
<td>1166/2251 (51.8%)</td>
<td>1.00</td>
<td>reference</td>
<td>1.00</td>
<td>reference</td>
<td>1.00</td>
<td>reference</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good PSGC</td>
<td>271/542 (50.0%)</td>
<td>0.94</td>
<td>(0.78–1.14)</td>
<td>0.54</td>
<td>0.84</td>
<td>(0.68–1.04)</td>
<td>0.12</td>
<td>0.84</td>
<td>(0.67–1.04)</td>
<td>0.11</td>
</tr>
<tr>
<td>Fair PSGC</td>
<td>204/470 (43.4%)</td>
<td>0.72</td>
<td>(0.59–0.88)</td>
<td>0.001</td>
<td>0.77</td>
<td>(0.61–0.97)</td>
<td>0.03</td>
<td>0.76</td>
<td>(0.59–0.98)</td>
<td>0.04</td>
</tr>
<tr>
<td>Poor PSGC</td>
<td>136/364 (37.4%)</td>
<td>0.53</td>
<td>(0.42–0.67)</td>
<td>&lt;0.001</td>
<td>0.52</td>
<td>(0.40–0.68)</td>
<td>&lt;0.001</td>
<td>0.50</td>
<td>(0.36–0.71)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

P for trend<0.001<0.001<0.001

| Neurological deterioration | | | | | | | | | | |
| Excellent PSGC   | 218/2251 (9.7%)  | 1.00 | reference    | 1.00 | reference | 1.00 | reference |
| Good PSGC        | 59/542 (10.9%)  | 1.12 | (0.82–1.52)  | 0.46 | 1.01 | (0.69–1.45)  | 0.95 | 1.02 | (0.70–1.46)  | 0.93 |
| Fair PSGC        | 71/470 (15.1%)  | 1.70 | (1.27–2.27)  | <0.001| 1.71 | (1.19–2.41)  | 0.004| 1.66 | (1.12–2.43)  | 0.01 |
| Poor PSGC        | 57/364 (15.7%)  | 2.04 | (1.47–2.79)  | <0.001| 2.37 | (1.62–3.43)  | <0.001| 2.32 | (1.39–3.83)  | 0.001|

P for the trend<0.001<0.001<0.001

| Death | | | | | | | | | | |
| Excellent PSGC   | 72/2251 (3.2%)  | 1.00 | reference    | 1.00 | reference | 1.00 | reference |
| Good PSGC        | 12/542 (2.2%)  | 1.49 | (0.93–2.30)  | 0.19 | 1.64 | (1.12–2.26)  | 0.05 | 1.59 | (1.13–2.23)  | 0.007 |
| Fair PSGC        | 17/470 (3.6%)  | 2.12 | (0.51–8.72)  | 0.59 | 1.04 | (0.50–2.17)  | 0.92 | 1.02 | (0.46–2.45)  | 0.97 |
| Poor PSGC        | 13/364 (3.6%)  | 0.72 | (0.49–1.05)  | 0.30 | 0.67 | (0.42–1.10)  | 0.14 | 0.65 | (0.41–1.06)  | 0.06 |

P for trend0.380.280.46

| Poor functional outcome | | | | | | | | | | |
| Death (or dependency) | | | | | | | | | | |
| Excellent PSGC   | 1025/2251 (45.5%)| 1.00 | reference    | 1.00 | reference | 1.00 | reference |
| Good PSGC        | 266/542 (49.1%)  | 1.12 | (0.91–1.36)  | 0.26 | 1.20 | (0.93–1.56)  | 0.16 | 1.16 | (0.90–1.51)  | 0.25 |
| Fair PSGC        | 246/470 (52.3%)  | 1.35 | (1.10–1.66)  | 0.004| 1.34 | (1.02–1.77)  | 0.04 | 1.26 | (0.94–1.71)  | 0.12 |
| Poor PSGC        | 202/364 (55.5%)  | 1.84 | (1.46–2.32)  | <0.001| 2.52 | (1.88–3.39)  | <0.001| 2.30 | (1.56–3.40)  | <0.001|

P for trend<0.001<0.001<0.001

The multivariate model 1 included age, sex, baseline NIHSS in quartiles, stroke subtype (non-cardioembolic or cardioembolic), systolic blood pressure on admission, hypertension, dyslipidemia, atrial fibrillation, BMI, thrombolytic therapy, and infectious complications. The multivariate model 2 included the same variables in the model 1 and casual blood glucose on admission.

PSGC indicates prestroke glycemic control; OR, odds ratio; CI, confidence interval.

