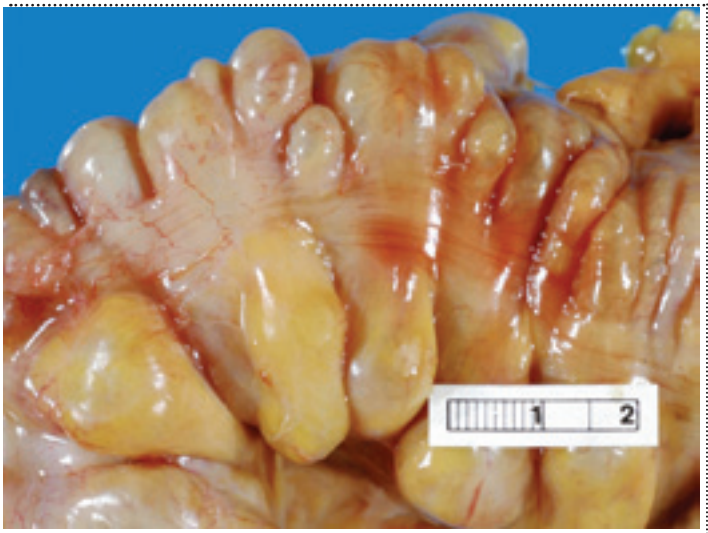
COLONIC DIVERTICULOSIS: A REVIEW

Tyara Banerjee, Suman Verma, Matthew W. Johnson



Colonic Diverticulosis: A Review
Good Clinical Care

Abstract

Diverticular disease is a common condition that is encountered regularly by both medical and surgical specialties. These patients frequently present acutely via A&E. It is therefore essential for Foundation Year doctors to understand the pathogenesis, clinical presentations and management of this disease.

Definitions

The term "diverticulum" is a Latin word meaning side-branch or pouch. Diverticulae are herniations of the colonic mucosa and submucosa, through the muscularis propria of the bowel wall. Diverticular disease and diverticulosis have been used as interchangeable terms to describe a condition where multiple diverticulae are seen within the bowel. For several years now the specialists in the field have sought a new classification system for this disorder, to assist diagnosis and research, as the present definitions are slightly confusing. With so many people in the Western world developing colonic diverticulae, and a vast majority of them being asymptomatic, it does not seem appropriate to call this anatomical development "a disease". The British Society of Gastroenterologists has recommended that the term "diverticulosis" is reserved for those with asymptomatic uncomplicated colonic diverticulae. The diagnosis of "diverticular disease" should be used in those who become symptomatic from their colonic diverticulae, whilst the term "diverticulitis" should be reserved for those who develop inflammation associated with their diverticulae[1]. This terminology is what we will adhere to for the rest of this article.

Introduction

The cause of diverticulosis is unknown, but the aetiology is likely to be multifactorial. Contributing factors include dietary changes, abnormal colonic motility and changes in the bowel wall. Diverticulosis is said to affect two thirds of the elderly population in the UK. The vast majority of patients with diverticulosis are asymptomatic. Only a small fraction of individuals (<25%) with diverticulosis develop symptoms. Despite this, diverticular disease and its complications are responsible for 68000 hospital admissions and 2000 deaths per year in the UK[2]. The symptoms can be quite disabling. The main complications of diverticulosis include diverticulitis, diverticular haemorrhage, stricturing and perforation. The mainstay of treatment is conservative management with surgery for those who fail to respond to medical therapy. This article gives an overview of diverticulosis, from the pathogenesis to the diagnosis and management of this common condition and its complications.

Epidemiology

The first description of diverticular disease was in the 18th century, but diverticulosis was not fully appreciated until the 20th century [3].

The prevalence of colonic diverticulae is similar between sexes and has been shown to increase with age. The distribution of diverticulae around the colon varies throughout the world. Diverticulosis is more common in Western countries where it is predominantly found on the left side (especially the sigmoid colon). In Eastern countries the distribution is largely right sided[4]. In the West, the condition occurs with prevalence rates of 5% by age 40, 30% by age 60, and 65% by age 85 [5]. The majority (75%) remain asymptomatic. Of those that do suffer complications, 25% develop bleeding and 75% develop diverticulitis [6].

