Acquired small bowel diverticular disease: a review

Acquired diverticular disease, common in the large bowel, can also occur throughout the small bowel. This article reviews the prevalence, various presentations and management options for acquired small bowel diverticular disease.

Acquired small bowel diverticulosis was first described at autopsy in 1794 (Surov and Stock, 2005) but it was not until Sir Astley Cooper's publication of 1807 that acquired small bowel diverticulosis was described in the English language. Acquired small bowel diverticulosis can occur in the duodenum, jejunum and ileum, and is characterized by herniation of mucosa and submucosa through the muscular layer of the bowel wall, i.e. a pseudo-diverticulum (Kassahun et al, 2007), much like large bowel diverticulae. This is in contrast to a true diverticulum, which is an out-pouching of all the layers of the bowel wall (including muscle). They are typically congenital, and include Meckel's diverticulae. True small bowel diverticulae are not included in this article.

Clinically acquired small bowel diverticulosis can present either acutely or chronically. Acutely these patients present to the surgeons, but chronically acquired small bowel diverticulosis can present to either physicians or surgeons as a result of the various symptoms. This article presents a review of the current literature (using a Medline search) including articles from 1966 to the present. All relevant articles have been reviewed and references checked for further relevant articles. The majority of articles were case reports or case series.

Prevalence

The actual prevalence of acquired small bowel diverticulosis is difficult to ascertain as many patients are asymptomatic and are unaware of their condition. Estimation of prevalence of acquired small bowel diverticulosis by small bowel contrast studies ranges from 0.5–2.3% (Maglinte et al, 1986), although one Japanese small bowel contrast study quoted rates of 0.01–0.2% (Matsumoto et al, 2000), suggesting an element of geographical variation. Larger studies account for the lower prevalence figures above, with the largest sample studied (553 715 patients undergoing gastrointestinal studies between 1975 and 1990) revealing a prevalence of 0.26% (Tsiotos et al, 1994). At autopsy, 0.26–4.6% of patients are found to have acquired small bowel diverticulosis (Surov and Stock, 2005).

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It has been suggested that many surgeons fail to document acquired small bowel diverticulosis at laparotomy because of its perceived lack of clinical significance (Kassahun et al, 2007). Furthermore, acquired small bowel diverticulosis typically occurs at the points of perforating blood vessels, and is therefore obscured by the small bowel mesentery, and so not seen during laparotomy (De Peuter et al, 2009).

Small bowel diverticulae are typically multiple, and tend to be larger and more numerous in the proximal jejunum, becoming smaller and fewer towards the terminal ileum (De Bree et al, 1998): 55–80% occur in the jejunum, 15–38% in the ileum, and 5–7% in both (Woods et al, 2008). Co-existent diverticulae are found in the colon in 20–70% of patients, in the duodenum of 10–40% of patients, and in the oesophagus and stomach in 2% of cases (Kassahun et al, 2007).

Aetiology

Acquired small bowel diverticulosis is essentially a disease of the elderly. Documented asymptomatic acquired small bowel diverticulosis occurs almost exclusively in patients over 40 years of age (Maglinte et al, 1986). The incidence of complicated acquired small bowel diverticulosis increases with age, and peaks in the sixth and seventh decades (Woods et al, 2008). There is some debate as to whether acquired small bowel diverticulosis is more common in males or females (Patel et al, 2008; Woods et al, 2008), but the prevalence appears roughly equal. These observations suggest a similar aetiology of acquired small bowel diverticulosis to the more common acquired large bowel diverticular disease. Studies of jejunal diverticula have suggested abnormalities in either the smooth muscle or the myenteric plexus result in disordered bowel contraction (jejuno-ileal dyskinesia), causing increased intraluminal pressures (Krishnamurthy et al, 1983; Fass and Colonval, 2007) similar to the aetiology of large bowel diverticular disease. This results in protrusion of the mucosa and submucosa through the muscular wall. Furthermore, this typically occurs at the sites of

perforating blood vessels where the wall is already weakened. However, this does not account for the increased frequency of proximal acquired small bowel diverticulosis as compared to large bowel diverticular disease, which occurs in the distal bowel as a result of repeated exposure to higher intra-luminal pressures.

Clinical manifestations

As the exact prevalence of acquired small bowel diverticulosis is unknown, the percentage of patients with acquired small bowel diverticulosis developing acute symptoms (incidence) is impossible to ascertain for certain. However, it is estimated that the majority of cases of acquired small bowel diverticulosis are clinically silent, with 60–70% of patients remaining asymptomatic (El-Haddawi and Civil, 2003; De Peuter et al, 2009). Another study (Fuerxer et al, 2005) suggested 6–20% of patients with acquired small bowel diverticulosis become symptomatic, while a further study of 553 715 patients with acquired small bowel diverticulosis found 18% developed symptoms attributed to acquired small bowel diverticulosis (Tsiotos et al, 1994). Of those patients who experience symptomatic disease, 10–38% of cases appear to require surgical intervention (Maglinte et al, 1986; Kassahun et al, 2007). Symptomatic patients can present chronically or acutely.

Chronic disease

Chronic symptoms of acquired small bowel diverticulosis are typically vague, and consist of abdominal pain, post-prandial bloating, cramping, weight loss, anaemia and malabsorbtion symptoms (Fuerxer et al, 2005; Surov and Stock, 2005). These symptoms have been attributed, in part, to intestinal dyskinesia (Woods et al, 2008). Treatment is with antispasmodics, antacids, analgesia and dietary modifications. Vitamin B_{12} malabsorbtion syndromes occur secondary to stagnation and bacterial overgrowth in large diverticulae. Steatorrhoea and megaloblastic anaemia results from the consumption of vitamin B_{12} and deconjugation of bile salts by bacteria. Treatment is with antibiotics and vitamin B_{12} supplementation. Chronic haemorrhage may occur, and presents with lower gastrointestinal bleeding with or without anaemia (Altemeier et al, 1963). Chronic intestinal pseudo-obstruction, a condition characterized by repeated episodes of clinical and radiological small bowel obstruction in the absence of true mechanical obstruction, has also been reported (Maglinte et al, 1986).

Owing to the vague nature of most chronic symptoms of acquired small bowel diverticulosis, diagnosis is usually delayed. Patients may be misdiagnosed with irritable bowel syndrome, symptomatic cholelithiasis, peptic ulcer disease or other such diseases. Tsiotos et al (1994)'s study showed an average time from becoming symptomatic to diagnosis of 22 months. Furthermore, the diagnosis of symptomatic acquired small bowel diverticulosis is often only made with certainty when small bowel resection results in alleviation of symptoms.

Acute complications

Acquired small bowel diverticulosis may present acutely with diverticulitis (2.3–6.4% of all patients with acquired small bowel diverticulosis), acute haemorrhage (3.4–8.1%) and intestinal obstruction (2.3–4.6%) (Woods et al, 2008).

Diverticulitis and perforation

Small bowel diverticulitis (acute inflammation) can present with varying degrees of severity, from mild abdominal discomfort to frank sepsis. As there are no pathognomic symptoms or signs of small bowel diverticulitis, it is most often misdiagnosed as acute appendicitis, followed by colonic diverticulitis, cholecystitis or perforated duodenal ulcer (Greenstein et al, 1986). Typically, patients are acutely unwell, and require aggressive resuscitation and antibiotics before surgery. Often the diagnosis is only made at laparotomy or laparoscopy. Even then inconspicuous diverticula can be missed when buried in the small bowel mesentery (Roses et al, 1976).

Simple diverticulitis can progress to full thickness necrosis and perforation. The majority of perforations result from these necrotizing inflammatory reactions (82%), although blunt trauma (12%) and foreign body impaction into the diverticulum (6%) are other reported causes (Herrington, 1962). Perforation of inflamed acquired small bowel diverticulosis is serious, carrying a mortality of up to 40% in some series (Roses et al, 1976; Chendrasekhar and Timberlake, 1995). Treatment of perforation (with or without localized abscess formation) involves resection of the affected bowel segment, via an open or laparoscopic approach (Cross and Snyder, 1993), usually with a primary anastomosis. Other small bowel diverticulae can be left alone if not actively involved in the disease process (De Peuter et al, 2009). The possible exception is where the length of resection needs only to be slightly increased to include all diverticulae (Woods et al, 2008). Non-surgical treatments with intravenous antibiotic therapy or radiologically-guided percutaneous drainage have been reported (Novak et al, 1997), but should only be undertaken in patients with localized perforations without general peritonitis or bowel leak.

In the acute patient, preoperative computed tomography has the highest diagnostic value when compared with other imaging modalities, and may demonstrate acquired small bowel diverticulosis or associated localized pathology (*Figure 1*) (Greenstein et al, 1986; Fass and Colonval, 2007) although it is often most useful in ruling out other diagnoses (such as renal calculi). Large diverticulae can be demonstrated on computed tomography, but smaller diverticulae may not be visualized. If they are visible they are not infrequently overlooked as colon or other sections of small bowel. On retrospective review of the images, previously missed acquired small bowel diverticulosis can often be identified (Greenstein et al, 1986; Woods et al, 2008). Inflammatory masses

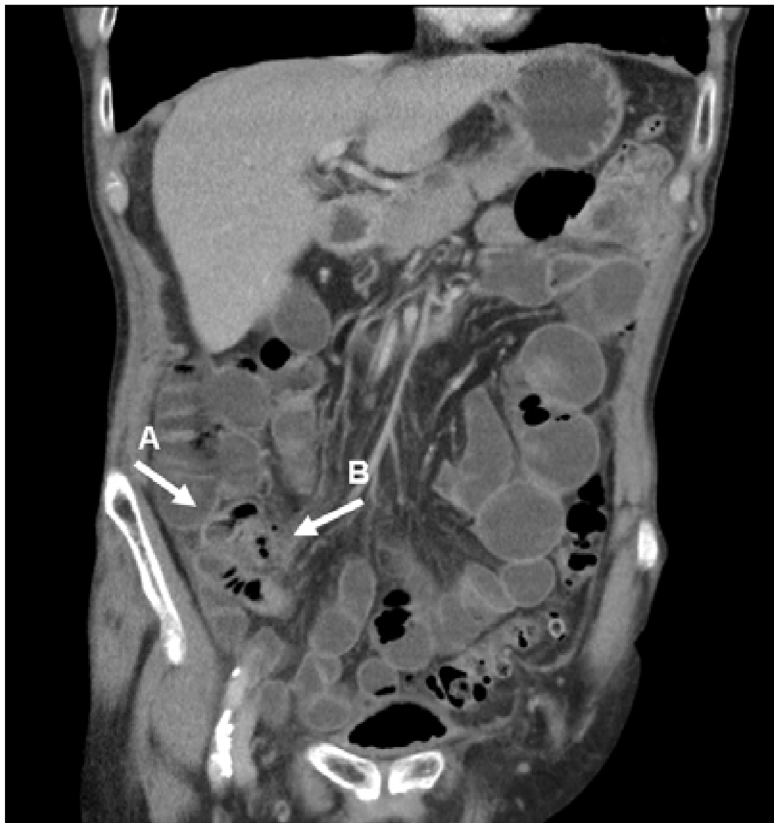
may be demonstrated with bowel wall thickening, abscess formation and fat stranding (a non-specific sign on computed tomography suggesting a localized inflammatory process; Pereira et al, 2004). Plain film radiology is generally considered unhelpful in diagnosis (Greenstein et al, 1986), although it may demonstrate a pneumoperitoneum in the case of acute perforation. Acquired small bowel diverticulosis can cause asymptomatic pneumoperitoneum in the chronic setting, which is not an indication for surgical intervention.

