Diverticular disease guidelines

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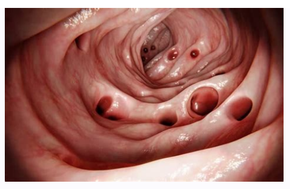
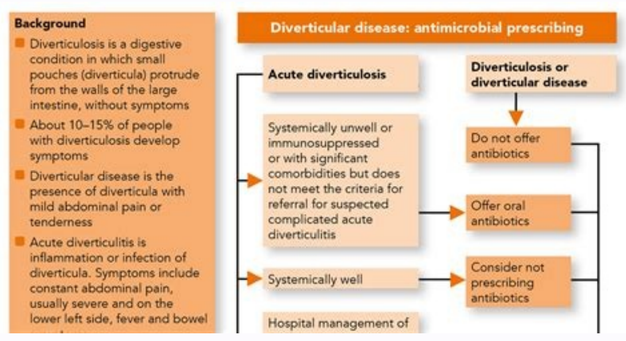
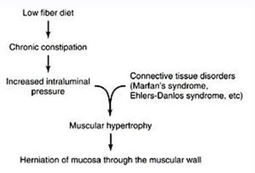
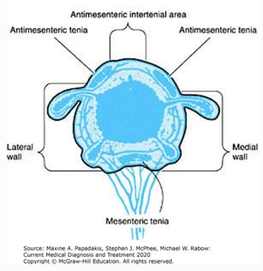


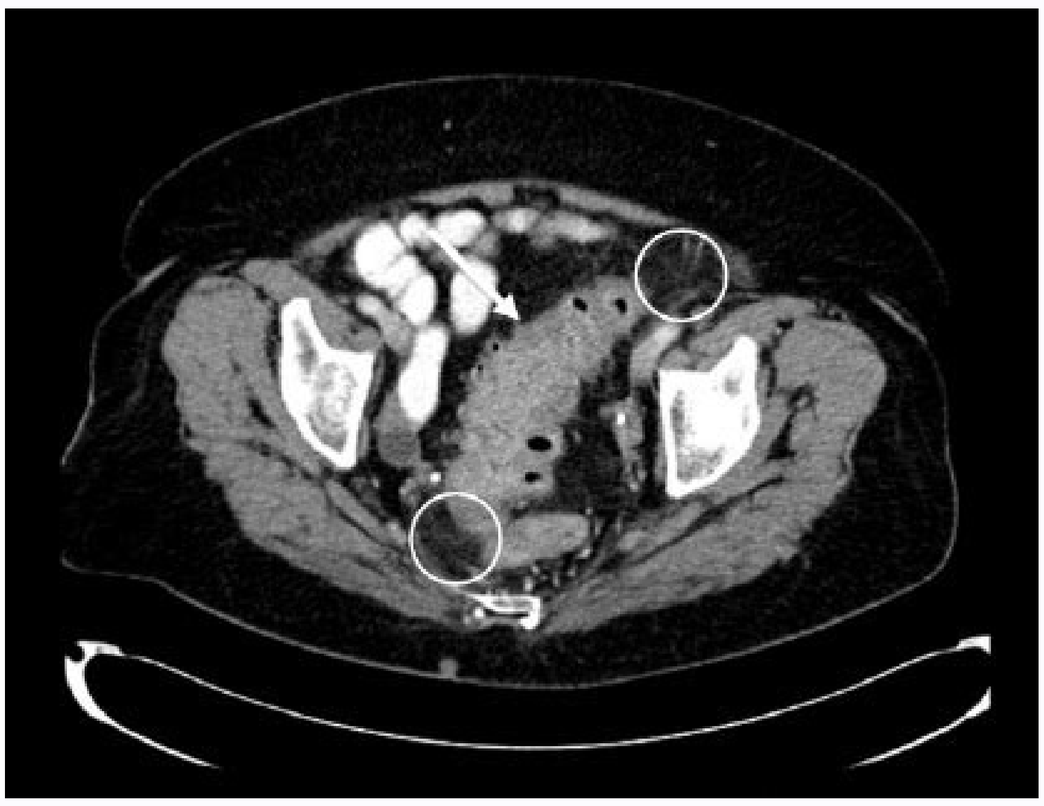






Epidemiología

- La prevalencia de esta patología es difícil de evaluar
  - La mayoría de los estudios han incluido a personas sintomáticas
  - La mayor parte de las personas que la presentan son asintomáticas y el diagnóstico se realiza en forma incidental, en el curso de estudios de imágenes y endoscópicos.
