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Aorto-Mesenteric Clamp Syndrome: A Case Report

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Abstract: Case Report

The aorto-mesenteric clamp syndrome “AMCS” or Wilkie's syndrome, is a duodenal obstruction secondary to an anatomically acquired clamp which results from the compression of the third duodenum by the superior mesenteric artery “SMA” in front, and the aorta behind following the disappearance of the perivascular fatty tissue. Advanced states of malnutrition are often the cause, and AMCS is clinically manifested by vomiting, fullness and post-prandial pain. Treatment is medical, but if this fails, surgery is required. We report a case of ACMS in a 21-year-old female patient who underwent gastrojejunal anastomosis with good postoperative outcome.

Keywords: Aorto-mesenteric clamp syndrome, Wilkie syndrome, duodenal obstruction.

INTRODUCTION

The syndrome of the aorto-mesenteric clamp (AMCS) or Wilkie's syndrome is defined by extrinsic compression of the third portion of the duodenum by the superior mesenteric artery and the aorta between which it passes [1]. The symptomatology resulting from this duodenal obstruction is that of a high occlusion [2]. Treatment is initially medical, but if this fails, surgery is required [2].

PATIENT AND OBSERVATION

A 21-year-old female patient with no previous history of epigastralgia was admitted to hospital with a one-year history of epigastralgia complicated by food vomiting, which occurred mainly in the morning on rising. The patient was in fairly good general condition, with no signs of dehydration. Examination of the abdomen revealed chattering on an empty stomach. Fibroscopy showed a bulbar ulcer with duodenal stenosis at D3. Gastric biopsies showed moderate chronic atrophic gastritis with the presence of Helicobacter pylori, for which eradication treatment was initiated.

A CT scan was used to diagnose ACMS. Surgery was decided upon, with obvious compression of D3 by the aorto-mesenteric clamp. The procedure consisted of a gastrojejunal anastomosis. The immediate post-operative course was straightforward and the long-term outcome was satisfactory.

DISCUSSION

Aorto-mesenteric clamp syndrome was first described in 1842 by Carl Von Rokitansky. In 1927 Wilkie published the first series of 75 patients, and the syndrome has since been named after him [1]. This syndrome results from compression of the duodenum by the SMA. In the normal state, the duodenum is protected by perivascular

fatty tissue, and it is during rapid weight loss (often caused by decompensation of a pre-existing defect) that AMCS occurs [2-4]. In our patient, the assessment of rapid weight loss was subjective, and the patient had a critically low BMI.

ACMS manifests itself, as in duodenal obstruction, by early postprandial vomiting, abdominal pain and satiety; in other cases, the symptomatology is more chronic with repeated postprandial fullness and intermittent vomiting [1, 2-4].

Advances in imaging technology mean that the diagnosis can be made pre-operatively. Standard radiography confirms high obstruction, and CT calculates the angle between the SMA and the aorta, which is reduced from 7° to 22°, compared with the normal range of 25° to 60°. The aorto-mesenteric distance is also reduced and measures 2-8 mm, whereas the normal distance is 10-28 mm [1-6]. In our patient, the angle between the SMA and the aorta calculated on the CT images was 18°. Duodenal obstruction leads to acute dehydration and worsens undernutrition, maintaining an aggravating vicious circle that treatment aims to break [4].

AMCS is initially treated medically, by inserting a nasogastric tube to decompress the stomach and duodenum, placing the patient in the left lateral position, and above all to compensate for hydro-electrolytic disorders and introduce a double high-calorie diet, enteral via a naso-jejunal tube and parenteral [1-3]. The success rate in this case is around 72%, but recurrences are around 30% [7, 8].

Medical treatment is considered to have failed if there is no improvement in symptoms. There is no time limit for failure, although treatment must be maintained for between 2 and 12 days, although a successful treatment lasting 169 days has been reported in a child [1, 7]. In our patient, it was difficult to keep the gastric tube in place because of the state of dementia, and the left postural position was impossible. In our case, a delay of three days was sufficient for the medical treatment to have failed.

Surgical treatment consists of either a gastro-jejunostomy or duodeno-jejunostomy bypass [1, 3], which can be performed laparoscopically [1], or modify the anatomical conditions by mobilising and decreasing the duodeno-jejunal angle by positioning the jejunum to the right of the SMA after sectioning Treitz's muscle using Strong's procedure. The best results are obtained with duodeno-jejuno-anastomosis [1, 3]. Gastro-jejuno-anastomosis is effective on gastric distension, but less so on the duodenum, resulting in an end to vomiting but persistence of epigastric fullness [9, 10], It is not feasible in all patients due to adhesions and duodenal distension, with recourse to gastro-jejuno-anastomosis or duodeno-jejuno-anastomosis in the event of failure [1-3].

CONCLUSION

Aorto-mesenteric clamp syndrome is a rare and poorly understood condition, posing diagnostic and therapeutic difficulties. Injected abdominal CT is a simple and permanent diagnostic tool. The first-line treatment is medical. Surgery should only be considered if medical treatment fails.

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