Ischemic enterocolitis examined by colonoscopy and selective angiography

Lei He, He-Sheng Luo

AIM: To study the value of colonoscopy and selective angiography in diagnosing ischemic enterocolitis.

METHODS: Among the 16 cases under study, 10 cases had hypertension and a history of coronary artery disease (one was hospitalized for sub-ventricular-wall infarction). The blood pressure of 10 of the 16 cases ranged from 13.9-23.8 to 13.3-14.6 kPa (170-180/100-110 mmHg). Two cases had chronic auricular fibrillation, and in four cases, a cardiogram showed left-front branch conduction block. Sixteen patients were examined by colonoscopy. Among them, 14 cases had a long course of angiocardiologic disease, and were further examined by selective mesenteric inferior angiography.

RESULTS: The colonoscopy revealed local mucous hyperemia edema and blood on contact. Lesions were found in the sigmoid colon in four cases, in the descending colon in eight cases and in splenic flexure in four cases, which suggests that the lesion always appeared in the left part of colon. Lesions had local mucous hyperemia, edema and blood on contact. Biopsy showed that there was inflammatory cell infiltration, submucous bleeding, edema, fibro-embolism and hemosiderosis in all 16 cases. Eight specimens showed hyperplasia of granular tissue and fibrosis. Selective mesenteric inferior angiography was performed in 14 cases. The results showed that there was fragmentation of the mesenteric inferior circulation in two patients. In the other 12 cases, the angiographic image was not complete and the ends of some branches even cannot be seen.

CONCLUSION: The colonoscopy and the selective mesenteric inferior angiography are both helpful in the diagnosis of ischemic enterocolitis.

© 2005 The WJG Press and Elsevier Inc. All rights reserved.

Key words: Ischemic enterocolitis; Colonoscopy; Selective angiography
in blood vessels 2 cm away from the inferior mesenteric artery and a lack of local circulation. There were several irregular collateral circulations toward the left part of colon along the superior fragmentation in three cases. In other cases, parts of the cavities of the inferior mesenteric artery became slim. There was obvious narrowing in primary part and related branches, so the radiographic image was not complete and the ends of some branches even cannot be seen.

**DISCUSSION**

Ischemic enterocolitis is caused by ischemia leading to colon wall mucous necrosis, exfoliation, bleeding or inflammation which produces abdominal pain, hematochezia, mucous stool with blood, and fever\(^{[3]}\). Non-characteristic symptoms make it extremely difficult to distinguish between enterocolitis, chronic ulcerative colitis, intestinal parasitosis and even early colon tumors. Four patients were diagnosed as having “enterocolitis” and “gastrointestinal tract infection” before they were referred to our hospital, where the diagnosis of “ischemic enterocolitis” was proven by colonoscopy. In the other 12 cases, four patients were diagnosed as having ischemic enterocolitis when they were first seen, and the others were hospitalized for “enterocolitis” and “gastrointestinal tract infection” or other diagnoses. All of their diagnoses were confirmed by colonoscopic examination and biopsy. Cases all showed a great deal of fibro-embolism and hemosiderosis, symptoms characteristic of the disease which may be used to distinguish it from other kinds of enterocolitis\(^{[2]}\).

Ischemic enterocolitis is induced by poor blood supply to the colon wall which results in ischemic change. One potent treatment is to vasodilate the surrounding blood vessels to recover blood supply. This acts to resolve the colon wall ischemia such as to reduce clinical manifestations, to reduce perforation, and to prevent colon wall necrosis such as to reduce clinical manifestations, to reduce perforation, and to prevent colon wall necrosis which occurs when local ischemia develops into general ischemia\(^{[5]}\). Sixteen patients were treated with papaverine, propranolol, and glucagon. After 2 days of medication, some patients felt better. Patients’ signs and symptoms essentially disappeared after 10-14 d medication. Patients were reinspected by colonoscopy after treatment to identify whether their colon wall lesion recovery was in line with their improved symptoms. Twelve patients’ initial ischemic colon wall and mucous were essentially recovered; the appearance of four patients’ colon wall mucous was normal. This suggests that symptoms disappear quickly and that lesions recover quickly after blood supply is improved. This fast recovery distinguishes ischemic enterocolitis from other inflammatory and non-specific intestinal diseases.

