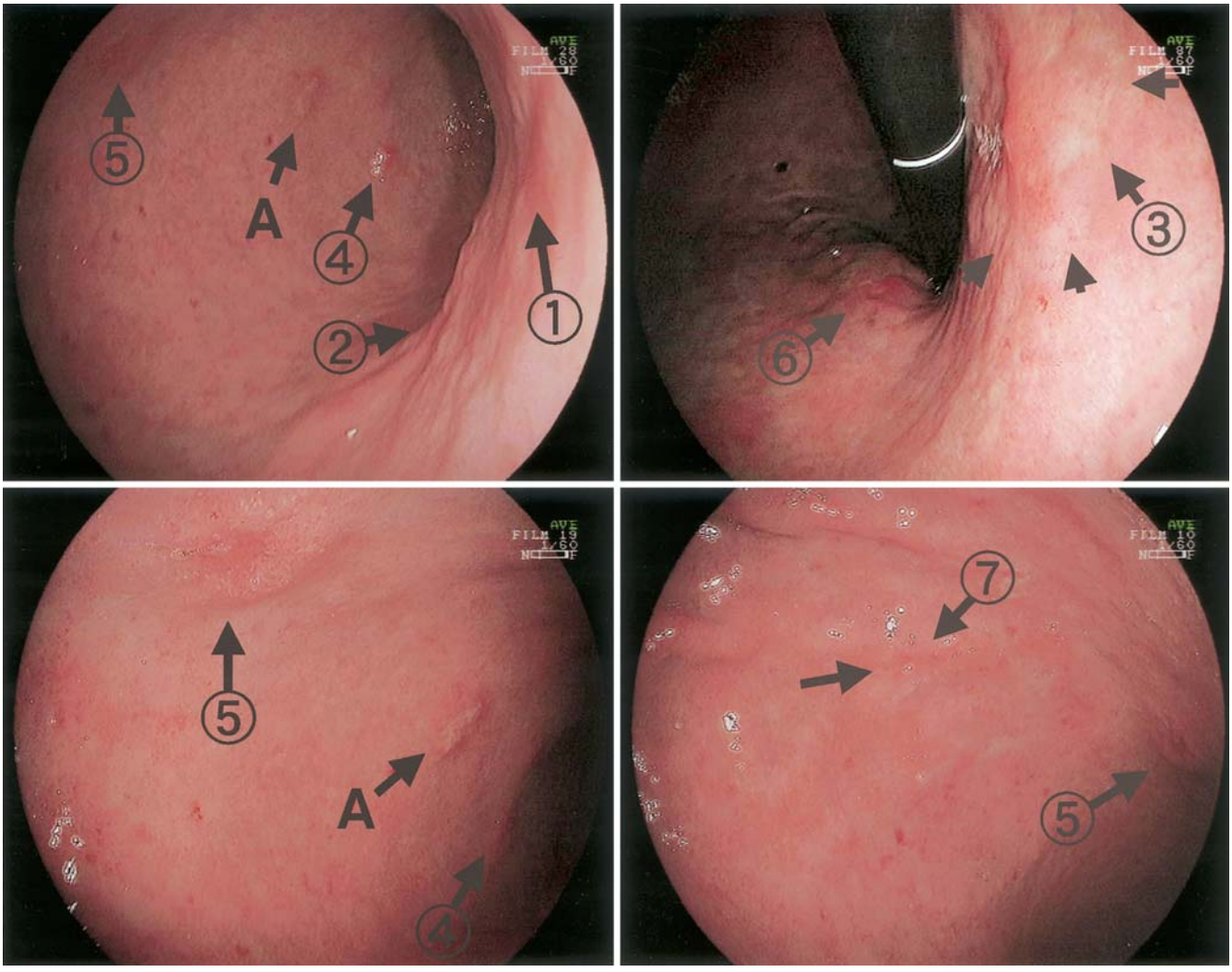

Fig. 1. Endoscopic appearance of the stomach. Six type II c (superficial depressed type) lesions (*1–3,5–7*), one type II a lesion (superficial elevated type) (*4*), and an adenoma (lesion *A*) were observed

A case of ischemic colitis during pregnancy

Several reports of ischemic colitis in young adults have appeared in the literature.[1–3] However, cases of ischemic colitis associated with pregnancy are very rare.[4,5] We report a case of ischemic colitis occurring in the eighth week of pregnancy.

A 27-year-old woman was referred to our hospital because of abdominal pain and bloody diarrhea that had lasted for 2 days. She usually had a bowel movement every 4 to 5 days. She was 8 weeks pregnant. She had had a normal delivery a year earlier and had no history of spontaneous abortion. Three weeks before coming to our hospital, she had experienced a similar episode, with a self-limited course, during this pregnancy. On admission, her vital signs were stable. Emergency colonoscopy showed a longitudinal ulcer demarcated from the surrounding mucosa, which was hyperemic and edematous in the descending colon (Fig. 1). Biopsy samples were examined histologically. The samples showed transmural inflammation, including degeneration of the muscularis mucosae and submucosa. The crypts were partly destroyed, with a ghost-like appearance characteristic of ischemic colitis (Fig. 2a). In the small capillaries in the submucosal layer, some fibrin thrombi were observed, showing a blue color with phosphotungstic acid-hematoxylin (PTAH) staining (Fig. 2b). Having ruled out an infectious cause, we diagnosed her illness as ischemic colitis. She was treated conservatively by fasting and intravenous hydration. She recovered and was discharged on the seventh hospital day. After a normal delivery, she remains asymptomatic, at 11 months after discharge.

Ischemic colitis is a disorder of older adults, who frequently show generalized atherosclerosis as a predisposing condition. However, it is being recognized more frequently in young healthy adults, in whom transient ischemic colitis typically presents with an abrupt onset of left-side abdominal pain, occasional nausea and vomiting, and bloody diarrhea.[3] In a young adult thought to have transient ischemic colitis, the illness is generally benign and self-limited.[3] Most patients have only a single episode of transient

ischemic colitis. However, recurrence, which seemed to develop in our patient, does occur in a few patients.[1,5,6]

Some younger patients affected by this condition have underlying vascular disorders or a predisposing condition, including vasculitis, diabetes, or coagulopathy, or a history of taking drugs such as vasopressin, ergotamine, cocaine, and oral contraceptives.[3]

In young women with transient ischemic colitis, the condition is often associated with the use of oral contraceptives. Deana and Dean[2] estimated a sixfold relative risk increase of transient ischemic colitis in women taking oral contraceptives. In a review of the literature, Gurbuz et al.[6] documented 67 cases of ischemic colitis associated with oral contraceptive use; 7 cases involved colitis induced by Premarin (a mixture of equine conjugated estrogens). Although the role of estrogen in ischemic colitis has not been definitely established, the proposed mechanism of estrogen-induced colitis is the induction of a hypercoagulable state.[1,7]

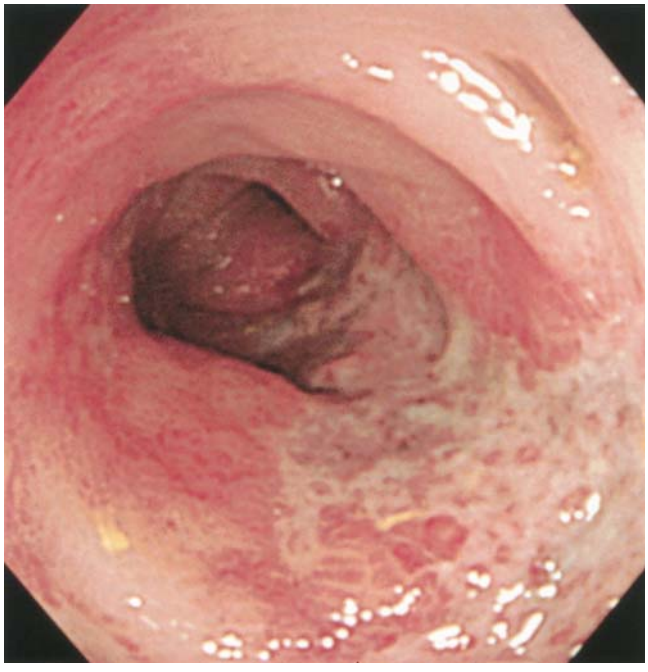

Fig. 1. Colonoscopy, showing a longitudinal ulcer demarcated from the surrounding mucosa, which was hyperemic and edematous

Our patient had no signs of vasculitis: she had normal fasting blood glucose levels, and no history suggesting coagulopathy, such as antiphospholipid syndrome. In addition, she had not taken any drugs before the onset of ischemic colitis. Although she had not taken any drugs such as oral contraceptives, her serum estrogen levels would have been elevated due to pregnancy. Thus, a relatively high level of circulating estrogens might have been involved in the ischemic episode in our patient.

It has been reported that chronic constipation is one of the risk factors for ischemic colitis.[8] Our patient usually had mild constipation. Although she had defecated 1 day before the onset of the ischemic episode, chronic constipation might have been involved in the illness.

We cannot clearly explain the reason that so few cases of ischemic colitis during pregnancy have been reported. The benign self-limited course of the disorder and the risk of abortion or teratism could make physicians hesitate to perform examinations such as colonoscopy or barium enema.

In treating pregnant women with abdominal pain and bloody diarrhea, ischemic colitis should be considered in the differential diagnosis. Colonoscopy and mucosal biopsy are effective methods of confirming the diagnosis of ischemic colitis.

Yasuo Okamoto, Masatoshi Fujii, Shinpei Tateiwa,
Toshiyuki Sakai, Fukashi Ochi, Masahiko Sugano,
Kiyoshi Oshiro, and Yoshinori Okabayashi
Department of Internal Medicine, Kobe Red Cross Hospital
(formerly Suma Red Cross Hospital), Wakinohama-Kaigandori,
Chuo-ku, Kobe 651-0073, Japan

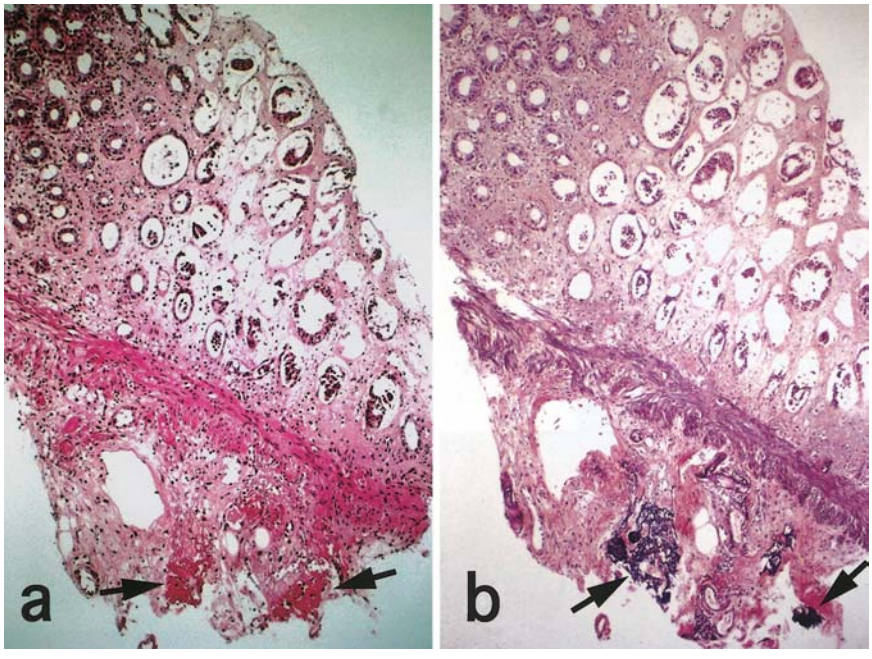

Fig. 2a,b. Mucosal biopsy sample, showing ghost-like cryptic necrosis with loss of the surface epithelium. Dilated capillaries and capillary thrombosis are present in the submucosal layer (*arrows*). **a** H&E ×200; **b** Phosphotungstic acid-hematoxylin (PTAH), ×200

Sakan Maeda
Department of Molecular Pathology, Kobe University Graduate School of Medicine, Kobe, Japan

References

1. Barcewicz PA, Welch JP. Ischemic colitis in young adult patients. Dis Colon Rectum 1980:23:109–14.
2. Deana DG, Dean PJ. Reversible ischemic colitis in young women: association with oral contraceptive use. Am J Surg Pathol 1995;19:454–62.
3. Newell AM, Deckert JJ. Transient ischemic colitis in young adults. Am Fam Physician 1997;15:1103–8.
4. Clark AW, Lloyd-Mostyn RH, Sadler MR. Ischemic colitis in young adults. BMJ 1972;14:70–2.
5. Frossard JL, Spahr L, Queneau PE, Armenian B, Brundler M-A, Hadengue A. Ischemic colitis during pregnancy and contraceptive medication. Digestion 2001;64:125–7.
6. Gurbuz AK, Gurbuz B, Salas L, Rosenshein NB, Donowitz M, Giardiello FM. Premarin-induced ischemic colitis. J Clin Gastroenterol 1994;19:108–11.
7. Bonnar J. Coagulation effects of oral contraception. Am J Obstet Gynecol 1987;157:1042–8.
8. Habu Y, Tahashi Y, Kiyota K, Matsumura K, Hirota M, Inokuchi H, et al. Reevaluation of clinical features of ischemic colitis. Scand J Gastroenterol 1996;31:881–6.

Received: January 14, 2003 / Accepted: June 13, 2003
Reprint requests to: M. Fujii
DOI 10.1007/s00535-003-1232-6

Hepatosplenic abscess due to candida infection effectively treated by the intraarterial injection of an antimycotic agent using an implanted reservoir

To the Editor: A mycotic infection constitutes a greave prognostic factor for patients in an immunodeficient state. A liver abscess is often refractory and can present serious clinical problems. We report a case of hepatosplenic abscess due to candida infection, which was effectively treated by the intraarterial injection of an antimycotic agent, using an implanted reservoir.

In May 1999, a 40-year-old man was admitted to our hospital with acute myelocytic leukemia, and complete remission was induced by two courses of idarubicin + cytarabine. During the subsequent consolidation therapy, he developed a fever (temperature of 39°C) on November 25 and right hypochondralgia on December 4, associated with a decreased white blood cell count (400/μl) and an increased C-reactive protein level (21.78 mg/dl). Hepatosplenomegaly was absent on physical examination, and serum levels of aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and γ-glutamyltransferase were normal. Abdominal computed tomography exhibited multiple low-density lesions in the liver and spleen (Fig. 1A). On December 17, the lesion was punctured, and milky white pus was collected, from which *Candida tropicalis* was identified. A diagnosis of hepatosplenic abscess due to candida infection was made, and intravenous administration of miconazole (800 mg/day) was started. Although the dose was raised to 1200 mg/day, the symptoms were not relived. Therefore, on January 4, 2000, intravenous infusion of 25 mg/day of amphotericin-B was initiated, but the fever and right hypochondralgia persisted. In addition, hypokalemia (2.1 mEq/l), an adverse effect of amphotericin-B, developed 2 weeks after the initiation of amphotericin-B treatment. On February 16, the drug delivery method was switched to an implanted reservoir. Hepatic arteriography showed a faint staining in the part corresponding to the liver abscess. Because a replaced right hepatic artery was recognized as an anatomical variation, this vessel and the gastroduodenal artery was embolized, by using coils, to alter the blood flow. The end of the catheter was occluded while side openings were created. Imaging, using an indwelling catheter, confirmed the position of both hepatic lobes and the spleen. After the intraarterial infusion of amphotericin-B (25 mg/day), the fever and right hypochondralgia abated. On March 7, the dose was reduced to 10 mg/day. The subsequent clinical course was satisfactory, and the hypokalemia was relieved. On March 18, the dose was further reduced and then maintained at 5 mg/day.

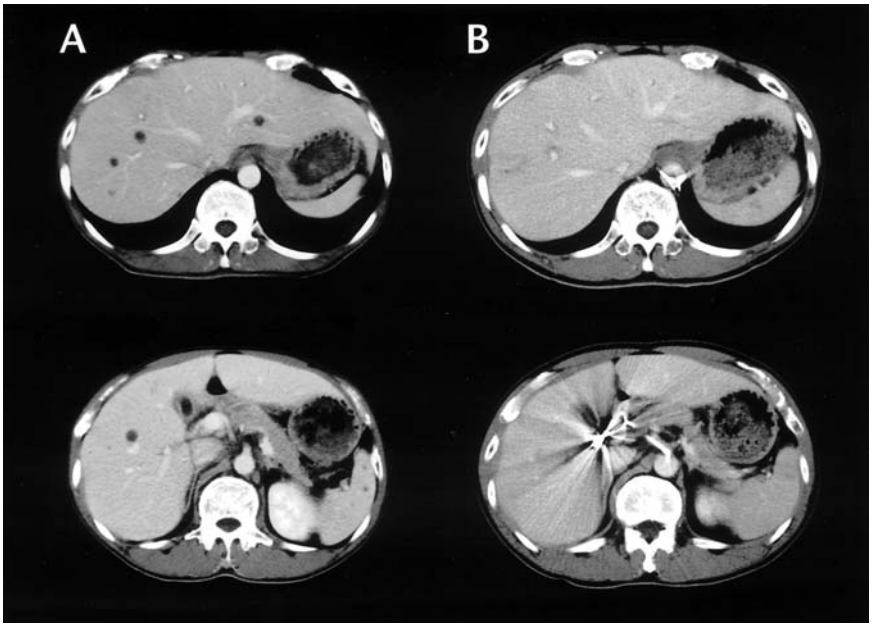

Fig. 1A,B. Enhanced computed tomography. **A** Before antifungal therapy, numerous small low-density areas were seen in the liver and spleen. **B** After a few months of treatment with intraarterial infusions of amphotericin-B, the low-density areas had disappeared