

Hypoglycemia a Rare Presentation of Superior Mesenteric Artery Syndrome

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Abstract

Background: Vomiting is a common presentation seen in pediatrics with wide range of causes. It is the most common presenting symptom of Superior Mesenteric Artery Syndrome. The concurrence of diabetes mellitus and Superior mesenteric artery syndrome is rare, especially in pediatrics. Both diseases can lead to recurrent vomiting whether as part of gastroparesis in diabetes mellitus or as part of intestinal obstruction in superior mesenteric artery syndrome.

Case Presentation: Here we describe a case of Superior mesenteric artery syndrome in 12 years old Type 1 diabetic patient who presented with recurrent attacks of vomiting associated with hypoglycemia and weight loss and the steps of diagnosis and its management.

Conclusion: This is the first case diagnosed with superior mesenteric artery syndrome who presented with multiple attacks of hypoglycemia and vomiting with concurrence of diabetes mellitus type 1. It targets to raise the awareness of superior mesenteric artery syndrome occurrence and its precipitating factors especially weight loss and optimal management steps, either conservative through dietary modifications or surgical intervention.

Keywords: Superior Mesenteric Artery Syndrome; Diabetes Mellitus; Vomiting and Hypoglycemia

Background

Diabetes mellitus (DM) is a multi-systemic disease with wide range of complications. It can result in a gastrointestinal symptom like nausea, vomiting, postprandial fullness, and anorexia secondary to gastroparesis as result of autonomic neuropathy [1]. In the same time these symptoms can be as result of other causes such as acute infection (e.g. acute gastroenteritis) or chronic causes (e.g. Peptic ulcer disease or gastritis) [2]. One of the rare causes of recurrent vomiting and weight loss is Superior Mesenteric artery Syndrome. Here we describe a diabetic patient (Type 1) with superior mesenteric artery syndrome.

Case Presentation

This is 12 years old Saudi girl known case of diabetes mellitus since age of 6 years old, since that time she was on multiple doses insulin regimen with poor compliance to both insulin and diet.

She presented with recurrent attacks of vomiting, non-projectile, mainly post prandial with no diurnal variation, for about 6 weeks associated with nausea, abdominal pain and decrease in appetite with weight loss which was significant about 5 kg. As a result, hypoglycemia has been developed with vomiting attacks that result in hospitalizations twice. There were no others attributing factors, such as overdose of insulin or exercise. As a result of the frequent attacks of hypoglycemia mother stopped insulin twice that lead to two attacks of hyperglycemia and development of diabetic ketoacidosis and requirement of intensive care admissions.

The past medical history reviewed, revealed a history of *H. pylori* infection one year back with similar presentation and responded well to treatment regimen for *H. Pylori* infection. So, patient was reinvestigated again and came negative for evidence of *H. pylori* through urea breath test, stool analysis and upper gastrointestinal endoscopy. She received several times anti emetics (metoclopramide) as symptomatic treatment with partial improvement in some vomiting attacks. There was no history of fever, diarrhoea, haematochezia, head trauma or any urinary symptoms or use of any other medications apart from insulin. On examination, patient was generally well not dysmorphic. Vital signs were normal with weight 32 kg (was 37 Kg) and height = 133 cm with BMI was 18.1 (above 25th centile which drop from 50th centile). Physical examination was unremarkable except for hepatomegaly. All haematological and biochemical profile was within normal. Her Hb_{A1C} was 9.8%. Abdominal ultrasound with doppler done twice in two different admissions revealed only hepatomegaly with no identified other abnormalities. Brain imaging was done to roll out space occupying lesion and revealed a normal study. Upper GI study done and was normal. Finally Abdominal CT scan with contrast done for her and found to have a markedly enlarged liver about 20 cm, with aorto-mesenteric angle 18 (normally > 25), aorto-mesenteric distance 8 cm (normally > 10 cm) and narrowed 3rd part of duodenum lumen which is suggestive of Superior Mesenteric Artery Syndrome (Figure 1 and 2). So, by addressing the final diagnosis patient was managed conservatively for six weeks by dietary counselling and regular follow up plan for the assessment of weight increment and improvement of symptoms.



Figure 1: This CT scan of abdomen with contrast Sagittal section showed depressed aorto-mesenteric angle (18), aorto-mesenteric distance (8 cm).

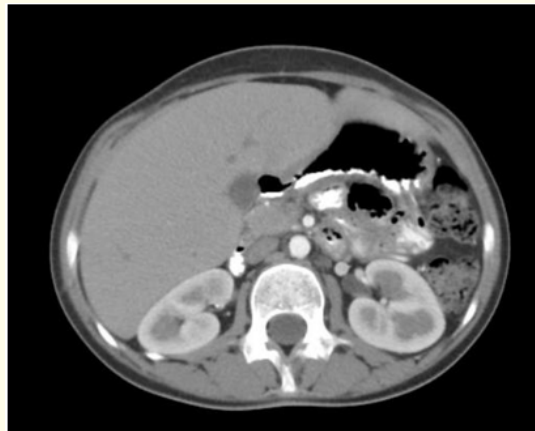


Figure 2: CT scan of abdomen with contrast coronal section showed: hepatomegaly around 20 cm with compressed third part of duodenum due to depressed aorto-mesenteric angle and distance.

