Chronic giardiasis of the stomach

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Abstract

Two cases of chronic giardiasis of the stomach diagnosed from gastric mucosal biopsy specimens are reported. The first case was associated with an acute-on-chronic gastritis and Helicobacter-like organisms, and the second with an adenocarcinoma of the stomach. In both cases the trophozoites had been missed in earlier biopsy specimens. As far as is known this is the first report of giardiasis of the stomach.

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Case reports

Both patients were participating in a screening programme to detect early gastric carcinoma by endoscopic surveillance. Neither patient had any known underlying immune deficiency.

CASE 1

A 70 year old man presented with a 12 month history of epigastric pain, anorexia, and nausea. Endoscopic examination showed an inflamed, friable, mucosa, especially in the antrum, and pronounced bile reflux. His initial symptoms were relieved by sucralfate, and at the time of writing was asymptomatic and receiving no medication.

CASE 2

A 74 year old man presented with a history of many years of indigestion with mild epigastric pain. Endoscopic examination initially showed only antral gastritis, but at the most recent endoscopy a malignant-looking ulcer was identified in the distal antrum. Gastric adenocarcinoma was confirmed by gastric biopsy, and subsequent liver biopsy of a space occupying lesion detected by ultrasound examination showed adenocarcinoma. In view of the metastatic disease, his age, and frailty, operative intervention was considered inappropriate.

Methods

Gastric biopsy specimens were fixed in buffered formalin, routinely processed to paraffin wax, and stained with haematoxylin and eosin. Sections were also stained by a modified Giemsa technique¹ to aid identification of Helicobacter-like organisms. Histological sections were examined independently by two pathologists (PDJ and CQ) for evidence of

trophozoites, bacteria, degree and type of inflammation, dysplasia, intestinal metaplasia and "bile reflux" changes.² The numbers of Helicobacter-like organisms on modified Giemsa stained sections were assessed as follows for each biopsy fragment: + = scanty, difficult to find, ++ = intermediate, +++ = numerous, continuous along surface of epithelium. The absolute number of giardia trophozoites was counted for each biopsy fragment. Polymorph numbers were assessed as follows: +/- = scanty in lamina propria, + = few in lamina propria, ++ = moderate numbers in lamina propria and involving gland walls and surface epithelium, +++ = larger numbers with gland abscesses. Intestinal metaplasia and lymphoid follicles were classified as being either present (+) or absent (-).

Results

For case 1 five sets of gastric biopsy specimens, each containing antral or body type gastric mucosa, were taken over 32 months; in case 2 two sets were taken over 14 month periods (table). Giardia trophozoites were easily identified in the haematoxylin and eosin stained sections from each set of biopsy specimens. The trophozoites were seen in detached surface mucus, within gastric pits, and adjacent to both metaplastic intestinal and gastric antral-type surface epithelium, suggesting adherence to these cells (figure). No trophozoites were seen in gastric body-type mucosa.

Helicobacter-like organisms were present in varying numbers in all the antral and fundal biopsy specimens from case 1 but absent from those from case 2. There was no particular relation between the distribution of Helicobacter-like organisms and the giardia in case 1, but they were observed together in places. There was no association between the site of either organism and the presence of either acute or chronic inflammatory cells, or the numbers of organisms and the severity of inflammation. Lymphoid follicles were present in the lamina propria of some biopsy specimens in case 1 but these showed no particular relation to the presence or absence of bacteria or trophozoites. Changes attributable to bile reflux were observed in both cases and were florid in the most recent biopsies. Biopsy fragments from case 2 showed foci of adenocarcinoma in situ in the most recent biopsy specimens; trophozoites were not observed in these areas.

