Case report

Superior mesenteric artery syndrome and its ramifications

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Abstract

The superior mesenteric artery extends anteriorly and inferiorly off the aorta at the level of the first lumbar vertebrae. The duodenal sweep and left renal vein are located in the aorto-mesenteric angle space. A decrease from the normal angle may compress these two structures. The case presented here discusses a unique patient with significant compression of the third portion of the duodenum and possible enlargement of the proximal left renal vein as these two structures cross the aorto-mesenteric angle.

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1. Case Report

A previously healthy 13-year-old boy presented with a one-day history of peri-umbilical abdominal pain and multiple episodes of bilious vomiting after ‘overeating’ at a family barbeque. The patient stated he had recently lost 26 pounds. The remainder of the clinical history was unremarkable. The patient denied any past medical or surgical history. On physical exam, the patient was noted to be tall and thin and had diffuse abdominal tenderness.

Abdominal radiographs were obtained which showed a markedly distended, fluid-filled stomach. CT scan of the abdomen and pelvis was performed with intravenous and oral contrast. This showed a grossly distended duodenum to the third portion where it is abruptly tapered and collapses completely between the superior mesenteric artery (SMA) and the inferior vena cava (Fig. 1). The patient was also noted to have partial compression of the left renal vein (LRV) by the superior mesenteric artery with more proximal dilation (Fig. 2).

The laboratory results on presentation revealed a BUN 25 and a creatinine 1.7. The patient was afebrile and vital signs were stable. After 1400 cm³ of emesis was noted, a nasogastric tube was placed and 2500 cm³ of gastric fluid was obtained overnight. Contrast upper GI study was performed that demonstrated an abrupt cut-off in the third portion of the duodenum with a massively distended stomach extending into the pelvis (Fig. 3). Greater than 1000 cm³ of nasogastric tube drainage was noted for the next few consecutive nights.

Conservative management was planned and parental nutrition was started. The patient was well hydrated with intravenous fluids and the creatinine level declined to the normal range in less than one week. The obstructive symptoms resolved with the nutritional support. The nasogastric tube was removed. Oral caloric and nutritional supplementation was started and the patient was discharged from the hospital. The patient continues to do well.

2. Discussion

The origin of the SMA is at the first lumbar vertebrae. The SMA extends inferiorly at an acute angle with the aorta. The third portion of the duodenum crosses the aortomesenteric angle at the third lumbar vertebral body. In the same respect, the LRV normally crosses the aortomesenteric angle, below the duodenum.

The normal aortomesenteric angle has been previously noted as 38–65⁰ [1]. The normal distance from the aorta to the SMA at the duodenal crossing is 13–34 mm [1]. When there is a relative decrease in space between the aorta and the SMA, there is a possibility of compression on
the third portion of the duodenum. Diagnostic criteria for SMA syndrome include a decrease in the aorto-mesenteric angle to 6–16° and a decrease in the aorta–SMA distance to 5–11 mm.

Anatomic causes include exaggerated lumbar lordosis, abnormally high fixation of the duodenojejunal flexure of the ligament of Treitz, an unusually low origin of the SMA, or a decrease in retroperitoneal fat in the aorto-mesenteric angle. The decrease in fat and lymphatic tissue in this region may be seen in patients who have either a severe wasting disease, including cancer or burns, causing a hypermetabolic state, severe injuries, deformity or trauma to the spine or aorta, dietary disorders and the postoperative state [1,2]. These syndromes usually occur as chronic illnesses, however, it is not uncommon to present with acute symptoms.

Typical symptoms of the SMA syndrome are postprandial upper abdominal pain and distress, early fullness, nausea, and vomiting. Plain abdominal radiograph illustrates marked gastric dilation. Diagnosis, until the advent of CT, has been the barium UGI series that showed an abrupt vertical or oblique compression of the third portion of the duodenum with proximal dilation [2]. There is continuous active peristalsis and delay in overall transit time of the barium meal. During the UGI series, placing the patient in the knee–chest position (Hayes maneuver) should relieve the duodenal obstruction [3]. CT, and the now less used arteriography, demonstrates the close proximity of the SMA and the aorta. CT also clearly illustrates the caliber change of the duodenum.

The primary treatment is conservative with fluid resuscitation and proper positioning of the patient during feeding, which includes the modified knee–chest, prone or left side down positioning, which increases the aorto-mesenteric...
angle. If necessary, the passage of a tube past the point of obstruction, jejunostomy placement or Strong’s operation (mobilization of the ligament of Treitz) may be needed [1]. Surgical bypass is reserved when conservative measures have failed.

The nutcracker syndrome, like the SMA syndrome, is due to the compression of the LRV between the aorta and the SMA. The elevated LRV pressure overtime produces renal hilar varices and the development of collateral veins. Collateral veins should relieve the increased LRV pressure. Collateral venous circulation including the gonadal, ascending lumbar, adrenal, ureteral and capsular veins has been well documented [4].

The typical clinical features are varying degrees of microscopic or macroscopic hematuria, orthostatic proteinuria and left flank pain. The hematuria is thought to be secondary to LRV hypertension with rupture of the small renal vessels [5]. Proteinuria has been shown to be slightly greater during a recumbent posture as compared with an upright posture or during exercise [6]. There is more reliance on ultrasound, CT, or MRI for diagnosis and less reliance of the more invasive venography, which measures the pressure gradient between the LRV and the inferior vena cava. A pressure gradient of 3 mm Hg during venography has been found to be significant [4]. The AP diameter and the duplex US peak systolic velocities of the LRV are measured at the renal hilum and in the aorto-mesenteric angle. An AP diameter ratio of greater than 5 has a sensitivity of 69% and a specificity of 89% for LRV nutcracker syndrome. A peak systolic velocity ratio greater than 5 has a sensitivity of 80% and a specificity of 94%. Together, the combined AP diameter and peak systolic velocity ratios have sensitivity to 90% and specificity to 100% [5,7]. CT is useful to show the LRV compression and formation of collaterals.

Treatment is usually conservative. Without worsening clinical symptoms or rising creatinine levels, only supportive care may be necessary. If the symptoms worsen, the treatment becomes surgical, including pre-aortic fibrous tissue resection, renocaval venous implantation or placement of a synthetic wedge into the bifurcation of the SMA [8]. If severe damage is done to the left kidney, nephrectomy may need to be performed [7].

3. Summary

The anatomic variations in this patient not only produced compression on the third portion of the duodenum with gastrointestinal obstructive symptoms but also compression on the left renal vein with proximal dilatation. However, in this patient, we did not see the clinical symptoms and radiographic signs of the LRV syndrome and we could not conclude that the decrease in the aorto-mesenteric angle seen here is producing both the SMA and LRV syndrome.

References


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