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Topical Budesonide for Treating Giant Rectal Pseudopolyposis

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Abstract. *Pseudopolyps are a frequent finding in the course of inflammatory bowel disease. They are non-neoplastic lesions resulting from a regenerative and healing process that leaves inflamed colonic mucosa in polypoid configuration. Data about their management is lacking. "Giant" pseudopolyps can be mistaken for adenocarcinomas and, as they rarely regress with medical management alone, a surgical resection is often required. A case of giant pseudopolyposis treated non-surgically, in a patient with concomitant ulcerative colitis and chronic hepatitis B, is reported, representing a co-morbidity complicating an eventual conservative treatment. The clinical implementation of topical budesonide was originally tested, resulting in clinical, endoscopic and histological remission. Budesonide seems a promising therapy for IBD, particularly when a comorbidity with viral hepatitis exist.*

The term pseudopolyp (or inflammatory polyp) is used to describe non-neoplastic polypoid lesions resulting from a regenerative and healing process that leaves inflamed colonic mucosa in polypoid configuration (1). The formation of pseudopolyps in the course of inflammatory bowel disease (IBD) and particularly of ulcerative colitis (UC) is frequent, with a reported incidence in older series varying from 12.5% to 74% (2). They grossly appear as small filiform lesions and very rarely as giant protruding colonic masses mimicking adenocarcinomas. Histologically, they are characterized by minimal alterations related to the underlying inflammation.

Data about the management of inflammatory polyps is unsufficient. Giant pseudopolyps rarely regress with medical management alone and often require surgical resection (3). To our knowledge, this is the first report of giant pseudopolyposis in a patient with concomitant IBD and chronic hepatitis B, representing a co-morbidity complicating an eventual conservative treatment.

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Case Report

A 45-year-old male patient was admitted to our service in July 2002 for acute rectal bleeding. All laboratory tests were normal, except an increase of aminotransferases level (>10 N) and a serological profile consistent with acute hepatitis B or a flare of chronic hepatitis B (HbsAg +, HbsAb –, HbeAg –, HbeAb +, HbcAb-IgM +). The patient underwent lower gastro-intestinal endoscopy and multiple biopsies were taken from a bulky rectal mass lesion (Figure 1). Histological study revealed alterations compatible with inflammatory bowel disease (IBD) with no dysplasia (Figure 2). Because of the atypical macroscopic features and the high suspicion of rectal adenocarcinoma, repeated endoscopic and histological studies were subsequently performed, which established the diagnosis of a giant pseudopolyp with no dysplasia, in a patient with IBD and acute or HBeAg-negative chronic hepatitis B.

The patient was initially given 5-aminosalicylate (5-ASA) both orally (2 g/d) and in enemas (4 g/d) for 6 weeks, resulting in both clinical and endoscopic failure. Budesonide enemas (2.3 g/d) replaced the initial therapy of 5-ASA for 6 more weeks, resulting in control of bleeding with both macroscopical and histological remission (Figure 3). The patient is followed-up annually and receives topical treatment with 5-ASA in suppositories (500 mg tiw).

In January 2003, both the initial serological profile and the abnormal aminotransferases levels persisted, thus imposing a liver biopsy. Chronic hepatitis with moderate fibrosis and moderate necroinflammatory activity was microscopically evidenced (Figure 4). The patient was given lamivudine (100 mg/d) orally and responded biochemically. Twenty months after his hospitalization, the patient maintains the above-mentioned therapy and remains asymptomatic with normal liver function tests.

Discussion

Inflammatory polyps complicating IBD are more commonly found in cases of pancolitis than left-sided colitis and seem related to the chronicity of the disease (4, 5). From this point of view, the case reported herein, with giant pseudopolyps as the primary anatomical expression of limited proctitis, is

