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Symptomatic postprandial hypotension in high paraplegia. Case report

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Symptomatic postprandial decrease in blood pressure has been described in patients with various autonomic disorders, but not in patients with spinal injuries. Presented herein is a 31 year old female patient with traumatic complete paraplegia under the T3 level, in whom postprandial hypotension (PPH) was observed. The PPH was preceded by an increase in insulin level and was followed by an acceleration of heart rate. Oral caffeine prevented the hypotension and alleviated the symptoms. It is suggested that the PPH might be manifested as a result of damage to an upper thoracic spinal baroreflex. Clinical investigation of PPH is recommended for patients with high paraplegia.

Key words: PPH – postprandial hypotension; high paraplegia; spinal baroreflex; caffeine.

Postprandial hypotension (PPH) may occur in patients with impaired autonomic function;[1] sometimes the decrease in blood pressure (BP) is symptomatic.[2] PPH was demonstrated in association with various conditions of autonomic dysfunction, including autonomic failure,[3] diabetes mellitus,[4] tabes dorsalis,[5] ganglionic blockage[6] and old age.[7]

Although patients with complete cervical spinal cord lesion have severe sympathetic autonomic impairment, they did not show a reduction in BP after food ingestion in a previous study.[8] To the best of our knowledge, this is the first report of a paraplegic patient with symptomatic PPH.

Case report

A 31 year old female with traumatic paraplegia under the level of the third thoracic segment (T3) since 1976 was admitted to the Spinal Department of Loewenstein Rehabilitation Hospital. She suffered from dizziness, drowsiness, weakness and palpitations in the sitting position, immediately after meals. These symptoms sometimes prevented her from getting up from bed, and she avoided eating when she planned activities that were not possible in the supine position. The patient in general ate little and was of slight build.

On physical examination, her general condition was good. Blood pressure was 95/60 mmHg and pulse rate (PR) was 60/min. Examination of lungs, heart and chest revealed no abnormalities. Neurological examination revealed complete spastic paraplegia below the T3 level. Routine laboratory tests (blood count, ESR, electrolytes, liver and kidney functions) were within normal limits.

To discover the cause of her complaint, BP, PR and some biochemical and hormonal plasma constituents were measured before and after the ingestion of a meal. Results were compatible with a diagnosis of PPH. The hypotensive effect disappeared when the tests were repeated with the oral administration of 250 mg caffeine before the meal.

Materials and methods

The tests were conducted after a 12 hr fast. During testing the patient lay flat on a tilt bed, held with wide bands fixed around chest, hip girdle and knees. No pressure was applied to the abdomen. A cannula was inserted into a cubital vein and irrigated

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with heparin diluted to 50 u/ml. A meal consisting of 60 g Isocal and 55 g Maxigul in 300 ml of milk was administered (Table I). BP and PR were monitored at least twice every 10 min from 76 min before the meal to 105 min after the meal. The bed was tilted to 35 deg for 10 min periods, at 25 min before the meal and at 45 and 95 min following the meal. Blood samples for glucose, insulin, plasma renin activity (PRA), sodium (Na) and potassium (K) testing were drawn through the cannula before and after the meal, in supine and tilted positions.

The procedure was repeated 24 hours later. Measurements were begun at 56 min before the meal; at 30 min before the meal 250 mg caffeine was ingested. Blood sampling was omitted.

Results

Basal haemodynamic and humoral measurements

On the first day of testing, before the meal, and with the patient in the supine position, systolic blood pressure fluctuated between 90 and 101 mmHg and diastolic between 50 and 64 mmHg; MBP was 66–80 mmHg. Pulse rate was 57–66 bpm (Fig 1a, b). Plasma Na was 137 mEq/l, K 4.3 mEq/lit, glucose 63–67 mg/100 ml, PRA 1.0–1.1 ng/ml/hr, and insulin 5.0–5.8 μu/ml (Fig 1c, d, e). Basal blood pressure and heart rate were lower than those of healthy subjects but similar to those of previously studied tetraplegic patients.[9] The Na, K and PRA were within normal limits, but the glucose and insulin levels were close to those reported after 24 hr fasting in healthy subjects.[10]