Pathogenesis

The development of diverticular disease is likely to be multifactorial although the specific cause is unknown. Contributing factors include increased luminal pressure, prolonged intestinal transit time, small stool volume, increasing age, hypersegmentation, mychosis, structural wall abnormalities and a lack of dietary fibre.

a) Hypersegmentation

Diverticulae develop in areas of weakness in the bowel wall, between the taeniae coli. Commonly this occurs where the vasa recta penetrates through the muscularis propria. Segmentation is a process by which a short segment of the circular colonic muscle contracts in a non-propulsive manner

COLONIC DIVERTICULOSIS: A REVIEW

Tyara Banerjee, Suman Verma, Matthew W. Johnson

to produce a closed segment of colon with increased luminal pressure. This process encourages an increase in water and electrolyte absorption through the colonic mucosa. This increase in luminal pressure is believed to predispose to herniation and hence the development of diverticulae at sites of colonic weakness. The process of segmentation is exaggerated in individuals with diverticulosis[7]. The law of Laplace states that the transmural pressure gradient equals the wall tension divided by the radius. As the sigmoid colon has the smallest diameter and therefore the highest intra-luminal pressure, this may explain why diverticulae are more commonly seen in the sigmoid colon. Diverticulae do not occur in the rectum because the taeniae coalesce at the rectum to form the circumferential longitudinal muscle layer.

b) Mychosis

Mychosis is a form of muscular thickening caused by increased deposition of collagen and elastin within the taeniae, rather than from hypertrophy or hyperplasia of the bowel wall[8]. The subsequent thickening of the circular and longitudinal muscle layers and the shortening of the taeniae coli leads to narrowing of the lumen and increases in the luminal pressures.

c) Structural wall abnormality

Diverticulae commonly occur in patients with connective tissue disorders such as polycystic kidney disease, Marfan's syndrome and Ehlers-Danlos syndrome. This suggests that structural changes in the colonic wall can predispose patients to diverticulosis[9]. The tensile strength of the collagen and muscle fibres of the colonic wall decreases with age due to increased cross linking of abnormal collagen fibres and the deposition of elastin in all layers of the colonic wall. The structural changes seen in patients with diverticulosis seem to be similar to but more extensive than those typically seen in the older population[10].

d) Dietary factors

The increased incidence of diverticulosis in the Western population implies a role for diet and other lifestyle factors[11]. Diverticulae are rarely seen in Africa and parts of rural Asia where diets are high in low residue (or soluble) fibre such as fruit and vegetables. A diet rich in high residue (insoluble) fibre can lead to increased stool bulk and a larger colonic diameter. Impaired segmental contractions are seen in wider colons and this leads to higher intraluminal pressures and the development of diverticulae[12]. A lack of physical activity and increasing obesity may also be risk factors for the development of diverticular disease[13,14].

Uncomplicated diverticulosis

Seventy five percent of patients with diverticulosis are said to be asymptomatic at diagnosis and the condition is found incidentally[15]. However, a majority of these patients, when questioned, will describe constipation, bloating, flatulence and colicky left abdominal pain, relieved by defaecation. It is unclear whether these symptoms are secondary to the diverticulosis itself or to the constipation associated functional bowel syndrome. Right sided abdominal pain, rectal bleeding and recent onset altered bowel habits should not be blindly attributed to diverticular disease without further investigation to exclude neoplastic changes.

Diverticulosis can be diagnosed using a range of investigations including barium enema, contrast CT scan, CT colonography and colonoscopy (Figures 1 and 2).

