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Superior mesenteric artery syndrome caused by surgery and radiation therapy for a brain tumor: A case report

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Abstract. Superior mesenteric artery syndrome (SMAS) is defined as an obstruction of the third part of duodenum due to compression by the superior mesenteric artery. Although traumatic brain injury is a risk factor for SMAS, few cases of SMAS resulting from brain surgery have been reported. SMAS has been observed to occur following neurosurgical surgery in pediatric patients but, to the best of our knowledge, no such cases have been reported in adults. The present study reports the case of a 21-year-old female patient who developed SMAS after persistent vomiting and prolonged weight loss following cerebellar tumor resection and cranial irradiation. The SMAS was confirmed by computed tomography and resolved following successful nutritional management.

Introduction

Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome, was first reported by Rokitansky in 1842 (1). It is defined as a compression of the third part of duodenum by the superior mesenteric artery (SMA). Numerous risk factors can lead to SMAS, including weight loss, spasticity, prolonged recumbency and severe traumatic brain injury (2). Although SMAS frequently occurs after traumatic brain injury, no cases of SMAS following brain surgery and cranial radiation have been reported. To increase awareness of this rare treatable effect of neurosurgery, the case of a patient who developed SMAS following cerebellar tumor resection and cranial irradiation is described in the present study.

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Case report

In March 2013, a 21-year-old woman underwent brain surgery following the diagnosis of neuroglioma at Jiangsu Provincial People's Hospital (Nanjing, China). The patient was also received Gamma Knife treatment (local brain radiotherapy) postoperatively, which stabilized her condition for a while. The patient, who had an unremarkable medical history, was admitted to a local hospital in May 2014, with a 3-day history of nausea and vomiting after eating. Symptom relief was achieved 1 week later following the treatment of influenza. The patient further developed chronic intermittent abdominal pain, repeatedly with no known cause. On initial examination, computed tomography (CT) plain and enhancement scanning of the abdomen showed duodenal stasis. The patient was given enteral and parenteral nutritional support treatment for >10 days. However, the patient exhibited no improvement and on July 13, 2014 was transferred to Jinling Hospital (Nanjing, China) for continuing management.

On admission to the hospital, the patient was generally unwell, exhibiting physical decline and loss of appetite. Clinical examination was unremarkable. The patient had undergone significant weight loss since the start of the illness (~10 kg), with a Patient-Generated Subjective Global Assessment (PG-SGA) classification of grade C and a Nutritional Risk Screening (NRS)-2002 score of 6. The patient was 1.63 m in height and weighed 34.5 kg, corresponding to a body mass index of 12.99 kg/m^2. Her resting energy expenditure (REE) rate was measured to be 1,229 kcal/day by indirect calorimetry. Levels of total protein (57.7 g/l), albumin (37.1 g/l) and prealbumin (154 mg/l) were low. The patient underwent magnetic resonance imaging (MRI) scanning, which revealed an annular focal high-intensity signal in the region of the right cerebellar hemisphere (Fig. 1). Based on the history of treatment, tumor recurrence was considered. On July 31, 2014, a CT scan confirmed compression of the duodenum (Fig. 2), and the diagnosis of SMAS was made by multidetector computed tomography angiography (CTA; Figs. 3 and 4).

Once the diagnosis of SMAS was established, treatment with nutrition support was initiated. For this patient, combined enteral and parenteral nutrition support was the preferred method. A percutaneous endoscopic gastric jejunum colostomy was performed for the subsequent management of the

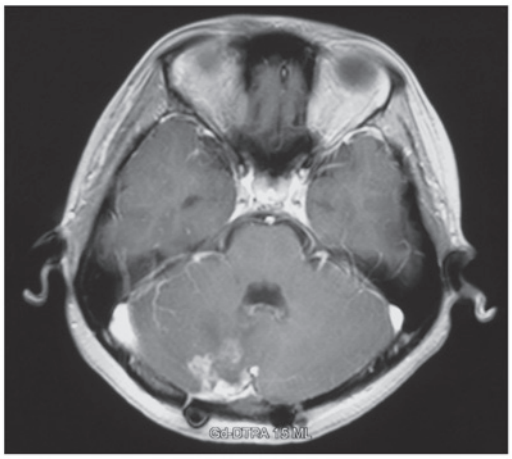

Figure 1. Axial magnetic resonance imaging scan of the brain shows an annular focal high-intensity signal in the region of the right cerebellar hemisphere.

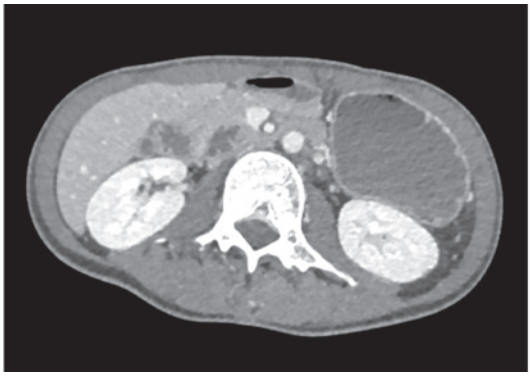

Figure 2. Axial computed tomography scan of the abdomen shows a narrowed aortomesenteric distance and a small, dilated proximal duodenum.

