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CASE STUDY

D1-D2 JUNCTION THICKNING A LANDMARK SUGESTING MEDIAL ARCUATE LIGAMENT SYNDROME: A CASE REPORT

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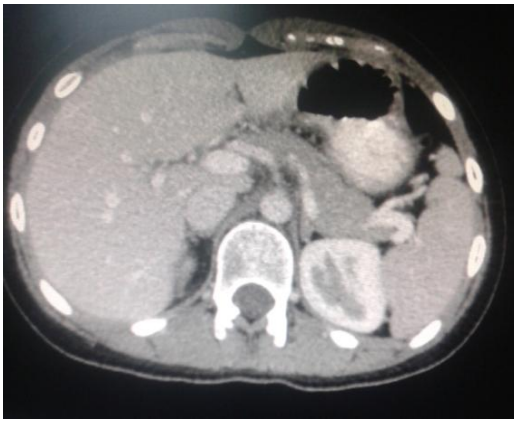
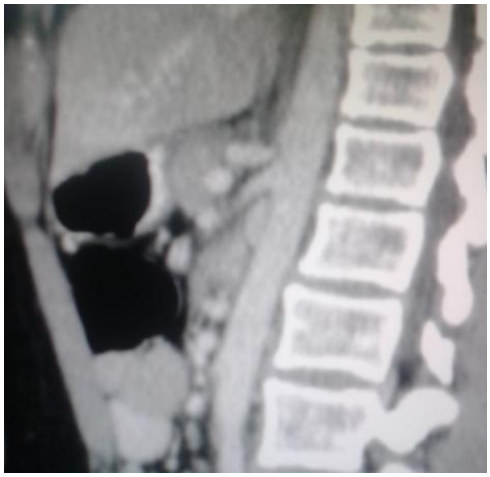
ABSTRACT

The median arcuate ligament syndrome (MALS, also known as celiac artery compression syndrome, celiac axis syndrome, celiac trunk compression syndrome or Dunbar syndrome) is a condition characterized by abdominal pain attributed to compression of the celiac artery and possibly the celiac ganglia by the median arcuate ligament. Median arcuate ligament usually passes superior to the origin of the celiac artery near the first lumbar vertebra. In the general population, 10-24% of people may have indentation caused by an abnormally low ligament. Duffy *et al.* (2009) We present the case of a patient with median arcuate ligament syndrome that caused abdominal pain associated with nausea and emesis.

INTRODUCTION

An 18 yr old female was referred to CMC for pain in abdomen. Pain was aggravated after meals. Even the smallest meal like half a sandwitch would aggravate the pain. Pain was mainly in epigastrium sharp and non radiating in nature. There were episodes of vomiting sometimes hametemesis which were small in quantity and subsided spontaneously. There was history of weight loss however psychiatric evaluation for anorexia nervosa was normal. She denied any alcohol, any illicit drug, or any tobacco product use. Physical examination revealed a temperature and vitals were normal. The patient appeared chronically ill thin but alert. Abdomen had generalised tenderness. There were no masses or organomegaly. There was no audible bruit and bowel sounds were present. The fasting blood sugar was 81 PT was normal and Leucocyte count was 5000 with a normal differential count. Her serum albumin levels were 4 mg/dl with a normal albumin globulin ratio. Her amylase lipase levels were 24 and 18 respectively. Urine microscopy, Stool microscopy all were within normal limits. Gastroduodenal, gall bladder, colon and small bowel roentgenograms revealed no abnormalities. Ultrasound of abdomen with colour flow Doppler sonographic evaluation was normal. Esophagoduodenoscopy was done which showed circumferential thickening around D1-D2 junction ad D2-and D3 were hugely dilated. Colonoscopy showed granularity of the terminal ileum. Biopsies of both endoscopies did not show any significant abnormalities





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A CT scan was done which showed decreased calibre of celiac artery (0.8mm), however splenic, left gastric and common hepatic artery were normal in calibre. SMA was normal in its course.

A diagnosis of Coeliac artery compression syndrome was made.She was taken up for surgery for the same. Thick muscular and ligamentous elements crossing across the aorta and compressing celiac axis, right up to pancreatic head were noted. All structures were divided and celiac axis clearly visualised. A bruit was palpable on celiac axis. Ligamentous bands were divided. Bruit disappeared after division of bands. All surrounding tissues including sympathetic ganglia were cleared. Satisfactory dissection of aorta (anteriorly) and proper exposure of celiac axis done.

