Control of Diabetes during Rehabilitation for Diabetic Stroke

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Sato, T. and Hiwatari, M. Control of Diabetes during Rehabilitation for Diabetic Stroke. Tohoku J. Exp. Med., 1994, 173 (4), 399-404 — Changes of diabetic control during rehabilitation therapy was studied retrospectively in 33 cases of diabetic stroke. Diabetic control was not deteriorated in any cases during rehabilitation therapy. In 21 cases, treatment of diabetes was not changed during rehabilitation therapy and the levels of fasting plasma glucose were decreased in all of them. Doses of hypoglycemic agents was reduced without significant deterioration of diabetic control in 9 cases whose diabetic control was excellent, and in 2 cases who experienced hypoglycemic attack during rehabilitation therapy. In these cases, oral hypoglycemic agent was reduced from 2 to 1 tablet and insulin from 32 to 21 units per day on average. These results indicate that training in the stroke rehabilitation moderately improves diabetic control.

The incidence of cerebrovascular accident is higher in the diabetics (Shafer et al. 1974), and the initial appearance of hyperglycemia or diagnosis of diabetes may occur as a result of stroke (Seltzer 1983). In the acute phase, severely uncontrolled diabetes is a frequently accompanying feature of diabetic stroke and intensive care is often necessary to control plasma glucose. By the time when active rehabilitation therapy for stroke is started, metabolic derangement is improved in most cases. Initiation of exercise in rehabilitation therapy following immobilization may improve insulin sensitivity (Pedersen et al. 1980; Heath et al. 1983; Bonen et al. 1986; Mikines et al. 198; Richter et al. 1989), and provoke hypoglycemia in the diabetics on insulin or oral hypoglycemic agents. And neurological deficit of varying severity and duration may follow hypoglycemia (Lawrence 1967; Foster and Hart 1987; Pell and Frier 1990; Duarte et al. 1993). On the contrary, intensive exercise may make diabetic control worse, especially when diabetic control is poor (Michel et al. 1984; Bonen et al. 1985; Jensen and Miles 1986). Hyperglycemia also could lead to complications such as infection (Bybee and Rogers 1964) and disturbance of water balance. Therefore, it is essential for effective rehabilitation in diabetic stroke to control diabetes well,
avoiding hypoglycemia or hyperglycemia. However, there has been few reports (Gandhavadi 1981) on control of diabetes during rehabilitation therapy for diabetic stroke.

In this study, changes of diabetic control by rehabilitation therapy for diabetic stroke was elucidated.

**Subjects and Methods**

In order to evaluate effect of rehabilitation therapy on diabetic control, changes of fasting plasma glucose were studied retrospectively on 33 cases of noninsulin dependent diabetes mellitus who were admitted for rehabilitation of cerebrovascular accidents to Narugo-Branch Hospital of Tohoku University School of Medicine, from 1983 to 1989. Patients were divided into three groups according to the treatment of diabetes; diet group, oral hypoglycemic agents group and insulin group. In each group, 0.65 to 5 mg of glybenclamide or 8 to 50 units of intermediate insulin was used.

Clinical pictures of the cases were summarized in Table 1. Mean age of the three groups ranged from 61.3 to 65.5 years old. Duration of diabetes showed tendency to be longer in the insulin group than in diet, oral hypoglycemic agent groups. There was no significant difference among the three groups in body mass index, which ranged from 22.5 to 23.8, or time from onset of stroke to the admission. Time from onset of stroke to admission to our hospital (TOA) was 2.9 to 5.8 months, and diabetic control was stable at admission in most cases. Rehabilitation therapy was started following about a week of functional evaluation. Usually, rehabilitation therapy was carried out in the departments of physical, occupational and speech therapy for 1 to 2 hr each day, and in the ward 6 days a week for about 2 months. They were placed on diet of approximately 28 kcal per kg of ideal body weight.

The plasma glucose levels of the blood samples drawn before breakfast were determined by enzymatic method and monitored about twice a week in the insulin group, once a week in the oral hypoglycemic agents group, and once every two

<table>
<thead>
<tr>
<th>Treatment of diabetes</th>
<th>Sex</th>
<th>Age (y/o)</th>
<th>Duration of diabetes (years)</th>
<th>BMI</th>
<th>TOA (months)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet</td>
<td>M/F</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=13)</td>
<td>7/6</td>
<td>63.7</td>
<td>3.3</td>
<td>22.5</td>
<td>3.5</td>
<td>9</td>
</tr>
<tr>
<td>OHA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=12)</td>
<td>8/4</td>
<td>61.3</td>
<td>± 5.3</td>
<td>± 3.3</td>
<td>± 3.6</td>
<td>9</td>
</tr>
<tr>
<td>Insulin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=8)</td>
<td>5/3</td>
<td>65.5</td>
<td>± 9.0</td>
<td>± 4.6</td>
<td>± 8.6</td>
<td>6</td>
</tr>
</tbody>
</table>

BMI, body mass index. TOA, time from onset to admission; CI, cerebral infarction; CH, cerebral hemorrhage; OHA, oral hypoglycemic agents. (Mean±S.D.)
weeks in the diet group, on average. The doses of oral hypoglycemic agents or insulin were adjusted to control the plasma glucose levels at least below 140 mg/100 ml. The fasting plasma glucose levels after approximately 2 months of rehabilitation therapy were compared with those before rehabilitation therapy. Statistical analysis was performed by Student's t-test. P value was based on two-sided test, and p < 0.05 was considered significant.