- Prevalencia según la edad
  - 40 años → 5%
  - 60 años → 30%
  - 80 años → 65%
- Prevalencia según el género
  - < 50 años → Más común en el sexo masculino
  - 50–70 años → Leve preponderancia femenina
  - > 70 años → Más común en el sexo femenino



Diverticular disease management guidelines. Diverticular disease guidelines aga. Colonic diverticular disease guidelines. Diverticular disease treatment guidelines. Diverticular disease guidelines australia. Diverticular disease guidelines nice. Diverticular disease guidelines 2021. Diverticular disease guidelines 2019.

World Gastroenterology Organisation Practice GuidelinesDr. T. Murphy Prof. R.H. Hunt Prof. M. Fried Dr. J.H. Krabshuis(Click to expand section) Diverticulum: A sac-like protrusion of mucosa through the muscular colonic wall [1]. Protrusion occurs in weak areas of the bowel wall through which blood vessels can penetrate. Typically 5–10 mm in size. Diverticula are really pseudodiverticular (false diverticula), as they contain only mucosa and submucosa covered by serosa. Diverticular disease consists of: Diverticulosis: the presence of diverticula within the colon Diverticulitis: inflammation of a diverticulum Diverticular bleeding Types of diverticular disease: Simple (75%), with no complications Complicated (25%), with abscesses, fistula, obstruction, peritonitis, and sepsis Prevalence by age [1]: Age 40: 5% Age 60: 30% Age 80: 65% Prevalence by sex: Age < 50: more common in males Age 50–70: slight preponderance in women Age > 70: more common in women Diverticular disease in the young (< 40) Diverticular disease is far more frequent in older people, with only 2–5% of cases occurring in those under 40 years of age. In this younger age group, diverticular disease occurs more frequently in males, with obesity being a major risk factor (present in 84–96% of cases) [2,3]. The diverticula are usually located in the sigmoid and/or descending colon. Management of this subset of diverticular disease patients remains somewhat controversial. The concept of diverticular disease being a more virulent condition in the young remains widely debated. The natural history still shows a trend towards recurrent symptoms [4] and an increased incidence of poor outcomes ultimately requiring surgery [5]. Surgery is often the treatment of choice for young symptomatic patients (approximately 50% compared with 30% for all patients). In young patients with no co-morbid conditions, elective surgery after a single episode of diverticulitis is still a reasonable recommendation. Low alimentary fiber was first described as a possible etiologic agent for the development of diverticular disease by Painter and Burkitt in the late 1960s [6,7]. Although this initially met with some resistance, its role in the condition was demonstrated by publications such as the Health Care Professionals Follow-Up Study [8]. The relative risk of developing diverticular disease is 0.58 for men with low alimentary fiber. Diverticular disease is less common in vegetarians [9]. The present theory that fiber is a protective agent against the development of diverticula and subsequent diverticulitis holds that insoluble fiber causes the formation of more bulky stool, which leads to decreased effectiveness in colonic segmentation. The overall result is that intracolonic pressure remains close to the normal range during colonic peristalsis [1,10]. Development of diverticular disease. There is no evidence of a relationship between the development of diverticula and smoking, caffeine, and alcohol consumption. However, an increased risk of developing diverticular disease is associated with a diet that is high in red meat and total fat content. This risk can be reduced by a diet high in fiber content, especially if it is of a cellulose origin (fruits and vegetables) [11]. Risk of complications. Complicated diverticular disease has been noted with increased frequency in patients who smoke, use non-steroidal anti-inflammatory drugs and acetaminophen (especially paracetamol) and those who are obese and have low-fiber diets [12]. Complicated diverticular disease is not more common in patients who drink alcohol or caffeinated beverages. Location of diverticular disease. The most typical form is a pseudodiverticulum or pulsion diverticulum (a diverticulum does not contain all the layers of the colonic wall; the mucosa