Polypoid and pseudopolypoid lesions of inflammatory bowel disease : diagnosis on double-contrast enema

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Abstract

The radiological and pathological features of ulcerative colitis (UC) and Crohn's disease (CD) are well known for most radiologists and gastroenterologists but on double-contrast enema, polypoid and pseudopolypoid manifestations of inflammatory bowel disease (IBD) often remain a source of major confusion. Inflammatory polyps project above the level of the surrounding mucosa. Pseudopolyposis is seen when extensive ulceration of the mucosa down to the submucosa results in scattered circumscribed islands of relatively normal mucosal remnants. Postinflammatory polyps reflect a non-specific healing of undermined mucosal and submucosal remnants and ulcers, and are mostly multiple. They have no malignant potential. Patients with long-standing UC and CD are at increased risk for developing colorectal carcinoma. Dysplasia is a precancerous histologic finding and is frequently seen in colitic colons at high risk for carcinoma. Dysplasia may be found in a radiographically normal appearing mucosa or it may be accompanied by a slightly raised mucosal lesion, a so-called dysplasia-associated lesion or mass (DALM lesion) and as a consequence radiographically detectable. Because differentiation of adenocarcinoma and dysplasia from inflammatory or postinflammatory polyps is sometimes difficult or impossible on double-contrast enema, endoscopy and biopsy are necessary for making a final diagnosis. (Acta gastroenterol. belg., **1999**, 62, **190-195**).

Key words : inflammatory bowel disease, pseudopolyposis, inflammatory polyps, postinflammatory polyps, dysplasia, neoplasia.

Introduction

The radiological and pathological features of idiopathic IBD (UC and CD) have been well described in the radiological literature (1-3) but the radiological evaluation of polypoid and pseudopolypoid lesions in patients with IBD and the terminology used are frequently a source of major confusion. The radiological spectrum of pseudopolyposis, inflammatory polyps, postinflammatory polyps, dysplasia, and neoplasia is reviewed, discussed and illustrated.

Active IBD : inflammatory polyps and pseudopolyps

A *polyp* is a mass that protrudes into the colonic lumen above the level of the mucosa. A polyp can be a neoplasm (adenoma or carcinoma) or a nonneoplastic lesion such as a hamartoma or a focal inflammatory process. A polyp may be seen in active IBD when inflammatory cellular infiltrates, mucosal and submucosal edema, or granulation tissue develops more pronounced in a focal area. If the resulting lesion projects above the level of the surrounding mucosa the term *inflammatory polyp* is used (4). The inflammatory polyp can be solitary or multiple and is seen against a background of granular mucosa (Fig. 1) (5). It has no malignant potential. Distinguishing isolated inflammatory polyp from dysplasia-associated lesion or mass (DALM lesion), adenoma or even carcinoma may be difficult or impossible on double-contrast enema. Therefore, differential diagnosis mostly requires endoscopic resection and histological examination.

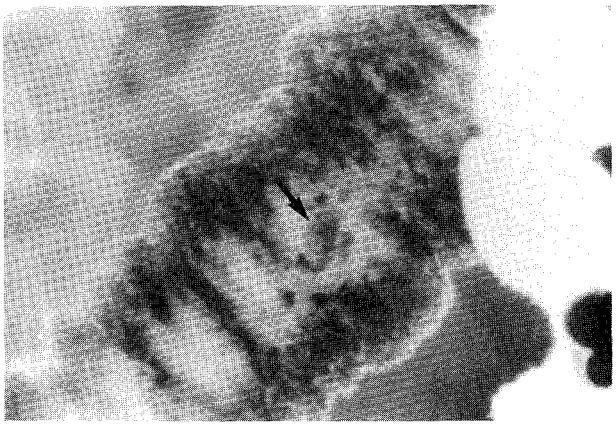

Fig. 1. — Inflammatory polyp in a patient with active ulcerative colitis (arrow). Filling defects with radiographic evidence of inflammatory changes are seen.

