

# Diverticular Disease: Diagnosis and Treatment

HOLLY SALZMAN, M.D., and DUSTIN LILLIE, M.D.

University of California, San Diego, School of Medicine, San Diego, California

Diverticular disease refers to symptomatic and asymptomatic disease with an underlying pathology of colonic diverticula. Predisposing factors for the formation of diverticula include a low-fiber diet and physical inactivity. Approximately 85 percent of patients with diverticula are believed to remain asymptomatic. Symptomatic disease without inflammation is a diagnosis of exclusion requiring colonoscopy because imaging studies cannot discern the significance of diverticula. Fiber supplementation may prevent progression to symptomatic disease or improve symptoms in patients without inflammation. Computed tomography is recommended for diagnosis when inflammation is present. Antibiotic therapy aimed at anaerobes and gram-negative rods is first-line treatment for diverticulitis. Whether treatment is administered on an inpatient or outpatient basis is determined by the clinical status of the patient and his or her ability to tolerate oral intake. Surgical consultation is indicated for disease that does not respond to medical management or for repeated attacks that may be less likely to respond to medical therapy and have a higher mortality rate. Prompt surgical consultation also should be obtained when there is evidence of abscess formation, fistula formation, obstruction, or free perforation. (*Am Fam Physician* 2005;72:1229-34, 1241-2. Copyright © 2005 American Academy of Family Physicians.)



ILLUSTRATION BY CYNTHIA TURNER

► **Patient information:** A handout on diverticular disease, written by the authors of this article, is provided on page 1241.

**D**iverticular disease includes a spectrum of conditions sharing the underlying pathology of acquired diverticula of the colon. Because diverticular disease occurs almost exclusively in developed countries, it has been dubbed a “disease of Western Civilization.”<sup>1</sup>

Acquired diverticula form through the relative weakness in the muscle wall of the colon at the site where arteries (the vasa recta) penetrate the muscularis layer to reach the mucosa and submucosa. Diverticula generally are multiple. Each diverticulum is typically 5 to 10 mm in diameter, but at times they can exceed 20 mm. The most common site is the sigmoid colon, although diverticula can occur throughout the large bowel, with right-sided disease being more common in Asians and in patients younger than 60 years.<sup>2,3</sup> Vegetarians and others who consume large amounts of dietary fiber have a lower incidence of diverticula. Although the pathogenic mechanisms of diverticular disease are poorly understood, they are clearly related to complex interac-

tions of colon structure, intestinal motility, diet, and genetic features.<sup>4</sup>

The true prevalence of diverticula is unknown, but in one large observational study<sup>5</sup> of 9,086 consecutive patients undergoing colonoscopy for all indications, the overall prevalence of diverticulosis was 27 percent and increased with patient age. Studies performed in the 1970s suggest that the prevalence of diverticula may be as high as 60 percent in patients older than 80 years, with no clear gender predilection. Of patients with diverticula, 80 to 85 percent are believed to remain asymptomatic. Three fourths of the remaining 15 to 20 percent of patients have symptomatic diverticular disease with colicky abdominal pain but no inflammation. The remaining one fourth (or approximately 5 percent of all patients with diverticula) develop diverticulitis, and a small number will develop complications of diverticulitis such as abscess formation, fistulas, obstruction, or hemorrhage.<sup>6</sup> *Table 1* compares the various diverticular syndromes.

**SORT: KEY RECOMMENDATIONS FOR PRACTICE**

<i>Clinical recommendation</i>	<i>Evidence rating</i>	<i>References</i>
Patients with asymptomatic diverticulosis should eat a high-fiber diet to prevent symptomatic diverticular disease.	C	6
All patients with symptomatic diverticular disease should undergo colonoscopy to exclude underlying neoplasm.	C	6
Patients with suspected diverticulitis should undergo computed tomography with intravenous and oral contrast rather than other diagnostic modalities such as endoscopy or contrast radiography.	C	13, 17
To provide adequate coverage of gram-negative rods and anaerobic bacteria, patients with acute diverticulitis treated as outpatients should receive metronidazole (Flagyl) combined with a quinolone or with trimethoprim-sulfamethoxazole (Bactrim, Septra) or amoxicillin-clavulanate (Augmentin).	C	6
Patients hospitalized with acute diverticulitis should receive metronidazole or clindamycin (Cleocin) combined with an aminoglycoside, a monobactam, or a third-generation cephalosporin.	C	6

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see page 1154 or <http://www.aafp.org/afpsort.xml>.

**Diverticulosis**

Diverticulosis is an anatomic diagnosis that describes the presence of one or more diverticula. Uncomplicated, asymptomatic diverticulosis typically is diagnosed incidentally and does not require further work-up. Consensus guidelines<sup>6</sup> recommend a high-fiber diet in these patients to prevent symptomatic diverticular disease, although there are no randomized controlled trials to support or refute this strategy.

**Symptomatic Diverticular Disease**

Symptomatic diverticular disease, also known as symptomatic diverticulosis, is characterized by nonspecific attacks of abdominal pain without evidence of an inflammatory process. This pain typically is colicky in nature, but can be steady, and often is relieved by passing flatus or having a bowel movement. Patients often note that attacks are precipitated by eating. Bloating and changes in bowel habits also can occur, and constipation is more common than diarrhea. Fullness or tenderness in the left lower quadrant, or occasionally a tender palpable loop of sigmoid colon, often is appreciated on physical examination. These nonspecific symptoms overlap considerably with those of irritable bowel syndrome; disorders of inhibitory control of neuromuscular function may play a role in the development of both diseases.<sup>7</sup>

**PATHOGENESIS**

For unknown reasons, only a small percentage of patients with diverticulosis develop symptomatic diverticular disease. Some researchers have proposed that more painful disease is associated with an increased motility index and increased intraluminal pressure.<sup>8</sup> Prospective studies<sup>9,10</sup> in men have shown no connection between symptomatic

diverticular disease and smoking, caffeine, or alcohol intake, although lack of physical activity appears to play at least an indirect role. In a large prospective study<sup>11</sup> of 43,881 U.S. male health care professionals, an inverse relationship was noted between the consumption of insoluble dietary fiber and the development of symptomatic diverticular disease.

**DIAGNOSIS**

The diagnosis of symptomatic diverticular disease is one of exclusion in patients with diverticula (*Table 2*). Traditionally, diverticula are identified by barium enema, and additional investigations are performed as indicated to rule out other potential causes of symptoms in individual patients. Although computed tomography (CT) has been used commonly in the investigation of diverticular disease, the morphology of diverticular segments makes the diagnosis of underlying cancer difficult even with ideal technique.<sup>12</sup> Consensus expert opinion recommends colonoscopy in all patients who present with symptomatic diverticular disease to exclude underlying neoplastic disease.<sup>6</sup> CT colonography is a promising modality that may supplant colonoscopy once further data demonstrating its effectiveness are available.

**MANAGEMENT**

Several uncontrolled studies<sup>11</sup> suggest that fiber supplementation benefits patients with symptomatic diverticular disease, but no large placebo-controlled trials have been performed. Nevertheless, a high-fiber diet, fiber supplementation, or both generally are recommended and are likely to do no harm. Historically, physicians have recommended avoiding ingestion of seeds, corn, popcorn, and nuts for fear that they might become entrapped in

**TABLE 1**  
**Comparison of Diverticular Syndromes**

<i>Disease</i>	<i>Features</i>	<i>Treatment</i>
Asymptomatic	Diverticula in the absence of clinical symptoms	High-fiber diet
Symptomatic	Diverticula and abdominal pain, with or without change in bowel habits; no inflammation	High-fiber diet
Diverticulitis: uncomplicated (in stable patients)	Abdominal pain, fever, leukocytosis; able to tolerate oral fluids	Oral antibiotics (to cover anaerobes and gram-negative rods); clear liquid diet; avoid morphine (Duramorph) if possible because of risk of increasing intracolonic pressure
Diverticulitis: uncomplicated (in older or ill patients)	Abdominal pain, fever, leukocytosis; able to tolerate oral fluids, or patient is older than 85 years	IV antibiotics (to cover anaerobes and gram-negative rods); IV fluids; bowel rest, nothing by mouth; meperidine (Demerol)
Diverticulitis: complicated	Abdominal pain, fever, leukocytosis; with or without sepsis, perforation, abscess, fistula, obstruction	Stabilization with fluids and antibiotics; surgical consultation; percutaneous drainage

IV = intravenous.

diverticula, but no controlled studies support this dietary restriction. Similarly, no evidence supports the use of antispasmodic agents, despite the cramping and bloating often associated with symptomatic diverticular disease.<sup>3</sup>

### Diverticulitis

Classically, diverticulitis is characterized by acute, constant abdominal pain most often occurring in the left lower quadrant. The location varies depending on the site of the involved diverticulum. Fever and leukocytosis generally are present. Other commonly associated symptoms include nausea, vomiting, and constipation or diarrhea. Some patients may complain of dysuria and frequency, reflecting what has been called “sympathetic cystitis” induced by bladder irritation from the adjacent inflamed colon. On physical examination, localized tenderness generally is found in the left lower quadrant and may be associated with guarding and rebound tenderness. Right-sided pain, however, does not preclude diverticulitis because some patients have redundant sigmoid colons, and right-sided diverticula can occur, particularly in Asian populations. Bowel sounds often are decreased but may be normal early in the condition or increased in the presence of obstruction. Hematochezia is rare and should suggest other diagnoses.<sup>3</sup>

### PATHOGENESIS

Diverticulitis is believed to develop as the result of a micro- or macro-perforation of a diverticulum, which may be caused by erosion of the luminal wall by increased intraluminal pressure or thickened fecal material in the neck of the diverticulum. After a micro-perforation, infection generally is contained by pericolic fat, mes-

entery, or adjacent organs, and a localized phlegmon occurs. With macro-perforation, the resultant infection is less restricted, and peritonitis or a pericolic abscess can occur. If this septic process erodes into adjacent structures, it may result in fistula formation.

### DIAGNOSIS

The diagnosis of diverticulitis is suspected most often on the basis of clinical history and physical examination. Laboratory studies and imaging can be used judiciously to confirm a diagnosis of diverticulitis and to exclude other potential causes of similar symptoms (*Table 2*). The white blood cell count usually is elevated with a predominance of polymorphonuclear cells. Immature band forms may be present. An acute abdominal radiographic series should be obtained in all patients with significant abdominal pain

**TABLE 2**  
**Differential Diagnosis of Symptomatic Diverticular Disease and Diverticulitis**

Acute appendicitis	Ovarian cyst, abscess, or neoplasm
Colorectal cancer	Ovarian torsion
Complicated ulcer disease	Pancreatic disease
Crohn's disease	Pelvic inflammatory disease
Cystitis	Peritonitis
Ectopic pregnancy	Pseudomembranous colitis
Gallbladder disease	Renal disease
Incarcerated hernia	Small bowel obstruction
Ischemic colitis	Ulcerative colitis
Mesenteric infarction	

## Diverticular Disease

and suspected diverticulitis to identify pneumoperitoneum if macro-perforation has occurred. Otherwise, findings often are nonspecific and include small or large bowel dilation or ileus, or evidence of bowel obstruction.

CT with intravenous and oral contrast is the test of choice to confirm a suspected diagnosis of diverticulitis.<sup>13</sup> The finding of pericolic fat infiltration (*Figure 1*) is diagnostic.<sup>14</sup> Other common findings include thickened fascia (78.9 percent), muscular hypertrophy (26.3 percent), and an “arrowhead sign” (23.7 percent). The arrowhead sign consists of focal thickening of the colonic wall with an arrowhead-shaped lumen pointing to the inflamed diverticula (*Figure 2*).<sup>14</sup> In one large prospective study,<sup>15</sup> investigators demonstrated the ability of CT to accurately predict medical treatment failure or risk of complications by determining the presence of severe disease (abscess or extraluminal air or contrast). Although the sensitivity is excellent (97 percent), a normal CT does not preclude the diagnosis.

Uncommonly, ultrasonography is used to confirm the diagnosis, primarily in patients with right-sided pain in whom other diseases (e.g., ovarian pathology) are suspected. In such cases, ultrasonography reveals a hypoechoic or anechoic structure protruding from a segmentally thickened colonic wall. Ultrasonography was found to have a specificity of 99.8 percent in one large retrospective study.<sup>16</sup>

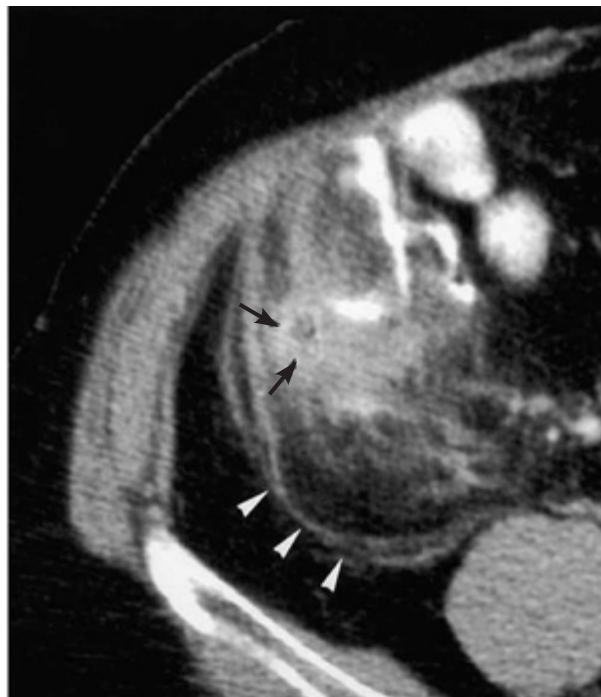
Although barium enema was used commonly in the past, it is no longer recommended because of the risk of extravasation of contrast material if perforation has occurred. If barium enema is performed, water-soluble contrast should be used. Typical findings include spiculation of the mucosa, spasm, abscess, or evidence of frank perforation.

### The Authors

HOLLY SALZMAN, M.D., is associate professor in the Department of Family and Preventive Medicine at the University of California, San Diego (UCSD), School of Medicine. She received her medical degree from the University of California, Los Angeles, School of Medicine, and completed a residency in family medicine at Long Beach (Calif.) Memorial Medical Center.

DUSTIN LILLIE, M.D., is a faculty member in the Department of Family and Preventive Medicine at the UCSD School of Medicine. He received his medical degree from the Keck School of Medicine at the University of Southern California, Los Angeles, and completed a residency in family medicine at the UCSD School of Medicine.

Address correspondence to Holly Salzman, M.D., UCSD Scripps Ranch Family Medicine, 9909 Mira Mesa Blvd., Suite 200, San Diego, CA 92131 (e-mail: hsalzman@ucsd.edu). Reprints are not available from the authors.



**Figure 1.** Computed tomographic scan revealing marked eccentric thickening of the wall of the ascending colon and pericolic inflammatory fat and fascia (*white arrowheads*). Fecal material is seen at the center of the inflammatory complex indicating an inflamed diverticulum (*black arrows*).

Reprinted with permission from Shen SH, Chen JD, Tiu CM, Chou YH, Chang CY, Yu C. Colonic diverticulitis diagnosed by computed tomography in the ED. *Am J Emerg Med* 2002;20:552.

Because of the theoretic potential to exacerbate perforation, endoscopic examination is acutely contraindicated unless inflammatory bowel disease, ischemic colitis, or carcinoma is highly suspected.<sup>17</sup> Colonoscopy always should be performed six to eight weeks after recovery from acute diverticulitis to exclude coexisting neoplastic disease.

### MANAGEMENT

The severity of the inflammatory and infectious processes, as well as the underlying health of the patient, determines the appropriate treatment for patients with diverticulitis. In patients with uncomplicated diverticulitis who are clinically stable and able to tolerate fluids, outpatient treatment with broad-spectrum antibiotics covering anaerobes and gram-negative rods is appropriate (*Table 3<sup>6</sup>*). Common choices are metronidazole (Flagyl) plus a quinolone; metronidazole plus trimethoprim-sulfamethoxazole (Bactrim, Septra); or amoxicillin-clavulanic acid (Augmentin).<sup>6</sup> Patients also should follow a clear liquid diet. Morphine (Duramorph) should be avoided if possible because of its propensity to increase intracolonic pressure.<sup>3,10</sup> Patients should improve within 48 to 72 hours, at which time the diet may be advanced cautiously. Close follow-up is recommended, and hospitalization should be considered if the patient experiences



**Figure 2.** Computed tomographic scan showing eccentric thickening of the colon wall with arrowhead-shaped lumen contrasted by air (arrow).

Reprinted with permission from Shen SH, Chen JD, Tiu CM, Chou YH, Chang CY, Yu C. Colonic diverticulitis diagnosed by computed tomography in the ED. *Am J Emerg Med* 2002;20:553.

increasing pain, fever, or an inability to tolerate fluids. Antibiotic treatment should be continued for seven to 10 days.

Hospitalization is recommended if patients show signs of significant inflammation, are unable to take oral fluids, are older than 85 years, or have significant comorbid conditions. These patients should be placed on bowel rest and treated with intravenous fluids and intravenous antibiotics (Table 3<sup>6</sup>). Appropriate choices, based on expert consensus rather than randomized trials, include anaerobic coverage with metronidazole or clindamycin (Cleocin) and gram-negative coverage with an aminoglycoside, a monobactam, or a third-generation cephalosporin.<sup>6</sup> Single-agent coverage with intravenous second-generation cephalosporins (i.e., cefoxitin [Mefoxin] or cefotetan [Cefotan]), or beta-lactamase inhibitor combinations such as ampicillin-sulbactam (Unasyn) or ticarcillin-clavulanate (Timentin) also have been advocated.<sup>3</sup> Meperidine (Demerol) is the opiate of choice because it has been shown to decrease intraluminal pressure. Nasogastric suction is not indicated unless there is significant ileus. As with patients receiving outpatient therapy, hospitalized patients should improve within 48 to 72 hours. The diet then can be advanced, and the patient discharged to complete a seven- to 10-day course of oral antibiotics.

Most patients with acute diverticulitis respond to conservative medical management, although 15 to 30 percent may require surgery during hospital admission because of lack of response to treatment or because of development of complications.<sup>18</sup> Surgery typically is not indicated after an uncomplicated first episode because only 7 to 35 percent of patients experience a recurrent episode. After a second episode, the prob-

ability of a third episode surpasses 50 percent, and subsequent attacks are less likely to respond to medical therapy and also have a higher mortality rate.<sup>19</sup> Surgical referral should be considered for patients with recurrent episodes or aggressive disease at a young age.

Several surgical options are available, including resection with primary anastomosis, resection with colostomy and closure of the rectal stump, transverse colostomy and drainage, and laparoscopic colectomy. The decision about which option to choose is best guided by an experienced surgeon and the patient's preference.

### COMPLICATIONS

Complications of diverticulitis include abscess, fistula, bowel obstruction, and free perforation. These complications all require surgical consultation.

Abscesses occur when the pericolic tissues fail to control the spread of the inflammatory process. Abscess formation should be suspected when fever, leukocytosis, or both persist despite an adequate trial of appropriate antibiotics. A tender mass may be palpable on physical examination. If right upper quadrant pain or elevated transaminase levels occur, evidence of a pyogenic liver

**TABLE 3**  
**Consensus Antibiotic Treatment for Patients with Diverticulitis**

#### Outpatient

Amoxicillin-clavulanate (Augmentin)  
Trimethoprim-sulfamethoxazole (Bactrim, Septra) and metronidazole (Flagyl)  
Fluoroquinolone and metronidazole

#### Inpatient

Metronidazole or clindamycin (Cleocin)  
*plus*  
Aminoglycoside (gentamicin [Garamycin] or tobramycin [Tobrex])  
*or*  
Monobactam (aztreonam [Azactam])  
*or*  
Third-generation cephalosporin (ceftriaxone [Rocephin], ceftazidime [Fortaz], cefotaxime [Claforan])  
*alternatively*  
Second-generation cephalosporin (cefoxitin [Mefoxin], cefotetan [Cefotan])  
Beta-lactamase inhibitor combinations (ampicillin-sulbactam [Unasyn], ticarcillin-clavulanate [Timentin])

Information from reference 6.

## Diverticular Disease

abscess should be sought. CT-guided percutaneous drainage may be appropriate for small abscesses or while patients with sepsis are being stabilized for surgery.<sup>3,6</sup>

Peridiverticular abscesses can progress to form fistulas between the colon and surrounding structures in up to 10 percent of patients. Colovesical fistulas are the most common variety and require surgery for treatment. Fistulas involving the bladder are more common in men; in women, the uterus is interposed between the colon and the bladder.

Intestinal obstruction is uncommon in diverticulitis, occurring in approximately 2 percent of patients. The small bowel is affected most often, and obstruction usually is caused by adhesions. The colon can become obstructed because of luminal narrowing caused by inflammation or compression by an abscess. Multiple attacks can lead to progressive fibrosis and stricture of the colonic wall. Obstruction generally is self-limited and responds to conservative therapy. If persistent, obstruction of the colon can be treated by a variety of endoscopic and surgical techniques.

Free perforation with peritonitis is rare, but it carries a mortality rate as high as 35 percent and requires urgent surgical consultation. If generalized peritonitis develops, the mortality rate is even higher. Perforation has been linked to nonsteroidal anti-inflammatory drug (NSAID) use in case-control studies.<sup>20</sup> Glucocorticoids may increase this risk. Steroids also may mask symptoms and delay appropriate therapy. Because of this, NSAIDs and glucocorticoids should be used with caution in patients who have known diverticular disease.

### Diverticular Hemorrhage

Diverticular hemorrhage is a possible complication of diverticulosis, and it is the most common cause of major lower gastrointestinal (GI) bleeding. It is arterial in nature and is attributed to medial thinning of the vasa recta as they cross over the dome of a diverticulum. In as many as 16 percent of patients with diverticular hemorrhage, bleeding may be the first sign of diverticular disease and is abrupt, voluminous, and painless in onset. The diagnosis and treatment of lower GI bleeds require a coordinated approach. Following fluid resuscitation, angiography, nuclear bleeding scans, and colonoscopy may be therapeutically useful in patients with ongoing bleeding. Surgery may be required for patients in whom medical management is unsuccessful.<sup>6</sup>

Author disclosure: Nothing to disclose.

Members of various family medicine departments develop articles for "Practical Therapeutics." This article is one in a series coordinated by the Department of Family and Preventive Medicine at the University of California, San Diego. The coordinator of the series is Tyson Ikeda, M.D.