Discussion

The superior mesenteric artery (SMA) originates from the abdominal aorta, just inferior to the origin of the celiac trunk at the level of 1st to the 2nd lumbar vertebrae. It is covered by adipose and lymphatic tissue. It runs in anterior or inferior direction and enters the small intestine tether and make an angle with the abdominal aorta which range from 38 to 56 degrees which is maintained by a fat pad, with a distance of 10 to 28 mm between these blood vessels. The superior mesenteric artery Syndrome (SMAS) is defined as proximal intestinal obstruction secondary of depressed aorto-mesenteric angle due to loss of retroperitoneal fat and vesical fat in the abdomen. This loss in body fat can be caused by either high energy demanding conditions such as chronic infection or chronic diseases like metabolic diseases, defect in anabolic factors such what seen with poor controlled diabetes mellitus and lack of insulin effect, or mechanical factors like trauma, post-surgeries or a high-degree of lumbar lordosis [3,4]. As a result, there will be compression on the duodenum, mainly the 3rd part, leading to intermittent obstruction. This leads to vomiting, early satiety and bloating sensation after meals secondary to delayed emptying of duodenum and stomach [1].

Diabetic patient at the risk of weight loss due to lack of insulin as it is considered an anabolic hormone. This is observed here, poor control of her blood sugar level, as it reflected by high hemoglobinA1c, secondary to mal adherence to insulin and diet predispose her to weight loss, then losses of retroperitoneal fat and development of SMAS. Also, this poor glycemic control explains the presence of hepatomegaly [4].

Clinical presentation of SMAS resemble that of diabetic gastroparesis and other common causes of recurrent vomiting, including symptoms of postprandial fullness, nausea, vomiting, and bloating up to the degree of acute intestinal obstruction [2,5].

Usually gastroparesis secondary to DM respond well to medications such as prokinetics e.g. metoclopramide and domperidone, or erythromycin. In comparison, our patient did not respond very well to prokinetic agents alone as what would be expected in gastroparesis [2,6,7].

There are varieties of radiological investigations aids in diagnosis of SMAS. Plain radiographs can show nonspecific signs of intestinal obstruction as gastric dilatation. Doppler Abdominal ultrasound can be diagnostic, but it is operator dependent, as in this case it failed initially in the diagnosis. For confirmation of diagnosis three criteria should be met. First, evidence of intestinal obstruction at the duodenal

level, mainly the third part of it. Then a depressed aortomesenteric artery angle of $\leq 25^\circ$. Finally, anomalies of superior Mesenteric artery or its origin, or high fixation of duodenum by ligament of Treitz. All this conformed here by abdominal CT scan with contrast as it is the gold standard radiological investigations which showed a decrease in aorto-mesenteric angle 18 (normally from 38 to 56 degrees), aorto-mesenteric distance 8 cm (normally from 10 - 28 cm) and narrowed 3rd part of duodenum lumen [8].

The management of SMAS can be either medical or surgical. Initially, a medical management consist of two steps. First, the resuscitation one includes fluid resuscitation and correction of electrolytes derangement. Then, weight gaining process to the fat pad which relieve the duodenal obstruction. The weight increment achieved mainly by dietary modification through consuming high caloric meals in small divided portions with some post prandial positioning include left lateral position, knee chest position or prone position which can improve symptoms. If the symptoms persist, stomach decompression can give some benefits through nasogastric tube and enteral feeding. Finally, if no improvement with enteral feeding, parenteral feeding can be initiated. The addition of proton pump inhibitors and/or prokinetics help to alleviate the symptoms. Evidence showed that medical management should be carried on before considering surgical intervention at least for 6weeks [9] that's was applied to our patient and after 6 weeks there were improvements of both symptoms i.e. there were no hypoglycemic attacks, nausea, or vomiting and there was weight gain.

In Meneghini, *et al.* reported a patient with type 1 diabetes mellitus diagnosed with SMAS. The presentation in both our patient and their were similar inform of recurrent vomiting and weight loos. Also, triggering factor was the same, which is weight loss secondary to poorly controlled blood sugar levels. The differences that the symptoms of SMAS were much more sever in our patient as it led to hypoglycemic attacks with an earlier age of presentation and lower glycemic index. Also, there was no hypoglycemia as it seen in our patient [1].

This is the first case reported in literature to be diagnosed with Superior Mesenteric Artery Syndrome through the presentation with hypoglycemia and concurrent illness with Diabetes Mellitus Type 1.

Conclusion

SMAS is a rare disease with a vague presentation resemble many other common diseases. To diagnose SMAS it needs a high index of suspicion specially if it was associated with other chronic illness share the same clinical signs and symptoms as it is seen with diabetic gastroparesis.

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