Because the local lumen was slim, biopsies were performed to rule out spasm causes, and the results showed that there was fibrous connective tissue hyperplasia in the biopsied tissue. Therefore, it is believed that the narrowing of the lumen was closely related to fibrous connective tissue hyperplasia. Pathologically, fibrous connective tissue hyperplasia in tissue reflects long-term ischemia. Whether or not the narrowing of the lumen is caused by fibrous connective tissue hyperplasia needs further observations. Eighty percent of ischemic enterocolitis patients are older than 50\(^{[6-10]}\). Ischemic enterocolitis is induced by poor blood supply to the colon wall which results in ischemic change. One potent treatment is to vasodilate the surrounding blood vessels to recover blood supply. This acts to resolve the colon wall ischemia such as to reduce clinical manifestations, to reduce perforation, and to prevent colon wall necrosis which occurs when local ischemia develops into general ischemia\(^{[5]}\). Sixteen patients were treated with papaverine, propranolol, and glucagon. After 2 days of medication, some patients felt better. Patients’ signs and symptoms essentially disappeared after 10-14 d medication. Patients were reinspected by colonoscopy after treatment to identify whether their colon wall lesion recovery was in line with their improved symptoms. Twelve patients’ initial ischemic colon wall and mucous were essentially recovered; the appearance of four patients’ colon wall mucous was normal. This suggests that symptoms disappear quickly and that lesions recover quickly after blood supply is improved. This fast recovery distinguishes ischemic enterocolitis from other inflammatory and non-specific intestinal diseases.

Because the local lumen was slim, biopsies were performed to rule out spasm causes, and the results showed that there was fibrous connective tissue hyperplasia in the biopsied tissue. Therefore, it is believed that the narrowing of the lumen was closely related to fibrous connective tissue hyperplasia. Pathologically, fibrous connective tissue hyperplasia in tissue reflects long-term ischemia. Whether or not the narrowing of the lumen is caused by fibrous connective tissue hyperplasia needs further observations. Eighty percent of ischemic enterocolitis patients are older than 50\(^{[6-10]}\). Ischemic enterocolitis is induced by poor blood supply to the colon wall which results in ischemic change. One potent treatment is to vasodilate the surrounding blood vessels to recover blood supply. This acts to resolve the colon wall ischemia such as to reduce clinical manifestations, to reduce perforation, and to prevent colon wall necrosis which occurs when local ischemia develops into general ischemia\(^{[5]}\). Sixteen patients were treated with papaverine, propranolol, and glucagon. After 2 days of medication, some patients felt better. Patients’ signs and symptoms essentially disappeared after 10-14 d medication. Patients were reinspected by colonoscopy after treatment to identify whether their colon wall lesion recovery was in line with their improved symptoms. Twelve patients’ initial ischemic colon wall and mucous were essentially recovered; the appearance of four patients’ colon wall mucous was normal. This suggests that symptoms disappear quickly and that lesions recover quickly after blood supply is improved. This fast recovery distinguishes ischemic enterocolitis from other inflammatory and non-specific intestinal diseases.