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Histological details of gastric mucosal biopsy specimens

Date	Mucosal type	Intestinal metaplasia	Bacteria	No of giardia	Polymorphs	Lymphoid follicles
Case 1:						
March 88	Antral	+	+	19	+++	-
	Antral	+	+	131	+/-	-
	Antral	+	+	47	+++	+
Oct 88	Antral	-	+	1	++	-
	Antral	+	+	29	++	-
	Antral	+	+++	-	+++	-
	Body	-	+++	-	+	+
	Antral	+	++	-	+++	+
	Antral	+	++	49	+	-
	Antral	+	+	-	+/-	-
April 89	Body	-	+++	-	++	-
	Body	-	+++	-	++	-
	Antral	+	+	-	+/-	-
	Antral	+	++	30	+/-	-
	Antral	+	++	11	++	-
Nov 89	Antral	+	+++	3	+	-
	Antral	+	+	36	+++	-
	Antral	+	+	54	+	-
	Antral	-	+	-	+	-
Nov 90	Antral	-	+	40	++	+
	Antral	+	+	69	+	+
	Antral	+	+	139	+	-
	Antral	+	++	180	+	-
Case 2:						
May 90	Antral	-	-	-	-	-
	Antral	-	-	-	-	-
	Antral	-	-	21	+	-
	Antral	+	-	41	+	-
July 91	Antral	-	-	46	+	-
	Antral	-	-	5	++	-
	Antral	-	-	-	+	-
	Antral	-	-	-	-	-
	Antral	-	-	-	-	-
	Antral	+	-	> 200	-	-
	Antral	+	-	90	-	-

Discussion

In man giardia trophozoites seem to be found principally in the duodenum and jejunum,^{3 4 5} although they may occasionally colonise the biliary tree and gall bladder.⁶ No attempts have been made to isolate them from the remainder of the gastrointestinal tract in man, although in mice they have not been identified in the stomach or caecum.⁷

The pH of the gastrointestinal tract is clearly important in the life cycle of giardia. Excystation occurs in the acidic conditions of the stomach and is maximal at pH 1.3–2.7⁸ and the “optimal” pH for the trophozoites is often quoted as 6.38–7.02.⁹ There have not been any studies, however, investigating the tolerance of trophozoites to acidic environments. Evidence of bile reflux was present in both of these cases

Giardia trophozoites (arrows) seen adjacent to gastric surface epithelium.

histologically and also endoscopically in case 1, which would lead to an increase in gastric pH. This, together with the position of the trophozoites within the protective surface mucus of the stomach, may enable the trophozoites to survive in the gastric environment.

It is known that bile is a primary growth stimulant for giardia⁵ and that bile salts are important in facilitating the uptake of lecithin from bile which is essential for the synthesis of membrane phospholipids.^{3,5,10} The usual location of the trophozoites in the small intestine ensures a plentiful supply of bile.

Histological changes in the duodenal mucosa in giardiasis include acute and chronic inflammation, epithelial damage, and lymphoid hyperplasia.^{3,4} The gastric biopsy specimens from case 1 revealed an active-chronic gastritis, changes due to bile reflux and the presence of both *Helicobacter*-like organisms and trophozoites. As the patient has remained asymptomatic, he has not been treated for either organism. It is therefore impossible to relate the mucosal abnormalities to giardia infection because of the association of *Helicobacter*-like organisms with gastric inflammation¹¹ and lymphoid hyperplasia.¹² In case 2, however, no *Helicobacter*-like organisms were present and inflammation was only slight and not related to the site of the trophozoites.

We cannot be certain whether the trophozoites were entering passively or were actually colonising the stomach. Unfortunately no biopsy material was available in either case from the duodenum to assess intestinal colonisation. If the organisms were colonising the stomach, the process of gastroduodenal reflux

may provide the necessary pH and bile constituents for trophozoite survival within it.

This is, to our knowledge, the first reported case of chronic gastric giardiasis. It is important to note that the giardia were missed on all previous sets of biopsy specimens from both patients, which were reported by a number of different pathologists. The clinical importance of finding giardia in the stomach is unknown in terms of the symptoms and possible pathological effects it may have at this site.

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