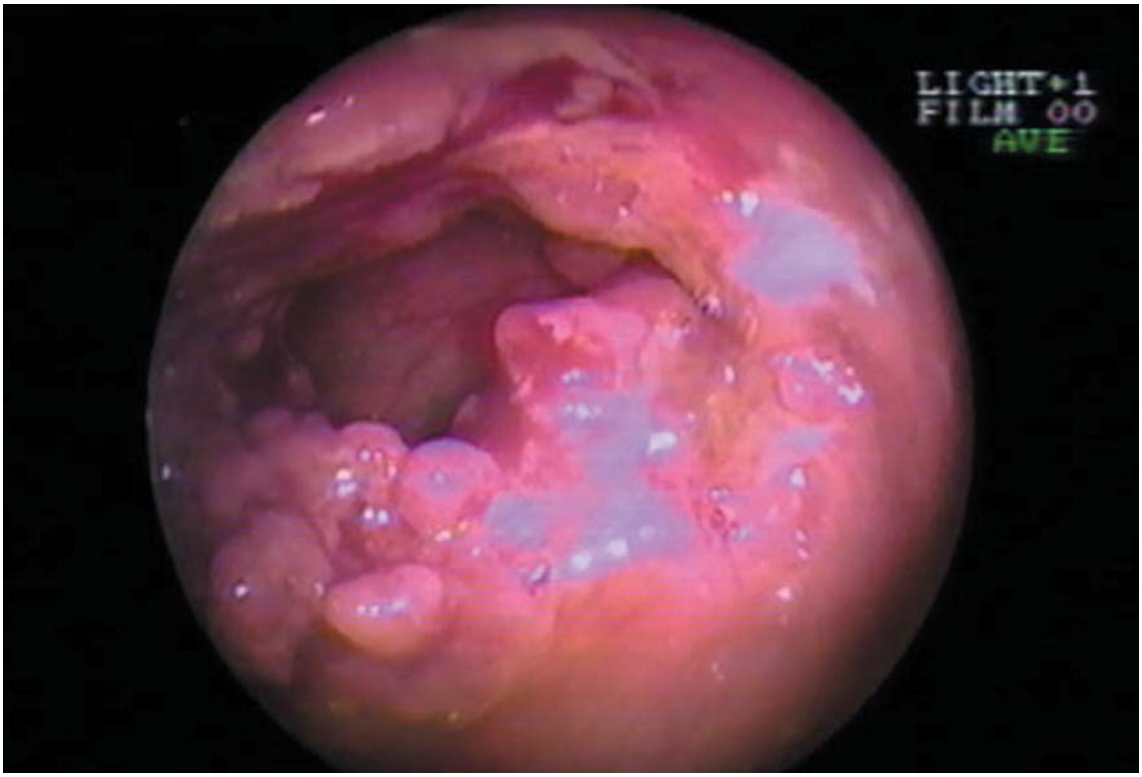

Figure 1. *Endoscopic view of rectum: giant pseudopolyp partially obstructing rectal lumen.*

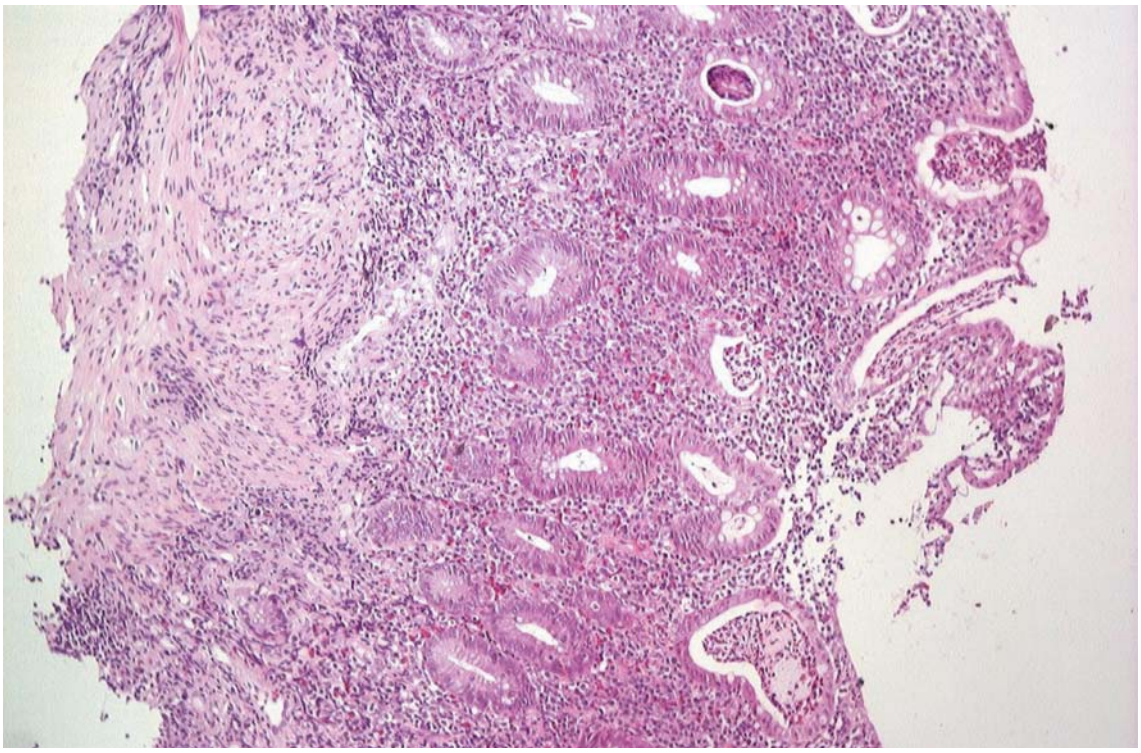

Figure 2. *Histological features of the colonic mucosa before treatment: epithelial ulceration with prominent crypt distortion, cryptic abscesses and lymphocytes infiltration.*

