LETTERS TO THE EDITOR

Campylobacter-like organisms in heterotopic gastric mucosa of the upper oesophagus

Campylobacter-like organisms (CLO) are strongly associated in the stomach with active type B chronic gastritis, and several different studies have suggested a role for CLO in the natural history of the disease. CLO are closely apposed to gastric mucin secreting cells in the stomach, and they can also be found in areas of gastric metaplasia in the duodenum where they are supposed to be a key factor in the tendency of duodenal ulcers to relapse.[1] CLO can also be observed in Barrett's oesophagus, but their incidence must probably be low, with a minor role in the natural history of the disease.[2] We wondered, therefore, whether CLO could be detected in heterotopic gastric mucosa of the upper oesophagus in adults, and what relation, if any, CLO might show to active inflammation in heterotopic gastric mucosa. Heterotopic gastric mucosa is a benign and usually asymptomatic condition, the congenital nature of which has recently been questioned.[3]

We reviewed 56 cases of heterotopic gastric mucosa from our files; the histological and histochemical aspects of 24 of these cases have already been published.[3] The heterotopic gastric mucosa was classified as antral or body type, and signs of inflammation were scored on sections stained with haematoxylin and eosin according to the criteria of Marshall[4]: grades 0 to 1, normal histological features; grade 2, increase in mononuclear cells; grade 3, increase of mononuclear and polymorphonuclear cells with intraepithelial invasion of polymorphonuclear cells. Modified Giemsa stained slides were used for the detection of CLO.

Six heterotopic gastric mucosa consisted of antral type mucosa, the remaining 50 had body-type glands; 23 showed signs of inflammation—18 cases with grade 2 and three cases with grade 3. CLO were observed in three out of the 56 (5·3%) heterotopic gastric mucosa, all three with body-type mucosa, the latter being normal in one case and of grade 3 inflammation in two cases. In addition to heterotopic gastric mucosa, gastric biopsy specimens obtained in 18 of the 56 patients during the same endoscopy, were also examined for inflammation and CLO. Two of the three patients with CLO in their heterotopic gastric mucosa had concomitant gastric biopsy specimens which also showed active chronic gastritis with CLO. Among 16 patients with CLO negative heterotopic gastric mucosa, 13 had normal concomitant gastric biopsy specimens without CLO, and three had concomitant CLO associated active chronic gastritis.

Our results show that CLO can be rarely observed in heterotopic gastric mucosa. As *Helicobacter pylori* has never been cultured from the oropharynx,[5] it is likely to be transmitted from the stomach up into the cervical oesophagus by gastro-oesophageal reflux of contaminated gastric juice. Our results in patients with heterotopic gastric mucosa from whom gastric biopsy specimens were taken would support this hypothesis. Some of our data also suggest that CLO could be responsible for the inflammatory changes which are rarely observed in heterotopic gastric mucosa. CLO could therefore have a role in the rare complications of heterotopic gastric mucosa such as inflammation, ulceration, or stenosis, in the same way as it has been shown in duodenal ulcer,[1] and suggested in Barrett's oesophagus.[2]

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Superior stain for Helicobacter pylori using toluidine O

In any histology laboratory that receives gastric biopsy specimens, staining for *Helicobacter pylori* should be part of the routine procedure. In our laboratory we are now using a toluidine blue stain (GURR) and consider this to be very effective, simple, and fast with excellent histological contrast.

As *H pylori* infection is described as producing a superficial active chronic gastritis, toluidine blue is a most satisfactory stain for the demonstration of neutrophilic infiltration between mucosa producing cells and in the lamina propria. The nuclear lobes of the neutrophils stand out against the clear unstained space of the neutrophil cytoplasm, and with proper dehydration, the mucus producing cells stain a very light, clean blue, allowing for easy examination and identification of *H pylori*.[1-4]

A maximum of 10–15 minutes in buffered toluidine blue is all that is required for very positive results. We found that the best buffer for toluidine blue was a veronal acetate buffer at pH 4·5.[5] This buffer contains a barbiturate and because of the complications involved with acquiring such we changed to Sorensen's phosphate buffer at pH 6·8[5] and found that it also works quite effectively. The unique quality of the toluidine blue O stain is its ability to give good staining results at varying pH ranges and with different buffer solutions.

Staining method

TOLUIDINE BLUE USING VERONAL ACETATE BUFFER

Veronal acetate solution:
Sodium acetate 1·943 g
Sodium barbiturate 2·943 g
Distilled water to 100 ml

For pH 4·5, make up in the proportions of 10 ml of stock veronal acetate solution, 22 ml of M/10 hydrochloric acid, and 18 ml of distilled water. To this add 1 ml of 1% toluidine blue made up in distilled water.

1 Sections are cut at 6 μm.
2 Bring sections to water.
3 Stain in toluidine blue buffered solution for 10–15 minutes.
4 Wash well in water.
5 Dehydrate, clear, and mount.

TOLUIDINE BLUE USING SORENSEN'S PHOSPHATE BUFFER

Buffer solution:
M/15 sodium phosphate dibasic; Na2HPO4. Dissolve 9·465 g in distilled water and make up to one litre.
M/15 potassium acid phosphate; KH2PO4. Dissolve 9·08 g in distilled water and make up to one litre.

For pH 6·8, add 25 ml of each solution together and to this add 1 ml of 1% stock toluidine blue solution. The staining technique for this method is the same as above.

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Thrombotic thrombocytopenic purpura (TTP) complicating leptospirosis: a previously undescribed association

Thrombotic thrombocytopenic purpura is a rare condition which can occur without an identifiable precipitant,[1] although it has been described in association with a variety of infective and therapeutic agents.[2] It is characterised by a combination of thrombocytopenic purpura, microangiopathic haemolytic anaemia, renal impairment, a neurological deficit and fever. Unlike disseminated intravascular coagulation, there is a minimal disturbance of coagulation, and histology shows characteristic granular hyaline thrombi within small vessels, an appearance not seen in disseminated intravascular coagulation.[3]