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LETTER TO THE EDITOR

Multidinous fundic gland polyps: An yet-underscribed association

Dear Editor,

We read with great interest the paper by Lam and Lau,[1] which recently appeared on your journal. In their paper, the authors report about two patients with a large number (52 and 147, respectively) of fundic gland polyps (FGPs) without any known association with previous therapies or genetic familial syndromes, which they aptly named "idiopathic" (literally "without known cause") multidinous FGPs.

FGPs are small sessile polyps (2–3 mm), usually multiple, of the acid-secreting gastric mucosa. They are characterized by shortened gastric pits and cystic dilations, both superficial and deep, bordered by columnar mucous, chief, and parietal cells. They are almost always negative for *Helicobacter pylori* colonization.[2] Although they show the same histology, they have been described as sporadic,[3] associated with familial adenomatous polyposis (FAP),[4] attenuated FAP,[5] Zollinger–Ellison syndrome,[6,7,8] MUTYH-associated polyposis,[9] gastric adenocarcinoma and proximal polyposis of the stomach (GAAPS),[10,11] and absorptive hypercalciuria[12] (Fig. 1). Although recently Kővári *et al.*[13] described subtle differences between sporadic, FAP-associated, and GAAPS-associated FGPs, so far the only difference between the histology of fundic polyps has been in the frequency of dysplasia, which is rare in sporadic FGPs (1%), frequent in FAP-associated polyps (30–50%), and almost universal in GAAPS-associated polyps.

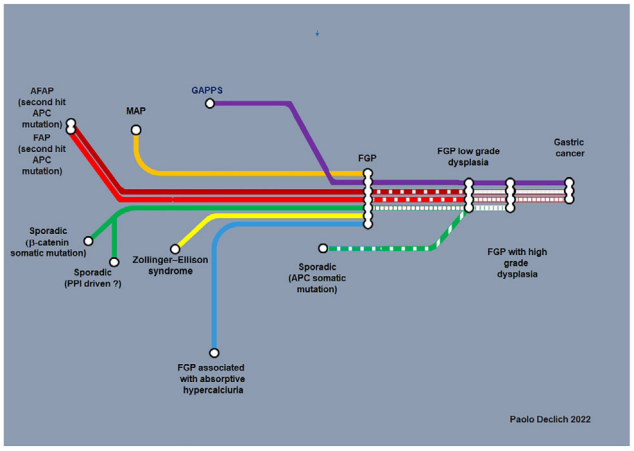

Figure 1 Diagram showing the clinical association of fundic gland polyps with their pathogenesis in brackets, where known. AFAP, attenuated familial adenomatous polyposis; APC, adenomatous polyposis coli; FAP, familial adenomatous polyposis; FGP, fundic gland polyp; GAPPS, gastric adenocarcinoma and proximal polyposis of the stomach; MAP, MUTHY associated polyposis; PPI, Proton pump inhibitors.

Lam and Lau[1] were clearly faced with a management dilemma—how to treat such unusual patients. They never received a long-term treatment with proton pump inhibitors (if such a treatment promotes polyps at all), nor had a positive family history for genetic syndromes with a known risk for FGP development. In absence of clear guidelines for such a situation, the authors decided to endoscopically remove all polyps and to submit them to histologic examination. Quite surprisingly, not a single polyp showed dysplasia.

Given such results, we would suggest to Lam and Lau to submit their polyps to molecular biology examination, in a search for possible new mechanisms driving such an exceptional multidinous polyposis.

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