Annals of Colorectal Research

What Makes You Tic: A Comprehensive Review of GI Tract Diverticular Disease

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Abstract

Context: Diverticula are acquired or congenital outpouchings of the gastrointestinal tract, normally occurring at points of weakness in the wall of the alimentary canal. They can be present at any point from the esophagus to the anus, with colonic diverticulosis accounting for the majority of cases. While often asymptomatic, diverticula of the gastrointestinal (GI) tract are clinically significant due to potential life-threatening complications. Familiarity with key epidemiologic, etiologic, clinical, imaging, and therapeutic features of various diverticula is therefore important. This review is meant to serve as a concise guide highlighting the distribution, epidemiology, presentation, classic imaging findings, and treatment for the spectrum of GI diverticula.

Evidence Acquisition: We reviewed the fluoroscopic, computed tomography (CT), and magnetic resonance imaging (MRI) features of the most commonly encountered GI diverticula. Diverticula that are anatomically adjacent often share features such as symptomatology, optimal diagnostic imaging modality, and management. Thus, we categorized them by location along the GI tract. Esophageal (Zenker's, Killian-Jamieson, midthoracic, epiphrenic), false and true gastric, small bowel (duodenal, jejunoileal, and Meckel's), and large bowel (appendiceal, cecal, ascending/descending/sigmoid colonic) diverticula are discussed.

Results and Conclusions: Although commonly incidental, diverticula of the GI tract can be clinically significant due to complications. This pictorial essay describes the epidemiologic, etiologic, clinical, and therapeutic features as well as imaging findings associated with the range of GI diverticula encountered in clinical practice.

Keywords: Diverticula, Fluoroscopy, Gastrointestinal, Esophageal, Colonic, MR

Introduction

Diverticula are congenital or acquired outpouchings of the gastrointestinal (GI) tract. Acquired diverticula normally occur at points of weakness in the wall of the alimentary canal secondary to traction (pulling) or pulsion (increased intraluminal pressure) forces. Congenital diverticula are true diverticula, involving all three layers of the bowel wall: mucosa, smooth muscle, and serosa. Acquired diverticula are characteristically false diverticula that do not involve all three layers, i.e., a mucosal outpouching through a defect in the muscular layer (1). Diverticula can be present throughout the GI tract from the esophagus to the anus (Figure 1). While often asymptomatic, the complications related to GI diverticula can cause significant morbidity. Computed tomography (CT), magnetic resonance imaging (MRI), fluoroscopy, and nuclear scintigraphy each have specific roles and limitations in diagnosis. It is important for primary care providers, surgeons, gastroenterologists, and radiologists to be familiar with the spectrum of GI diverticular disease and its diagnostic challenges in order to guide appropriate evaluation and management.

This pictorial essay describes the epidemiologic, etiologic, clinical, and therapeutic features as well as imaging findings associated with GI diverticula encountered in clinical practice.

Hypopharyngeal/Esophageal Diverticula

Fluoroscopic esophagram is the gold standard imaging modality for multiple upper GI diverticula including Zenker's, Killian-Jamieson, midesophageal, and epiphrenic diverticula. These diverticula are often incidentally discovered on fluoroscopic modified barium swallow studies performed for other indications. Small diverticula may be easily missed on fluoroscopy in the event that they are obscured by superposition on the main column of barium in the esophagus. This can be avoided by rotating the patient or x-ray beam and obtaining multiple projections (2).

Upper Esophagus

Zenker's Diverticulum

Zenker's diverticulum (ZD) is a false hypopharyngeal pulsion diverticulum that occurs in the Killian triangle, between the fibers of the cricopharyngeus and the inferior constrictor (Figure 2, A and B) (3, 4). It is the most common diverticulum of the upper GI tract, with a prevalence of 0.01-0.11% in the general population (2, 5). Pathophysiology is related to high intraluminal pressures relative to the retropharyngeal space, which can be secondary to decreased upper esophageal sphincter (UES) compliance or failed coordination of the UES
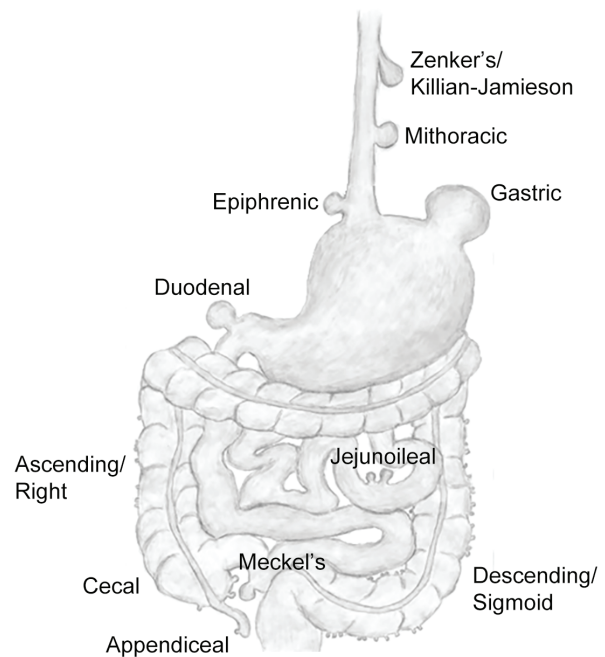

Figure 1: Diverticula of the GI tract, Overview. Overview illustration depicting common locations of gastrointestinal diverticula

with pharyngeal contraction (3, 6). The common presentation is dysphagia and cervical borborygmus in the presence of a palpable neck lump, hoarseness/dysphonia, cough, regurgitation, malnutrition, and halitosis (3, 7). Sudden worsening of symptoms or development of local pain, hemoptysis, or hematemesis are important signs of a diverticular complications such as ulceration or squamous cell carcinoma (3). Other serious complications include aspiration pneumonia, perforation during endoscopy, or rarely, perforation during nasogastric tube placement (3).

With few exceptions, treatment is indicated for symptomatic ZD with one of a variety of open surgical or endoscopic techniques (2, 3, 7). Open surgical techniques involve some combination of myotomy with either diverticulopexy, diverticular inversion, or diverticulectomy (3, 7). Less invasive transoral rigid or flexible endoscopic procedures such as peroral endoscopic myotomy (POEM) involve dividing the septum between the esophagus and the diverticulum (Figure 2, F and G). The decision of surgical procedure depends on the size of the diverticulum, surgical candidacy, local expertise, and the amount of endoscopic exposure (2, 7, 8).

Killian-Jamieson Diverticulum

Killian-Jamieson Diverticulum (KJD) is a false diverticulum that, while often confused with the more superior and posterior ZD, is anatomically distinct in that it occurs inferior to the cricopharyngeus and lateral to the longitudinal esophageal muscle fibers in an area known as the Killian-Jamieson area (Figure 2, C) (3, 4). Incidence of KJD has been reported to be one-fourth that of ZD and carries a lower association with gastroesophageal
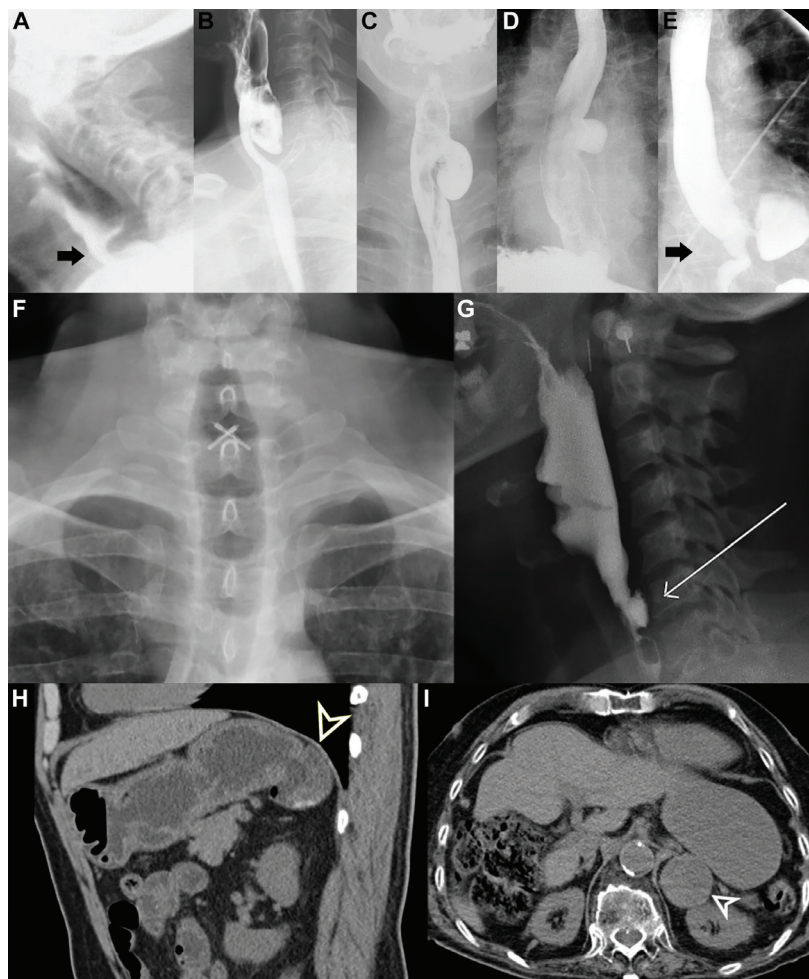
Figure 2: Esophageal and Gastric Diverticula. (A) Lateral view esophagram demonstrating a hypertrophied cricopharyngeal bar (arrow). (B) Lateral view esophagram with Zenker's diverticulum, which typically presents posteriorly with its sac above the cricopharyngeal bar. (C) AP view barium esophagram showing a Killian-Jamieson diverticulum. (D) AP view barium esophagram showing a midthoracic diverticulum. (E) AP view barium esophagram of an epiphrenic diverticulum just superior to the crux of the diaphragm (arrow). (F) AP view of the neck demonstrating surgical clips from treatment of a Zenker's Diverticulum with peroral endoscopic myotomy (Z-POEM). One year follow-up with esophogram (G) demonstrates recurrent Zenker's diverticulum (arrow). (H) CT sagittal image shows an outpouching at the posterior wall of the gastric fundus consistent with a gastric diverticulum (arrowhead in H). (I) CT axial image of a gastric diverticulum (arrowhead in I), which can be mistaken for an adrenal adenoma or other retroperitoneal mass depending on the location and intraluminal contents.

reflux or aspiration pneumonia risk (4, 9). Surgical management involving excision of the diverticulum and esophageal myotomy is indicated for large and symptomatic cases of KJD. Due to the proximity of KJD to the recurrent laryngeal nerve and lack of established safety, endoscopic procedures are generally avoided (6, 9).

Differentiation of ZD from KJD is generally achieved radiographically (4, 6). Barium esophagram should be obtained in both anteroposterior (AP) and lateral projections with the patient supine and upright (6). While ZD will present posteriorly with its sac above the cricopharyngeal bar (Figure 2, A), KJD is seen below the cricopharyngeus and will be located lateral to the cervical esophagus on frontal images and overlapping the anterior wall of the cervical esophagus on lateral images (4, 6). Furthermore, ZD is more likely to show reflux of barium from the diverticulum into the hypopharynx with overflow aspiration (4); it is also generally larger than KJD, and can become so large that it begins to extend

laterally. In this case, CT of the neck may be obtained for further evaluation (4, 6).

Middle and Distal Esophagus

Midesophageal Diverticulum

The prevalence of midesophageal diverticula (MED) is unclear, as they are often asymptomatic and found incidentally (10, 11). While traction MED can exist due to mediastinal disease, recent medical consensus suggests esophageal dysmotility and pulsion forces are the more common etiology (10, 11). In fact, underlying motility disorders are thought to account for the majority of symptoms and complications associated with MED, including retrosternal pain, dysphagia, regurgitation, epigastric pain, and recurrent pulmonary infections (12). On fluoroscopic esophagram, these diverticula are usually small and have a wide mouth (Figure 2, D) (12).

Surgical correction is usually not required but can be done via thoracoscopy or thoracotomy (12, 13).

If necessary, myotomy along with diverticulectomy is performed to treat the underlying motility disorder (11).

Epiphrenic Diverticulum

Epiphrenic diverticula (EPD) are rare false pulsion diverticula occurring in the distal 10 cm of the esophagus, usually arising from the right posterior wall (Figure 2, E) (14-16). These diverticula account for less than 10% of all esophageal diverticula, with an estimated incidence of 1:500,000 (17). It is estimated that 75-100% of cases occur in conjunction with a primary esophageal motility disorder such as achalasia or diffuse esophageal spasm (15). Pathophysiology is related to contractile discoordination between the distal esophagus and lower esophageal sphincter (LES), leading to increased intraluminal pressure and subsequent outpouching (14). While EPD are commonly asymptomatic, they may present with symptoms such as dysphagia, chest pain, weight loss, and regurgitation, many of which are related to the primary esophageal motility disorder (13, 14, 16). Important complications include nocturnal aspiration, aspiration pneumonia, perforation, and rare progression to carcinoma (13, 14, 17).

Given the high association with esophageal motility disorders, fluoroscopic esophagram in EPD is important for identifying disordered distal esophageal contractions, achalasia, and diffuse esophageal spasm (14, 15). Upper endoscopy can be helpful to exclude neoplastic processes and mucosal lesions. Intervention is indicated for symptomatic patients, particularly those with debilitating dysphagia, regurgitation, and aspiration (14, 15). Diverticulectomy is accompanied by cardiomyotomy to correct the underlying motility disorder, as well as partial fundoplication to prevent postoperative reflux (2). Both open and minimally invasive approaches are available depending on the patient's surgical candidacy (15, 17). Non-surgical candidates may opt for botulinum toxin injections placed at the LES and esophagus distal to the diverticulum (16).

Gastric Diverticulum

Gastric diverticula (GD) are rare, with prevalence ranging from 0.01%-2.6% (18, 19). These diverticula are usually less than 4 cm in size and are more commonly congenital than acquired (18, 19). Most congenital GD are located in the posterior wall of the gastric fundus, approximately 2 cm below the esophagogastric junction and 3 cm from the lesser curvature of the stomach (18, 19). Acquired GD are due to traction or pulsion forces, commonly located in the gastric antrum; these diverticula usually present in patients with peptic ulcer disease, malignancy, pancreatitis, gastric outlet obstruction, or history of gastric surgery (18, 19). While most GD are asymptomatic, some can cause a full, painful, or uncomfortable sensation in the upper abdomen, as well as dyspepsia, nausea, or emesis (18, 19). Diagnosis is often incidental, though

patients can present with complications of GD such as bleeding or diverticulitis (18, 19). Furthermore, ectopic mucosa within GD can undergo malignant transformation to adenocarcinoma (18, 19).

Detection of GD is usually accomplished by upper GI barium fluoroscopy or esophagogastroduodenoscopy (EGD). A combined approach is preferred, as GD with a narrow neck may be missed by either modality (18, 19). The EGD procedure allows for concomitant biopsy of any visualized pathology, and may also predict response to surgical treatment if symptoms can be recreated via distention of the diverticulum (18, 19). While CT imaging can also be used to evaluate GD, the diverticulum may be mistakenly interpreted as a retroperitoneal mass (Figure 2, H and I) (18, 20, 21). Medical management of GD with proton pump inhibitors may help with symptomatic relief but is less effective for diverticula greater than 4 cm (18, 19). Laparoscopic surgical resection is indicated for symptomatic patients with large diverticula (18, 19).

Small Bowel Diverticulum

Most diverticula of the small bowel are believed to occur at focal weaknesses in the bowel wall where mesenteric vessels penetrate the muscular layer; these diverticula are presumably linked to abnormalities in intestinal peristalsis, intestinal dyskinesia, and high intraluminal pressure, with most being asymptomatic and occurring after the age of 40 (22). When symptoms do occur, they can include abdominal cramping, diarrhea, and those related to complications such as diverticulitis, abscess, malabsorption, volvulus, bacterial overgrowth, obstruction, perforation, hemorrhage, as well as pancreatic and biliary disease in the case of the duodenum (22). The rate of mortality from complications is associated with the age at presentation (22).

Duodenal Diverticulum

Duodenal diverticula (DD) are the second most common GI diverticula after colonic diverticula, with a population prevalence of 5-22%. Duodenal diverticula can be single or multiple, and are classified as true, false, or intraluminal (23). True DD are less common than false DD, and are thought to result from failed duodenal recanalization in utero. They are generally located at the medial aspect of the second and third portions of the duodenum (23). False DD are acquired secondary to pulsion, or less commonly, to traction from conditions such as duodenal ulcers or cholecystitis (23). False DD occur in a similar distribution as true DD, often at the ampulla (23, 24). Intraluminal DD are duodenal mucosal webs by histopathology and occur in the second or third portions of the duodenum (23). These are fenestrated diaphragms spanning the inner circumference of the duodenum due to incomplete recanalization during
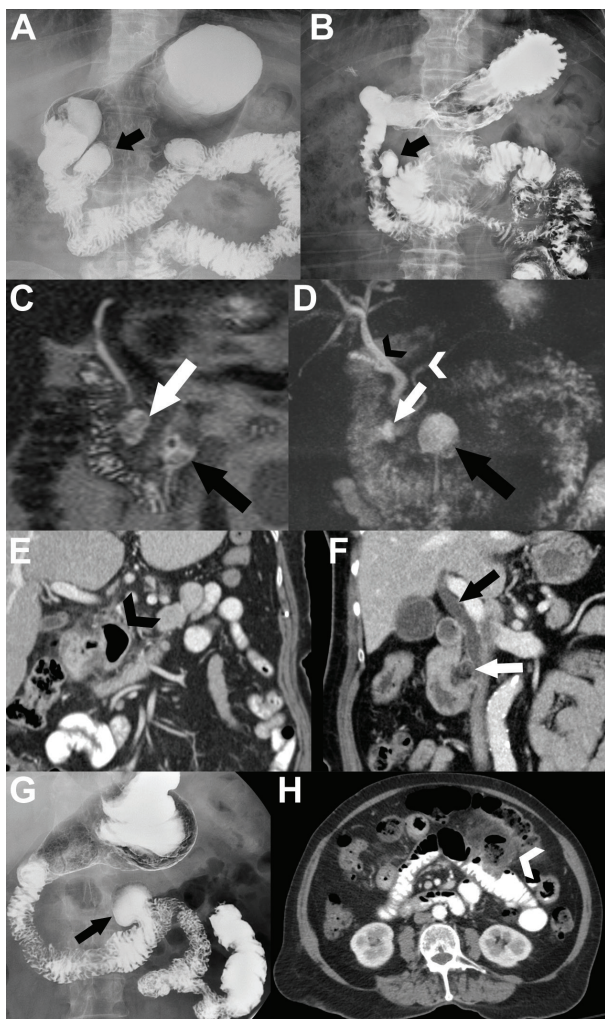
Figure 3: Diverticula of the Small Bowel. (A and B) Two examples of upper GI fluoroscopy AP views showing an outpouching (arrows) off the second portion of the duodenum consistent with duodenal diverticula. (C and D) Duodenal diverticula on MR T2-weighted sequence (C) and MR maximum intensity projection (D) images. White arrows in C and D indicate a small periampullary diverticulum near the confluence of the common bile duct (black arrowhead in D) and pancreatic duct (white arrowhead in D). The black arrows in C and D indicate a large diverticulum along the third portion of the duodenum. (E) CT coronal image showing a gas-filled duodenal diverticulum (black arrowhead in E) with surrounding fat stranding consistent with duodenal diverticulitis. (F) CT coronal image showing a dilated common bile duct (black arrow in F) secondary to duodenal diverticulum (white arrow in F) with intraluminal debris. (G) AP view fluoroscopy showing an outpouching (black arrow in G) of the proximal jejunum, consistent with a large jejunal diverticulum. (H) CT axial view with oral contrast with a large outpouching (white arrowhead in H) in an opacified loop of jejunum (normal appearance of the jejunal folds) with surrounding inflammatory changes consistent with jejunal diverticulitis.

development, resulting in a segment that is prone to ballooning in the setting of increased pressure (23). Intraluminal DD are associated with Down syndrome, choledochoceles, and annular pancreas (23, 24).

Duodenal diverticula appear as saccular outpouchings on barium fluoroscopy (Figure 3, A and B). On CT or MRI, they often appear as a thin-walled, rounded collection of gas or debris with gas-

fluid levels (Figure 3, C and D) (23, 25). Intraluminal DD are classically described as having a "windsock" appearance on fluoroscopy, with a radiolucent line surrounding a barium filled sac. On CT with oral contrast, they have a target or halo appearance (23, 24). Ultrasound (US) may show a bright linear or concave echo that obscures visualization of the pancreatic head (23). These diverticula are often incidentally found and reported in view of the potential technical challenges they can pose during endoscopic procedures. Duodenal diverticula must sometimes be differentiated from solid or cystic neoplasms of the pancreatic head, and magnetic resonance cholangiopancreatography (MRCP) may aid in further evaluation (23, 25).

In cases of duodenal diverticulitis (Figure 3, E), CT may show fat stranding, wall thickening, and occasionally, a periduodenal abscess (23). In rare cases, periampullary DD can cause obstruction of the common bile duct, which can be seen on MRCP or CT (Figure 3, F) (23, 24). For complicated cases of DD, conservative or endoscopic management is attempted before surgical management to avoid perforation (24).

Jejunoileal Diverticulum

Jejunoileal diverticula (JID) are usually acquired, false pulsion diverticula, which are approximately five times less common than DD (22, 26). Pathogenesis is related to increased intraluminal pressure and subsequent weakening of the bowel wall with herniation of mucosa and submucosa through the muscular layer (Figure 3, G) (26). These diverticula may also occur secondary to abdominal surgery or inflammatory/infectious conditions such as Crohn's disease or tuberculosis (26). The majority of JID occur in the jejunum. Less commonly, they can occur in the ileum; simultaneous occurrence in both the jejunum and ileum is even less frequent (26-28). Although JID are mostly asymptomatic, presentation is possible with chronic non-specific symptoms such as abdominal pain, constipation, diarrhea, or malnutrition (26).

As the jejunum is difficult to examine endoscopically, imaging is the diagnostic modality of choice (26, 29). The mainstay of diagnostic imaging is CT of the abdomen and pelvis (26, 29). Oral contrast is helpful to determine the extent of diverticulosis and IV contrast can assist in the evaluation of diverticulitis (Figure 3, H) (29). However, CT may underestimate the severity of disease as diverticula may be obscured in the mesentery or mistaken for small bowel loops (29). An upper GI series with small bowel follow through may be able to identify JID; however, evaluation is limited in the setting of wide mouth diverticula or if the diverticula are obscured by adjacent bowel loops (28, 30). Enteroclysis, especially with double contrast technique, offers higher sensitivity especially for wide-mouthed JID,

but offers poor patient satisfaction (28, 30).

Complications of JID can be serious and require prompt diagnosis; they include diverticulitis with or without frank perforation, hemorrhage, intussusception, and intestinal obstruction (31). In the case of diverticulitis, CT or MRI should be scrutinized carefully for free fluid and extraluminal gas, which signify complication (32). Heterogeneous enhancement of the diverticular wall correlates poorly with disease severity (32). Note that a small amount of pneumoperitoneum may not necessarily indicate true perforation, as gas can travel through distended diverticular mucosa (32).

Treatment of complicated JID can be conservative or surgical depending on the severity of disease. Conservative management involves antibiotics and bowel rest, while surgical management includes resection of the involved small bowel (26). Note that small bowel resection risks short bowel syndrome and anastomotic leak, while failing to prevent recurrence in the remaining small bowel (26).
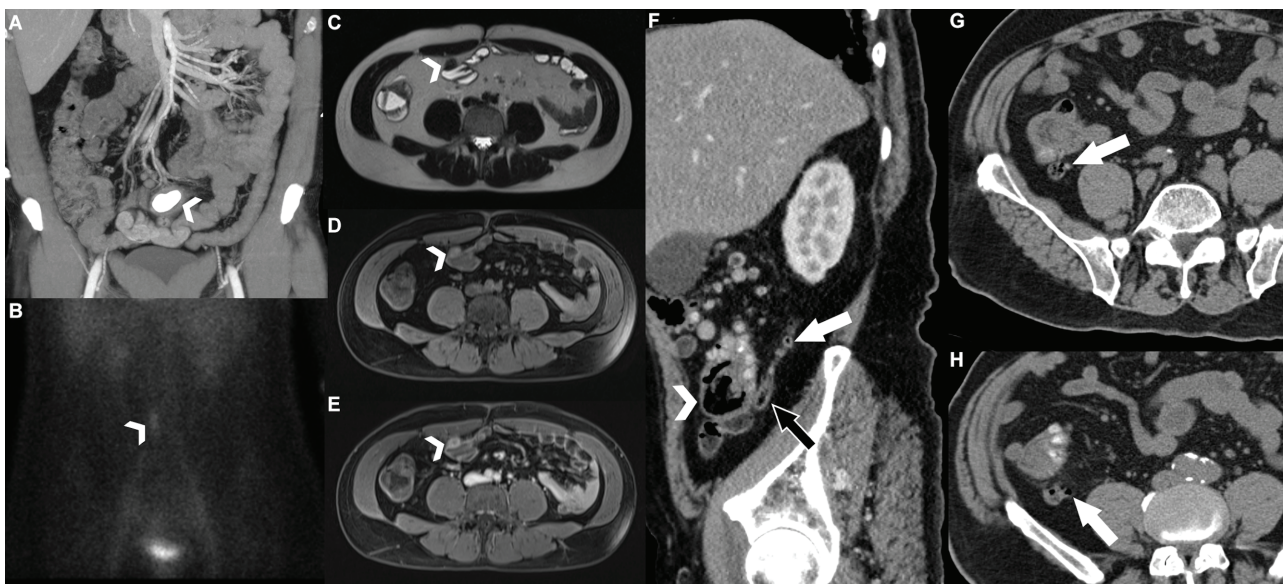
Meckel's Diverticulum

Meckel's diverticula (MD) are the most common congenital diverticula of the GI tract. As true diverticula, MD result from a persistent omphalomesenteric duct (also called vitelline duct) and classically occur in about 2% of the population; they are approximately 2 inches in length, occur within 2 feet of the ileocecal valve, and present before the age of 2 (the "Rule of 2s") (33). Clinical manifestations are more common in males and include gastrointestinal bleeding, vomiting, abdominal pain and distention related to obstruction or perforation, as well as fever related to

diverticulitis (33). Lifetime complications associated with MD occur at a rate of 4.2-6.4% and include bleeding, intussusception, obstruction, diverticulitis with abscess or perforation, peptic ulceration due to ectopic gastric mucosa, and neoplasm (33). Diverticular neoplasms often present at a mean age of 60 years with bleeding or intestinal obstruction and include carcinoid tumors or, less commonly, lipoma, neurofibroma, angioma, leiomyoma, gastrointestinal stromal tumors, leiomyosarcoma, and metastatic adenocarcinoma (33, 34).

Meckel's diverticula can be evaluated with multiple imaging modalities. In general, anti-mesenteric location will differentiate MD from ileal diverticulitis, while the presence of a normal appendix will exclude appendicitis (33). For pediatric patients, US can be utilized, demonstrating a blind-ending, thick-walled structure; pressure can be applied to express contents and prove communication with the ileum, excluding an enteric duplication cyst (33). Barium studies will show a blind-ending, tubular or saccular structure in the terminal ileum, directed away from the root of the mesentery, sometimes with mucosal irregularity due to ectopic mucosa or filling defects due to blood clots (33). Contrast-enhanced CT is important for identifying associated complications, including abscess, obstruction, perforation, neoplasm, and bleeding (33). Digital subtraction angiography can be used if bleeding from a persistent vitellointestinal artery requires embolization (33).

A nuclear medicine "Meckel's scan" (Figure 4, A-E) with technetium-99 pertechnetate localizes to mucus-producing cells of ectopic gastric mucosa in the MD (33, 34). The specificity and sensitivity of a Meckel's scan in symptomatic patients is respectively

Figure 4: Meckel's and Appendiceal Diverticula. (A) CT coronal maximum intensity projection image shows a Meckel's diverticulum (MD) containing retained oral contrast material (arrowhead in A). (B) A nuclear medicine Meckel's scan showing uptake in a MD lined with gastric mucosa (arrowhead in B). (C) T2-weighted MR image showing a small hypointensity off the distal ileum (arrowhead in C). (D) This area is hyperintense on T1-weighted images with fat saturation (arrowhead in D). (E) Enhancement (arrowhead in E) on postcontrast T1-weighted imaging is consistent with an MD. (F) CT sagittal view of the cecum (white arrowhead in F) with a retrocecal appendix (black arrow in F). A small appendiceal diverticulum (white arrows in F-H) is visualized on the sagittal view and can be better seen on the axial views (G&H).
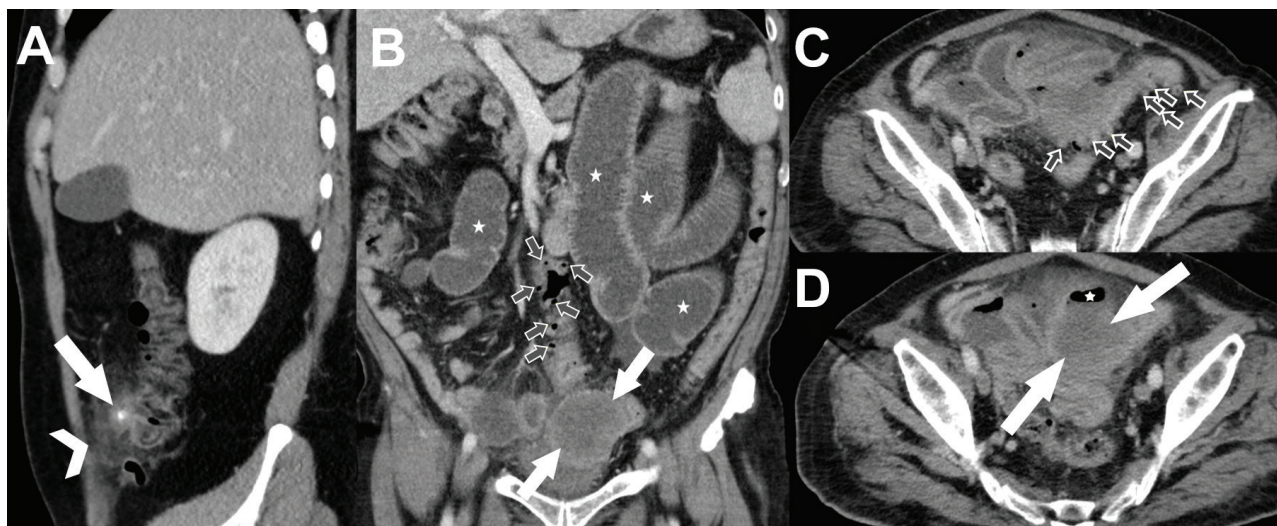
Figure 5: Diverticula of the Large Bowel. (A) Atypical location of diverticulitis in the cecum (white arrowhead in A). There is fat stranding around a small hyperdensity (white arrow in A) which represents an obstructive fecalith. (B-D) CT sagittal and axial images showing extensive diverticular disease of the sigmoid colon (gray arrows in B and C) with complicated sigmoid diverticulitis (Hinchey stage 1). There is a developing pericolic abscess (white arrows in images B&D) superior to the bladder dome containing free air (star in D). There is a reactive ileus with multiple loops of dilated small bowel (stars in B).