UC presents with superficial ulcers of the colonic mucosa which become deeper with further progression of the disease. The ulcerative process extends into the relatively vulnerable submucosa but is limited by the resistant, more deeply lying muscle. This leads to lateral undermining beneath the relatively resistant mucosal membrane and the formation of typical collar-button ulcers. These undermining ulcerations may join together in an interlacing network. With further progression

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Paper presented at Xth Belgian Week of Gastroenterology, Knokke, March 7th, 1998.

large areas of denuded mucosa and submucosa are seen. The remaining scattered islands of edematous mucosa and re-epithelialized granulation tissue result in the presence of *pseudopolyps or pseudopolyposis* which cause filling defects on Barium enema (4,6,7). In toxic megacolon edematous mucosal remnants visible as soft-tissue nodules consistent with pseudopolyps may be seen on plain film of the abdomen (Fig. 2).

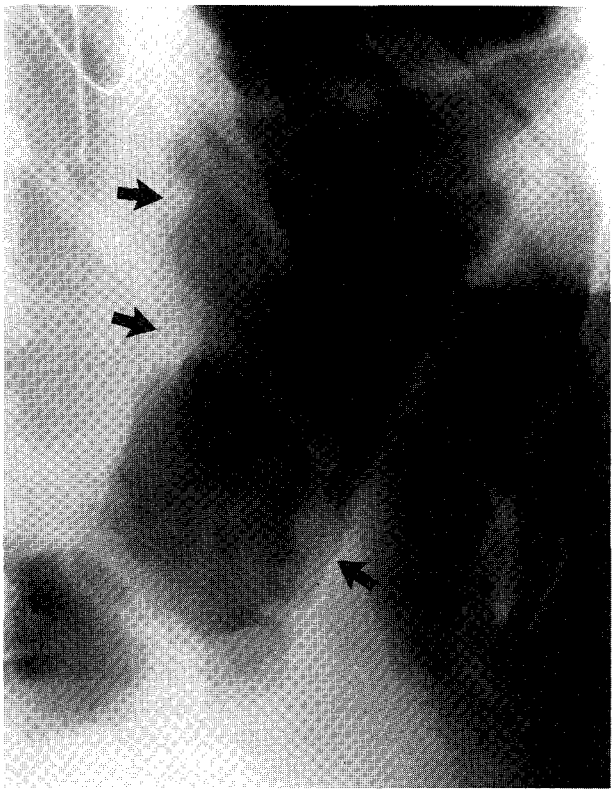


Fig. 2. — Pseudopolyposis in a patient with ulcerative colitis and symptoms of toxic megacolon. Plain film of the left upper abdomen shows markedly distended and irregular colon, consistent with toxic megacolon. Within the air-filled splenic flexure protruding nodules are seen (arrows).

CD produces aphthoid ulcers which become deeper and more irregular as Crohn's colitis progresses. Deep, linear transverse and longitudinal ulcers often separate intervening areas of edematous, but nonulcerated, mucosa producing a similar appearance of pseudopolyposis. For CD the term cobblestoning is usually used (6).

Remission : Postinflammatory polyposis (PIP)

PIP, also termed filiform polyposis, is most commonly seen in the colon, of UC and CD patients which are in clinical remission. It is also reported as a sequel of other colitides such as ischemic colitis, pseudomembranous colitis, infectious colitis and has been described in the stomach and small bowel in patients with CD (1). It is the most frequent local sequel of UC, occurring in nearly 20% of the patients with total or subtotal involvement of the colon. PIP is a nonspecific sequel of severe diffuse mucosal inflammation with undermining ulceration, due to regeneration of the epithelium along the undersurface of the undermined mucosal and submucosal remnants and along the base of the ulcer. This results in fingerlike projections of submucosa covered by mucosa and thus true polyps. Mucosal bridges are seen when a bridge of mucosa survives between islands of mucosa surrounded by ulceration. With remission, the underside of the mucosal bridge and the underlying ulcer re-epithelialize (4,8,9). PIP is a sign of ongoing mucosal healing following one or more episodes of severe colitis and should not be confused with the manifestation of active disease. PIP may regress in patients with clinically quiescent disease and has no malignant potential. PIP alone is not an indication for bowel resection but complications, such as massive haemorrhage or intestinal obstruction, may necessitate surgical intervention (8). PIP has a characteristic radiographic appearance : small sessile and filiform polyps resembling the stalks of polyps without the heads. Some may be joined by mucosal bridges or have a radiating or branching pattern (Fig. 3 and 4). PIP is visualized against a background of relatively