and submucosa herniate through the muscle layer and are covered by serosa). There are four well-defined points around the circumference of the bowel at which the vasa recta penetrate the circular muscle layer. The vessels enter the wall on each side of the mesenteric teniae and on the mesenteric border of two antimesenteric teniae. Diverticula do not form distal to the rectosigmoid junction, below which the teniae coalesce to form a longitudinal muscle layer. Distribution [1]. There is sigmoid involvement in 95% of cases; involvement of the sigmoid alone in 65%; involvement of the entire colon in 7%; and the diverticulum is located near the sigmoid (but with a normal sigmoid) in 4% of cases. Natural history. Diverticulosis is symptomatic in 70% of cases; leads to diverticulitis in 15–25%; and is associated with bleeding in 5–15% [1]. The vasa recta penetrating the bowel wall create areas of weakness through which a portion of the colonic mucosa and submucosa (covered by serosa) can herniate. Segmentation can occur as a result of raised intracolonic pressure in certain areas of the colon. Such segmentation represents strong muscular contractions of the colonic wall, which serve to propel the luminal content or to halt the passage of material. Individual chamber pressures are temporarily elevated above those found when the colonic lumen is unsegmented. Segmentation in diverticulosis is exaggerated, causing occlusion of both “ends” of the chamber and resulting in high intra-chamber pressures [1]. The sigmoid is commonly affected, probably due to its small diameter. Laplace’s law explains their development, with the equation P = kT/R. Most complications are therefore also located in this area. In the sigmoid and other segments, the bowel becomes non-compliant in diverticular disease through several mechanisms: Mycosis — thickened circular muscle layer, shortening of the teniae, and luminal narrowing. Elastin — increased elastin deposition between muscle cells and teniae coli. Elastin is also laid down in a contracted form that causes shortening of THE teniae and bunching of THE circular muscle. Collagen — connective-tissue diseases such as Ehlers–Danlos syndrome, Marfan’s syndrome, and autosomal-dominant polycystic kidney disease result in structural changes in the bowel wall, leading to decreased resistance of the wall to intraluminal pressures and thus allowing protrusion of diverticula. Diverticulitis This term represents a spectrum of inflammatory changes, ranging from subclinical local inflammation to generalized peritonitis with free perforation. The mechanism of developing diverticulitis centers around the perforation of a diverticulum, whether it be microscopic or macroscopic. The old luminal obstruction concept is probably a rare occurrence. Increased intraluminal pressure or inspissated food particles may erode the diverticular wall with resultant inflammation and focal necrosis, leading to perforation (micro/macro). The clinical manifestation of the perforation depends on the size of the perforation and how vigorously it is walled off by the body. Perforations that are well controlled result in the formation of an abscess, while incomplete localization may present with free perforation. Simple diverticulitis: 75% of cases Complicated diverticulitis: 25% of cases (abscess, fistula, or perforation) Diagnosis. The majority of patients have left lower quadrant pain. An element of rebound tenderness implies some degree of peritoneal involvement. Fever and leukocytosis are other important but nonspecific findings. Examination. The examination may be relatively unremarkable, but most commonly reveals abdominal tenderness or a mass. Urinary symptoms may further suggest a pelvic phlegmon. Differential diagnosis: Carcinoma of the bowel — pyelonephritis Inflammatory bowel disease — appendicitis Ischemic colitis Irritable bowel syndrome Pelvic inflammatory disease Investigations: Chest/abdominal radiography usually shows no specific findings for diverticular disease, but a pneumoperitoneum can be seen in 11% of patients with acute diverticulitis. The abdominal radiograph is found to be abnormal in 30–50% of patients with acute diverticulitis. The most common findings include: — Small-bowel and large-bowel dilation or ileus — Bowel obstruction — Soft-tissue densities suggestive of abscesses [13,14] A diagnosis that is made solely on a clinical basis will be incorrect in 33% of cases. From the investigational standpoint, computed tomography (CT) is better than ultrasound. Diverticulitis is often regarded as a predominantly extraluminal disorder, and CT offers the benefit of evaluating both the bowel and the mesentery with a sensitivity of 69–98% and a specificity of 75–100%. The CT findings most commonly noted in acute diverticulitis include: Thickening of the bowel wall Streaky mesenteric fat Associated abscess [1] In a series of 42 patients with diverticulitis, the following CT findings were noted [15]: Inflamed periodic fat: 98% Diverticula: 84% Thickened bowel wall: 70% Pericolic abscess: 35% Peritonitis: 16% Fistula: 14% Colonic obstruction: 12% Intramural sinus tracts: 9% Other investigations: Ultrasound findings may include thickening of the colonic wall and cystic masses. Contrast enema: the use of a contrast enema in the acute setting is mainly reserved for situations in which the diagnosis is not clear. The enema has a sensitivity of 62–94%, with a false-negative rate of 2–15%. Meglumine diatrizoate is a hyperosmolar contrast agent that may assist in relieving partial obstruction if present. Endoscopy, proctosigmoidoscopy, flexible sigmoidoscopy. The use of endoscopy, involving air insufflation, is relatively contraindicated in the acute setting due to the increased chance of perforation. Obstruction Complete colonic obstruction due to diverticular disease is relatively rare, accounting for approximately 10% of large-bowel obstructions. Partial obstruction is a more common finding and results from a combination of edema, bowel spasm, and chronic inflammatory changes. Acute diverticulitis can lead to partial bowel obstruction due to edema (colonic, pericolonic) or compression from an abscess. Recurrent progressive fibrosis and/or stricturing of the bowel may lead to high-grade or complete obstruction (it is often difficult, but important, to distinguish between a diverticulum-induced stricture and neoplasm). Abscess The formation of a complicated diverticular abscess depends on the ability of the pericolic tissues to control (localize) the spread of the inflammatory process. In general, intra-abdominal abscesses are formed by: Anastomosis leakage: 35% Diverticular disease: 23% Limited spread of the perforation forms a phlegmon, while further (but still localized) progression creates an abscess. Signs and symptoms: fever with or without leukocytosis despite adequate antibiotics, tender mass. Treatment: Small pericolic abscess: 90% will respond to antibiotics and conservative management alone. Percutaneous abscess drainage (PAD) is the treatment of choice for small, simple, well-defined collections. A group at the University of Minnesota published an overall success rate for PAD of 76%. 100% of simple unilocular abscesses resolved with PAD and antibiotic therapy. Factors identified as limiting the success of this management strategy include: multilocular collection, abscesses associated with enteric fistulas, and abscesses containing solid or semisolid material [16]. Perforation (free perforation) Free perforation is fortunately uncommon. It occurs more frequently in the immunocompromised patient. Free perforation is associated with the highest mortality rate, in up to 35% of cases. Urgent surgical intervention is required in most cases. Fistulas Fistulas occur in 2% of patients with complicated diverticular disease. The formation of the fistula results from a local inflammatory process that results in an abscess, which spontaneously decompresses by perforating into an adjacent viscus or through the skin. The fistulous tract is commonly single, but multiple tracts are found in 8% of patients. Fistula are more frequent in men than in women (2 : 1); in patients with previous abdominal surgery; and in immunocompromised patients. Types of fistula related to diverticular disease: Colovesical: 65% Colovaginal: 25% Colocutaneous: data not available Coloentero: data not available Diagnosis. The diagnosis of fistulas may require multiple investigations, but they are most commonly identified with CT, barium enema, vaginoscopy, cystoscopy, or fistulography. Trends. A group at Yale noted the following trends with regard to intra-abdominal fistulae: Fistula from diverticular disease — older patients with pneumaturia