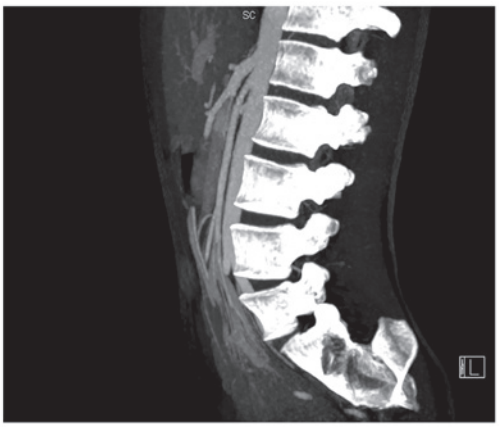

Figure 3. Sagittal slice of a computed tomography angiography scan of the abdomen shows that the superior mesenteric artery-aorta angle is small.

patient on August 11, 2014, and a step-by step process was used to enable a gradual transition to the provision of total enteral nutritional support, with the aim of inducing the patient to gain weight. The fluid and electrolyte balance of the patient were also regulated. The patient was discharged to be cared for by her family on August 18, 2014, and continued to receive enteral nutritional support. At the last follow-up 3 months later, the patient's nutritional status had improved.

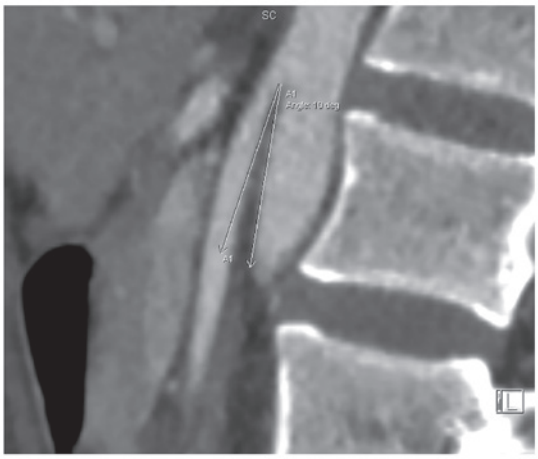

Figure 4. Sagittal slice of the computed tomography angiography scan of the abdomen shows that the superior mesenteric artery-aorta angle in the patient is ~10˚.

Discussion

SMAS is a relatively rare cause of duodenal outlet obstruction resulting from compression of the third part of the duodenum between the SMA and the aorta (1,3). In 1982, the prevalence of SMAS in the general population was reported to be between 0.013 and 0.3% (4). It has also been reported that SMAS occurs most frequently in older children and young adults, with a higher incidence in females (5,6).

There are numerous potential causes of SMAS, including congenital and acquired factors. Congenital factors are mainly anatomical anomalies, such as a low take-off position of the SMA and an abnormally high origin of the ligament of Treitz (7). Acquired factors include numerous severe wasting diseases (such as cancer, trauma or burns), disorders resulting from malnutrition (such as malabsorption or anorexia nervosa) and prolonged vomiting (2). It is worthy of note that the postoperative state is also a common cause of SMAS. A study in 2012 reported a case of SMAS resulting from minimally invasive correction of pectus excavatum (8), which implies that postoperative malnutrition resulting from non-abdominal surgery may be a potential cause of SMAS. In neurosurgery, postoperative malnutrition and vomiting are very common following brain tumor surgery. In 2008, Bhattacharya *et al* described the case of a 3-year-old female child with pilocytic astrocytoma who had persistent vomiting following neurosurgical procedures for posterior fossa astrocytoma; during the recovery period, the patient was diagnosed with SMAS (9). As early as in the 1990s, certain scholars had observed that severe traumatic brain injury is a cause of SMAS (2,10). Brain injury and brain surgery frequently present the same symptoms; managements such as craniotomy, tumor resection and radiotherapy in patients with brain tumors are a cause of brain injury. It may be speculated that in the present patient, a thin younger woman who underwent brain tumor surgery and radiotherapy, it is likely that prolonged vomiting and postoperative malnutrition contributed to weight loss, resulting in the obstruction of the duodenum.

The clinical symptoms of SMAS are usually nonspecific, and include epigastric pain, bilious vomiting, nausea, fullness, postprandial bloating and weight loss (3,11). The diagnosis is mainly based on the history and clinical features and can be confirmed by radiological examination of the abdomen, for example, with CT and a barium meal. The angle between the abdominal aorta and SMA is usually 38-60˚, and the normal distance between those two blood vessels averages 10-28 mm (12). However, the angle between the abdominal aorta and SMA in patients with SMAS is usually <22˚ and the distance between vessels is <8 mm (13,14). The angle between the abdominal aorta and SMA observed in the CTA scan of the present patient was ~10˚, which also supports the diagnosis of SMAS. The choice of treatment is usually dependent upon the cause of SMAS and degree of obstruction. Conservative therapeutic approaches include nasogastric decompression, hyperalimentation (15), maintenance of fluid and electrolyte balance (3) and nutritional therapy. Surgical procedures are indicated if there is no response to conservative treatment.

In conclusion, to the best of our knowledge, the present patient is the first reported case of SMAS in an adult following neurosurgical surgery and cranial irradiation. In the present case, a progressive decline in body weight occurred during the postoperative period following brain tumor surgery, but in the absence of effective nutritional therapy, weight loss persisted, finally resulting in the development of SMAS. When prolonged vomiting and acute weight loss are observed postoperatively in patients who have undergone brain surgery or cranial irradiation, neurosurgeons should consider the possibility of SMAS. A combination of parenteral and enteral nutritional therapy is recommended.

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