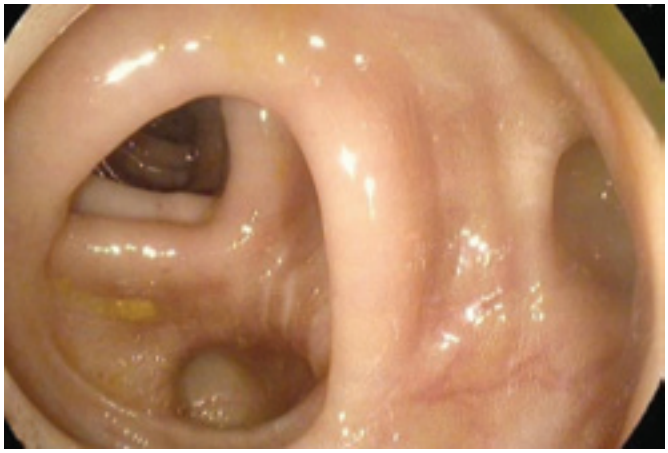


Figure 1: Diverticulosis as observed on endoscopic examination

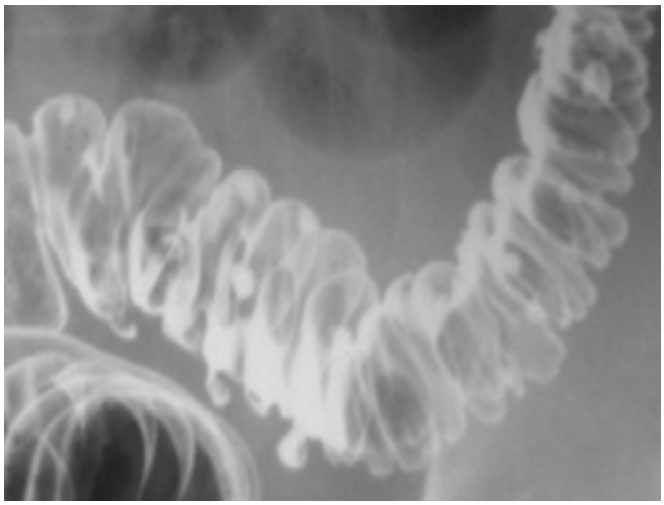


Figure 2: Diverticulae seen on a barium enema study

The medical management of patients with uncomplicated diverticular disease should be aimed at reducing the development of further diverticulae and preventing the associated complications. This can be achieved by softening the consistency of the stool. Patients should be encouraged to take a high fluid intake of over two litres a day, and may benefit from adopting a low residue (soluble) fibrous diet, whilst avoiding high residue (insoluble) roughage foods[16]. Existing diverticulae, however, do not regress. Traditionally, patients have been advised to avoid indigestible foods such

COLONIC DIVERTICULOSIS: A REVIEW

Tyara Banerjee, Suman Verma, Matthew W. Johnson

as those containing seeds which may increase the risk of complications by getting caught inside the diverticulae causing secondary inflammation. Many patients find osmotic laxatives helpful and antispasmodics can be beneficial in those who present predominantly with abdominal pain. In general it is recommended that stimulant laxatives should be avoided, as they can worsen pain by increasing colonic pressure. Constipating medications such as opiates and loperamide should also be avoided.

Complications

Of those who develop complications secondary to diverticulosis, 75% suffer from diverticulitis and 25% experience rectal haemorrhage. In turn, of those suffering from diverticulitis a further 25% of these patients can go on to suffer from abscess formation, stricturing, obstruction, fistula formation and perforation[6].

Diverticulitis

The exact pathophysiological process involved in developing diverticulitis is unknown. It is believed to start with the inspissation of faecal material stuck within the narrow neck of a diverticulum. The resulting obstruction at the diverticular neck leads to increased pressure on the epithelial lining, focal ischaemia and a break down of barrier function. This allows bacterial overgrowth and translocation to occur into the lamina propria where they cause acute inflammation, attracting pus cells from the circulating blood and creating micro-abscesses. As the inflammation resolves, fibrotic scarring can be laid down with secondary muscular hypertrophy and motor abnormalities.

Diverticulitis should be suspected in those patients presenting with lower abdominal pain, fever, leucocytosis and an elevated ESR or CRP. The abdominal pain is usually gradual in onset, present for several days and localised to the left lower quadrant. Other symptoms include diarrhoea, constipation, nausea, vomiting and urinary symptoms. Differential diagnoses to be considered include appendicitis (from right sided diverticulitis), colitis, cholecystitis, pancreatitis and bowel obstruction.

On physical examination, there can be fever and abdominal tenderness, usually in the left lower quadrant. Be aware if the patient presents with diffuse abdominal tenderness as this may indicate perforation and assessments would then be needed to exclude an acute abdomen. A tender mass in the left iliac fossa may indicate a pericolic abscess.[17]

Routine blood tests may reveal a leucocytosis with raised CRP or ESR. The amylase may be elevated in peritonitis or perforation. Urinalysis may reveal a sterile pyuria secondary to inflammation of the adjacent sigmoid colon. Colonic debris and flora may be present on culture in patients with a colovesical fistula. Plain abdominal radiographs usually demonstrate proximal faecal loading. If an obstruction is present, proximal bowel dilatation may be visible. In patients with intraperitoneal perforation, free air may be visible under the diaphragm on erect chest radiographs. CT colography is the gold standard in the diagnosis of diverticulosis as well as complications such as abscess formation, fistulae, peritonitis and obstruction. CT findings in acute diverticulitis include soft tissue density of the pericolic fat, bowel wall

thickening of more than 4mm, phlegmons or pericolic fluid in the tissue surrounding the diverticulae [18]. Endoscopy is contraindicated in the acute setting due to the associated risk of perforation.

