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LETTER TO THE EDITORS

J. Stachura

Gastric carcinoma risk index in patients infected with *Helicobacter pylori*

Sirs: I read the paper by Meining A. et al. (1998) in the recent issue of *Virchows Archiv* with interest. It is postulated that computation of this risk index might be a simple method of identifying patients infected with *Helicobacter pylori* and carrying a higher risk for gastric cancer. The authors studied gastric mucosa in patients bearing an early gastric cancer. *H. pylori*-infected duodenal ulcer patients were used for comparison. They listed three features: chronic active corporeal gastritis (neutrophils present) and corporeal and antral intestinal metaplasia. With a score of 3 (all three features present) the predictive value for the presence of gastric carcinoma was 0.94.

I doubt whether this index is of real practical value in surgical patholgy.

1. The first feature considered in the index is chronic active corporeal gastritis (neutrophils present). This limits the index to patients currently infected with *H. pylori*, whereas in severe atrophic gastritis bacteria may be long gone.
2. Patients with atrophic pangastritis and intestinal metaplasia are known to be in the higher risk group for gastric carcinoma anyway, with no need to compute the index.
3. Duodenal ulcer patients are not the best control group for such a study. Most duodenal ulcer patients are hypersecretors. In infected persons without ulceration the maximal acid output (MAO) tends to be normal or diminished. Increased MAO with increased parietal cell mass is typical for duodenal ulcer patients, associated with hyposomatostatinaemia and hypergastrinaemia. ECI cell density in duodenal ulcer patients is three times that in non-ulcer-infected individuals. Duodenal ulcer patients also develop an active non-atrophic antral gastritis, mostly chronic, without corporeal involvement.

Global infection with *H. pylori* is associated with the global incidence of gastritis, ulcer disease and gastric cancer. The incidence of these diseases varies, however. *H. pylori* infection persists for years. Possibly all individuals harbouring the microrganism develop chronic active gastritis but the majority remain asymptomatic, while a minority suffer from gastric or duodenal ulcer and fewer still develop gastric cancer.

H. pylori has been postulated as a group 1 carcinogen, but the mechanism of gastric carcinogenesis remains to be elucidated. It is believed that gastric cancer morphogenesis follows Correa's paradigm of a gastritis–atrophy–metaplasia–dysplasia–cancer sequence. We have investigated noncancerous gastric mucosa in 40 patients operated on for early gastric cancer (Stachura et al. 1981). In all cases of well-differentiated adenocarcinoma, mucosal atrophy and intestinal metaplasia were present in noncancerous gastric mucosa. Atrophy was never entirely diffuse and was separated by spared mucosal areas. In addition, focal glandular hyperplasia was present in 83.3% of cases and hyperplastic surface epithelium was present in 50% of cases. Mucosal atrophy and intestinal metaplasia were seen in 90.5% of poorly differentiated adenocarcinoma. This was accompanied by surface epithelium hyperplasia in 66.7% and glandular hyperplasia in 47.6% of cases. In a diffuse early gastric cancer atrophy was present in 71.4% and intestinal metaplasia in 57.1%, while hyperplasia (either surface epithelium or glandular) was present in 42.8%.

Since the introduction of Genta's "strict definition" of gastric mucosal atrophy (Genta 1997) it has been clear that atrophic gastric mucosa is not just a matter of simple atrophy but can be compared to a cirrhotic organ. Our study documents surface and glandular hyperplasia in the atrophic gastric mucosa leading to mucosal nodularity just like the hyperplasia that accompanies cirrhosis in other organs. Cirrhotic mucosa is assoiated especially with well-differentiated gastric adenocarcinoma.

In summary, it is chronic atrophic pangastritis with intestinal metaplasia and focal hyperplasia that should identify patients carrying a higher risk for gastric carcinoma. By the way, I share the author's admiration of Professor Dr. Kurt Elster.

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