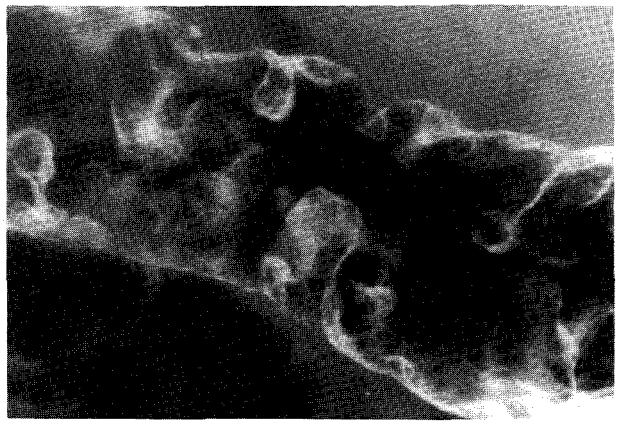


Fig. 3. — Postinflammatory polyps in a patient with ulcerative colitis.

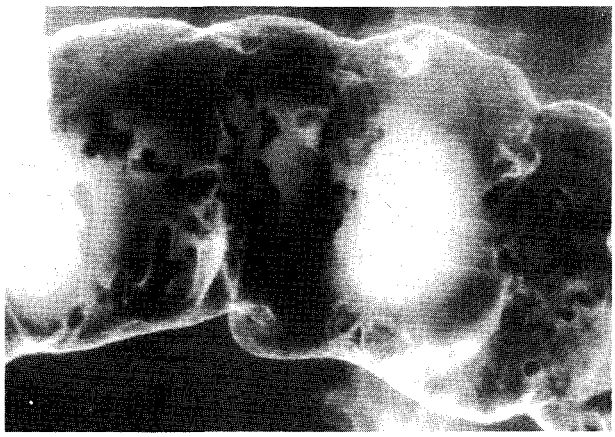


Fig. 4. — Typical postinflammatory polyposis. Note variable shape, clubbed ends and branching. The mucosa has a smooth appearance indicating that the disease is quiescent.

normal, nonulcerated colonic mucosa (1,8,9,10,11,12). PIP is nearly always multiple, but it may be solitary. Radiological appearance is variable and four presentations are described (13). 1. *localized multiple polyposis* characterized by individual polyps usually measuring less than 0.5 cm in diameter and localized in one segment of the colon. 2. *localized giant polyposis* characterized by large polyps measuring up to 2 cm in diameter. 3. *generalized polyposis* characterized by numerous postinflammatory polyps of varying sizes and shapes distributed throughout the colon from the rectum to the cecum. 4. *filiform polyposis* characterized by long, finger-like postinflammatory polyps that can occur either as solitary polyps or as diffuse polyposis distributed over large areas of colonic mucosa. Filiform polyps are usually 2-5 cm in length and 0.5 cm in diameter.

Conglomerates of giant polyps may simulate a mass lesion, especially a villous adenoma and adenocarcinoma (10,12) (Fig. 5). Uncommonly, this collection of polyps and adherent fecal material may obstruct the colonic lumen, or may eventually cause intermittent colonic intussusception or haemorrhage (12). On Barium enema, a large, localized, filling defect is seen which may partially or completely obliterate the lumen of the bowel. Linear, filiform strands are visible on the surface of the mass. The lesion has the shaggy features of a villous tumor and may be compressible as well as changeable in shape. The transition between the localized polyps and the more normal mucosa is usually gradual. Other polyps are often present elsewhere in the colon (14). A change in shape and contour and the presence of other areas of postinflammatory polyps around giant postinflammatory polyposis mostly allows differentiation with villous adenoma and adenocarcinoma. Villous neoplasms have typically sharp margins. A carcinoma is rarely polypoid but is either flat and ulcerated or constricting (4,14,15). In questionable cases, biopsy is required to estabish the diagnosis.

Fig. 5. — Giant postinflammatory polyposis in a patient with ulcerative colitis (arrows). Barium enema demonstrates partial obstruction of the lumen by a conglomerate of postinflammatory polyps. This lesion may be confused with an adenoma or adenocarcinoma. The presence of polyps in the more distal colon sustains a correct diagnosis.

Dysplasia and neoplasia

Patients with IBD, either UC or CD, are at increased risk for the development of gastrointestinal *carcinoma.* The colonic carcinomas that develop in patients with UC are more likely to be multifocal and to occur at earlier ages than the colonic carcinomas in the general population (4). The two best-established risk factors for developing colorectal cancer in UC are disease duration and anatomic extent (15). Colorectal cancer is rarely encountered when the total duration of colitis is less than 8 to 10 years, but thereafter the risk of cancer rises at a rate of approximately 0.5% to 1.0% per year. Patients with extensive colitis (defined as disease proximal to the splenic flexure) and to a lesser degree patients with left-sided colitis are at risk, whereas those with only proctitis have virtually no risk for colorectal cancer. There is no agreement between different authors concerning the additional risk for colorectal cancer in case of onset of colitis at a young age (15). A true colonic stricture in UC is considered as a major risk factor while primary sclerosing cholangitis and folate depletion have been reported as rather additional risk factor for colorectal cancer (3,16,17).

The detection of the carcinomas complicating UC and the differentiation from benign strictures is often difficult because they are, in many cases, flat and infiltrating (18). They may present with a scirrhous or linitis plastica appearance, characterized by infiltration of submucosa and subjacent muscular wall with marked thickening and rigidity of the bowel wall and by lack of significant mucosal and intraluminal component. Occasionally postinflammatory polyps may be large and multilobulated and indistinguishable from carcinoma (18) (Fig. 6). CD patients have a similar risk for carcinoma as UC patients and the clinical and pathologic features of CD associated carcinoma are comparable to those of UC associated cancers (19). Similarities include long duration of disease, relatively young age at diagnosis of cancer compared with sporadic colorectal cancer, frequent multiple tumours at presentation, usual predominance of cancer in the

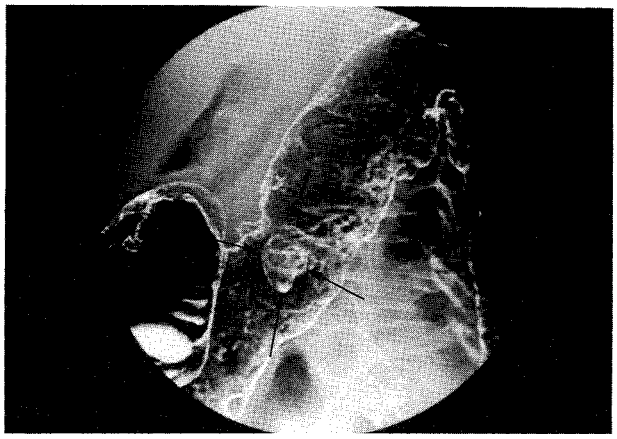


Fig. 6. — Postinflammatory polyp versus adenoma and adenocarcinoma in a patient with ulcerative colitis. Double-contrast Barium enema reveals postinflammatory polyposis of the transverse colon and a 2 cm multilobulated sessile polyp (arrows). After endoscopic biopsy the lesion was classified as a postinflammatory polyp.

area of macroscopic disease, prevalence of associated dysplasia, high frequency of mucinous and signet-ring cell tumours, and a poor survival rate (20). Strictures and chronic fistulas in longstanding active disease have been labeled as carrying a high risk for carcinoma (4).

