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Progression of gastric enterochromaffin-like cells growth in Zollinger-Ellison syndrome and atrophic body gastritis patients.

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BACKGROUND: Enterochromaffin-like cell hyperplasia of the gastric body mucosa occurs in hypergastrinaemic conditions such as atrophic body gastritis and Zollinger-Ellison syndrome. However, the time course of change or factors involved are not known. **AIMS:** To compare the rate of change of enterochromaffin-like cell proliferation in patients with atrophic body gastritis and Zollinger-Ellison syndrome. **PATIENTS:** From a consecutive series of atrophic body gastritis and Zollinger-Ellison syndrome patients, studied at the time of first diagnosis, 10 atrophic body gastritis (4 with pernicious anaemia) and 14 Zollinger-Ellison syndrome (4 with multiple endocrine neoplasia type 1) patients were followed-up for a median time of 48 months. **METHODS:** At entry and during follow-up patients underwent: plasma gastrin determination, endoscopic sampling of body mucosa for qualitative assessment of enterochromaffin-like cell hyperplasia pattern and degree of glandular atrophy, qualitative and morphometric analyses of body mucosa endocrine cells. **RESULTS:** At time of diagnosis, enterochromaffin-like cell lesions were more severe in atrophic body gastritis than in Zollinger-Ellison syndrome. During follow-up, no significant variations were observed in gastrin values, enterochromaffin-like cell patterns and grade of body mucosa atrophy in atrophic body gastritis. In contrast, gastrin levels were significantly increased [median 1200 (235-2625) vs 1947 (225-5200) pg/ml; $p < 0.001$] as was total volume density of enterochromaffin-like cells [median 1.60 (0.53-4.06) vs 3.18 (1.35-21.13)% of mucosal epithelial component; ($p < 0.005$)] in Zollinger-Ellison syndrome. Micronodular hyperplasia of enterochromaffin-like cells, present in only one patient at diagnosis, was observed in 8 Zollinger-Ellison syndrome patients at follow-up. **CONCLUSIONS:** These data suggest that the progression of enterochromaffin-like cell growth in human gastric mucosa requires an increase of and/or a prolonged exposure to severe hypergastrinaemia.