Haemorrhage

Haemorrhage usually presents as bleeding per rectum, although haematemesis has been reported (Patel et al, 2008). Jejunal diverticulae are reported to be more likely to bleed than ileal diverticulae (Longo and Vernava, 1992), although massive haemorrhage is most likely to occur with ileal diverticulae (Matsumoto et al, 2000). Bleeding results from an erosion of the diverticulum through the adjacent perforator artery, as with large bowel diverticulae (Patel et al, 2008). The investigation of choice depends on whether the patient is stable. Upper and lower endoscopies are generally non-diagnostic (Woods et al, 2008).

Further examinations including small bowel contrast studies, computed tomography, mesenteric arteriography and technetium red cell-tagged scans have been used (Patel et al, 2008). Technetium 99-labelled red cell scintigraphy has largely been replaced by computed tomo-graphy but, if used, it can be useful in localizing slow, intermittent bleeding, with good results obtained with blood loss as small as 0.1 ml/min (Smith et al, 1987). It does, however, fail to accurately localize the precise point in more than 50% of cases (Suzman et al, 1996). Mesenteric arteriography can specifically localize bleeding sites, and also has the added advantage of allowing embolization to be used. If embolization is performed, the coil used to embolize the artery allows specific identification of the area of affected small bowel at laparotomy, should resection be required. The disadvantage of embolization is that it can result in intestinal ischaemia and perforation requiring resection. Lastly, if the patient is haemodynamically compromised, urgent laparotomy is warranted. Even at laparotomy, the precise bleeding source may be difficult to identify (Matsumoto et al, 2000).

Figure 1. Coronal computed tomography showing a 'C'-shaped section of terminal ileum (A), with an associated inflammatory mass (B), initially thought to represent a perforated appendix. At laparotomy this was found to be a perforated acquired small bowel diverticulosis.



Obstruction

Acute intestinal obstruction may be caused by extrinsic compression from a nearby small bowel loop containing a large diverticulum, intussusception, adhesions, enterolith formation or by non-mechanical small bowel dyskinesia (Patel et al, 2008; Woods et al, 2008). Enterolith formation can occur de novo or around a nidus such as a seed or undigested vegetable matter (Hayee et al, 2003). Enteroliths are rarely seen on radiographs unless calcified, and are most frequently discovered at laparotomy (El-Haddawi and Civil, 2003; Hayee et al, 2003). Steenvoorde et al (2003) reported on the surgical options for treating enterolith ileus, which involved enterotomy in 71% of cases, segmental resection in 23% of cases, and a combination of crushing and milking the enterolith distally in 6% of cases.

Conclusions

The majority of patients with acquired small bowel diverticulosis are asymptomatic. However, acquired small bowel diverticulosis may present with a variety of symptoms, from the chronically unwell patient to one who is profoundly ill. Despite advances in imaging technology, it appears that accurate diagnoses are rarely made except during laparotomy. Understanding of acquired small bowel diverticulosis and its complications is important to ensure timely diagnosis of such patients, with appropriate surgical and medical treatment. BJHM

Conflict of interest: none.

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KEY POINTS

- Acquired small bowel diverticulae occur throughout the small bowel and, in contrast to Meckel's diverticulae, contain only the mucosa and submucosa.
- Acquired small bowel diverticulae are rare, and the majority remain clinically silent.
- Acquired small bowel diverticulae can present acutely or chronically, with chronic symptoms being typically vague and difficult to diagnose.
- Acute acquired small bowel diverticulosis can present with acute diverticulitis, bleeding or obstruction.
- Management of acute acquired small bowel diverticulae varies, but typically requires surgery with resection and primary anastomosis.