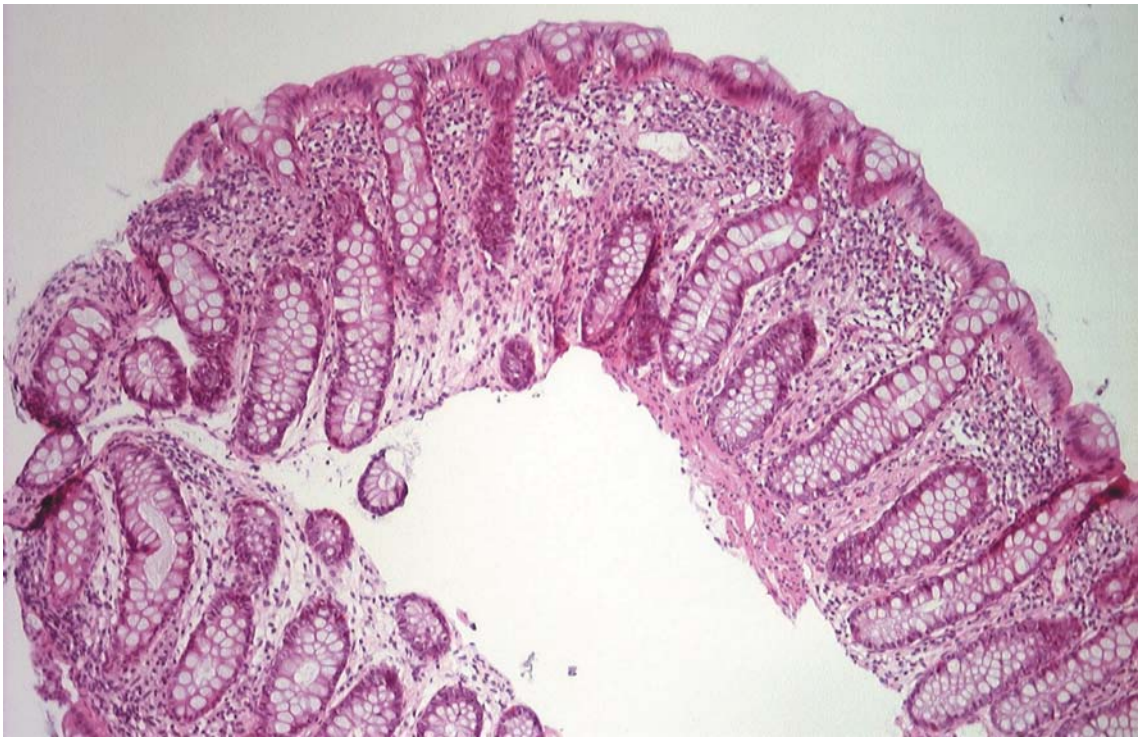

Figure 3. *Histological restitution "ad integrum" (regression of all lesions) of the colonic mucosa after 12 weeks of topical treatment.*

