ABSTRACT
Gastric outlet obstruction (GOO) presenting with symptoms like nausea, vomiting, abdominal bloating, pain abdomen and distension of the abdomen can arise as a consequence of acid peptic disease, gastric polyps, malignancy of the stomach or gall stones. Superior mesenteric syndrome is a rare entity causing obstruction of the gastric outlet triggered by narrowing of aorta and mesenteric angle and shortening of aortomesenteric distance. We report a rare case of superior mesenteric syndrome presenting as gastric outlet obstruction to stress the importance of considering superior mesenteric artery syndrome as a differential diagnosis for GOO.

INTRODUCTION:
Superior mesenteric syndrome also known as Wilkie’s syndrome is a rare disease entity characterized by compression of third part of the duodenum between aorta and superior mesenteric artery (SMA) due to acute angulation of SMA resulting in partial or complete duodenal obstruction. It’s a disease of exclusion which can be life threatening at times. It usually occurs in debilitated patients and presents with upper abdominal symptoms like nausea, vomiting, pain abdomen and distension. Even though duodenal obstruction can be demonstrated by simple imaging like X-ray, confirmatory diagnosis requires multidetector CT or CT angiography to establish the superior mesenteric artery syndrome. Patients can be managed conservatively when presents with short history, moderate symptoms and incomplete duodenal obstructed. When patient remains symptomatic despite the conservative modalities surgical or laparoscopic procedures should be opted. High index of suspicion, timely diagnosis and appropriate treatment is of paramount importance in patients with Wilkie’s syndrome.

CASE REPORT:
A 44 year old male presented with complaints of colicky type abdominal pain, aggravated with food intake, abdominal distension and bilious vomiting which had undigested food particles. Patient also had early satiety and weight loss of about 12 kilogram in past 4 months. On general examination, patient was emaciated and malnourished. A vague mass of 17*8cm extending from epigastrium to umbilicus was palpated per abdomen. Visible gastric peristalsis and succussion splash were present. Laboratory investigations revealed hypoproteinemia and hypokalemia (s.protein-3 gram /dl and s.potassium -2.2meq/l).On imaging, X-ray abdomen showed dilated gastric lumen. Barium study was also done which showed similar findings. Upper GI endoscopy showed dilated duodenum with lot of bile. On contrast enhanced computed tomography of abdomen, proximal duodenum was distended. Third part of duodenum was seen compressed between aorta and Superior mesenteric artery with mesenteric angle less than 20 . Patient was managed conservatively with Nasogastric decompression, correction of fluid and electrolyte imbalances and protein supplementation in the form of Total parenteral nutrition. Patient gradually improved and was discharged on 15th day in good condition. He was followed up at 1 month and is doing well currently.

DISCUSSION:
Superior mesenteric artery syndrome also known as Wilkie’s syndrome...
The syndrome, aortomesentric duodenal compression syndrome, cast syndrome or chronic duodenal ileus was first described by Von Rokitansky in 1861 and was studied later in detail by Wilkie. On reviewing the literature, incidence of SMA syndrome is found to be in the range of 0.013-0.78%. It occurs due to the narrowing of aortomesentric angle causing compression of third part of duodenum. The normal angle formed by the SMA with abdominal aorta and third part of duodenum is approximately 45˚ (38-56˚). Narrowing of this angle to < 25˚ and shortening of the aortomesentric distance to < 10 mm causes duodenal compression. The predisposing factors for SMA syndrome can be congenital or acquired which include: malrotation of the gut, paraduodenal hernias, thin built body, exaggerated lumbar lordosis, lax abdominal wall, stress ulceration conditions involving rapid and severe weight loss, spinal surgeries and abnormally high position of ligament of Trietz. Wilkie’s syndrome poses a diagnostic dilemma and can be diagnosed clinically by exclusion. The symptoms are usually insidious in onset and include, post prandial epigastric discomfort, nausea, bilious vomiting, abdominal bloating and distension. Knee to chest posture may relieve the small bowel mesenteric tension and thus produce symptomatic relief. Hayes maneuver may also be useful to relieve subacute symptoms. Multidetector contrast CT imaging is the best modality for diagnosis of SMA and is superior to barium studies. Aortomesentric angle of < 22˚ and aortomesentric distance of < 8-10 mm confirms the diagnosis. X-ray abdomen shows dilated, fluid or gas filled stomach and fluoroscopy shows abrupt narrowing of 3rd part of duodenum with dilated 1st and 2nd part of duodenum, delayed gastroduodenal emptying and anti peristaltic waves proximal to the obstruction. CT angiography, MR angiography, conventional angiography and upper GI endoscopy are other imaging modalities which aid in the diagnosis. Management is usually conservative which includes nasogastric tube insertion, parenteral nutrition, correcting dehydration and electrolyte imbalance and mobilization of the patient in left lateral decubitus position. If the patient remains symptomatic after 2-12 days of conservative management, surgical intervention should be considered. Surgical modalities of superior mesenteric artery syndrome include, Strong’s operation i.e. division of Trietz ligament, subtotal gastrectomy, gastrojejunostomy, duodenojejunostomy and anterior reposition of the duodenum. Duodenojejunostomy is the most frequently conducted surgical procedure with a success rate of 90%. Laparoscopic procedures are also technically superior and feasible for the patients.

CONCLUSION:
Gastric outlet obstruction can rarely occur as a consequence of Wilkie’s syndrome. High amount of suspicion, timely diagnosis and appropriate management are of prime importance in managing the patients with this syndrome.

REFERENCES: