Celiac disease is an autoimmune disorder that causes inflammation and destruction in the small intestine in response to the ingestion of gluten and related proteins. Although long considered a pediatric condition, it is now recognized as a condition that affects all ages, with significant complications if it goes untreated. Adults are most likely to present for imaging without a diagnosis for unspecified abdominal symptoms. Imaging the small intestine is complicated. Although the advent of multidetector computed tomography (CT) helps, the relative newness of its use, combined with the relative scarcity of imaging studies of celiac disease, make CT imaging of celiac disease a constantly evolving arena.

Celiac disease, also known as gluten-sensitive enteropathy or celiac sprue, is an autoimmune disease that causes inflammation and destruction in the small intestine (specifically the jejunum) in response to the ingestion of gluten (from wheat) and related proteins from rye and barley. The term “gluten” is now used as a catchall phrase for all the trigger proteins, regardless of source. Although the inflammatory trigger is environmental, like many autoimmune conditions, the disease occurs in people with a genetic predisposition. Among whites, the prevalence of proven disease is 1%, though the number of undiagnosed cases is thought to be much higher.

Although children most often present with certain classic symptoms, the presentation of celiac disease in adults is more varied and often far less specific. This article primarily focuses on computed tomography (CT) imaging in adults because they are most likely to present for CT without a diagnosis for unspecified abdominal symptoms or systemic symptoms that point to an abdominal origin. In addition, adults often present with complications of long-term, ongoing disease processes that are uncommon in children. As the people performing the CT examination, radiologic technologists may be the first to notice suspicious findings. If they recognize signs that raise suspicion of celiac disease, technologists can aid the radiologist in directing further diagnostic testing. Because untreated celiac disease increases the risk of early death in adult patients, timely diagnosis is essential.

Anatomy of the Small Intestine

The small intestine is divided into 3 parts:

- Duodenum – the shortest part (about 20 cm long) and the passage from the stomach to the rest of the small intestine.
- Jejunum – starts at the point where the duodenum angles.
Ileum – the small intestine’s third and longest part; there is no clear demarcation between the jejunum and the ileum.

Customarily the jejunum, located in the left quadrant, is thought of as two-fifths of the small intestine; the ileum is the remaining three-fifths. The total length of the small intestine is 270 cm to 290 cm. The internal layers of the small intestine include the mucosa (further divided into the muscularis mucosa, lamina propria, and epithelial layers) and the submucosa (see Figure 1). The submucosa consists of connective tissue, blood vessels, and nerve fibers. It is the strongest layer of the small intestine. The muscularis mucosa, the base layer of the mucosa, separates the submucosa from the lamina propria, a layer rich with immune system components (eg, mast cells, macrophages, and lymphocytes). This mucosal layer is the defense barrier of the small intestine, and it plays a crucial role because of the small intestine’s daily contact with a large variety of pathogens.

Epithelial cells serve various functions depending on their location in the small intestine. Epithelial cells lining the crypts (the tubular glands between the villi) produce new epithelium that migrates up to the villi and secretes ions and hormones that aid in digestion. Villi epithelium helps the villi absorb nutrients. Goblet cells are specialized cells found in villi epithelium that secrete mucus.

The small intestine is connected to the abdominal wall via a pleated fan-shaped membrane — the mesentery. Auh et al described the mesentery’s appearance on CT as “a fat containing structure that is inseparable from the other fat containing peritoneal folds.” The fat density of the mesentery is -100 HU to -160 HU. Blood and lymphatic vessels pass through the mesentery, feeding and draining the small intestine.

The small intestine is a major portal for a variety of pathogens ingested daily, so it is rich in immune factors and supplied by a large network of lymph nodes and lymphatic drainage vessels. Lymphoid tissue is concentrated in the Peyer patches of the small intestine, which are basically unencapsulated lymph nodes in the lamina propria. Numerous scattered lymphocytes also are found in the intraepithelial space of the mucosal layer. The mesentery adjacent to the wall of the small intestine is rich in lymph nodes easily visualized on CT, especially with contrast enhancement and on coronal images.

The nutrients required for continued well-being are mostly absorbed through the small intestine. For that reason, the wall of the small intestine is covered in projections, or villi, that extend into the lumen and greatly increase the surface area of the small intestine (see Figure 2). Destruction of the villi impedes nutrient absorption and is the cause of many of the signs and symptoms of celiac disease.

The normal thickness of the small intestine’s wall on CT is 2 mm to 3 mm, except in the terminal ileum, where normal thickness is up to 5 mm. Wall thickening is a mark of many diseases and conditions of the small intestine and should be looked for and noted in CT imaging.

Causes

Prolamins are storage proteins found in the seeds of certain grains. Gliadins, belonging to the prolamin group, are alcohol-soluble proteins in wheat gluten. Gliadins and prolamins from rye and barley — all containing high levels of the amino acid proline — are the triggers for the autoimmune response that characterizes celiac disease. Oat prolamins, by contrast, contain high levels of glutamine and rarely cause a problem in people with celiac disease.

The pathogenesis of celiac disease is unclear. Like other autoimmune conditions, it is thought to result from an interaction between genetic and environmental factors. Two particular human leukocyte antigen (HLA) molecules are strongly associated with celiac disease:
Risk Factors

First-degree relatives of celiac disease patients are at an increased risk for the disease themselves. Screening studies uncover many such cases — approximately 15% of first-degree relatives are diagnosed with celiac disease. The concordance among identical twins approaches 100%. Certain genetic conditions, such as Down and Turner syndromes, are associated with a higher risk of celiac disease. Although other autoimmune diseases commonly occur with celiac disease (notably diabetes mellitus type 1), establishing a gluten-free diet does not affect the course of these other diseases. Therefore, even though people with certain autoimmune conditions are at an increased risk for developing celiac disease, and these conditions may have a common genetic background, the exact nature of the relationship between celiac disease and other autoimmune diseases is unclear.

Symptoms

Young children and some adults with celiac disease present with classic signs of malabsorption, including chronic diarrhea, vomiting, and wasting (loss of muscle mass, resulting in weight loss). A common presentation across all ages is iron-deficiency anemia that does not respond to iron supplementation. This condition also results from malabsorption in the damaged small intestine.

Additional systemic effects of malabsorption can be seen in celiac disease and include seizures, myopathy, neuropathies, short stature (this may also appear in adults with long-standing silent disease), and osteoporosis. When osteoporosis is present in children diagnosed with celiac disease, adherence to a gluten-free diet results in a return of normal bone density.

It is unclear what causes the variety of presentations and severity of signs and symptoms among celiac disease patients. The disease itself can have a course ranging from silent to symptomatic, with several varieties in between. Silent celiac disease is often found when first-degree relatives of celiac disease patients are screened for the disease through antibody studies. People with silent celiac disease:
- Show no signs or symptoms.
- Are antibody-positive for antitissue transglutaminase (anti-tTG) and antiendomysium (anti-EMA) IgA antibodies.
- Show the characteristic damage from celiac disease from a biopsy of their small intestine.\(^7\)

Individuals with latent celiac disease have normal intestinal biopsies but at some point in their lives experience gluten-dependent intestinal problems. In addition, some people with typical celiac disease antibodies and normal intestinal biopsies have never experienced gluten-related intestinal problems. These people might develop celiac disease in the future.\(^7\)

**Diagnosis**

The definitive test for diagnosing celiac disease is a tissue biopsy of the small intestine showing characteristic damage that may include villous atrophy (the most common finding), crypt enlargement, inflammation, and an increase in intraepithelial lymphocytes (see **Figure 3**).\(^1\) These changes should improve, although they may not completely be reversed, with adherence to a gluten-free diet. Such clinical improvement confirms the celiac disease diagnosis, according to several medical societies.\(^1\)

Alongside characteristic findings on the biopsy, immunoassays that attempt to locate anti-tTG and anti-EMA IgA antibodies also assist in the diagnosis. Tissue transglutaminase (tTG) is an enzyme that modifies proteins, including the prolamines responsible for triggering celiac disease. Tests for anti-tTG IgAs have a sensitivity of 90% and specificity approaching 100% for celiac disease.\(^1\) Tests for anti-EMA IgAs are as sensitive but somewhat less specific.\(^1\) These tests also can be used to monitor disease response to a gluten-free diet and adherence to such a diet if symptoms recur. They are not suitable for children younger than 2 years of age or individuals with IgA deficiency.\(^7\)

**Treatment and Prognosis**

The only known and effective treatment for celiac disease is the complete elimination of gluten, or products containing wheat, rye, and barley in any form, from the diet. Although oats are safe for most people with celiac disease, only people on a stable, completely gluten-free diet should try them so any adverse effects of oats can be identified easily and quickly.\(^7\) In addition to the rare individual with celiac disease who also is sensitive to oat, sometimes oat grain or oat-containing products are contaminated with gluten during harvesting, preparation, or shipping, so caution must be used.\(^7\)

According to Mooney et al, “in reality it is not possible to completely avoid gluten” because the international definition of gluten-free is 20 parts per million (ppm) or less.\(^8\) The Codex Alimentarius Commission set this level in 2008, having been lowered from a previous 200 ppm.\(^*\) Most celiac disease patients are able to tolerate such low levels with no ill effects.

Dietary adherence is a difficult adjustment for many people, and a healthy adherence — one that resolves symptoms and maintains health — ranges between 42% and 91% of patients.\(^*\) Less than 5% of patients do not adhere to a gluten-free diet. Participation in celiac-related advocacy or support groups improves dietary adherence and the patient’s overall health.

In children, symptom resolution may take as few as 2 weeks.\(^1\) The process is longer in adults, and histological recovery can take much longer than symptom resolution.

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**Figure 3.** Normal mucosa (A) and signs of celiac disease in the small intestine, including inflammation (intraepithelial lymphocytosis) (B), partial villous atrophy (C), and total villous atrophy (D). Photomicrographs obtained from Prof Åke Ost, previous chairman of the Swedish National Steering Group for Small Intestinal Pathology. Reprinted under the Creative Commons License from Ludvigsson JF, Brandt L, Montgomery SM, Granath F, Ekborn A. Validation study of villous atrophy and small intestinal inflammation in Swedish biopsy registers. BMC Gastroenterol. 2009;9:19.
In 1 review of 114 adults with celiac disease, despite the disappearance of symptoms and negative antibody tests in all patients, a complete recovery of the small intestine at 2 years was seen in only 17.5% of patients.⁸

People with celiac disease have a higher risk of early mortality, especially if diagnosis is delayed or the disease does not respond to treatment.⁷ The most common cause of early death is non-Hodgkin lymphoma, specifically enteropathy-associated T-cell lymphoma (EATL), though other complications also contribute to this increased risk.⁴,⁷

**Complications**

**Refractory Celiac Disease**

In refractory celiac disease, signs and symptoms persist for 12 or more months despite strict adherence to a gluten-free diet.⁹ The “strict adherence” part of the definition is significant because often cases thought to be refractory celiac disease turn out to be the result of inconsistent dietary habits or from dietary mistakes. Some processed foods are labeled poorly, and some patients have an incomplete understanding of what constitutes gluten-containing foods.⁸ Although most people with celiac disease can tolerate a small amount of gluten with no ill effects, reports exist of patients in whom mucosal abnormalities in the small intestine were seen with as little as 10 mg a day of gluten ingestion. According to Mooney et al, a regular diet in a healthy person contains around 13 g of gluten per day.⁸ If anti-EMA or anti-tTG antibodies still appear in blood tests, it is likely dietary inconsistencies are the cause of persistent symptoms, not refractory celiac disease.⁹

Other conditions such as irritable bowel syndrome or lactose intolerance also can cause symptoms that would appear to be refractory celiac disease. Irritable bowel syndrome in particular seems to appear in many celiac disease patients.⁸ Some of these conditions may actually result from damage to the small intestine caused by celiac disease. However, this does not qualify as refractory celiac disease, and correcting the condition itself (eg, IBS or lactose intolerance) will bring symptom resolution. If a gluten-free diet is verified through antibody tests, these other causes also should be investigated before making a diagnosis of refractory celiac disease.⁹

True refractory celiac disease is primary if there is never any improvement of symptoms with a gluten-free diet, or it is secondary if symptoms have improved for 12 or more months and then reappeared.

