Cap polyposis occurring in the postoperative course of pelvic surgery

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Abstract
The case is reported of a 42 year old woman who presented with the characteristic clinical, sigmoidoscopic, and histological features of cap polyposis occurring in the postoperative course of pelvic surgery. Pathogenesis of cap polyposis is unknown. In this patient, abnormal colonic motility was not a likely aetiological factor as suggested in previous cases. Despite some arguments favouring an infectious origin or participation, no specific viral or bacterial agent was identified. Cap polyposis remains a poorly recognised condition, which may be confused with inflammatory bowel disease.

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Cap polyposis, which was first described by Williams et al in 1985, has since remained an overlooked entity.1 Campbell et al recently described two new cases, which called attention to this condition.2 Cap polyposis has specific clinical, sigmoidoscopic, and histological features but its pathogenesis is unknown. It has been postulated that abnormal colonic motility leading to mucosal prolapse may be an important aetiological factor. We report a case of cap polyposis occurring in the postoperative course of pelvic surgery, which points in our opinion to a possible infectious origin or participation.

Case report
A 42 year old woman presented in February 1993 with a 14 month history of recurrent mucoid and bloody diarrhoea (more than eight stools per day) with abdominal pain. She was operated on for an uterine myoma in December 1991. The surgical report pointed out technical difficulties because of pelvic adhesions. Ampicillin (2 g/day) was started the day before surgery. Three days after operation, the patient complained of mucus and bloody diarrhoea accompanied with abdominal and rectal pain.

The first sigmoidoscopy performed in January 1992 showed a flushed oedematous mucosa with some abraded areas in the sigmoid colon sparing the rectum. Examination of stools for Clostridium difficile and toxin was negative. Biopsy specimens suggested non-specific colitis.

Treatment with 5 aminosalicylate (2 g/day) improved the patient’s condition (four stools per day still containing mucus). A repeat sigmoidoscopy in March 1992 showed some small sessile inflammatory polyps located 15 to 35 cm from the anal margin. Treatment with 5-aminosalicylate (5-ASA) was continued but the patient’s symptoms worsened in August 1992. Colonoscopy showed about 25 haemorrhagic sessile, ulcerated polyps, spread over 15 to 35 cm from the anal margin with a normal pattern of the rectum and of the left colon (Fig 1). Biopsy specimens again pointed to a non-specific inflammatory colitis.

Bacteriological analysis proved negative for...
Koch Bacillus. Treatment combining oral and rectal enemas of 5-ASA was continued, without improvement. Because the patient’s condition worsened (eight stools/day with mucus and blood), she was admitted to the hospital in February 1993. She was in good general condition with no weight loss, clinical examination was normal, and laboratory tests were unremarkable. A surgical treatment was decided upon because the symptoms persisted associated with the same lesions at endoscopy. Sigmoid colectomy was performed on 11 March 1993. Macroscopic examination showed haemorrhagic polyps at the apices of large mucosal folds separated by normal mucosa (Fig 2). The polyps consisted of hyperplastic colonic mucosa resting on an oedematous, lacy, well vascularised submucosa (Figs 3–4). Colonic glands were dilated and odd shaped, but seldom enfolded, and the epithelial cells were well differentiated and filled with mucus. In the luminal one fifth of the mucosa of some polyps, many glands were cystically dilated, had thin columnar epithelium that was only partially intact and contained detritus (Fig 5). The detritus consisted of granulocytes, degenerate epithelial cells, few round cells with granular eosinophilic cytoplasm, and mucus. These damaged glands occurred in a zone of inflammation comprising the luminal one third to one half of the mucosa. The inflammatory infiltrate consisted of chronic inflammatory cells, new blood vessels, and histiocytes – granulation tissue between the epithelial glands at the surface (Fig 6). In histological preparations, the cap was often artifactually displaced from the polyp. When special stains for virus inclusions bodies (Lundrum and Page Green) were applied to these lesions none were found. A Brown and Hoppe stain showed that the dilated cystic glands did not contain bacteria, and there were few bacteria in the fibropurulant cap. Giemsa and the periodic acid Schiff stains were unrewarding and a modified Steiner’s silver stain failed to show rickettsia, spirochetes, or intracellular campylobacter like...
Discussion

Our patient presented with the characteristic morphological and pathological features of cap polyposis as initially described in 15 patients by Williams et al.\(^1\) and recently recalled by Campbell et al.\(^2\) Cap polyposis affects patients of both sexes, ranging from 17 to 82 years. The common symptoms are mucoïd and bloody diarrhoea with abdominal pain and tenesmus. At endoscopy polyps of the rectosigmoid are red, sessile, and located at the apices of enlarged transverse mucosal folds with a normal intervening mucosa. Microscopic features include elongated hyperplastic looking glands with a mixed inflammatory infiltrate in the lamina propria. A cap of fibrinopurulent exudate covers the polyps. Symptoms are often relieved by polypectomy but, as in our patient, rectosigmoid resection may be required to control diarrhoea. The course of the disease is still uncertain but it is worth noting that cap polyposis can recur in the rectum left in place after left hemicolectomy as illustrated in one case of Campbell et al.\(^2\).

In his series Williams et al did not find any particular familial history or any strong association with other major colorectal diseases although two of 15 patients had chronic ulcerative colitis and one of 15 had sigmoid adenocarcinoma. Histological aspects similar to cap polyposis have been described in other disorders where mucosal prolapse is the underlying mechanism such as solitary rectal ulcer syndrome or prolapsed colostomies. It has thus been suggested that abnormal colonic motility may be a crucial aetiological factor: it could lead to prolapse of redundant mucosa at the apices of transverse mucosal folds resulting in a local ischaemia producing the characteristic histological appearances.\(^1\)\(^2\) Presence of intramucosal elastin at the edges of some of the cap polyps in one case described by Campbell et al.\(^2\) is another argument in favour of this hypothesis. Our patient had no evidence of abnormal colonic motility and orcein stain for elastin fibres was negative. An infectious origin or participation might be suggested in this case: (a) symptoms occurred in the postoperative course of pelvic surgery, a few days after antibiotics; (b) treatment with metronidazole improved her condition; (c) inflammatory cap polyps have been described in diverticular disease in which infectious agents might play a part. The search for virus or bacteria, however, in either dilatig cystic glands or fibrinopurulant cap was negative.