Complications of patients with acute diverticulitis include pericolic or pelvic abscess formation, perforation with purulent or feculent peritonitis, strictures and fistula formation. Colovesical and colovaginal fistulas are the most common forms of these fistulae. Seeding of infection can also occur to heart valves and the psoas muscle.

The Hinchey classification system is used to characterise the severity of an acute episode of diverticulitis and its associated mortality risks. There are four classes of increasing severity; Grade I describes a localised abscess (less than 5% mortality), grade 2 describes a larger abscess extending into the pelvis (less than 5% mortality), grade 3, purulent peritonitis (13% mortality) and grade 4, faeculant peritonitis (43% mortality)[19].

Management

Uncomplicated acute diverticulitis can be managed medically in 70-100% of cases.[20]. Mild cases of diverticulitis can be treated as an outpatient provided they have no signs of systemic infection or paralytic ileus. Treatment consists of oral antibiotics, bowel rest (clear fluids followed by a low residue diet) and avoidance of corticosteroids and NSAIDS. Antibiotics should be continued for 7 to 10 days and the choice is aimed to cover gram negative rods and anaerobes. Common regimes include metronidazole with ciprofloxacin or co-amoxiclav. Clinical improvement should be seen within 2-3 days. If the patients develop increasing pain, fever or are unable to tolerate oral fluids, they should be advised to seek medical attention. Patients who have recurrent attacks of diverticulitis can be empirically treated with antibiotics without repeating extensive investigations.

The in-patient management of acute diverticulitis is as above with antibiotics given intravenously. Surgery is indicated in the presence of diffuse peritonitis, obstruction and where there has been a failure to respond to medical therapy. The main objectives of surgical intervention are to adequately drain infected areas and to remove the affected region

COLONIC DIVERTICULOSIS: A REVIEW

Tyara Banerjee, Suman Verma, Matthew W. Johnson

of the colon. With the advancement of interventional radiology, 60-80% of abscesses can be now managed by percutaneous placement of catheters under ultrasound or Xray guidance, therefore providing another management option[21]. The type of surgical procedure undertaken depends on the severity of the disease and the status of the patient. In less severe disease, primary resection and anastomosis can be considered. In the presence of advanced peritonitis, a Hartmann's procedure is usually performed. This involves resection of the affected colon, closure of the rectal stump and the formation of a colostomy. In other circumstances, a double barrelled colostomy is formed. Most stomas can be reversed a few months later after anastomotic healing.

Diverticular Colitis

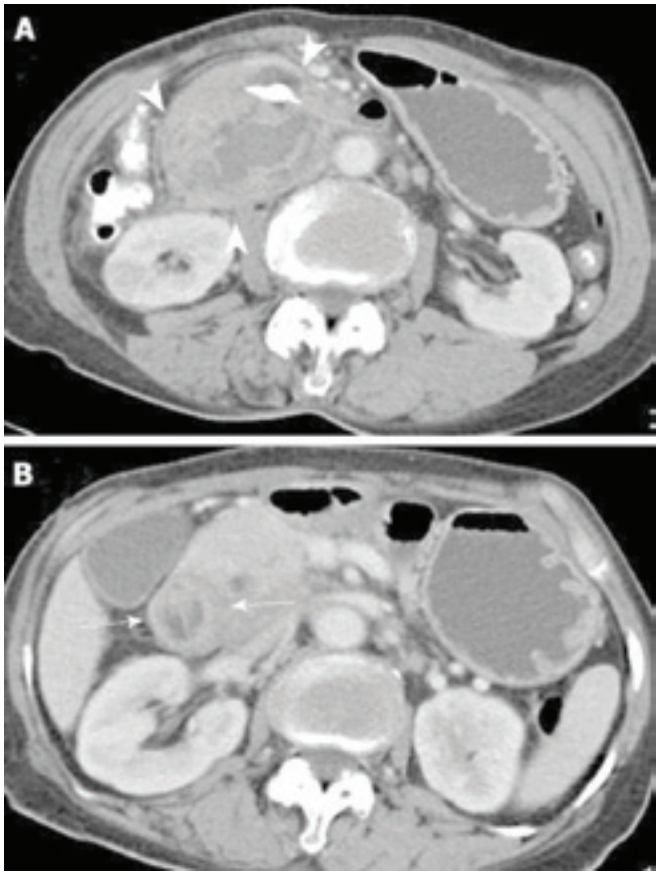
As well as inflammation of the diverticulae themselves, diverticulosis can be associated with colonic inflammation between the diverticulae, known as diverticular colitis, This is typically a segmental colitis, most commonly seen in the sigmoid colon[22]. Patients may present with bloody diarrhoea and abdominal pain, or be asymptomatic. Diverticular colitis can be difficult to differentiate from ulcerative colitis, both endoscopically and histologically. In these cases rectal biopsies may prove useful as a discriminating test, as diverticular colitis rarely, if ever, affects the rectum. Evidence suggests that this form of colitis can be effectively treated with short courses of mesalazines[23]. There may also be a role for aminosalicylates in the treatment of symptomatic diverticular disease or recurrent diverticulitis[2,24,25].

