CASE REPORT

Double Pylorus

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Summary

Double pylorus (DP) or duplication of the pylorus is an uncommon condition that is either congenital or acquired. Acquired double pylorus (DP) results from a peptic ulcer eroding through and creating a fistula between the duodenal bulb and the distal stomach. We report a case of an acquired double pylorus in an adult gentleman that resulted from the erosion of a duodenal and prepyloric ulcer.

Key Words: Double pylorus, Peptic ulcer disease

Introduction

Double channel pylorus or more frequently termed as double pylorus (DP) is an uncommon condition consisting of two communicating channels between the gastric antrum and the first part of the duodenum. Endoscopically, two separate openings of the pylorus can be seen leading to the duodenum. DP maybe congenital or acquired. True congenital form is rarely reported while on the other hand there have been more than 100 cases of the acquired form reported in the literature so far[1]. A few theories have been proposed to explain the acquired form. The most popular was quoted to have been that of Rokitansky who proposed that the acquired type is a form of gastroduodenal fistula resulting from the presence of two ulcers, one a distal gastric ulcer and the other a duodenal ulcer, eroding through towards each other and creating a communication between the two areas[1].

We report a case of a Chinese gentleman who was confirmed endoscopically to have both prepyloric and duodenal ulcers which subsequently eroded towards each other forming a double pylorus.

Case Report

A 59 year old Chinese construction worker with a history of upper abdominal dyspepsia for the past 4 years was admitted in December 2001 for symptomatic anaemia secondary to upper gastrointestinal bleeding. He was a heavy smoker and frequently took analgesics for arthralgia. An upper gastrointestinal endoscopy revealed a large prepyloric ulcer with another large ulcer situated in the first part of the duodenum. There was no stigmata of recent bleeding. Blood transfusion was given to correct his haemoglobin of 7.9 g/dl. Histopathological examination of biopsies taken at that time showed diffuse lymphoplasmacytic cells with neutrophilic infiltration seen within the

This article was accepted: 10 January 2003

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lamina propria and epithelium. Helicobacter pylori was seen. The findings were consistent with chronic active gastritis with moderate H. pylori activity. The patient was treated with one week's course of H. pylori eradication therapy followed by 6 weeks of ranitidine 150 mg BD. He remained asymptomatic thereafter and a repeat upper gastrointestinal endoscopy was performed 4 months later. This revealed a double pyloric opening into the duodenum. Both openings extruded bilious fluid and it was possible to pass the endoscope into the duodenum through both openings (Figure 1). The pyloric antrum and duodenum was mildly oedematous. No ulcers were seen and both channels were lined by normal epithelium. Since he was asymptomatic it was decided that no treatment was necessary and he was scheduled for a repeat endoscopy in about 6 month's time.

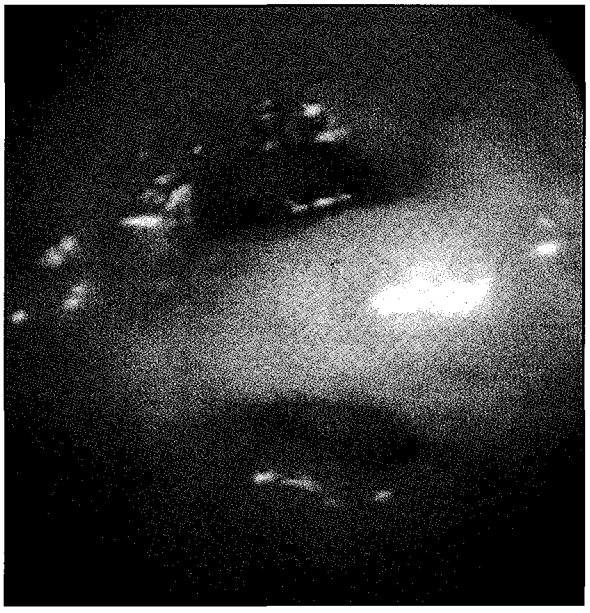

Fig. 1: Endoscopic picture of the double pylorus

Discussion

The presence of two ulcers in our patient supported the theory that the double pylorus occurred as a result of both ulcers being in close proximity and eroding towards each other to form a fistulous tract. Most of the fistulas in DP occur on the lesser curve aspect of the gastric antrum or duodenal bulb where the route of penetration is the shortest. It is now known that DP can also result from a single ulcer. The presence of this single peptic ulcer can cause an inflammatory adhesion, most commonly between the lesser curve of the distal stomach with the duodenum. Subsequently the ulcer erodes through both layers of muscle and creates a fistulous tract [2]. DP can also be due to a gastric cancer ulcerating and eroding through, thus creating a DP as reported by Matsumaya et al [3].

It is interesting to note that this patient was asymptomatic whilst on H. pylori eradication therapy and it was probably during this period and thereafter that the DP was developing. Hu et al. [1] noted the same observation and postulated that the lack of or relief of symptoms in patients with DP was most likely due to the fistulous opening functioning as a gastroduodenostomy that bypasses the natural pyloroduodenal opening that was oedematous. At the other end of the spectrum, some patients are troubled by persistent symptoms and recurrent ulcers. This is most likely due to the regurgitation of bile through the sphincterless fistula. This could explain the fact that the majority of patients had at least one symptomatic recurrence of peptic ulceration that responded poorly to repeated 3 month courses of antisecretory agents[1]. Disease states commonly associated with poor healing such as diabetes mellitus, chronic renal failure and chronic obstructive airway disease, together with continued use of ulcerogenic medication such as NSAIDS as well as poor compliance to antisecretory agents are thought to play a significant role in the poor healing of ulcers in patients with DP. In this patient, the ulcers had healed completely by the time the DP was diagnosed at endoscopy.

Diagnosis of DP by endoscopy is quite straightforward, however, the treatment strategy may not be so. The sequelae of a DP may be that

of complete closure of the fistulous tract, eroding through of the tissue bridge to create a single large and incompetent pyloroduodenal opening or persistence of the DP. Hu et al.[1] noted that majority of the cases of DP treated with eradication therapy for H. pylori remained open. Hence, it is important that repeat endoscopy is arranged for our patient to document the final outcome and consequences of the DP particularly if the DP remains as such indefinitely as it increases the risk of ulcer recurrence. Surgery would probably be the final option for patients with DP complicated by recurrent or non healing ulcers despite intensive anti-ulcer therapy.

References

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