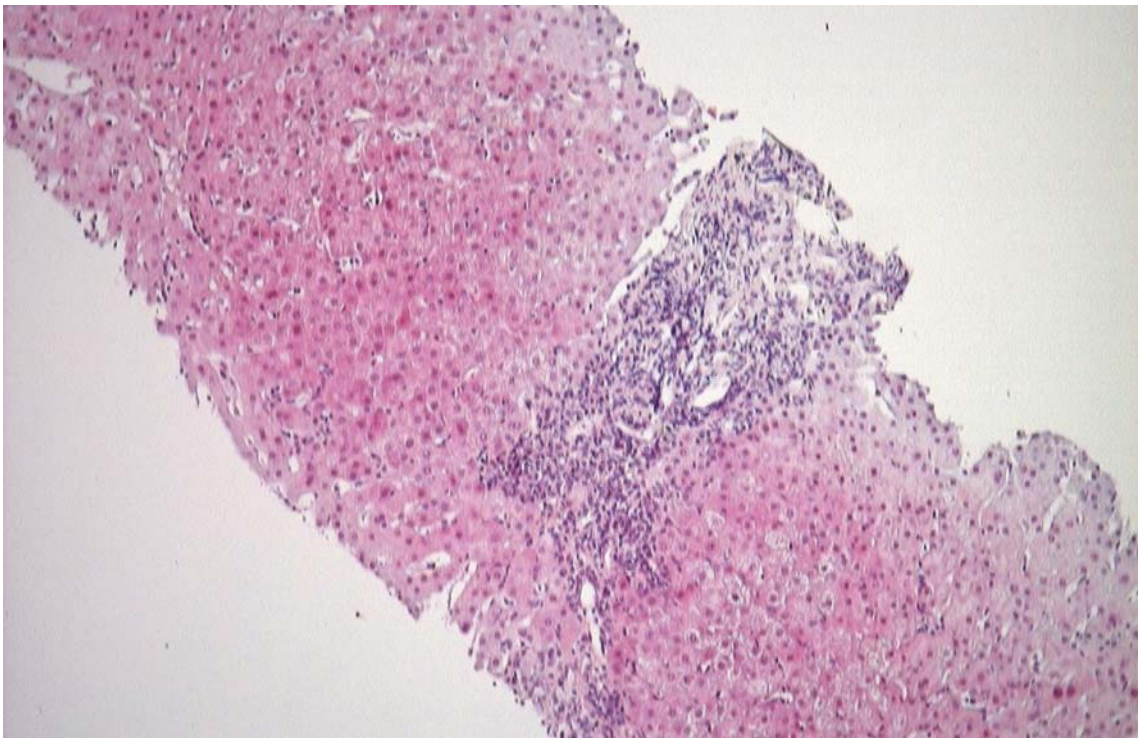

Figure 4. *Liver histology before lamivudine treatment. Active viral replication with moderate fibrosis and moderate necroinflammatory lesions.*

exceptional. Clinically, inflammatory polyps may be quiescent, manifested by symptoms related to the underlying IBD or even complicated by partial or complete colonic obstruction or intussusception requiring emergency surgery (3-7). The above-mentioned factors (extension, chronicity and acute complications) make total coloproctectomy with ileoanal anastomosis the most reasonable therapeutic choice, while some authors have performed local excision of the pseudopolyp, in a bowel-sparing method (6). However, in the reported patient whose IBD was limited to the rectum, in whom pseupoloyposis was the first manifestation of UC and who presented without a life-threatening complication, total coloproctectomy seemed disproportional to the severity of the disease. It was our opinion that a non-surgical treatment would be more appropriate.

Lesions located more distally than splenic flexure are accessible to local treatment. Topical formulations of mesalamine have proven efficacy and may be used as a first-line therapy (8). Non-response to local mesalamine is, however, possible and a second-line therapy of orally- or rectally-administered corticosteroids might be an adequate alternative (9). Because of the co-morbidity of UC and HBV infection, priority was given to rectal formulations. Budesonide, being the only corticosteroid available in enemas in Greece, induced both clinical and endoscopic remission in 6 weeks.

The co-morbidity of UC and chronic hepatitis B was the major problem in the management of our patient. Despite their proven effectiveness in treating distal colonic disease, various molecular forms of topical corticosteroids preserve various degrees of colonic absorption and thus their administration is not devoid of systemic side-effects, such as immunosuppression (10). The use of corticosteroids (even in topical formulations) in cases of viral hepatitis should be with caution, since they may lead to an enhanced viral replication and the widespread infection of hepatocytes (11). This is a concern, particularly in cases of concomitant IBD and hepatitis B, in which a fulminant liver failure is possible at the time of steroids withdrawal. Conversely, interferon (IFN-α) treatment for chronic viral hepatitis could exacerbate the clinical course of an IBD (12). This risk is more than theoretical, as Mavrogiannis reported a case of UC exacerbation following interferon treatment for chronic hepatitis C (13). Nevertheless, despite these concerns, in eight patients with concomitant Crohn's disease and chronic hepatitis C, Biancone *et al.* showed that corticosteroids (as well as other immuno-suppressors) and IFN-α do not counteract and should be both integrated in the same therapeutic strategy (14).

Being devoid of the immune modulating properties of IFN-α, lamivudine was chosen as an initial antiviral therapy. In cases of chronic HBV infection by a pre-core mutant, which is commonly found in Greece, there is indeed growing evidence supporting the efficacy, the safety and the excellent tolerability of lamivudine (15).

However, although topical budesonide and lamivudine are both proven safe and effective as first-line treatment, classic therapies including systemic corticosteroids and IFN-α still have a place in the therapeutic arsenal and might be subsequently implemented, in case of clinical aggravation or viral flare.

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