85% and 95% in children and 60% and 9% in adults (35). False positives can result from ectopic mucosa unrelated to MD or inflammatory disease of the bowel, while false negatives can be caused by delayed intestinal activity (33). Management of symptomatic or complicated MD is surgical resection via a laparoscopic diverticulectomy or segmental small bowel resection. However, management of asymptomatic MD incidentally noted during abdominal exploration remains controversial. In adults, MD may be resected if the patient is male, the patient age is less than 50 years, the diverticulum length is greater than 2 cm, or there is presence of ectopic tissue or abnormal features (33, 36). In children, incidental MD should be removed for patients under eight years of age (37).

Appendiceal Diverticulum

Appendiceal diverticula (AD) are congenital or acquired. Acquired AD are often seen in the distal third of the appendix at the mesenteric edge (Figure 4, F-H), and may be seen more commonly in males as well as people affected with Hirschsprung's disease or cystic fibrosis. Congenital AD are more commonly located on the antimesenteric edge and are associated with Patau syndrome (trisomy 13) (38). While relatively rare, AD is important to recognize given the complications of diverticulitis and association with malignancy. Appendiceal diverticulitis often presents with milder clinical symptoms and subtler imaging findings than acute appendicitis, and delayed diagnosis can result in perforation. A common clinical presentation is nonspecific chronic abdominal pain (38, 39). Appendiceal diverticula have also been associated with higher rates of malignancy, especially carcinoid tumors and mucinous adenocarcinomas (39, 40).

Prophylactic appendectomy is recommended for AD (39).

Colonic Diverticulum

Colonic diverticula (CD) are acquired pulsion diverticula traditionally thought to be a disease of Western societies secondary to low-fiber diets. In populations with traditionally high-fiber diets, CD affect less than 0.2% of patients (41). In European and American populations, CD are the most common diverticula of the GI tract involving the descending and sigmoid colon. Incidence increases with age, affecting approximately 50% of the population by age 50, and 70% of the population by age 80 (41). While ascending diverticular disease is uncommon in European and American populations (approximately 1-2%), it is much more prevalent in Asian populations (approximately 55-75%) (42). Right-sided diverticular disease can be misdiagnosed as appendicitis without adequate imaging. CT is the imaging modality of choice for right lower quadrant pain in adults and can reliably distinguish between acute appendicitis and cecal/ascending colonic diverticulitis (Figure 5, A). While right-sided diverticulitis is often managed medically, evaluation for underlying adenocarcinoma is important, especially in older patients (43). The disease burden of CD is evaluated on screening colonoscopy and may be graded using the Diverticular Inflammation and Complication Assessment (DICA), which factors in CD number, extension, inflammatory signs, and presence of complications (44).

The most common complications of CD are diverticular bleeding and acute diverticulitis. Factors that predispose patients to the development of acute diverticulitis include obesity, smoking, sedentary behavior, and medications such as non-steroidal

anti-inflammatory drugs (44). Mild cases of left lower quadrant pain in the appropriate age group are often managed as presumed diverticulitis and treated empirically with oral antibiotics. When symptoms are more severe or do not respond to empiric therapy, CT is used to confirm the diagnosis and assess for disease complications (45). Most diverticulitis is uncomplicated, with imaging features of peridiverticular fat stranding, reactive fluid, hyperemia in the affected part of the colon, fluid distention of the colon, and occasionally, reactive ileus. Complicated diverticulitis can be graded with the Hinchey or modified Hinchey classification systems: grade 1- presence of a pericolic abscess (Figure 5, B-D); grade 2- presence of a pelvic, intra-abdominal, or retroperitoneal abscess (2a if amenable to percutaneous drainage or 2b if complicated by associated fistula); grade 3- purulent peritonitis; or grade 4- feculent peritonitis (45, 46). Accurate characterization of diverticulitis is fundamental to determining treatment approach. Advanced cases may require sigmoid colectomy with primary anastomosis and, far less commonly, multistage surgery with an initial Hartmann's procedure and ostomy reversal several months after symptoms and inflammation have resolved (45, 47).

Conclusion

Clinicians and radiologists should be familiar with the spectrum of GI tract diverticular disease and the key epidemiologies, presentations, diagnostic findings, and treatments. While GI diverticula are often asymptomatic and incidentally found, complications are common and may be serious.

Acknowledgments

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