Recently, on the basis of experience with three AIDS patients—two with gastroduodenal cryptococcosis as the primary clinical manifestation of disease—Chalasani *et al*. (4) have emphasized that gastrointestinal cryptococcosis may be the initial presentation of disseminated cryptococcal infection. In our opinion, however, it may well be that gastrointestinal involvement by *Cryptococcus* is much more than just the primary clinical manifestation, inasmuch as cryptococcosis can actually start in the gastrointestinal tract and not only in the lung as usually asserted (1). Interestingly, an analogous opinion was advanced years ago for a nonimmunocompromised patient with a hard tumor-like mass located in the descending colon and consisting of large accumulations of histiocytes containing *Cryptococcus* cells (5). A similar opinion was also put forward in discussing a case of disseminated infection in a HIV-negative patient with extensive lesions of the colon, omentum, and skin, but without signs of lung or meningeal involvement (6). Furthermore, no evidence of lung involvement has been reported in the many cases of cryptococcosis of gastrointestinal organs, such as the omentum (7) or peritoneum (8, 9), affecting immunocompromised patients.

The histology of our case strongly supports the hypothesis of a primary infection in the bowel. The presence of hyperplastic lymphoid tissue with cryptococci engulfed in histiocytes, but without evidence of giant cells or granulomatous tissue deposition, rules out the possibility of an old lesion, which is reported to be characterized by granulomatous features (1). Moreover, the presence of yeast debris and partially degenerated cryptococci in the histiocytes lends evidence to their previously reported cytotoxicity (10). The unique portal of entry for cryptococcal infection in our case, *i.e.*, a peduncolated adenomatous polyp, enhances the interest of the finding.

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REFERENCES

1. Baker RD, Haugen RK. Tissue changes and tissue diagnosis in cryptococcosis. A study of 26 cases. Am J Clin Pathol 1955;25:14–24.
2. Mitchell TG, Perfect JR. Cryptococcosis in the era of AIDS—100 years after the discovery of *Cryptococcus neoformans*. Clin Microbiol Rev 1995;8:515–48.
3. Pinner RW, Hajjeh RA, Powderly WG. Prospects for preventing cryptococcosis in persons infected with human immunodeficiency virus. Clin Infect Dis 1995;21(suppl 1):S103–7.
4. Chalasani N, Wilcox CM, Hunter HT, et al. Endoscopic features of gastroduodenal cryptococcosis in AIDS. Gastrointest Endosc 1997;45:315–7.
5. Unat EK, Pars B, Kosyak JP. A case of cryptococcosis of the colon. Br Med J 1960;2:1501–2.
6. Daly JS, Porter KA, Chong FK, et al. Disseminated, nonmeningeal gastrointestinal cryptococcal infection in an HIV-negative patient. Am J Gastroenterol 1990;85:1421–4.
7. Chong PY, Panabokke RG, Chew KH. Omental cryptococcoma. An unusual presentation of cryptococcosis. Arch Pathol Lab Med 1986;110:239–41.
8. Clift SA, Bradsher RW, Chan CH. Peritonitis as an indicator of disseminated cryptococcal infection. Am J Gastroenterol 1982;77:922–4.
9. Poblete RB, Kirby BD. Cryptococcal peritonitis. Report of a case and review of the literature. Am J Med 1987;82:665–7.
10. Granger DL, Perfect JR, Durack DT. Macrophage-mediated fungistasis in vitro: Requirements for intracellular and extracellular cytotoxicity. J Immunol 1986;136:672–80.

TREATMENT OF CHRONIC POST-RADIATION PROCTITIS WITH ORAL ADMINISTRATION OF SUCRALFATE

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Several nonsurgical approaches to the treatment of postradiation proctitis have been described, but no effective conservative treatment has yet been established. As an alternative to the usual treatment, three cases of chronic postradiation proctitis with hemorrhage were successfully treated with oral administration of sucralfate, with resultant decreased bleeding in long term follow-up period. Oral sucralfate may provide a novel approach to the treatment of intractable postradiation proctitis. (Am J Gastroenterol 1998;93:1593–1595. © 1998 by Am. Coll. of Gastroenterology)

INTRODUCTION

Recurrent hemorrhage in patients with postradiation proctocolitis is often refractory to medical therapy (1). When conservative therapy fails to control the bleeding, surgical treatment may be required. However, this can be technically difficult, and the incidence of complications is high (2). Therefore, several nonsurgical approaches to the treatment of postradiation proctocolitis with severe hemorrhage have been described (1, 3–5), but no effective conservative treatment has been established to date.

An increasing number of small clinical trials have used sucralfate to treat ulceration of the colon; some beneficial effect of sucralfate enema has been suggested for ulcerative colitis (6) and solitary rectal ulcer (7). As an alternate treatment for postradiation proctitis with hemorrhage, we present three cases successfully treated by oral administration of sucralfate.

CASE REPORTS

Case 1

A 54-yr-old woman was treated for adenocarcinoma of the uterine corpus with radical hysterectomy in March 1989. The radiation was given over a period of 50 days to a total dose of 55 Gy. In April, 1990, the patient developed loose stools, abdominal pain, and moderate rectal bleeding. On colonoscopy, a diffusely oozing rectal mucosa covered with blood clots was seen, and biopsies confirmed changes consistent with radiation-induced proctitis. Although suppositories of sulfasalazine or β-methazone were administered for 4 wk, her symptoms remained resistant to the therapy. The patient was then treated with oral administration of sucralfate (3 g/day), leading to improvement of the symptoms. On colonoscopy performed in October 1990, the rectal mucosa was mildly active with patchy teleangiectasias, but spontaneous or

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contact bleeding was not observed. Since then, she has been well without significant rectal bleeding for >7 yr.

Case 2

A 40-yr-old woman was treated for cervical carcinoma of the uterus with radiotherapy to the total dose of 60 Gy, which was completed by August 1991. In October 1991, she suddenly complained of diarrhea and severe hematochezia. Colonoscopic examination demonstrated teleangiectasias and friability in the rectal mucosa, with spontaneous bleeding, and the histology of the biopsies was compatible with postradiation proctitis. Although sulfasalazine (3 g/day) and predonisolone (30 mg/day) were administered orally for 4 months and 2 months, respectively, her symptoms remained resistant to the therapy. In February 1992, intra-arterial infusion of predonisolone (40 mg) was performed, but without any improvement. For the next 8 months, persistent bleeding required 10 U of blood transfusion. In November 1992, oral administration of sucralfate (4 g/day) was started. During the next 2 months, her symptoms gradually subsided and the hematochezia disappeared. On follow-up colonoscopy in March 1993, the rectal mucosal erosions were healed, with an almost normal vascular pattern. Since then, she has been well, without hematochezia or fall of hematocrit for >4 yr.

Case 3

A 70-yr-old man with prostatic carcinoma underwent castration and radiotherapy to the total dose of 70 Gy in November 1992. One year after the radiation treatment, he complained of moderate hematochezia, and colonoscopic examination demonstrated teleangiectasias and spontaneous bleeding in the rectal mucosa with a histology compatible with postradiation proctitis. Encouraged by the experience of the previous cases, we started oral administration of sucralfate (4 g/day). During the following month, the hematochezia gradually disappeared. In June 1993, colonoscopy demonstrated that a portion of the rectum had undergone atrophic change, but without spontaneous or contact bleeding. Since then, the patient has been well without hematochezia for >4 yr.

DISCUSSION

Patients with postradiation proctocolitis present most frequently with hematochezia, bowel habit disturbances, and pain. Particularly, recurrent hemorrhage is often refractory to medical therapy and can be severe enough to warrant transfusion (1). Agents including corticosteroids (systemic or in enema form) and oral sulfasalazine have been reported to be successful in treating a small case series of patients with chronic postradiation proctitis (8), but no systematic investigation has been reported and generally these agents are not used to treat chronic postradiation proctitis. Furthermore, surgical treatment cannot always be recommended because of the high incidence of complications (2), because diverting colostomy without proctectomy often fails to diminish blood loss, and because proctectomy in patients with prior radiation can result in severe morbidity (1). Accordingly, there are several nonoperative approaches to the treatment described such as the use of Nd-YAG laser fiber to coagulate bleeding teleangiectasias (1, 4), instillation of formalin into the rectum (3), and hyperbaric oxygenation with 100% oxygen inhalation (5). However, these procedures have some disadvantages: they may be risky and cannot be performed at every institution because of the need for special equipment or endoscopic technique. As an alternate therapy, the use of sucralfate in enema form has been reported (9).

Sucralfate, a sulfated disaccharide, is a locally acting antiulcer agent that shields the base of peptic ulcers from the destructive effects of acid by the formation of a viscous coagulum (10). The application of sucralfate in enema form for inflammatory bowel diseases (6) and solitary rectal ulcer (7) has also been reported to have some beneficial therapeutic effect. Because sucralfate, when orally administered, binds selectively at the sites of mucosal ulceration in active inflammatory bowel disease (11), we evaluated the efficacy of oral administration of sucralfate in controlling hemorrhagic postradiation proctitis.

In the present report, three cases of postradiation proctitis with hemorrhage were treated with oral sucralfate (3–4 g daily) with resultant decreased bleeding in long-term follow-up. Although the precise mechanisms underlying the therapeutic effect of sucralfate have not been established, the cytoprotective effects of sucralfate on mucosal protection may include its binding to the colonic erosions or ulcerations (11) and a wide diversity of factors such as increased mucus secretion, proliferative zone stimulation and protection, and binding of epidermal growth factor (10). These effects may contribute to repair of the injured mucosa and thus accelerate recovery of the vascular changes in the postradiation proctitis by preventing inappropriate introduction of microorganisms or toxins into the submucosa.

The advantages of using sucralfate are its simplicity of administration (oral administration in this report) and the low frequency of adverse effects. Accordingly, oral sucralfate may provide a novel approach to the treatment for postradiation proctitis, and add to the prophylaxis of the disease as earlier documented (12). Double blind trials to determine the efficacy of this drug in managing hemorrhagic postradiation proctitis are awaited with interest.

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REFERENCES

1. Ahlquist DA, Gostout CJ, Viggiano TR, et al. Laser therapy for severe radiation-induced rectal bleeding. Mayo Clin Proc 1986;61:927–31.
2. Jao SW, Beart RW, Gunderson LL. Surgical treatment of radiation injuries of the colon and rectum. Am J Surg 1986;151:272–7.
3. Rubinstein E, Ibsen T, Rasmussen RB, et al. Formalin treatment of radiation-induced hemorrhagic proctitis. Am J Gastroenterol 1986;81: 44–5.
4. Berken CA. Nd:YAG Laser therapy for gastrointestinal bleeding due to radiation colitis. Am J Gastroenterol 1985;80:730–1.
5. Nakada T, Kubota Y, Sasagawa I, et al. Therapeutic experience of hyperbaric oxygenation in radiation colitis. Report of a case. Dis Colon Rectum 1993;36:962–5.
6. Ladas SD, Spiliades C, Hatzioannou J. Sucralfate enema in treatment of patients with distal ulcerative colitis refractory to steroid enemas. In: Clinical controversies in inflammatory bowel disease. Bologna: Tipografia Negri SRL, 1987:188.
7. Batman F, Arslan S, Telatar H. Effect of sucralfate enema in the treatment of solitary rectal ulcer (letter). Endoscopy 1988;20:128.
8. Goldstein F, Khoury J, Thornton JJ. Treatment of chronic radiation enteritis and colitis with salicylazosulfapyridine and systemic corticosteroids. A pilot study. Am J Gastroenterol 1976;65:201–8.

9. Ladas SD, Raptis SA. Sucralfate enemas in the treatment of chronic postradiation proctitis. Am J Gastroenterol 1989;84:1587–9.
10. Szabo S, Hollander D. Pathways of gastrointestinal protection and repair: mechanisms of action of sucralfate. Am J Med 1989;86 (suppl 6A):23–31.
11. Dawson DJ, Khan AN, Miller V, et al. Detection of inflammatory bowel disease in adults and children: evaluation of a new isotopic technique. Br Med J 1985;291:1227–30.
12. Henriksson R, Franzen L, Littbrand B. Effects of sucralfate on acute and late bowel discomfort following radiotherapy of pelvic cancer. J Clin Oncol 1992;10:969–75.

A SIMPLE TECHNIQUE TO REMOVE MIGRATED ESOPHAGEAL STENTS

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A 51-yr-old man with a tracheoesophageal fistula from an esophageal carcinoma had two expandable covered stents placed, which migrated distally. After several unsuccessful attempts to remove the stents, we fashioned a homemade snare to entrap and remove the stents under endoscopic and fluoroscopic guidance. (Am J Gastroenterol 1998;93:1595. © 1998 by Am. Coll. of Gastroenterology)

INTRODUCTION

Placement of expandable stents for esophageal cancer may be complicated by stent migration (1). Removal can be difficult or even impossible, making this a drawback of these stent systems. We report a new method for removal of migrated esophageal stents that may lessen this drawback.

CASE REPORT

A 51-yr-old man with progressive dysphagia to solids was evaluated at Jacobi Medical Center and found to have a squamous cell carcinoma of the esophagus, 5–7 cm in length, beginning 3 cm below the upper esophageal sphincter. A computed tomography scan revealed extensive local spread and extrinsic compression of the esophagus. The patient elected to have endoscopic dilation of the lesion while undergoing radiation therapy. Six wk later, he developed a cough and fever, and a barium esophagram demonstrated a tracheoesophageal fistula arising from within the lesion. After obtaining consent, we inserted a 14-cm covered Z-stent (Wilson-Cook, Inc., Winston-Salem, NC) under fluoroscopic guidance. A second stent was required at that time, after the first stent migrated distally and could not be repositioned to cover the fistula. He was discharged 2 days later, but returned 1 wk later with a fever, cough, and aspiration pneumonia. Chest radiographs demonstrated distal migration of the two stents, with one protruding into the stomach. During endoscopy, repeated attempts to reposition the stents were unsuccessful, using the "hook maneuver," jumbo forceps, a TTS balloon, and large snares. After advancing both stents into the stomach with a forceps, we removed the endoscope. We fashioned a homemade snare by passing a Geenen guidewire (Microvasive, Watertown, MA) down the biopsy channel of an upper endoscope (Olympus GIF-100), making a loop, and sending the end of the wire back up the channel; this snare could be expanded to any diameter desired. After reinserting the endoscope, we manipulated the ends of the wire coming out of the biopsy channel, facilitating placement around the end of one stent. By tightening the loop, we collapsed the stent, and it was easily withdrawn under fluoroscopic and endoscopic guidance. The second stent was removed in a similar fashion. A 15-cm Ultraflex covered stent (Microvasive, Watertown, MA) was inserted with good results, and the patient was discharged within several days. Several weeks later, he has no dysphagia, cough, or evidence of aspiration.

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DISCUSSION

If migration of esophageal stents occurs, repositioning or removal presents a problem for the endoscopist, and these stents sometimes are left in the stomach (2). Endoscopic removal of stents with exposed wires at their ends may cause damage to the instrument or the esophagus. Several methods used to remove or reposition migrated stents include the "hook" maneuver, snare retrieval, stent inversion through an overtube, and the use of rat tooth forceps (3). The "hook" maneuver can lead to kinking of the proximal end of the stent, which began to occur in our case (4). This maneuver also could potentially damage the endoscope, if the stent has exposed wires at its distal end. Snares may not have a large enough diameter to entrap larger sized stents. The inversion maneuver will not work with Z-stents, and forceps may not collapse stents adequately for removal.

In this patient, we were unable to entrap the ends of the stents with different snares. We overcame this problem by fashioning a homemade snare from a flexible guidewire, which we could enlarge to any size. Tightening the guidewire collapsed the end of the stents enabling us to extract them with the exposed ends trailing in a J-configuration.

In summary, if migrated stents cannot be repositioned easily using forceps or snares, we suggest fashioning a homemade snare to capture and remove the stent after manipulating it into the stomach. We do not recommend this technique to remove stents made with lateral barbs, *e.g.*, Gianturco-Rosch (Cook Inc.), which could potentially damage the endoscope (1, 5).

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REFERENCES

1. Angueira CE, Kadakia SC. Esophageal stents for inoperable cancer: Which to use? Am J Gastroenterol 1997;92:373–6.
2. Schmassmann A, Meyenberger C, Knuchel J, et al. Self-expanding metal stents in malignant esophageal obstruction: A comparison between two stent types. Am J Gastroenterol 1997;92:400–6.
3. Mallery S, Freeman ML. Removal of an incompletely expanded ultraflex esophageal stent. Gastrointest Endosc 1996;43:163–4.
4. Berkelhammer C, Roberts J, Steinecker G. Repositioning a migrated esophageal stent using a retroflexed endoscope: A note of caution. Gastrointest Endosc 1996;44:632–4.
5. Kozarek RA, Ball TJ, Brandabur JJ, et al. Expandable versus conventional esophageal prostheses: Easier insertion may not preclude subsequent stent-related problems. Gastrointest Endosc 1996;43:204–8.