Diverticular haemorrhage

Up to 15% of patients with diverticulosis will develop diverticular bleeding and this is said to account for up to 40% of all cases of lower gastrointestinal haemorrhage[26]. As mentioned previously, diverticulae form at the site of penetration of the vasa recta through the circular muscle layer. Bleeding occurs as a result of progressive injury to the arterioles passing through with the base of the diverticulum, which can be very thin walled. Right sided diverticulae are more likely to bleed as a longer portion of the arteriole is exposed to direct injury. This occurs due to the diverticulae having wider necks and domes. The right colonic wall is also thinner, which may also be a contributing factor[27].

Diverticular bleeding is usually painless and self limiting. The patient typically presents after passing moderate to large amounts of maroon blood or hematochezia per rectum. Bright red blood and clots are associated with a more distal source, whereas malaena is more likely to be coming from the right side of the colon. Clinical examination may be normal or the patient might exhibit signs of shock with massive haemorrhage. On initial presentation, the haemoglobin level can be normal. Low haemoglobin levels may reflect pre existing anaemia or a significant bleed that has been ongoing for several days.

Colonoscopy is the inital examination of choice for both diagnosis and treatment. Bleeding vessels can be treated with submucosal injections of adrenaline, endoclips or heater probe. If the source of bleeding



proves difficult to identify one could consider a radionuclide scan using technetium sulphur colloid or 99mTc pertechnetate-labeled autologous red blood cells. These techniques can detect bleeding at a rate of 0.1ml/min. The main disadvantage of both these red blood cell labeling scans is that they can only localise bleeding to an area of the abdomen rather than to a specific site in the colon, with estimated accuracy rates ranging from 24 to 91%[28]. Alternative diagnostic techniques include dynamic enhanced helical CT scans and angiography. The latter however requires a brisker rate of bleeding than the radionuclide scans at 0.5 ml/min, but offers the option of an "on-the-table" therapeutic embolisation.

Initial management for a patient with an acute diverticular bleed should include establishing intravenous access, cross matching blood and administering intravenous fluids until blood is available. Any coagulopathy should be corrected. Surgery may be required if bleeding cannot be controlled through the above means. Approximately, 75 – 90% of patients will stop bleeding spontaneously with a 10-40% risk of recurrent bleeding[29]. As the majority of patients are elderly with multiple comorbidities, the mortality and morbidity rate of a diverticular bleed is as high as 10 – 20%[30].

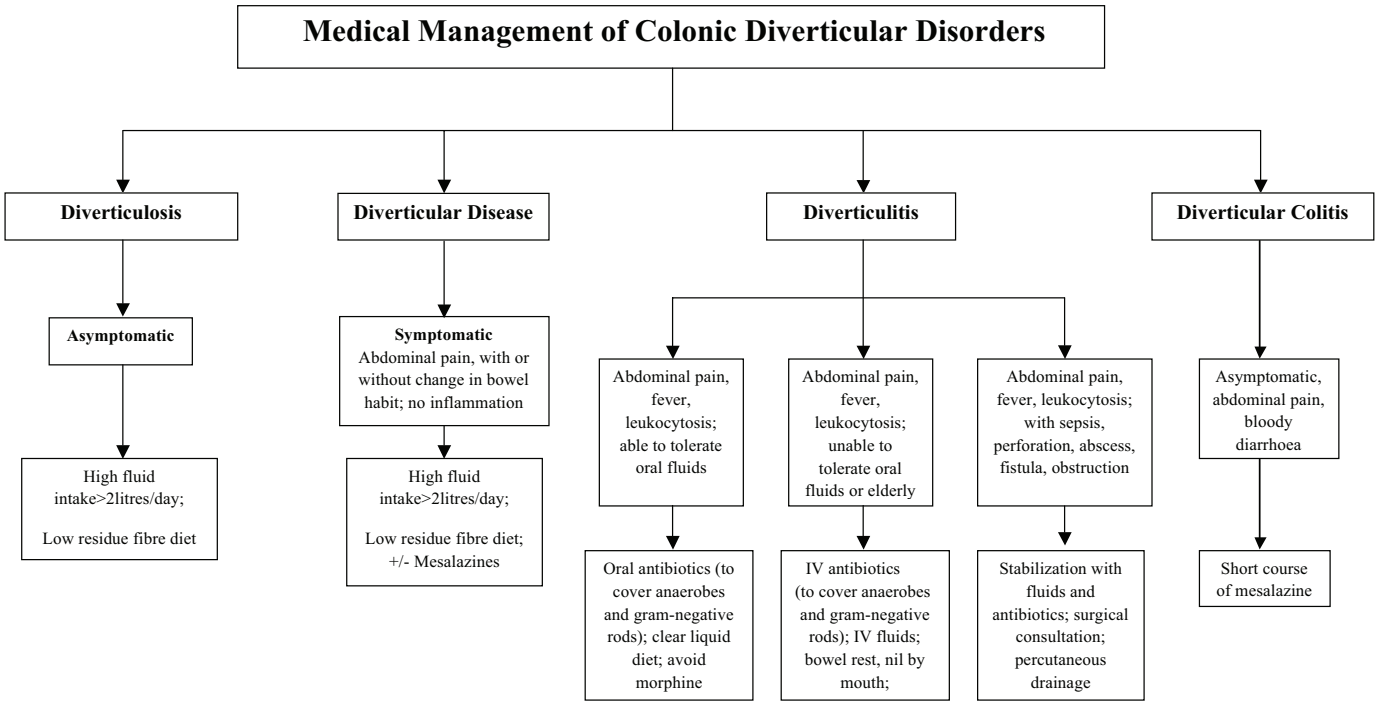


Figure 3: Flow-Chart depicting the Medical Management of Colonic Diverticular Disorders

Summary

The following flow chart summarises an evidenced based approach to the management of asymptomatic diverticulosis, symptomatic diverticular disease and diverticulitis. (Figure 3)

References

1. http://www.bsg.org.uk/patients/general/diverticular-disease.html
2. Spiller RC. Mechanistic RCT of Mesalazine in Symptomatic Diverticular Disease. Clinical Trial number NCT00663247.
3. Painter N, Burkitt D. Diverticular disease of the colon: A deficiency disease of the western civilization. British Medical Journal 1975; 2:450.
4. Chan CC, Lo KK, Chung EC, et al. Colonic diverticulosis in Hong Kong: distribution pattern and clinical significance. Clin Radiol 1998; 53:842.
5. Greenberger NJ, Blumberg R, Burakoff R. Diverticular disease of the colon. In: Current Diagnosis & Treatment: Gastroenterology, Hepatology & Endoscopy, 1st ed, McGraw-Hill, 2009;243.
6. Travis S, Ahmed T, Collier J, Steinhart A. Colonic Diverticular disease. In: Pocket Consultant Gastroenterology, 3rd ed, Blackwell Publishing, 2005; 312.
7. Painter NS, Truelove SC, Ardran GM, et al. Segmentation and the localisation of intraluminal pressures in the human colon, with special reference to the pathogenesis of colonic diverticula. Gastroenterology 1965; 49:169.
8. Whiteway J, Morson BC. Elastosis in diverticular disease of the sigmoid colon. Gut 1985; 26:258.
9. Scheff RT, Zuckerman G, Harter H, et al. Diverticular disease in patients with chronic renal failure due to polycystic kidney disease. Ann Intern Med 1980; 92:202.
10. Wess L, Eastwood MA, Wess TJ, et al. Cross linking of collagen is increased in colonic diverticulosis. Gut 1995; 37:91.
11. Greenberger NJ, Blumberg R, Burakoff R. Diverticular disease of the colon. In: Current Diagnosis & Treatment: Gastroenterology, Hepatology & Endoscopy, 1st ed, McGraw-Hill, 2009;243.
12. Painter NS, Burkitt DP. Diverticular disease of the colon, a 20th century problem. Clin Gastroenterol 1975; 4:3.
13. Aldoori WH, Giovannucci EL, Rimm EB, et al. Prospective study of physical activity and the risk of symptomatic diverticular disease in men. Gut 1995; 36:276.