When patients with chronic UC develop colon carcinoma, there are often scattered areas of epithelial *dysplasia* in the resected specimen. This association of adenocarcinoma with dysplasia in patients with IBD has been well established. The dysplasia seen in patients with UC and CD is essentially identical (21). Dysplasia is a histological term used to describe "unequivocal neoplastic epithelial proliferation" and is accepted to be a harbinger of malignancy. Dysplasia may be low or high grade carrying a corresponding risk of cancer (21). Dysplastic epithelium may be flat and invisible macroscopically and thus not visible on double-contrast barium enema or colonoscopy. Dysplasia is sometimes visible macroscopically as obvious nodular protrusions, irregular areas with slightly elevated mucosa and sharply angulated edges, a plaquelike lesion, a solitary polyp or a cluster of polyps (the so-called 'DALM lesion') and consequently visible on double-contrast barium enema and colonoscopy (4,18) (Fig. 7,8,9a and b). When dysplasia presents as a DALM lesion the risk of carcinoma is very high and total proctocolectomy is usually recommended whether the dysplasia is of high or low grade (4). Histological diagnosis or suitably ordered differential diagnosis remains essential because a simple adenomatous polyp only requires a colonoscopic polypectomy (4).

Because carcinoma associated with UC is simultaneously or metachronously accompanied by dysplasia, the main aim of surveillance by colonoscopy is the detection of these precancerous lesions. In patients with colitis dysplastic areas or carcinoma are hardly recognizable while in normal colon abnormal areas are in contrast with the smooth pale background of normal mucosa. Patients with UC show nondysplastic mucosal abnormalities due to chronic and recurrent inflammation so that not all areas of dysplasia are necessarily recognized on colonoscopy. Therefore multiple samples throughout the colorectum must be obtained every 10 cm and biopsies are performed on all visible macroscopic lesions to rule out dysplasia (22). Depending on the risk factors involved as well as on the data from previous biopsies, these procedures are performed anywhere from 6 months to a few years apart. Radiological studies play a secondary role in detection of dysplasia and carcinoma. Radiography may provide better information about the gross mucosal appearance and the presence of changes in contour and strictures than colonoscopy and seems therefore to be valuable for determining the area within the colon that should be carefully surveyed at colonoscopy and biopsy. It has been reported that cancers associated with UC occur predominantly in the rectosigmoid colon (23,24). On the basis of this finding proctosigmoidoscopy was reported to be more valuable than colonoscopy of the entire colon in a surveillance program (24). Matsumoto *et al.* (18) reported that radiography can depict dysplasia, especially in the proximal colon and concluded