Fistula from neoplasms — fecaluria, gastrointestinal symptoms, and hematuria Fistula from Crohn’s disease — younger patients, pain, abdominal mass, pneumaturia [16] Bleeding Apart from hemorrhoids and other nonneoplastic perianal disorders, colorectal cancer is the most common cause of lower gastrointestinal bleeding. Diverticular disease remains the most common cause of massive lower gastrointestinal bleeding, accounting for 30–50% of cases. It is estimated that 15% of patients with diverticulosis will bleed at some time in their lives. The bleeding is usually abrupt, painless, and large in volume, with 33% being massive, requiring emergency transfusion [1]. Despite this, bleeding stops spontaneously in 70–80% of cases. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown to increase the risk of bleeding from diverticular disease, with over 50% of patients with bleeding diverticula receiving NSAID treatment at the time of presentation. Angiodysplasia accounts for 20–30% of lower intestinal bleeding. Mechanism. Diverticular disease is responsible for colonic bleeding because as a diverticulum herniates, the penetrating vessels responsible for the bowel wall weakness become draped over the dome of the diverticulum. In this configuration, these vessels are only separated from the bowel lumen by a thin mucosal lining. The artery is thus exposed to injury from luminal contents, and bleeding occurs [1]. Histologic examination of these ruptured vessels reveals an architecture in keeping with this theory of diverticular bleeding. Asymmetric rupture of the vas rectum (the vessel draped over the diverticulum) occurs towards the lumen of the diverticulum at its dome on the antimesenteric margin. Injurious factors within the colonic lumen produce asymmetric damage on the luminal aspect of the underlying vas rectum, resulting in segmental weakness of the artery and predisposition to rupture into the lumen. Rupture is associated with eccentric thickening of the intima of the vessels and thinning of the media near the bleeding point. There is also a notable absence of inflammation (diverticulitis) in this process [1]. Although the anatomic relationship between the penetrating vessels and the diverticula is similar on both the right and left sides of the colon, the right colon is the source of bleeding in 49–90% of patients [17–19]. In those with an initial episode of bleeding, 30% go on to have a second bleed, and of those 50% will have a third bleed. The source of bleeding is not identified in up to 30–40% of cases. Methods of locating the area of hemorrhage include: Selective angiography: The minimum rate needed is 1.0–1.3 mL/min. This modality has the advantage of allowing interventional therapy to be carried out in the form of vasopressin, somatostatin; embolization; and marking the area with methylene blue for future investigation. Radioisotope scanning: Bleeding can be detected at rates as low as 0.1 mL/min. Several types of isotope can be used, including: 99mtechnetium-labeled sulfur colloid, which clears within minutes, pools in the lumen,

and has the advantage of only taking a short time to complete the study; and labeled red blood cells, which have a longer circulating half-life and allow scans to be repeated for up to 24–36 hours. The accuracy of bleeding studies varies widely, from 24% to 91%. Colonoscopy: Colonoscopy is best reserved for self-limited bleeding. In patients with moderate bleeding that has stopped, colonoscopy can be safely done within 12–24 hours. In patients with less severe bleeding, colonoscopy is a reasonable option as an outpatient procedure. Colonoscopy continues to be an important investigation for excluding neoplasm (32%) and carcinoma (19%) as the source of bleeding. Emergency colonoscopy after aggressive bowel lavage has been suggested by several authors [20,21]. Therapeutic interventions, with local injection of epinephrine or sclerosant or thermocoagulation of specific bleeding diverticula that are identified may lead to decreased rebleeding rates in the early phase. The presence of other diverticula and their inherent propensity to bleed makes acute endoscopic intervention unlikely to affect the overall rebleeding rates in the longer term. Urgent surgery for bleeding. Urgent surgery for diverticulum-related bleeding only controls bleeding in 90% of patients. Indications for urgent surgical intervention include: Hemodynamic instability not responsive to conventional resuscitation techniques Transfusion of blood > 2000 mL (approximately six units) Recurrent massive hemorrhage Outpatient treatment. Patients with mild abdominal pain/tenderness and no systemic symptoms: Acute low-residue diet. Antibiotics for 7–14 days (amoxicillin/clavulanic acid, sulfamethoxazole-trimethoprim, or quinolone + metronidazole for 7–10 days). After initiation of therapy, expect improvement in 48–72 hours. It is important to cover for Escherichia coli and Bacteroides fragilis. If there is no improvement in 48–72 hours, look for a collection intra-abdominally. In-patient treatment: Patients with severe signs/symptoms (1–2% of cases): Admit the patient to hospital. Ensure bowel rest. Intravenous antibiotics (Gram-negative and anaerobic coverage) for 7–10 days Intravenous fluids Analgesia (meperidine). Meperidine is preferable to morphine, as the latter may lead to increased intracolonic pressure in the sigmoid. If there is improvement within 48 hours, then continue management, starting a low-residue diet in the acute period. Antibiotics may be switched to the oral form if the patient is afebrile for 24–48 hours and there is a decreasing white blood cell trend. If there is no improvement, phlegmon or a collection (abscess) should be suspected and investigated accordingly. Some 15–30% of patients admitted for the management of diverticulitis will require surgery during admission, with an associated mortality rate of 18%. Investigations Barium enema is inaccurate in 32% of cases of acute diverticulitis. Colonoscopy in the acute setting is associated with a theoretical increase in the risk of colonic perforation, due to insufflation of air during the procedure. For this reason, this investigation is often not used. Technical difficulties with colonoscopy in diverticular disease include: Bowel spasm Luminal narrowing due to prominent folds Fixation of the colon due to previous inflammation, pericolic fibrosis Surgical management (diverticulitis) Between 22% and 30% of patients with an initial episode of diverticulitis will go on to have a second episode [22]. Urgent surgical intervention is mandatory if complications occur, which include: Free perforation with generalized peritonitis Obstruction Abscess not amenable to percutaneous drainage Fistulas Clinical deterioration or failure to improve with conservative management [1] Elective surgery is a more common scenario. Surgery is undertaken after adequate bowel preparation has been performed. The indications for surgery most frequently reported include: Two or more episodes of diverticulitis severe enough to cause hospitalization Any episode of diverticulitis associated with contrast leakage (Ba), obstructive symptoms, or an inability to differentiate between diverticulitis and cancer Resection is usually carried out 6–8 weeks after any acute episode of inflammation. The surgical options vary depending on whether the indication is urgent or elective. Elective surgery most commonly involves resection of the sigmoid colon. Resection is performed after mechanical and antibiotic bowel preparation has been completed. The procedure can be performed either as an open procedure or laparoscopically. Inflammation and scarring may technically preclude the laparoscopic route. There are numerous options exist for urgent surgical intervention in patients with acute diverticulitis and its complications. Controversy with regard to the surgical options historically involved the need for primary resection at the initial operation and performing a staged procedure, as against a single operative plan. Primary resection is now the accepted standard and has been shown by a number of studies to be: Associated with a shorter hospital stay [23,24] Associated with reduced morbidity than with colostomy alone and drainage [25,26] Associated with a lower mortality than with colostomy alone versus resection (26% vs. 7%) Associated with a survival advantage [27] Hartmann’s procedure, originally described in 1923 [28], was initially intended for the treatment of cancer of the rectum. It represents a staged procedure in which the sigmoid colon is mobilized and resected, with the rectum being closed and a colostomy formed. The colostomy is closed at a later date (often around 3 months postoperatively), with