14. Strate LL, Liu YL, Aldoori WH, et al. Obesity increases the risks of diverticulitis and diverticular bleeding. Gastroenterology 2009; 136:115.

15. Stollman N, Raskin J. Diverticular disease of the colon. The Lancet 2004; 363:631.

16. Gear JS, Ware A, Fursdon P, et al. Symptomless diverticular disease and intake of dietary fibre. Lancet 1979; 1:511.

17. Parks TG. Natural history of diverticular disease of the colon. Clin Gastroenterol 1975; 4:53.

18. Hulnick DH, Megibow AJ, Balthazar EJ, Naidich DP, Bosniak MA. Computed tomography in the evaluation of diverticulitis. Radiology. 1984;152(2):491.

19. Hinchney EJ, Schaal PG, Richards GK. Treatment of perforated diverticular disease of the colon. Adv Surg. 1978; 12:85-109.

20. Frattini J, Longo WE. Diagnosis and treatment of chronic and recurrent diverticulitis. J Clin Gastroenterol. 2006; 40:S112-116.

21. Schechter S, Eisenstat TE, Oliver GC, Rubin RJ, Salvati EP. Computerized tomographic scan-guided drainage of intra-abdominal abscesses. Preoperative and postoperative modalities in colon and rectal surgery. Dis Colon Rectum. 1994;37(10):984.

22. Ludeman L, Shepherd NA. What is diverticular colitis? Pathology 2002; 34:568

23. Di Mario F, Comparato G, Fanigliulo L, Aragona G, Cavallaro LG, Cavestro GM, Franze A. Use of mesalazine in diverticular disease. J Clin. Gastroenterol. 2006; 40 Suppl 3: S1 55-9.

24. Comparoto G, Fanigliulo L, Cavallaro LG, et al. Prevention of complications and symptomatic recurrences in diverticular disease with mesalazine: a 12-month follow up. Dif Dis Sci 2007; 52: 2934-2941.

25. Tursi A, Brandimarte G, Giorgetti GM, et al. Continuous versus cyclical mesalazine therapy for patients affected by recurrent symptomatic uncomplicated diverticular disease of the colon. Dig Dis Sci 2007; 52:671-674.

26. Gostout CJ, Wang KK, Ahlquist DA, Clain JE, Hughes RW, Larson MV, Petersen BT, Schroeder KW, Tremaine WJ, Viggiano TR. Acute gastrointestinal bleeding. Experience of a specialized management team. J Clin Gastroenterol. 1992;14(3):260.

27. Imbembo AL, Bailey RW. Diverticular disease of the colon. In: Textbook of Surgery, 14th ed, Sabiston, DC Jr (Eds), Churchill Livingstone, 1992;910.

28. Greenberger NJ, Blumberg R, Burakoff R. Diverticular disease of the colon. In: Current Diagnosis & Treatment: Gastroenterology, Hepatology & Endoscopy, 1st ed, McGraw-Hill, 2009;253.

29. McGuire HH Jr. Bleeding colonic diverticula. A reappraisal of natural history and management. Ann Surg. 1994;220(5):653.

30. Udén P, Jiborn H, Jonsson K. Influence of selective mesenteric arteriography on the outcome of emergency surgery for massive, lower gastrointestinal hemorrhage. A 15-year experience. Dis Colon Rectum. 1986;29(9):561.

Authors

Tyara Banerjee MRCP MBBS MA (Oxon)
ST3 Gastroenterology,
Luton & Dunstable Hospital FT

Suman Verma MB BChir, BSc, MRCP, PhD
ST3 Gastroenterology,
Luton & Dunstable Hospital FT

Matt W. Johnson BSc MBBS MRCP MD
Lead Clinician for Gastroenterology,
Luton & Dunstable Hospital FT

Correspondence Author

Matt W. Johnson
matthew.johnson@ldh.nhs.uk