DISCUSSION

Compression of the celiac axis by extraluminal structures was first described in 1917 by the anatomist Lipshutz but the association with a clinical syndrome was initially described by Harjola. Duffy *et al.* (2009) in 1963, and by Dunbar *et al.* in 1965 and it is a well documented anatomic variant, reportedly seen in 12.5%-49.7% of patients (Dunbar *et al.,* 1965). Median arcuate ligament syndrome (also known as Dunbar syndrome or celiac artery compression syndrome) was first described by Harjola in 1963. Duffy *et al.* (2009) A patient who presented with postprandial abdominal pain and an epigastric bruit was found to have his celiac artery encased with thick ganglionic tissue at the time of surgery. The patient experienced full relief of symptoms following removal of this thick fibrotic tissue from the celiac artery.

The pathophysiology of the disease is external compression of the celiac artery by an abnormally low lying ligament. The compression worsens with expiration as the diaphragm moves caudally during expiration, causing compression of the celiac trunk. This compression leads to visceral ischemia and postprandial abdominal pain. Some also claim that this causes a steal phenomenon from blood flow being diverted away from the superior mesenteric artery via collaterals to the celiac axis, causing midgut ischemia. A-Cienfuegos *et al.* (2010) Overstimulation of the celiac ganglion is also believed to cause chronic pain in these patients. Sustained compression of the celiac axis may lead to changes in vascular layers such as intimal hyperplasia, proliferation of elastic fibers in the media, and disorganization of the adventitia.

Patients are usually young thin women between the ages of 30 and 50 and typically have had extensive workups for other sources of abdominal pain. A-Cienfuegos *et al.* (2010) However this patient was 18 years old .Pain was located in the epigastric area and worsens after meals, which was typical of MALS However association with exercise, or with leaning forward was noticed. The pain is also associated with nausea, emesis, bloating, and diarrhea. Patients may also experience sitophobia, or food fear, because of these symptoms. Patients may get transient relief of these symptoms by bringing their knees to their chest. This position decreases impingement of the arcuate ligament on the celiac artery by pushing it cephalad relative to the artery as expiration does. Epigastric pain may be present, and physical examination may reveal epigastric bruit in as many as 83% of patients. A-Cienfuegos *et al.* (2010) This bruit may increase on expiration. There was no bruit on auscultation present in our patient

Angiography has been the gold standard in the diagnosis of this disease in the past. A CT scan will be able to detect focal narrowing of the celiac axis, particularly in sagittal views. This narrowing has a characteristic hooked appearance similar to that seen in our patient's CT. Gastric tonometry has also been used to aid in the diagnosis. Faries et al saw a normalization on gastric pH (measured via tonometry catheter) upon release of the median arcuate ligament. In that study, a gastric pH of less than 7.32 indicated significant ischemia. A-Cienfuegos *et al.* (2010) Mensink *et al.* also used tonometry measurements that correlated well with relief of symptoms. Mensink *et al.* (2006) In that study, 83% of patients with abnormal gastric pH measurements obtained relief after operative release of the median arcuate ligament. Our patient was not subjected to gastric pH measurements because of the characteristic CT findings.

Surgical median arcuate ligament release has been the mainstay of treatment. Reilly *et al.* (1985) published the largest follow up study of open surgery in MALS patients in 1984. A total of 51 patients underwent surgery for median arcuate ligament syndrome: 16 patients underwent decompression only, 17 patients underwent decompression and dilatation, and 18 patients underwent decompression and reconstruction. At 10-year follow-up, 53% of patients who had decompression only had resolution of symptoms compared to 76% of patients with decompression and revascularization. Reilly *et al.* (1985) Patient characteristics that predicted relief of symptoms after surgery were postprandial pain, age 40-60 years, female gender, and weight loss greater than 20 pounds. Current results using laparoscopic techniques show relief in nearly 80% of patients undergoing this surgery. Typically, pain relief is immediate, but because postoperative pain can mimic preoperative symptoms and may take up to 6 weeks to resolve, it may take that long to determine if the procedure was successful. Persistent symptoms have been successfully treated with angioplasty. At follow-up in CTVS 2 weeks after surgery, the patient's postprandial abdominal pain had significantly improved. She tolerated a diet without difficulty and had no complaints of nausea, emesis, or bloating.

Conclusion

The MALS or CACS is a rare cause of postprandial pain and weight loss. Such patient are often young have asthenic built. Whenever a patient complains of post prandial pain which subsides on sitting in knee chest position. A possibility of MALS should be considered if routine investigations of causes of abdominal pain are negative. CT angiography should be done to confirm the diagnosis. Open or laproscopic surgical decompression is the modality for management.

Abbreviations

MALS: Median Arcuate Ligament Syndrome

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

Dr K C Das, Dr sumeet and Dr Manish Lalwani were involved in the clinical assessment and writing the case report. All authors read and approved the final manuscript.

Consent

Full written consent was received for the manuscript to be published.

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