restoration of bowel continuity. This staged procedure posed problems, including a second operation, rectal scarring, and difficulty in completing the anastomosis. Transverse colostomy and drainage is another staged procedure (without primary resection) in which an initial colostomy is formed, followed by resection of the diseased segment and later closure of the colostomy. This procedure is associated with a morbidity rate of 12% and a mortality rate of 5–29% [27,29,30]. The concept of primary anastomosis arose out of the inherent problems with staged revision of the Hartmann procedure. Primary anastomosis is the preferred procedure in the majority of patients with adequate bowel preparation, but is contraindicated if the patient is unstable, has feculent peritonitis, or is severely malnourished or immunocompromised. Resection with primary anastomosis and proximal stoma is a modified procedure used on an individualized basis. It allows easier reversal of the colostomy via a less invasive second (staged) operation. A single-stage procedure with on-table gut lavage can also be used in the acute setting to allow for primary anastomosis of a less than ideally prepared bowel. Recurrent diverticulitis after resection is rare, ranging from 1% to 10%. In general, the progression of diverticular disease in the remaining colon is approximately 15%. The reoperation rate for diverticular disease ranges from 2% to 11% and depends on the procedure chosen at the time of resection. The use of the rectum as the distal margin decreases the rate of recurrence (in comparison with the sigmoid as the margin). Care must be taken to exclude other causes of symptoms/signs suggestive of diverticular disease, such as irritable bowel syndrome (IBS) or ischemic colitis. Important associations: Diverticulitis and Crohn’s disease — especially in the aged Diverticulosis and IBS Up to 30% of diverticular disease patients have IBS Right-sided diverticulitis Diverticulosis in Asia is predominantly a right-sided phenomenon, occurring in 35–84% of cases. The early age of onset suggests a genetic basis, although this is still under investigation. Right-sided diverticular disease is also more commonly associated with multiple diverticula, while right-sided diverticular disease in the Western hemisphere is usually a single diverticulum. Diagnosis. Right-sided symptomatic diverticular disease can be difficult to distinguish from appendicitis. It may present with: Right upper quadrant pain. Nausea, emesis, fever. An abdominal mass is found in 26–88% of patients on clinical examination Leukocytosis is commonly present, but is a nonspecific finding. CT is able to diagnose appendicitis with a sensitivity of 98% and a specificity of 98%. Treatment. The treatment of right-sided diverticular disease follows that outlined under the heading of medical management above (section 5). Surgical options are as outlined above, but may also include a diverticulotomy for disease confined to a focal area or a right hemicolectomy. Subacute diverticulitis Subacute diverticulitis represents moderate to severe episodes of diverticulitis with some resolution on antibiotics and conservative treatment, but which does not resolve completely. The problem continues in a smoldering fashion with low-grade fever, left lower quadrant pain, and altered bowel habit. Smoldering diverticulitis Smoldering diverticulitis consists of abdominal pain and a change in bowel habit, without obvious fever or leukocytosis. This condition can persist for 6–12 months. The condition is often diagnosed by the presence of: Chronic left lower quadrant pain Diverticulosis on history and investigations An absence of signs of diverticulitis Treatment. Sigmoid resection provides complete resolution in 70% of cases. Diverticular disease in the immunocompromised patient Conditions that represent an immunocompromised state include: Severe infection Steroids Diabetes mellitus Renal failure (45–50% of patients) Malignancy Cirrhosis Chemotherapy/immunosuppressive agents (13%) The clinical findings are usually very subtle. The condition is associated with: An increased rate of free perforation (43% vs. 14% in immunocompetent patients) An increased need for surgery (58% vs. 33%) Increased postoperative mortality (39% vs. 2%) Giant diverticulum (colon) This is a rare condition, first described by Bonvin and Bronte in 1942. Sex: as frequent in men as in women. Age: usually occurs in patients over the age of 50. Size: must have a diameter > 13 cm Location: the sigmoid is almost exclusively involved. Mechanism: there is a ball-valve effect, with air being trapped in the diverticulum Types: type 1 is a pseudodiverticulum, type 2 is a true diverticulum. In the developed world, the prevalence of diverticular disease ranges from 5% to 45%. The majority of this population (90%) is made up of patients with distal bowel disease. Only 1.5% of cases involve solely the right side of the large bowel [10]. In contrast, individuals in Africa and Asia who develop diverticular disease have predominantly right-colon involvement (70–74%), especially in the ascending colon. In Singapore, only 23% of patients have sigmoid involvement, and 70% of those with right-sided diverticulosis are under 40 years of age [22,31]. The early age of onset and the location suggest a genetic basis for the development of diverticular disease in Asia, but this requires further investigation. Despite the increasing westernization of the diet, Japan still has a higher prevalence of right-sided diverticular disease (although cases involving the left colon are increasing). Hong Kong still has a 76% prevalence of right-sided diverticulosis. Young-Fadok TM, Roberts PL, Spencer MP, Wolff BG. Colonic diverticular disease. Curr Prob Surg 2000;37:457–514 (PMID: 10932672). Schauer PR, Ramos P, Ghiatas AA, Sirinek KR. Virulent diverticular disease in young obese men. Am J Surg 1992;164:443–8 (PMID: 1443367). Konvolinka CW. Acute diverticulitis under age forty. Am J Surg 1994;167:562–5 (PMID: 8209928). Ambrosetti P, Robert JH, Witzig JA, Mirescu D, Mathey P, Borst F, et al. Acute left colonic diverticulitis in young patients. J Am Coll Surg 1994;179:156–60 (PMID: 8044384). Anderson DN, Driver CP, Davidson AI, Keenan RA. Diverticular disease in patients under 50 years of age. J R Coll Surg Edinb 1997;42:102–4 (PMID: 9114680). Painter NS, Burkitt DP. 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Am J Gastroenterol 1999;94:3110–21 (PMID: PMID: 10566700). This is a full overview of the topic for and on behalf of the Ad Hoc Practice Parameters Committee of the American College of Gastroenterology, dated July 1999. The document is available for free via the ACG web site at: Society for Surgery of the Alimentary Tract (SSAT): Surgical Treatment of Diverticulitis. This patient care guideline is written primarily for primary-care physicians to assist with decisions on whether or not to refer a patient for surgical consultation. It is a basic outline dealing with symptoms and diagnosis, treatment, risks, expected outcome and qualifications for performing surgery for diverticulitis. The document is available for free from the SSAT web site at: American Society of Colon and Rectal Surgeons (ASCRS) annual meeting in San Diego, California, 3 June 2001: webcast by Tonia Young-Fadok of the Mayo Medical School on “Core Subjects — Diverticular Disease”. Free registration at www.vioworks.com; go to the ASCRS 2001 Annual Conference lectures area and choose this one. Prof. R.N. Allan (Birmingham) Prof. Franco Bazzoli (Bologna) Dr. Philip Bornman (Cape Town) Dr. Ding-Shinn Chen (Taipei) Dr. Henry Cohen (Montevideo) Prof. A. Elewaut (Ghent) Dr. Suliman S. Fedail (Khartoum) Prof. Michael Fried (Zurich) Prof. Alfred Gangl (Vienna) Prof. Joseph E. Geenen (Milwaukee) Dr. Saeed S. Hamid (Karachi) Prof. Richard Hunt (Hamilton, Ontario) Prof. Günter J. Krejs (Graz) Prof. Shiu-Kum Lam (Hong Kong) Dr. Greger Lindberg (Stockholm) Dr. Hou Yu Liu (Shanghai) Prof. Juan-R. Malagelada (Barcelona) Prof. Peter Malfertheiner (Magdeburg) Prof. Roque Saenz (Santiago de Chile) Dr. Nobuhiro Sato (Tokyo) Prof. Mahesh V. Shah (Nairobi) Dr. Patreek Sharma (Kansas City) Dr. Jose D. Sollano (Manila) Prof. Alan B.R. Thomson (Edmonton) Prof. Guido N. J. Tytgat (Amsterdam) Dr. Nimish Vakil (Milwaukee) The Practice Guidelines Committee welcomes any comments and queries that readers may have. Do you feel we have neglected some aspects of the topic? Do you think that some procedures are associated with extra risk? Tell us about your own experience. You are welcome to click on the link below and let us know your views. guidelines@worldgastroenterology.org

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