codons with restriction enzymes for cleavage sites produced by the specific genetic alteration [9]. A mutation was found in exon 10, which alters codon 618 from TGC (Cys) to GAC (Ser). Cysteine codon 618 is associated with a milder course and less frequent involvement of the adrenal and parathyroid glands [3, 10].

Our findings raise the question of whether the separation of FMTC as a separate syndrome is warranted. It appears from our one example that the longer a family with presumed FMTC is followed, the greater the chance for the development of a phaeochromocytoma or parathyroid hyperplasia. The syndrome of FMTC appears to need a better definition to include the number of families that need to be examined, the length of the follow-up, the frequency of the follow-up examinations, and the screening methods for phaeochromocytomas and parathyroid hyperplasia. One alternative is to eliminate the designation and substitute in its place the use of FMTC and MEN 2A syndromes by their specific mutations.

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Gastric, Duodenal and Rectal Multifocal Malt Lymphoma: the Possible Co-existence of Two Different Cell Populations

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In the last few years several studies have reported regression of gastro-intestinal MALT lymphomas after anti-*Helicobacter pylori* therapy [1–4]. In this regard, the regression of intestinal MALT lymphomas originating outside the stomach is particularly interesting, suggesting that the association between *H. pylori* and MALT lymphoma might not be limited to the stomach [5–7]. In this letter, we report a case of multifocal MALT lymphoma, in which the co-existence of two different cell populations is possible, and the role of *H. pylori* infection seems to represent an important, but not the sole, pathogenetic event.

A 56-year-old male was found to be affected by concurrent gastric, duodenal and rectal low-grade MALT lymphoma. Despite a high serum titre of anti-*H. pylori* antibodies, routine sections of gastric, duodenal and rectal biopsies did not show the presence of *H. pylori*, and six courses of cyclophosphamide, doxorubicin, vincristine and prednisone (CHOP) were administered. Complete regression in the rectum and partial regression in the stomach and duodenum were achieved. However, the serum titre of anti-*H. pylori* antibodies increased after chemotherapy, and restaging gastric biopsies showed the presence of *H. pylori*, suggesting that the number of histological samples examined at the time of the diagnosis was probably inadequate. Eradication treatment consisting of Omeprazole, Clarithromycin and Tynidazole was administered for 7 days. *H. pylori* was eradicated and complete remission was endoscopically and histologically documented after 2 months. Twenty-eight weeks later, rectal, gastric and duodenal relapse was observed. Multiple biopsies taken from the gastric fundus, body and antrum excluded *H. pylori* recurrence, and the serum titre of anti-*H. pylori* antibodies was lower than that of previous assays. The patient was treated with chlorambucil and, at present, is clinically well.

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The different pattern of response to treatment of the three sites involved, as well as the relapse after *H. pylori* eradication suggests the presence of two cell populations in our patient. It is known that direct antigen stimulation may play an important role in the evolution of low-grade MALT lymphoma, particularly at an early stage. A tentative scheme for the pathogenesis might be that B and T cells are recruited as part of the immune response to antigen stimulation. Genetic changes in B cells, conferring a proliferative advantage, could result in a monoclonal lymphoproliferative lesion, escaping from T cell dependency and resulting in MALT lymphoma [8]. *H. pylori* is considered the antigen most frequently involved, but the cascade of MALT formation–B cell clonality–MALT lymphoma may not be uniquely associated with *H. pylori* infection [9]. Recently, a clonal link between concurrent gastric and intestinal MALT lymphomas has been shown, suggesting that the two lesions might be formed by two different tumour subclones derived from a common precursor clone [10]. In light of these observations, two hypotheses can be formulated as concerns our patient: (1) two different B cell clones may have developed as result of two antigen stimulations, the former being represented by *H. pylori* infection, and the latter by an antigen of unknown origin, not responding to anti-*H. pylori* therapy; or (2) a common precursor clone, triggered by *H. pylori*, infection, may have given rise to two subclones, one of which became disengaged from *H. pylori* stimulation.

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