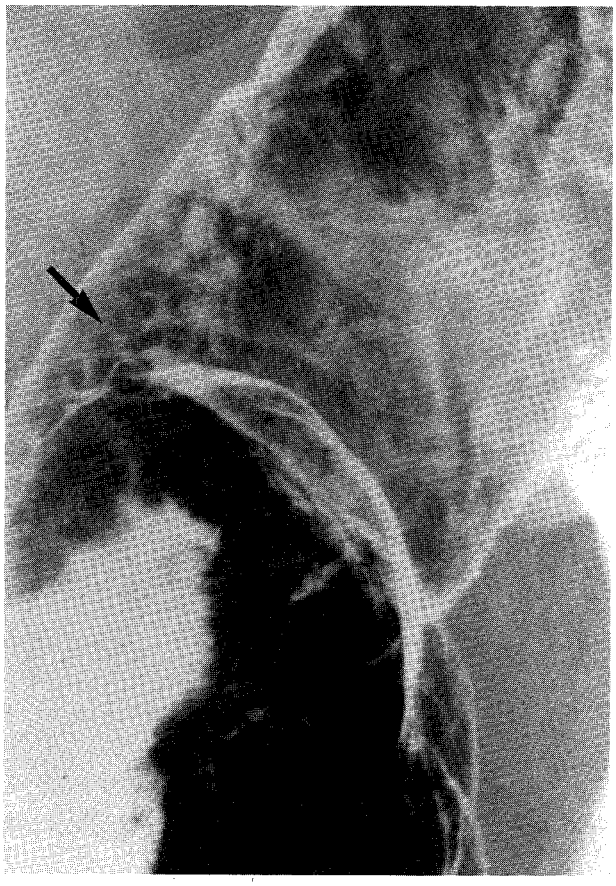


Fig. 7. — Double-contrast Barium enema shows a focal irregularity of the mucosal surface irregularity with multiple concomitant nodules representing dysplasia (arrow).

that the combined use of sigmoidoscopy in the distal colon and barium enema examination in the proximal colon may be an effective mean of surveillance in patients with UC. Even though radiography may be useful in the identification of dysplasia, subsequent colonoscopy and biopsy is mandatory for further diagnosis (4,18). Cancer surveillance in patients with CD at increased risk also includes periodic colonoscopy and biopsies in the area of macroscopic disease (20).

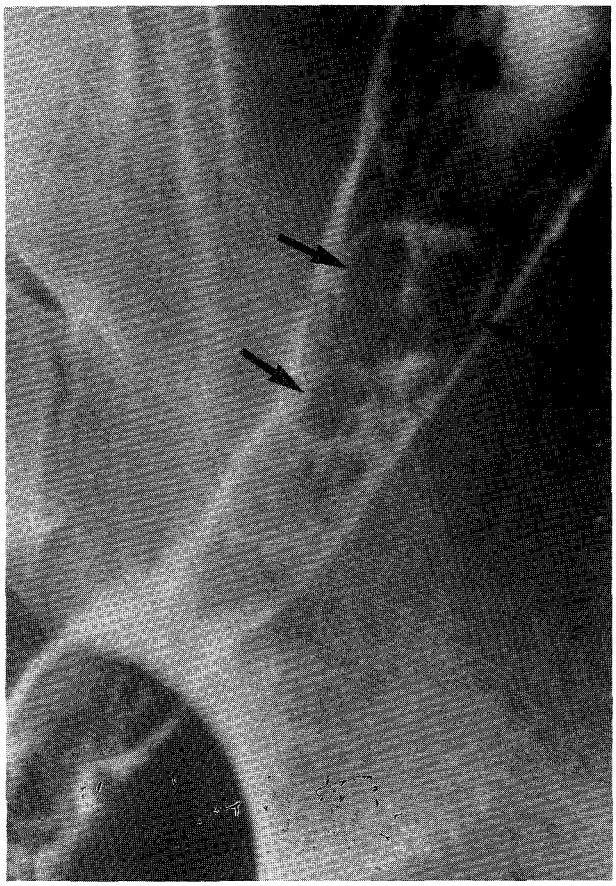
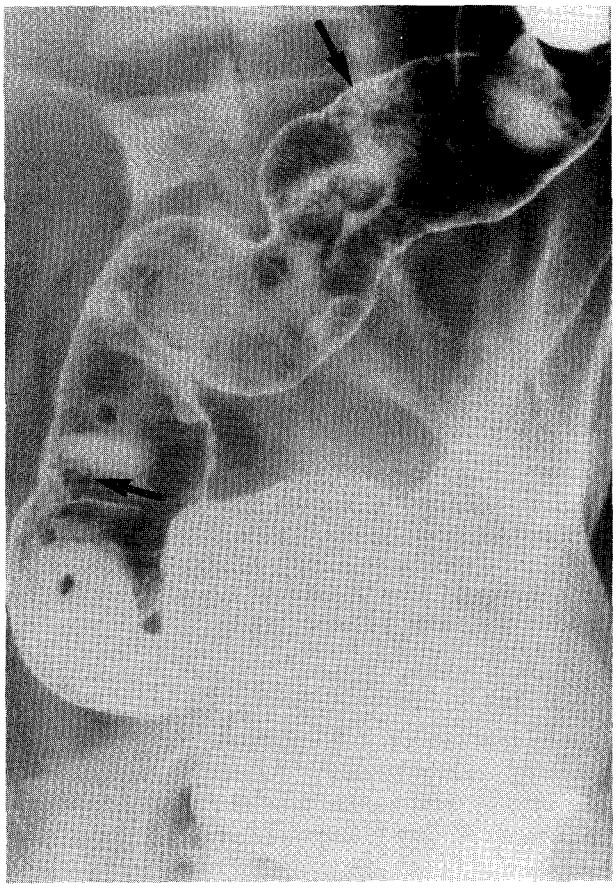