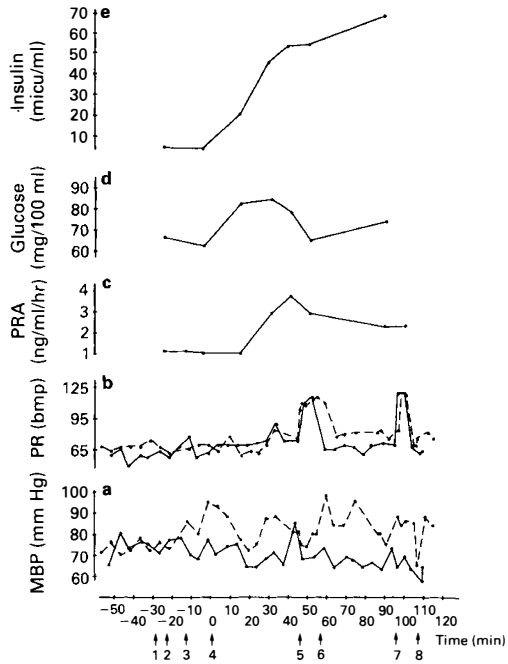

Figure 1 Haemodynamic and humoral variables before and after the meal, in supine and head-up tilt positions, without caffeine (continuous line) and with caffeine (segmented line). (a) Mean blood pressure (MBP). (b) Pulse rate (PR). (c) Plasma renin activity (PRA). (d) Plasma glucose level. (e) Plasma insulin level. *Arrows* 1: Caffeine ingestion (250 mg); 2, 5, 7: 35 deg tilt; 3, 6, 8: Bed supine; 4: Meal.

Haemodynamic and humoral effects of the meal

The mean supine post prandial MBP (68.8 mmHg, SD = 6.0) was significantly lower than the mean basal MBP (73.8 mmHg, SD = 3.9) ($p < 0.01$); this difference increased during the second hour

Table I Meal constituents

	Carbohydrates (g)	Fats (g)	Proteins (g)	Na (g)	Energy (kcal)
60 g Isocal	35.8	11.8	12.0	0.14	282
55 g Maxigul	54.9	—	—	0.13	220
300 ml milk	15.0	3.0	9.0	0.15	123
Total	105.7	14.8	21.0	0.42	625

after the meal (mean supine MBP = 66 mmHg, SD = 3.9) and was even more significant ($p < 0.0005$) (Fig 1a). The mean PR following food ingestion (70.1 bpm, SD = 4.9) was significantly higher than the mean basal PR (62.3 bpm, SD = 2.9) ($p < 0.0005$) (Fig 1b). Na and K levels remained unchanged after the meal (135–138 mEq/l and 4.2–4.6 mEq/l, respectively). The glucose level reached a peak of 85 mg/100 ml at 30 min after start of the meal, and then sloped down to 66–74 mg/100 ml (Fig 1d). Insulin reached a level of 46 μu/ml (about 8 times the basal level) when the glucose level was 85 mg/100 ml, and then continued to rise to 68 μu/ml (about 13 times the basal level) despite the reduction in the glucose level (Fig 1e). These insulin levels were much higher than expected at the patient's glucose levels.[11] PRA reached a peak value of 3.7 ng/ml/hr at 40 min after start of the meal and then decreased to 2.4 ng/ml/hr (Fig 1c).

Haemodynamic clinical and humoral effects of meal plus tilt

The tilt had little effect on BP, either before or after the meal (Fig 1A). On the other hand, tilting caused PR acceleration. The acceleration was slight prior to food ingestion (to 71–88 bpm), but very prominent after the meal (to 111–120 bpm) (Fig 1B). The patient was asymptomatic when tilted to 35 degrees during fasting, but she reported palpitations, dizziness, nausea and 'black spots' in the visual field when tilted after the meal. PRA was not elevated during tilt.