We further analyzed the association between the PSGC status and each outcome separately for noncardioembolic and cardioembolic infarctions. As shown in Figure 2, multivariate-adjusted ORs for neurological improvement significantly decreased with poorer PSGC status in patients with noncar-

### Figure 2

**Multivariate-adjusted ORs and 95% CIs for neurological improvement in subjects with non-cardioembolic (closed squares) and cardioembolic (open squares) infarctions according to PSGC status. The multivariate model included age, sex, baseline NIHSS in quartiles, systolic blood pressure on admission, casual blood glucose on admission, hypertension, dyslipidemia, atrial fibrillation, body mass index, thrombolytic therapy, and infectious complications. The abscissa is shown on a logarithmic scale. OR indicates odds ratio; CI, confidence interval; PSGC, prestroke glycemic control; NIHSS, National Institutes of Health Stroke Scale.**

<table>
<thead>
<tr>
<th>PSGC</th>
<th>Events (%)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-cardioembolic infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>813/1616 (50.3)</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>Good</td>
<td>177/377 (47.0)</td>
<td>0.79 (0.61–1.03)</td>
</tr>
<tr>
<td>Fair</td>
<td>151/363 (41.6)</td>
<td>0.74 (0.55–0.98)</td>
</tr>
<tr>
<td>Poor</td>
<td>109/304 (35.9)</td>
<td>0.50 (0.34–0.73)</td>
</tr>
<tr>
<td>Cardioembolic infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>353/635 (55.6)</td>
<td>1.00 (reference)</td>
</tr>
<tr>
<td>Good</td>
<td>94/165 (57.0)</td>
<td>1.00 (0.66–1.51)</td>
</tr>
<tr>
<td>Fair</td>
<td>53/107 (49.5)</td>
<td>0.81 (0.46–1.42)</td>
</tr>
<tr>
<td>Poor</td>
<td>27/60 (45.0)</td>
<td>0.60 (0.26–1.36)</td>
</tr>
</tbody>
</table>

P for trend<0.001

P for heterogeneity between subtypes = 0.29
dioembolic infarctions ($P<0.001$). A similar pattern was observed in patients with cardioembolic infarctions, but it did not reach the level of significance, probably because of the limited sample size ($P=0.29$). In any case, there was no evidence of heterogeneity between ORs in noncardioembolic and in cardioembolic subtypes ($P$ for heterogeneity=0.29). As shown in Figure 3, the risks of neurological deterioration significantly increased with poorer PSGC status in both subtypes, and no evidence of heterogeneity between subtypes was found ($P$ for heterogeneity=0.93).

**Association Between PSGC Status and Short-Term Functional Outcomes**

The risks of poor functional outcome were higher in patients with poorer PSGC status in all logistic models (Table 2; $P<0.001$). In both age- and sex-adjusted and multivariate model 1, the risk of poor functional outcome was significantly higher in both the fair and poor PSGC groups than in the excellent PSGC group. However, the fair PSGC group failed to show a significant association with functional outcome in the multivariate model 2. As shown in Figure 4, the risk of poor functional outcome significantly increased with poorer PSGC status in patients with noncardioembolic infarctions ($P=0.001$). A similar, but nonsignificant, association was observed in patients with cardioembolic infarctions ($P=0.15$), and there was no evidence of heterogeneity between subtypes ($P$ for heterogeneity=0.55).

**Discussion**

It remains to be fully elucidated whether PSGC status affects the clinical course of ischemic stroke. A previous study with 99 ischemic stroke patients suggested that prestroke blood glucose level did not have any predictive value for stroke outcome.9 However, the present study has shown that poor glycemic control before stroke occurrence may be detrimental to clinical course after onset, and consequently lead to poor functional outcome of ischemic stroke.

Because noncardioembolic infarction is mainly caused by atherosclerosis, and diabetes is a major risk factor for atherosclerosis, the effects of PSGC status on clinical outcomes might differ among stroke subtypes. However, we found no evidence of heterogeneity between ORs for each outcome in the noncardioembolic and cardioembolic subtypes. Therefore, PSGC status has similar effects on the clinical course and short-term outcome, regardless of stroke subtype.

The mechanism by which poor glycemic control before onset is associated with unfavorable outcome of ischemic stroke is unclear. There are possible hypotheses regarding the
association between PSGC status and outcome. Many studies have shown that hyperglycemia after stroke onset has adverse effects on the clinical course of ischemic stroke,17–23 although other studies did not acknowledge this effect.6–8 Baird et al. reported that persistent hyperglycemia was associated with the expansion of infarct volume and worse functional outcome.21 Poppe et al. found that admission hyperglycemia was independently associated with outcome both in patients with and without diabetes.24 Hyperglycemia itself probably results in neurotoxicity and induces a procoagulant state.25 In this study, initial NIHSS was similar among the 4 PSGC groups defined by admission HbA1c. However, PSGC was found to be an independently significant predictor of neurological and clinical outcomes in the multivariate model 1, and this relationship remained significant even after adjusting for blood glucose levels in multivariate model 2. Because the HbA1c level at admission, which is considered to be an index of glucose control a few months before stroke onset, showed a positive correlation with casual blood glucose level at admission, it is possible that prestroke and poststroke hyperglycemia contributed to an exacerbation of the clinical course, and to poorer short-term outcome in patients with poor PSGC status.

