Management of Postprandial Hypotension in a Tetraplegic Patient after Cervical Spinal Cord Injury

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Our aim was to present a case of postprandial hypotension in a tetraplegic patient with a cervical spinal cord lesion. A 43-year-old woman presented in the emergency room with tetraplegia after a motor vehicle crash. Radiologic studies showed dislocation at the C4-C5 level. Promptly, under general anesthesia, she underwent closed reduction, anterior cervical discectomy, and fusion at the C4-C5 level for stabilization. From the second day postoperatively, her systolic blood pressure was reduced from 120-150 mmHg to 40-60 mmHg when she made a positional change or after she ate. We tried administering a very slow and continuous infusion rate of a liquid meal (60 cc/h), and it was very effective in controlling PPH. In rare cases of PPH in a tetraplegic patient with a cervical spinal cord lesion, a very slow and continuous infusion rate of a liquid meal (60 cc/h) may be one effective management method.

Key Words: Tetraplegia; Hypotension; Feeding methods

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INTRODUCTION

Postprandial hypotension (PPH) has been defined as a decrease in systolic blood pressure (BP) of 20 mmHg or a decrease <90 mmHg from a pressure of 100 mmHg within 2 h after a meal⁽⁹⁾. PPH occurs in elderly persons and in those with autonomic dysfunction, Parkinson disease, paraplegia, diabetes mellitus, tabes dorsalis, and ganglionic blockage^(3,6).

There is a previous report about symptomatic PPH in a paraplegic patient with complete neurological deficits below the T3 segment⁽³⁾ and an experimental article about the relationship between PPH and thoracic spinal cord damage⁽²⁾. These reports have stated that PPH cannot occur in tetraplegic patients with cervical cord injury. However, we report a case of symptomatic PPH in a tetraplegic patient after cervical cord injury and discuss its management.

CASE REPORT

A 43-year-old woman presented to our emergency room after a motor vehicle crash. On neurologic examination, she was tetraplegic. She had no pinprick or light touch sensation below the C4 level and had difficulty breathing. Plain radiographs showed facet dislocation at the C4-C5 level, suggesting a spinal

Fig. 1. Preoperative radiographs of the anteroposterior (A) and lateral (B) views showing dislocation of both facet joints at the C4-C5 level, and fracture of the spinous process and both lamina of C4.

cord lesion (Fig. 1). Magnetic resonance imaging (MRI) showed spinal cord contusion at the C3-C6 level (Fig. 2). Halo traction was gently applied with the patient under general anesthesia as the first step of treatment for closed reduction. After successful closed reduction, anterior discectomy at the C4-C5 level, anterior fusion with a cage filled with allo-bone graft, and anterior plate fixation at the C4-C5 vertebral body was performed (Fig. 3). Postoperatively, she was transferred to the intensive care

Fig. 2. Preoperative magnetic resonance images in the sagittal (A) and axial (B) views showing dislocation of both facet joints and spinal cord compression at the C4-C5 levels.

Fig. 3. Immediate postoperative radiographs in the anteroposterior (A) and lateral (B) views.

unit. To improve spinal cord perfusion, her BP was maintained between 140/80 mmHg and 120/60 mmHg with an infusion of dopamine (10–15 $\mu\text{g/kg/min}$). From the second day postoperatively, her systolic blood pressure (SBP) decreased from 120–150 mmHg to 40–60 mmHg, and she lost consciousness. This phenomenon occurred frequently after position change from supine to head elevation, defecation, or ingestion of liquid meal through a Levine tube (300 cc/h). Therefore, we could not taper the dose of dopamine. She had no self-respiration until 6 days postoperatively, thus tracheostomy was performed. A portable ventilator was used for transfer to the general ward. Until 20 days postoperatively, the fluctuation in BP occurred continuously after a positional change or eating a meal. At first, it may have been helpful to use midodrine, an α 1-receptor agonist, as a vasoconstrictor for postural hypotension and PPH. However, it had no effect on BP fluctuation after eating or positional changes. On 24 days postoperatively, when the patient's position was changed very gently, SBP was maintained with a decrease of about 20 mmHg. On 27 days postoperatively, we had decreased the continuous infusion rate of the liquid meal in stages until a modest rate for preventing a reduction in the SBP was identified. Ultimately, we determined that 60 cc/h was

the maximum infusion rate for maintaining BP. After infusing liquid meals at 60 cc/h, PPH did not occur anymore, and dopamine was tapered.

DISCUSSION

Mathias et al.¹⁰⁾ suggested that similar to patients with a dopamine β -hydroxylase deficiency, tetraplegic patients have the ability to activate vagal efferent pathways and may release peptides and other active vasoconstrictor substances from sympathetic nerve endings, thus PPH cannot occur in tetraplegic patients with a cervical cord lesion. Moreover, Catz et al.^{2,3)} reported that in their experiment, PPH could not occur in tetraplegic patients with a cervical cord lesion, but it can occur in paraplegic patients with a thoracic spinal cord lesion. They suggested that the normal hemodynamic reaction during a liquid meal is mediated by the mid-thoracic spinal cord rather than by signals descending through the cervical spinal cord. However, our case suggests that PPH can occur in paraplegic and tetraplegic patients. There is no definite mechanism for PPH in tetraplegic patients.

There are several theories about PPH in elderly persons, but the mechanism has not been fully described. In elderly persons, the bowel blood volume increases by approximately 20% after a meal, and this is maintained longer than that in young persons⁷⁾. The bowel blood volume is absent or blunted by the sympathetic response to systemic hypotension evoked by an intraduodenal glucose load^{5,11)}. Furthermore, insulin-induced blunting of the baroreflex sensitivity in elderly persons may affect BP¹⁾. Similar to PPH in elderly persons, PPH in tetraplegic patients with a cervical spinal cord lesion may be due to the blunted sympathetic response to the increase in splanchnic blood flow after meal ingestion. Bowel movements in autonomic failure may induce PPH because of the intrathoracic pressure elevation⁹⁾. Therefore, it is easier for tetraplegic patients who do not have spontaneous breathing to be affected by bowel movements. Additionally, we hypothesized that in our patient, the mechanism for a normal hemodynamic reaction during a liquid meal was mediated by signals descending from the cervical spinal cord, and the patient had no ability to activate vagal efferent pathways like patients with a dopamine β -hydroxylase deficiency.

Nonpharmacologic modification or pharmacotherapy can be beneficial for treating PPH⁸⁾. Nonpharmacologic modifications include drinking water before meals, frequently eating small meals, and maintaining a recumbent or sitting position after meals. Drinking water before meals to increase the intravascular volume and changing positions were not effective for our patient. Only slowing the infusion rate of the liquid meals was effective. Pharmacotherapy includes caffeine, α -glucosidase inhibitors, guar gum, and octreotide. However, α -glucosidase inhibitors

and guar gum can cause diarrhea, and diarrhea causes effects such as malnutrition and ileus through an electrolyte imbalance in tetraplegic patients. Octreotide can increase the splanchnic and peripheral vascular resistance⁴⁾, but it is expansive and has side effects of QT prolongation and pain at the injected location. The preprandial pressor effect of caffeine prevents PPH, and caffeine is nontoxic, inexpensive, and widely available^{6,8,11)}.

CONCLUSION

In tetraplegic patients with a cervical spinal cord injury, PPH can occur; however, the slow and continuous infusion of a liquid meal is effective treatment of PPH.

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