was an increase in the prevalence of neuropathy as determined by biothesiometry in patients with increasing duration of diabetes (duration <5 years = 6.8%, 5–10 years = 12.8%, >10 years = 17.8%) (trend χ^2 = 5.55, P = 0.018). In patients with abnormal biothesiometry, although neuropathic symptoms were present in 67%, there was poor correlation with the evidence of neuropathy as determined by MFT (in 33%) and also with abnormal podotrack (in 52%). In patients with abnormal MFT, there was good correlation with the neuropathic symptoms (in 77%), abnormal biothesiometry (in 69%), and abnormal podotrack (in 62%). Kumar et al. (5) had reported that the monofilaments were most sensitive (100%) but less specific (77.7%) in identifying patients who had foot ulcers compared with biothesiometry, which was less sensitive (78.6%) but more specific (93.4%).

Abnormal plantar foot pressure seems to exist in diabetic patients before there is evidence of neuropathy as determined by biothesiometry and MFT tests, two commonly used devices. Podotrack is a simple and inexpensive method that can be used as a screening test for abnormal plantar foot pressure in diabetic patients, and appropriate preventive measures can then be advised to such patients.

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The Efficacy of Voglibose on Glycemic Excursions in Non–Insulin-Treated NIDDM Patients

We read with interest in an article by Matsumoto et al. (1) that voglibose administration had a more beneficial effect on the daily glycemic excursions through the attenuation of postprandial hyperglycemia in non–insulin-treated NIDDM patients compared with either diet therapy alone or treatment with a sulfonylurea (SU) drug. We almost agreed with their results, which were consistent with our recent report (2). However, we were confused at the difference between their research design and ours. Of their study subjects receiving diet therapy alone or treatment with an SU drug, half were treated with voglibose for 4 weeks, and the remaining patients served as a control group. Because both the voglibose-treated group and the control group were composed of either diet therapy alone or SU drug treatment, the intrinsic insulin secretion varied among patients with diet therapy alone and SU treatment. The assessment of insulin secretion therefore should be done separately in treatment groups. Concerning the administration timing of voglibose, the patients were administered voglibose at the start of the study, when glycemic control was poor (fasting glucose levels >7.8 mmol/l and postprandial [2-h] glucose levels >11.1 mmol/l), and the dosage of the SU drug in use was modified to achieve good glycemic control (fasting glucose levels <7.8 mmol/l and postprandial glucose levels <11.1 mmol/l) during the 4-week study period. In our study, we administered voglibose to patients who achieved satisfactory fasting glucose levels (<7.8 mmol/l) but had unsatisfactory postprandial glucose levels (>11.1 mmol/l) even after 4 weeks of strict diet therapy combined with an SU drug. Half of the patients recruited in our study did not need voglibose in addition to an SU drug after 4 weeks of strict diet therapy, and the other half of the patients needed voglibose. Therefore, some patients who did not really need voglibose in addition to diet therapy or an SU treatment after 4 weeks of study period might have been included in their study. Indeed, voglibose improves glycemic control by lowering the daily glycemic excursions (1,2) and inhibits overwork of the pancreatic β-cells (1); we emphasize that α-glucosidase inhibitors should be administered only to patients with satisfactory fasting glucose levels but unsatisfactory postprandial glucose levels even after strict diet therapy.

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Response to Yoshioka et al.

Protection of the pancreatic β-cells by an α-glucosidase inhibitor

As Yoshioka et al. (1) mentioned, both the voglibose-treated group and the control group were composed of either diet therapy alone or a sulfonylurea

(SU) drug therapy in our study (2). According to their valuable comments, we assessed insulin secretion separately in the treatment group. In the control group, area under the curve of insulin ($AUC_{insulin}$) did not change by treatment in the patients treated with diet alone or with an SU drug. In the patients treated with voglibose plus diet therapy (n = 4), $AUC_{insulin}$ tended to decrease from (mean ± SD) 3,382.6 ± 2,449.0 to 2,179.6 ± 1,994.3 pmol · l^{-1} · h (P = 0.19). In the patients treated with voglibose plus SU drug (n = 10), $AUC_{insulin}$ decreased significantly from 1,759.8 ± 500.7 to 1,286.3 ± 522.0 pmol · l^{-1} · h (P = 0.02). Therefore, voglibose could decrease the levels of serum insulin both in the patients treated with diet alone and in those treated with an SU drug. These results agreed with previous reports from other investigators involving other α-glucosidase inhibitors (3,4).

It is important to limit the patients who really require voglibose in addition to diet or SU drug treatment. However, we demonstrated that voglibose therapy could inhibit overwork of the pancreatic β-cells in non–insulin-treated NIDDM patients (2). Kawamori et al. (5) reported that, in the NIDDM patients treated with an SU drug (gliclazide), the addition of α-glucosidase inhibitor could diminish the daily doses of gliclazide required for effective glycemic control. Therefore, we believe that combination therapy with an SU drug and voglibose may have a theoretical advantage in glucose metabolism. Early addition of α-glucosidase inhibitor may improve glycemic control and may inhibit overwork of the pancreatic β-cells. Thereafter, the doses of SU drug should be adjusted to achieve better glycemic control.

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