Other factors could also be involved in the mechanism(s). Blood pressure was significantly higher in patients with poor glycemic control compared with that in patients with good or better glycemic control. The acute blood pressure level is associated with neurological deterioration in patients with ischemic stroke.26 Therefore, high blood pressure may also be associated with poor outcome in patients with poor PSGC status. Although various factors may also be involved, PSGC status had a significant impact on short-term outcome even after adjustment for possible confounding factors including stroke subtypes, admission blood glucose, and admission blood pressure. These results indicate that PSGC is an independent predictor for poor clinical outcome in patients with ischemic stroke.

Recent randomized controlled trials have revealed that intensive glycemic control could not reduce cardiovascular risk in diabetic patients (median glycohemoglobin levels, 6.4% [ACCORD27] and 6.9% [VADT28]; median glycohemoglobin level 6.5% [ADVANCE29]) compared with conventional control. From this point of view, less intense therapy may be recommended for high-risk diabetic patients, especially elderly patients, to avoid adverse events such as hypoglycemia. Before intensifying the therapy regimen, patient life expectancy, risk of hypoglycemia, and the presence of cardiovascular diseases should be considered.30

It is plausible that poststroke blood glucose is higher in patients with poor PSGC, whereas it is better controlled in patients with better PSGC status. We did not verify whether prestroke intensive treatment of blood glucose affects clinical outcome after ischemic stroke. However, if poststroke hyperglycemia is detrimental to clinical outcome in ischemic stroke, a better PSGC status may be beneficial. Additional studies are needed to elucidate whether treatment to provide better glycemic control before onset improves clinical course and outcome in patients with ischemic stroke.

The present study had several strengths. The number of subjects was large, and they were recruited from multiple stroke care centers that treat patients with standardized criteria. In addition, possible confounding factors were extensively collected in all subjects and adjusted in multivariate analysis. However, there were also limitations to this study. There was a selection bias, since 1006 patients without HbA1c measurement data on admission were excluded from present analyses. The proportion of diabetic subjects was smaller among excluded patients (16.3%) than among patients included in the study (34.0%). Therefore, we consider that ORs shown in the patient analyses might have been underestimated, but this selection bias did not affect the study conclusions. Although we adjusted for blood glucose levels on admission as a confounding factor in multivariate model 2, the changes in blood glucose level and its management during hospitalization were not considered in the analyses. Since it was an observational study, and therefore treatment could not be controlled, the efficacy of treatment for blood glucose and the optimal range for glycemic control remain unclear. Moreover, the ORs for cardioembolic infarction did not reach statistically significant levels, probably because of the small sample size. Additional studies will be required to elucidate whether control of prestroke blood glucose improves clinical outcome after ischemic stroke.

Conclusions

In conclusion, higher HbA1c at onset was found to be associated with unfavorable clinical outcome of ischemic stroke. In comparison with excellent PSGC, fair or poor PSGC was associated with unfavorable neurological outcomes, and poor PSGC was found to be related to poorer functional outcome, whereas no association was observed between PSGC status and death during hospitalization. In high-risk diabetic patients, glycemic control should be reappraised from the standpoint of functional outcome after ischemic stroke.

Appendix

FSR Investigators
The participating Hospitals in the FSR were as follows: Kyushu University Hospital, National Hospital Organization Kyushu Medical Center, National Hospital Organization Fukuoka Higashi Medical Center, Fukuoka Red Cross Hospital, St. Mary’s Hospital, Nippon Steel Yawata Memorial Hospital, Japan Labor Health and Welfare Organization Kyushu Rosai Hospital.

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Disclosures

None.

References

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Prestroke Glycemic Control Is Associated With the Functional Outcome in Acute Ischemic Stroke — The Fukuoka Stroke Registry

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Abstract

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Background and Objective: Hyperglycemia is a risk factor for acute ischemic stroke (AIS), but the relationship between prestroke glycemic control and functional outcome is not clear. We assessed whether prestroke glycemic control was associated with functional outcome in AIS.

Methods: The Fukuoka Stroke Registry (FSR) is a large cohort of patients who have experienced AIS. We included all patients who were admitted to participating hospitals from January 1, 2003, to December 31, 2011. The relation between prestroke glycemic control and functional outcome was assessed using the Rankin scale.

Results: Among the 1,401 patients included in the analysis, 1,396 patients had a prestroke fasting plasma glucose level. The mean Rankin score was 2.3 (standard deviation, 0.8). The mean prestroke glycemic control level was 6.2±1.1 mmol/L. The patients were divided into four groups based on their prestroke glycemic control levels: normal (6.0-6.3 mmol/L), abnormal (6.4-6.7 mmol/L), and high (6.8-7.1 mmol/L). The functional outcome was better in the normal group than in the other groups.

Conclusion: Prestroke glycemic control is associated with functional outcome in AIS.