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LETTER TO THE EDITORS

M. Stolte · A. Meining · E. Seifert · H. Koop

Reply

We should like to thank Declich et al. for their comments on our case report [5].

They are, of course, perfectly correct in noting that parietal cell hypertrophy occurs not only in association with PPI treatment [6] but also in other conditions, for example Zollinger-Ellison syndrome. These other conditions, however, can readily be excluded by contacting the gastroenterologist and by additional clinical data and laboratory investigations.

They are not correct in stating that parietal cell hypertrophy has not so far been described in autoimmune gastritis. In an earlier study on "not-yet" atrophic autoimmune gastritis with detection of parietal cell antibodies in the serum, we were able to show that in addition to diffuse periglandular lymphocytic infiltration of the corpus mucosa and focal lymphocytic destruction of corpus glands, parietal cell hypertrophy is a frequently demonstrable diagnostic feature [7]. We suspect that this is a result of blockade of the proton pumps of the parietal cells leading to hyperchlorhydria, hypergastrinaemia and an associated increase in the secretory canaliculi in the parietal cells, but this has not yet been finally confirmed by prospective studies involving measurement of acid secretion capacity and the gastrin concentration in the serum.

In recent years, studies conducted by Kirchner's group [1, 2, 4] have shown that in some patients with *H. pylori* gastritis an antigastric autoimmunity may develop and that the antigen for the antigastric antibodies is, again, the proton pump in the parietal cells [3]. It may thus well be that, as in patients with "classical" autoimmune gastritis, identical histological changes of this gastritis subtype – that is, diffuse lymphocytic infiltration of the corpus mucosa with focal lymphocytic destruction of glands and hypertrophy of the parietal cells – also occur here. In these patients, eradication of the very few *H. pylori*, with subsequent reduction in *H. pylori* antibodies in the serum may result in healing of this form of *H. pylori*-induced autoimmune gastritis. In a retrospective pilot study (as yet unpublished) involving 80 patients with these histological features, we were able to show that such treatment was successful in 80% of cases. However, for final confirmation of this hypothesis and also of the usefulness of the histological features described, a prospective study with additional clinical, serological and immunohistochemical investigations needs to be carried out.

M. Stolte (✉)
Institute of Pathology, Klinikum Bayreuth, D-95445 Bayreuth, Germany

A. Meining
Medical Department II, Technical University Munich, Munich, Germany

E. Seifert
Medical Department, Hospital Kemperhof, Koblenz, Germany

H. Koop
Medical Department, Hospital Buch, Berlin, Germany

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