Dear Sir,

We have read with interest the article by Kuipers [1]. The paper is mainly focused on the development of corpus atrophic gastritis in H. pylori positive patients on long term PPI therapy. As such, it was not devoted to discuss exhaustively on other aspects.

Nonetheless the author find room for a brief comment about the (possible) link between PPI and fundic gland polyps (FGPs). We feel that this point deserve some more comment.

First of all we would like to deal with the histopathologic diagnosis of FGPs. We certainly agree that FGPs are characterized by “distorted glandular cysts lined with fundic-type epithelial cells”, but… it is not enough! They are also characterized by foveolar-type cysts. One should be very strict on this criterium, otherwise the distinction between FGPs and parietal cell hyperplasia (PCH) (or protrusion) is blurred [2, 3]. We published a case of PCH with deep cystic dilations stressing the subtle differences with the FGPs [4].

A second point is the debate about PPI and FGPs. There have been reports favouring [5, 6] and others denying any influence [2, 7, 8]. Kuipers seems to favour a positive influence, but in fact, the cited paper does not support this view [9]. Cats et al, in their reply, agreed with us that their findings had demonstrated a link between omeprazole-gastrin and parietal cell protrusions, and “that at this time one can only speculate about causal link between omeprazole-fundic gland cysts-fundic gland polyps” [10].

Moreover, a year later Klinkenberg-Knoll et al. [11], in a very large prospective study from the same institution, could not find any de-novo case of FGPs developing under omeprazole therapy [12].

Possibly, Vieth and Stolte [13] have definitely demonstrated that H. pylori negative patients, with and without PPI therapy show an almost identical prevalence of FGPs. As FGPs do not develop, or disappear with an infection by H. pylori [4, 15], one should compare patients in therapy with PPI with H. pylori negative controls. Bearing these facts in mind, H. pylori eradication and not omeprazole per se produces “de-novo” FGPs appearance. Recently, even an authoritative text of gastrointestinal pathology seems to have incorporated this view [16].

In conclusion, we can not exclude, as suggested by Burt, “that a small subset of patients is susceptible to forming fundic gland polyps when placed on acid suppression” [17], but we would like not to propagate a sort of “Mozart effect” of gastrointestinal pathology [18].

References

10. Declich P, Bellone S, Porcellati M et al: Parietal cell protrusions with fundic gland cysts and fundic gland polyps: are they related or simply similar but distinguishable? Hum Pathol 2000, 31, 1536-1537 (with authors’ reply).

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