Because the local lumen was slim, biopsies were performed to rule out spasm causes, and the results showed that there was fibrous connective tissue hyperplasia in the biopsied tissue. Therefore, it is believed that the narrowing of the lumen was closely related to fibrous connective tissue hyperplasia. Pathologically, fibrous connective tissue hyperplasia in tissue reflects long-term ischemia. Whether or not the narrowing of the lumen is caused by fibrous connective tissue hyperplasia needs further observations. Eighty percent of ischemic enterocolitis patients are older than 50\(^{[6-10]}\). Ischemic enterocolitis is induced by poor blood supply to the colon wall which results in ischemic change. One potent treatment is to vasodilate the surrounding blood vessels to recover blood supply. This acts to resolve the colon wall ischemia such as to reduce clinical manifestations, to reduce perforation, and to prevent colon wall necrosis which occurs when local ischemia develops into general ischemia\(^{[5]}\). Sixteen patients were treated with papaverine, propranolol, and glucagon. After 2 days of medication, some patients felt better. Patients’ signs and symptoms essentially disappeared after 10-14 d medication. Patients were reinspected by colonoscopy after treatment to identify whether their colon wall lesion recovery was in line with their improved symptoms. Twelve patients’ initial ischemic colon wall and mucous were essentially recovered; the appearance of four patients’ colon wall mucous was normal. This suggests that symptoms disappear quickly and that lesions recover quickly after blood supply is improved. This fast recovery distinguishes ischemic enterocolitis from other inflammatory and non-specific intestinal diseases.

There were several irregular collateral circulations toward the left part of colon along the superior fragmentation in three cases. In other cases, parts of the cavities of the inferior mesenteric artery became slim. There was obvious narrowing in primary part and related branches, so the radiographic image was not complete and the ends of some branches even cannot be seen.

**REFERENCES**

1. Liu BY, Liu QM. Etiological factors and pathologenesis of ischemic enterocolitis. *Shijie Huairen Xiaohua Zazhi* 2001; 9: 1424-1425
2. Shi SS. Diagnosis of ischemic enterocolitis. *Shijie Huairen Xiaohua Zazhi* 2001; 9: 1425-1426
3. Wei ZB, Zhou YX, Liu N. The diagnosis and treatment of intestine function disorder during ischemic enterocolitis. *Shijie Huairen Xiaohua Zazhi* 2001; 9: 1428-1429
4. Greenwald DA, Brandt LJ. Colonic ischemia. *J Clin Gastroenterol* 1998; 27: 122-128
5 Caprilli R, Viscido A, Frieri G, Latella G. Acute colitis following colonoscopy. *Endoscopy* 1998; 30: 428-431

6 Levine JS, Jacobson ED. Intestinal ischemic disorders. *Dig Dis* 1995; 13: 3-24

7 Chang RY, Tsai CH, Chou YS, Wu TC. Nonocclusive ischemic colitis following glycerin enema in a patient with coronary artery disease. A case report. *Angiology* 1995; 46: 747-752

8 Qin GL, Zhang AG, Niu YH. The relation between ischemic enterocolitis and cardiovascular lesion. *Shijie Huaren Xiaohua Zazhi* 2001; 9: 1429-1430

9 Brandt LJ, Boley SJ. Colonic ischemia. *Surg Clin North Am* 1992; 72: 203-229

10 McKinsey JF, Gewertz BL. Acute mesenteric ischemia. *Surg Clin North Am* 1997; 77: 307-318

11 Ranschaert E, Verhille R, Marchal G, Rigauts H, Ponette E. Sonographic diagnosis of ischemic colitis. *J Belge Radiol* 1994; 77: 166-168

12 Parish KL, Chapman WC, Williams LF. Ischemic colitis. An ever-changing spectrum? *Am Surg* 1991; 57: 118-121

13 Cheng TG, Zhang Y. Image analysis of ischemic enterocolitis. *Shijie Huaren Xiaohua Zazhi* 2001; 9: 1426-1427

14 Caroline DF, Friedman AC. The radiology of inflammatory bowel disease. *Med Clin North Am* 1994; 78: 1353-1385

15 Wolf EL, Sprayregen S, Bakal CW. Radiology in intestinal ischemia. Plain film, contrast, and other imaging studies. *Surg Clin North Am* 1992; 72: 107-124