### REFERENCES

1. Painter NS, Burkitt DP. Diverticular disease of the colon, a 20th century problem. *Clin Gastroenterol* 1975;4:3-21.
2. Farrell RJ, Farrell JJ, Morrin MM. Diverticular disease in the elderly. *Gastroenterol Clin North Am* 2001;30:475-96.
3. Reisman Y, Ziv Y, Kravrovitc D, Negri M, Wolloch Y, Halevy A. Diverticulitis: the effect of age and location on the course of disease. *Int J Colorectal Dis* 1999;14:250-4.
4. Simpson J, Scholefield JH, Spiller RC. Pathogenesis of colonic diverticula. *Br J Surg* 2002;89:546-54.
5. Loffeld RJ, Van Der Putten AB. Diverticular disease of the colon and concomitant abnormalities in patients undergoing endoscopic evaluation of the large bowel. *Colorectal Dis* 2002;4:189-92.
6. Stollman NH, Raskin JB. Diagnosis and management of diverticular disease of the colon in adults. *Am J Gastroenterol* 1999;94:3110-21.
7. Camilleri M, Lee JS, Viramontes B, Bharucha AE, Tangalos EG. Insights into the pathophysiology and mechanisms of constipation, irritable bowel syndrome, and diverticulosis in older people. *J Am Geriatr Soc* 2000;48:1142-50.
8. Cortesini C, Pantalone D. Usefulness of colonic motility study in identifying patients at risk for complicated diverticular disease. *Dis Colon Rectum* 1991;34:339-42.
9. Aldoori WH, Giovannucci EL, Rimm EB, Wing AL, Trichopoulos DV, Willett WC. A prospective study of alcohol, smoking, caffeine, and the risk of symptomatic diverticular disease in men. *Ann Epidemiol* 1995;5:221-8.
10. Aldoori WH, Giovannucci EL, Rimm EB, Ascherio A, Stampfer MJ, Colditz GA, et al. Prospective study of physical activity and the risk of symptomatic diverticular disease in men. *Gut* 1995;36:276-82.
11. Aldoori WH, Giovannucci EL, Rockett HR, Sampson L, Rimm EB, Willett WC. A prospective study of dietary fiber types and symptomatic diverticular disease in men. *J Nutr* 1998;128:714-9.
12. Halligan S, Saunders B. Imaging diverticular disease. *Best Pract Res Clin Gastroenterol* 2002;16:595-610.
13. Simmang CL, Shires GT. Diverticular disease of the colon. In: Feldman M, Friedman LS, Sleisenger MH, eds. *Sleisenger and Fordtran's Gastrointestinal and liver disease: pathophysiology, diagnosis, management*. 7th ed. Philadelphia: Saunders, 2002:2100-12.
14. Shen SH, Chen JD, Tiu CM, Chou YH, Chang CY, Yu C. Colonic diverticulitis diagnosed by computed tomography in the ED. *Am J Emerg Med* 2002;20:551-7.
15. Ambrosetti P, Grossholz M, Becker C, Terrier F, Morel P. Computed tomography in acute left colonic diverticulitis. *Br J Surg* 1997;84:532-4.
16. Chou YH, Chiou HJ, Tiu CM, Chen JD, Hsu CC, Lee CH, et al. Sonography of acute right side colonic diverticulitis. *Am J Surg* 2001;181:122-7.
17. Wilcox CM. Miscellaneous inflammatory diseases of the intestine. In: Cecil RL, Goldman L, Bennett JC, eds. *Cecil Textbook of medicine*. 21st ed. Philadelphia: Saunders, 2000:729-32.
18. Ferzoco LB, Raptopoulos V, Silen W. Acute diverticulitis. *N Engl J Med* 1998;338:1521-6.
19. Munson KD, Hensien MA, Jacob LN, Robinson AM, Liston WA. Diverticulitis. A comprehensive follow-up. *Dis Colon Rectum* 1996;39:318-22.
20. Goh H, Bourne R. Non-steroidal anti-inflammatory drugs and perforated diverticular disease: a case-control study. *Ann R Coll Surg Engl* 2002;84:93-6.