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Little weight loss related to short NPO period causing SMA syndrome in patient with ALS

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Introduction

Superior mesenteric artery (SMA) syndrome is a rare cause of small bowel obstruction, characterized by an extrinsic vascular compression of the 3rd portion of the duodenum, between the abdominal aorta and overlying SMA. The most common cause of SMA syndrome is known to be significant weight loss (commonly 33~55% of initial body weight) that leads to loss of mesenteric fat pad. Here we present a case of a male patient with amyotrophic lateral sclerosis (ALS), who developed SMA syndrome abruptly after 5 days of fasting.

Case

A 61 years old man who was diagnosed with ALS was admitted to the hospital for evaluation and management of blood backflow through the percutaneous radiologic gastrostomy (PRG) tube. He was 176cm tall and weighed 43kg (BMI 13.88 kg/m2). Esophagoduodenoscopy was performed and acute gastric ulcer with recent bleeding was identified. The irritation by PRG tube was thought to the cause of bleeding, therefore it was removed. Patient underwent therapeutic fasting and high dose proton pump inhibitor and 3rd generation cephalosporin was administered intravenously. After 5 days of fasting, patient resumed feeding through Levin tube and percutaneous endoscopic gastrostomy (PEG) tube was inserted 12 days after PRG tube removal. Feeding was resumed through PEG tube starting from 100cc and increased thereby. When the feeding amount reached 200cc, the patient presented vomiting, abdominal distention and tachycardia. Amount of the feeding was adjusted and maintained 100 cc. Body weight was re-measured it was reduced from 43kg (BMI of 13.88 kg/m2) to 38kg (BMI 12.26 kg/m2). We performed abdomen CT and found that the second portion of the duodenum is narrowed at the site between the aorta and the SMA and severe stomach distension above at the level which led to diagnosis of SMA syndrome. The patient underwent jejunal tube insertion, and the amount of feeding through the jenunal tube was gradually increased while maintaining parenteral nutrition. After 2 weeks, presenting symptoms were improved, follow up CT scan was performed to find improvement of duodenal narrowing and stomach distension. Tubogram showed passage of contrast agent through the duodenum without disturbance. Afterwards patient maintained diet by PEG. At discharge, the patient tolerated 500cc of diet and body weight was increased to 43kg.

Conclusion

In this case, we experienced a rare case of the patient with ALS who developed SMA syndrome despite a short period of fasting and relatively small weight loss. When ALS

patients with malnutrition state present vomiting or abdomen distention after feeding, possibility of SMA syndrome should be on the list of consideration and proper evaluations should be performed in order to provide proper nutritional support.