to test for H. pylori. Ideally, a $^{13}$C- or $^{14}$C-urea breath test, which assesses presence of H. pylori in the entire stomach, should be used to determine eradication. Furthermore, the in-house rapid urease test used should have been validated against commercially available urease tests in the post-eradication setting; it has been shown to be as good only when used as an initial diagnostic test when the number of organisms in the antrum is high.

Secondly, eradication of H. pylori infection in patients with nonulcer dyspepsia (NUD) is a controversial issue, with contradictory results in two recent trials. Studies in NUD have been criticised for failing to use validated outcome measures; a critique of 16 therapeutic trials in patients with H. pylori-positive NUD found at least one methodological weakness in each of them. While the authors of the present paper do not address the treatment outcome in these patients, this remains an important aspect of such a study in India. After all, as clinicians we should aim to treat patients and not bugs. The study would have been more meaningful if the authors had incorporated outcome measures such as a dyspepsia symptom score and an objective histology score.

The utility of data on reinfection is more relevant to patients with duodenal ulcer, as they correlate with recurrence rates, bleeding and perforation. The authors of the present study have included only three patients with ulcer disease, and hence their results are subject to revalidation in this group of patients.

Anjan Dhar
Department of Gastroenterology,
The Royal Hospital, Oman

References


Reply from the authors

We agree with Dr Dhar on both counts. There have been recent reports on the migration of H. pylori to the gastric corpus after attempts at eradicating the organism. This phenomenon was not well documented in literature at the time we conducted the study.

However, if this phenomenon did occur in our patients, we would have obtained a false-negative post-therapy result by testing in the antrum and also with the tests we performed there (histology and the rapid urease test, both of which are admittedly not as reliable in this context as a urea breath test). This could have resulted in a falsely high “reinfection” (actually, recurrence) rate. Instead, we obtained a “reinfection” rate of only 2.4% at one year. Dr Dhar’s contentions are therefore valid, but they do not seem to have affected the outcome in our study.

Regarding the issue of therapy for H. pylori infection in patients with nonulcer dyspepsia, we had clearly stated that there are no Indian recommendations for treatment in this group. Our viewpoint is similar but, as we stated in our article, our intention was only to study the outcome vis-à-vis the presence or absence of infection and not its effect on the host. We believe Dr Dhar will agree with us that there is no evidence yet that infection acquisition rates are likely to be different in patients with or without peptic ulcer.

Mukta R Bapati, Philip Abraham, Aniruddha Y Phadke
Department of Gastroenterology,
K E M Hospital, Mumbai 400 012

Blister pack ingestion resulting in esophago-pleural fistula

Most ingested foreign bodies (FB) that perforate the gastrointestinal tract (GIT) are lodged at the site of perforation. We report a patient who inadvertently swallowed a blister pack; this was passed out later per rectum but left behind an esophago-pleural fistula.

A 50-year-old man accidentally swallowed a 2 cm x 2 cm blister pack with sharp edges, made of plastic and aluminum foil (Fig). Following this, he developed severe retrosternal pain and dysphagia which lasted for a day, and was subsequently asymptomatic. Ten days later, he presented with features of right-sided pyopneumothorax. X-rays of the chest and neck showed a loculated hemothorax but no radio-opaque FB. Routine blood tests were normal except for leukocytosis. Pleural paracentesis revealed frank pus with protein 4.3 g/dL, glucose 8 mg/dL, and amylase 800 Somogyi units; culture grew Streptococcus spp and Candida albicans. An intercostal drain was placed and he was started on broad-spectrum intravenous antibiotics and analgesics. However, the drain output was persistently high (>1 L/day); cultures repeatedly grew mixed flora, including anaerobes, Staphylococcus aureus and Pseudomonas aeruginosa; and there was no significant clinical improvement.

Gastrografin study revealed leakage of contrast from the postero-lateral wall of the lower third of the esophagus into
Fig: Gastrografin study showing leakage of contrast from esophagus (long arrow) into right pleural cavity (short arrow) the right pleural cavity. He had dramatic improvement on stopping oral feeds and placement of a nasogastric tube. Serial Gastrografin and methylene blue dye studies showed a decreasing leak, which closed off in 4 weeks' time with development of pseudointestinal. Barium study and esophagography subsequently did not reveal any motility disorder or narrowing in the esophagus. Oral feeds were introduced gradually; he tolerated them well. The chest tube was removed; the rest of his hospital stay was uneventful. Sixteen weeks after discharge, he was doing well. On questioning, he revealed that he had passed the blister pack intact per rectum 5 days after its ingestion.

Ingested FB frequently (28%–68%) lodge in the esophagus, and only 1% result in esophageal perforation. However, with sharp FB, as in our patient, the frequency of perforation is as high as 35%. These FB usually get embedded in local tissues or migrate into nearby structures. Of the five patients with blister pack ingestion reported in English literature, three had intestinal perforation, one had gastric perforation, and one had esophageal impaction. None had esophageal perforation or passed it intact per rectum, as in our case.

We suggest that explicit instructions for use must be included with drugs dispensed in blister packs.

Usha Dutta, Narinder Mohan Gupta,* Biren Negi, Kartar Singh

Departments of Gastroenterology and *Surgery,
Postgraduate Institute of Medical Education and Research, Chandigarh 160012

References

Massive hemorrhage from colonic ulcers in typhoid fever

Intestinal hemorrhage is a well-known complication of typhoid fever that usually occurs during the third week of illness. Massive bleeding can occur from ulcers in the terminal ileum, cecum and rarely ascending colon.

A 26-year-old man presented with massive lower gastrointestinal hemorrhage requiring transfusion of 7 units of packed red cells. He had a 3-week history of fever, headache and muscle pains. He complained of ill-defined right lower quadrant discomfort, generalized weakness, anorexia and a recent 4-kg weight loss. Physical examination revealed a pale, diaphoretic man with pulse rate of 110/min and BP 84/50 mmHg. The abdomen was tender in the right lower quadrant.

Investigations: hemoglobin 6.4 g/dL, WBC 6.2 x 10^9/L. Titers of antibodies to O and H Salmonella antigens were 1:640 and 1:80, respectively. Blood culture was positive for S. typhi. Liver function and coagulation parameters were normal. Upper GI endoscopy was normal. Colonoscopy revealed multiple irregular, punched-out ulcers distributed diffusely in the ascending colon and cecum: they ranged in size from 0.5 cm to 1.5 cm. Microscopy of colon biopsy tissue revealed acute colitis with predominant polymorphic infiltrate and superficial ulcerations; periodic acid-Schiff stain showed no evidence of amebae. Stool cultures were negative for pathogens. Amoebic serology was negative.

The patient was managed with blood transfusions and antibiotics. He left the hospital in good health after 10 days. One month later, he was symptom-free and colonoscopy was normal.

In typhoid fever, the distribution of ulcers in the intestinal tract parallels the anatomic location of Peyer's patches: most hemorrhages from ulcerations therefore arise in the vicinity of the terminal ileum. However, colonic ulcers have also been reported: these occur in the proximal colon but may occur on the left side.

Colonoscopy can localize the bleeding source, and local endoscopic therapy in the form of endoclips or sclerotherapy can be considered before surgery, which has been the conventional treatment in such patients.

Ajit Sood, Vandhana Midha, Neema Sood*
Departments of Medicine and *Pathology, Dayanand Medical College and Hospital, Ludhiana, Punjab

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