Haemodynamic effects of meal plus caffeine

Ingestion of caffeine alone elevated the basal MBP from 70–79 mmHg to 95 mmHg. The mean supine MBP after the meal with caffeine (83.6 mmHg, SD = 8.3) remained higher than the mean basal MBP (73.7 mmHg, SD = 3.6). Even during tilt, the mean postprandial MBP (81.5 mmHg, SD = 5.7) was higher than the mean basal MBP (Fig 1a).

Caffeine ingestion did not prevent the postprandial tachycardia and palpitations (Fig 1b). It was quite clear from the patient's report, however, that significant symptomatic relief was achieved with caffeine ingestion.

Discussion

The study showed that in our present patient food was a factor inducing symptomatic hypotension. Her BP was significantly reduced after food intake, and symptoms that did not appear before the meal in either supine or tilt position appeared after the meal in tilt position. Ingestion of caffeine (250 mg) which prevented PPH in patients with autonomic failure,[12] also prevented the reduction in BP in our patient. The PPH followed an accessive increase in insulin level (Fig 1a, e). The reason for such an increase is not clear, but it is plausible that PPH was initiated by insulin-induced vasodilatation.[13]

Postprandial vasodilatation followed by increased intestinal blood flow is a physiological phenomenon.[14] PPH, however, is unusual in both healthy subjects and patients with complete tetraplegia.[7,8] In healthy subjects, the postprandial vasodilatation is probably buffered by vasoconstriction, induced mainly by the sympathetic baroreflexes, which allows physiological redistribution of the blood flow to the splanchnic[15] and hepatic[16] beds and prevents a decrease in BP.[5] In patients with complete tetraplegia PPH does not occur, probably because the vasoconstrictor buffering is not prevented by de-efferentation of the reflexes originating in the baroreceptors of the aortic arc and carotid sinus.[8] This could be explained by some of the mechanisms suggested as preventing orthostatic hypotension in chronic tetraplegics.[17]

One possible explanation for the symptomatic PPH in our patient and not in tetraplegics, is an upper thoracic spinal baroreflex that had been interrupted whilst preserving the upper sympathetic nerve supply to the cerebral vessels and a part of the heart. Such a purely spinal upper thoracic BP reflex was suggested by Gilliatt *et al*.[18] This hypothesis was supported by Brown and Malliani who showed a spinal reflex ho-

meostatic regulation of BP in cats, with increased sympathetic discharge through the white ramus at the third thoracic level.[19]

Preservation of the ability to activate cardiac nerves through cervical and uppermost thoracic spinal segments apparently allowed in this particular patient prominent reactive tachycardia, which is limited in complete tetraplegics.[5] Preservation of her sympathetic supply to cerebral vessels, could impair autoregulation of cerebral blood flow (CBF) at low BP, which is possible in patients with chronic tetraplegia.[20] Therefore, symptoms such as palpitations, dizziness and drowsiness were likely to appear in this patient in contrast to patients with complete cervical lesions. These suggestions, however, should be further evaluated in a group of patients with high paraplegia.

Although the compensatory mechanism for postprandial vasodilatation was damaged, the compensatory reaction to head-up tilt remained intact: PPH was demonstrated in the absence of orthostatic hypotension. Different responses to tilt and food administration were also expressed in the level of PRA. Renin release, which is probably dependent on the decrease in renal perfusion pressure,[21] remained unchanged in spite of the pre meal head-up tilting, but was enhanced in the supine position after the meal (Fig 1c). It seems, therefore, that the mechanism buffering the tilt effect may be different from that buffering the food effect. Caffeine intake caused an elevation in BP before and after the meal, so that BP was above the basal level most of the time. On the other hand, the effect of caffeine on PR was diverse and insignificant (Fig 1a, b). These results were probably caused by the combination of the direct cardio-accelerator effect and the secondary vagally-mediated bradycardia effect of caffeine.[12] The effect of caffeine on BP but not on PR may explain the relief of symptoms compatible with brain ischemia such as dizziness, nausea and visual disturbances, without relief of the palpitations, which followed tachycardia.

The findings in this study suggest that an upper thoracic spinal lesion may cause symptomatic PPH. Since caffeine may alleviate symptoms of PPH, clinical examination for the presence of PPH might be beneficial to these patients.

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