Postprandial Changes in Noradrenergic and Dopaminergic Activity in Patients with Essential Hypertension

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In order to elucidate the role of noradrenergic and dopaminergic activity in the pathogenesis of postprandial hypotension, the effect of feeding of ordinary diet on blood pressure, pulse rate, plasma catecholamine and other circulating vasoactive substances such as insulin were examined in mild essential hypertensive patients (EH) and their age-matched control subjects (N). Mean blood pressure significantly decreased in both N and EH after feeding, and the decrease tended to be greater in EH than in N. Feeding induced a marked increase in plasma norepinephrine in both N and EH. Plasma dopamine significantly increased following feeding was observed in N, while the increase in plasma dopamine following feeding was blunted in EH. The ratio of norepinephrine to dopamine following feeding in EH was significantly greater than that in N. From these results, it is suggested that the feeding-induced stimulation of noradrenergic activity may be a result from the decrease in blood pressure, and that the blunted response of dopaminergic activity in EH may reflect the enhanced conversion of dopamine to norepinephrine probably due to the enhanced activity of dopamine β-hydroxylase in the sympathetic nerves. (Hypertens Res 1995; 18 Suppl. I: S199–S200)

Key Words: dopamine, postprandial hypotension

Postprandial hypotension is frequently observed in patients with hypertension (1). It is supposed that sympathetic nervous system plays an important role in postprandial hypotension (2), but its mechanism has not been fully elucidated. The present study was designed to elucidate the effect of feeding on noradrenergic and dopaminergic activity with relation to postprandial hypotension in patients with mild essential hypertension.

Patients and Methods

Thirty-one patients with essential hypertension (EH) (mean age 53.2 ± 1.7) and 17 age-matched normal subjects (mean age 50.2 ± 3.3) were enrolled in this study. Patients with diabetes mellitus and cardiovascular disease were excluded. All subjects provided informed consent before this study. After measuring basal valuables, 450 kcal of ordinary diet containing 73 g of carbohydrate, 10 g of protein and 2.2 g of NaCl was ingested and blood pressure (BP) and pulse rate (PR) were monitored every 30 min for 3 h. Bloodsampling for the determination of plasma dopamine (DA), two oxidative metabolites of DA [3,4-dihydroxyphenylacetic acid (DOPAC) and homovanillic acid (HVA)], norepinephrine (NE), glucose (PG), immunoreactive insulin (IRI) and renin activity (PRA) was performed.

Results

A significant postprandial decrease in mean BP was observed in both groups at 30 min after ingestion (normal controls: -4.8 ± 0.8 mmHg, EH: -8.5 ± 0.7 mmHg, p < 0.05), and the decrease of mean BP tended to be greater and prolonged in EH patients (Fig. 1). Feeding induced a marked increase in PG and IRI, but the increase in EH patients was compatible with that in normal subjects. Plasma NE increased after feeding in both groups (Fig. 2, left panel). PRA increased after feeding in normal subjects, but no significant change in PRA was found in patients with EH (Fig. 2, right panel). The increase in plasma free and conjugated DA after feeding was found in normal controls, but not in EH (Fig. 3). Plasma DOPAC and HVA did not change after feeding.

Discussion

Postprandial hypotension is one of the annoying complications in hypertensive patients which may result from re-distribution of circulating blood by the dilation of intestinal blood vessels (3). Dopamine is known to be a depressor catecholamine and to be released from the sympathetic nerve endings. From our results, it is suggested that the increase in plasma dopamine following feeding is accompanied...
by the increase in plasma norepinephrine in normal subjects. This concomitant increase of these two catecholamines are disturbed in essential hypertensive patients. This may indicate that the enzyme activity responsible for the synthesis of neuronal catecholamine is different between N and EH. Since the ratio of norepinephrine to dopamine following feeding was greater in EH than in N, it is speculated that the dopamine β-hydroxylase activation induced by decreasing blood pressure is accelerated in EH. Although dopamine has a potency of vasodilation via stimulation of vascular dopamine (D1-like) receptors, it is unlikely that dopamine play a predominant role in the cause of postprandial hypotension. It is because that the feeding-induced decrease in blood pressure was not related to the increase in plasma dopamine. In conclusion, postprandial decrease in blood pressure was enhanced in EH where the peripheral dopaminergic system may not have important role. In spite of the relative enhanced activity of neuronal dopamine β-hydroxylase activity, vascular responsiveness to circulating norepinephrine may be insufficient in EH.

References