Fig. 8. — Dysplasia in chronic ulcerative colitis demonstrated by double-contrast enema. Flat polyps in a benign stricture in the distal colon were biopsy proven dysplastic lesions. Patient underwent total proctocolectomy. In the resected specimen a carcinoma in situ was found.



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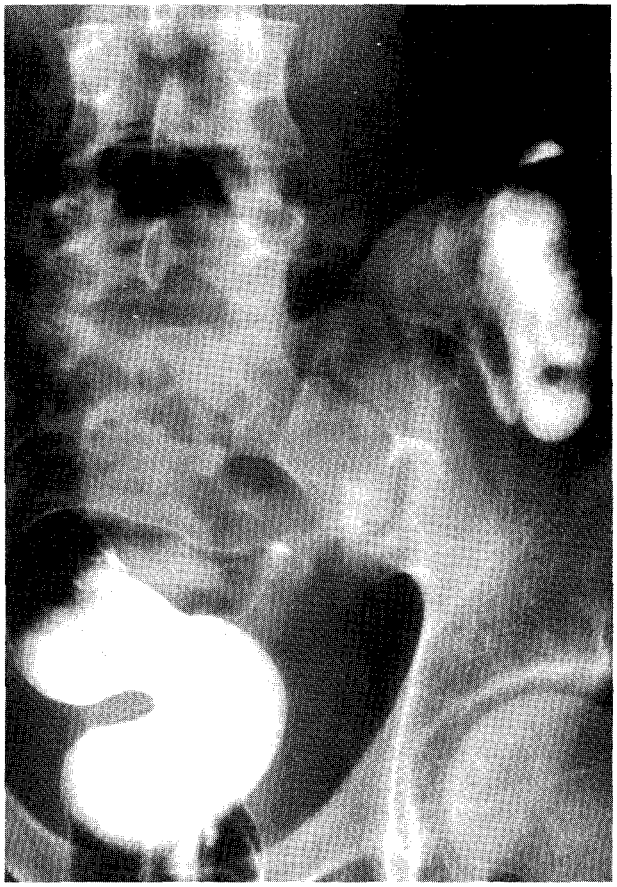

b

Fig. 9a and b. — Development of carcinoma in a patient with chronic ulcerative colitis and dysplasia. (a) Double-contrast barium enema of the distal colon shows two areas of elevated mucosa (arrows) and postinflammatory polyposis. Biopsies of these areas demonstrated moderate dysplasia. Patient refused total proctocolectomy and further follow-up. (b) Three years later symptoms of distal colonic obstruction occurred due to stenosing adenocarcinoma of the sigmoid.

Conclusion

Polypoid and pseudopolypoid lesions frequently occur in patients with IBD. Inflammatory polyps and pseudopolyps are characteristic of active disease. Postinflammatory polyps are common, almost always multiple and usually have a unique appearance. They have no malignant potential. Patients with IBD are at increased risk for the development of gastrointestinal carcinoma. Dysplasia is a histologic marker for the risk of carcinoma and sometimes visible macroscopically. It may be difficult or impossible to differentiate inflammatory or postinflammatory polyps from polyps containing dysplasia or carcinoma. For the definitive diagnosis of any suspicious lesion the radiologist should recommend endoscopy and biopsy.

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