RESULTS

Changes of the fasting plasma glucose levels by rehabilitation therapy were illustrated in Fig. 1 and Table 2. The fasting plasma glucose levels were improved in most cases, and initiation of treatment with oral hypoglycemic agents or increase of insulin dose was necessary in only 3 cases (9%). In 9 cases, doses of oral hypoglycemic agents or insulin was reduced by rehabilitation therapy, mainly during the first month of rehabilitation, and their fasting plasma glucose levels increased slightly in 7 cases of them (78%). No cases showed deterioration of diabetic control by rehabilitation therapy without reduction of doses of

![Fig. 1. Changes of fasting plasma glucose by rehabilitation therapy for diabetic stroke; treatment of diabetes was unchanged (●—●), doses of hypoglycemic agent were increased (●—●), doses of hypoglycemic agents were decreased (○—○).]
hypoglycemic agents. As a whole, the fasting plasma glucose levels decreased from 131 ± 6 mg/100 ml to 114 ± 20 mg/100 ml by rehabilitation therapy for diabetic stroke (p < 0.01).

In the diet group, fasting plasma glucose was lower than 140 mg/dl in 6 cases and decreased by rehabilitation therapy in all of them (group a). Among 7 cases, whose fasting plasma glucose levels were over 140 mg/100 ml, 5 cases showed improvement of diabetic control (group b) by rehabilitation for stroke, and 2 cases needed initiation of treatment with glybenclamide to control diabetes (group c).

In the oral agent group, fasting plasma glucose was at good level before rehabilitation therapy. Doses of oral agents were decreased by one tablet on average in 2 cases whose fasting plasma glucose levels were about 100 mg/100 ml and one case who had hypoglycemic attack during rehabilitation (group d). By reduction of oral hypoglycemic agents, their fasting plasma glucose increased by approximately 20 mg/100 ml. In 9 cases, doses of oral hypoglycemic agents were same during rehabilitation therapy, and the levels of fasting plasma glucose decreased from 125 to 109 mg/100 ml on average.

In the insulin group, insulin dose was increased in only one case to control diabetes, and other cases showed good diabetic control and doses of insulin were decreased in 6 cases (group f), including one case who showed hypoglycemic attack during rehabilitation therapy. Average reduction of insulin doses was 11 units, and their fasting plasma glucose levels were increased slightly from 97 ± 23 to 117 ± 23 mg/100 ml.
DISCUSSION

Intensive physical exercise may elevate the plasma glucose levels as a result of increase of counter-regulatory hormones and the hepatic glucose production (Michel et al. 1984; Bonen et al. 1985). However, diabetic control was not deteriorated by stroke rehabilitation therapy in any cases of this study, except 7 cases whose insulin or oral hypoglycemic agents were reduced. Our results suggest that increase of physical activity in stroke rehabilitation does not deteriorate diabetic control.

When insulin levels are sufficient to control diabetes, acute exercise can lower the plasma glucose levels and reduce insulin requirement (Heath et al. 1983; Mikines et al. 1989). Exercise in stroke rehabilitation, following the acute stage when physical activity is restricted, may improve insulin sensitivity and cause hypoglycemia in the diabetics especially on insulin or oral hypoglycemic agents. Gandhavadi (1981) reported an woman of brittle diabetes with stroke. Her blood glucose levels were very sensitive to emotional status and exercise. Brittleness of diabetes was very disturbing to her, and destructive to the rehabilitation process. The amount of exercise throughout the weekdays was standardized and she received 2 units less of insulin during the weekdays, when she exercised more than on the weekends.

We should be cautious to prevent hypoglycemia, because it can provoke even hemiparesis (Foster and Hart 1987; Pell and Frier 1990). Actually, diabetic control was improved in most cases in our study, and 2 cases showed hypoglycemic attack. The doses of oral hypoglycemic agents or insulin should be changed according to the plasma glucose levels. In this study, oral hypoglycemic agent was reduced from 2 to 1 tablet, and insulin from 32 to 21 units per day on an average. The levels of fasting plasma glucose was increased by approximately 20 mg/100 ml in 7 cases of them, however they were still at the good levels. These reduction of hypoglycemic agents will be rather excess, however near to optimal for the well-controlled mild diabetics.

Diabetic control was improved in most cases of the diet group in this study. This indicates that initiation of drug therapy for moderate hyperglycemia should be withheld at the beginning of rehabilitation therapy for diabetic stroke.

References

presenting as acute tetraplegia. *Stroke*, 24, 143.