There are 2 types of refractory celiac disease, and they differ histologically by the appearance of their intraepithelial lymphocytes (IELs). IELs are specialized T cells found in the epithelial lining of several organs, including the biliary tract, the oral cavity, the upper respiratory tract and lungs, and the reproductive system. The largest group of IELs is found in the small intestine. These lymphocytes become activated and migrate from the crypts at the base of the villi up to the villous tip.

Type I refractory celiac disease has normal IELs, while the IELs in type II refractory celiac disease are abnormal in the types of receptors they carry and express.⁹ The distinction is important because adults with type II refractory celiac disease are at a significantly increased risk of EATL, and some reports consider type II refractory celiac disease itself to be low-grade lymphoma.⁸ Regardless, there is a clear risk for patients with type II refractory celiac disease to develop EATL (a significant cause of mortality among people with celiac disease), and they should be monitored carefully.

Type II refractory celiac disease is a rare condition. Because it causes a constant autoimmune response, the T and B lymphocytes (the main immune factors) are made and activated constantly. The increased risk of lymphoma in patients with this condition is likely because of this constant activation of the lymphocytes.¹⁰

Refractory celiac disease patients suffer from malnutrition because of the constant damage to their small intestine, resulting in the malabsorption of essential nutrients. They require nutritional support or correction in the form of supplements, and in advanced cases of malnutrition, total parenteral nutrition.⁸ Patients with type I refractory celiac disease have shown good response to steroid treatments, and although some type II patients have shown symptom relief on steroids, mucosal healing was not achieved.⁹ Because refractory celiac disease is so rare, studies of treatments are small, and no accepted treatment for this condition exists.

**Intussusception**

Intussusception is the “telescoping” or sliding of 1 portion of the intestine (the intussuscipiens) into another (the intussusciens). In the general population, intussusception is mostly a pediatric condition,
peaking in the first year of life. It is rare in adults. In general, the condition can occur in the small or large intestine and can cause intestinal obstruction although in celiac disease it is typically a “transient, nonobstructing condition” limited to short segments of the jejunum.\textsuperscript{11} In children, the condition is mostly unknown in origin, or idiopathic; in adults, there often is an underlying medical cause — and celiac disease is one such cause.\textsuperscript{11} Intussusception in celiac disease is thought to be the result of poor muscle tone in the damaged intestine.\textsuperscript{11}

**Ulcerative Jejunoileitis**

This condition, usually diagnosed in people older than 40 years of age, is considered a precursor lesion of EATL. It appears frequently in patients with type II refractory celiac disease.\textsuperscript{9} As the name implies, the jejunum is the part most frequently affected, followed by the ileum.

The mucosa in ulcerative jejunoileitis is chronically ulcerated and remains so, even in the absence of gluten in the diet.\textsuperscript{11} Presenting symptoms are similar to those of gastrointestinal (GI) lymphoma — loss of appetite, weight loss, and abdominal pain. Fever and night sweats also may be present.\textsuperscript{9} Ulcerative jejunoileitis can lead to hemorrhaging and intestinal perforation, and it may require surgery to repair such life-threatening complications.\textsuperscript{11} Ulcerative jejunoileitis shows the same abnormal IELs seen in type II refractory celiac disease, but unlike type II, the IELs in ulcerative jejunoileitis are only found in the ulcerated parts, instead of spread throughout the small intestine.\textsuperscript{9}

**Enteropathy-associated T-cell Lymphoma**

EATL is an aggressive and rare type of lymphoma, accounting for less than 1% of all non-Hodgkin lymphomas and less than 5% of all primary GI lymphomas.\textsuperscript{9} The 2 forms of EATL differ in their malignant cell type and presentation. Both forms arise from the IELs, but only EATL type I is associated with celiac disease; EATL type II has no known connection with celiac disease. Both types, however, have a poor prognosis, with 5-year survival rates not exceeding 20%\textsuperscript{9}.

EATL type I is specifically associated with a diagnosis of celiac disease in adulthood, and the mean age at cancer diagnosis is 64 years.\textsuperscript{9} Although in general this is a rare cancer, approximately 50% of people with type II refractory celiac disease develop EATL type I within 5 years of diagnosis, and EATL is a leading cause of mortality in adults with celiac disease.\textsuperscript{9}

There may be a common genetic association between type II refractory celiac disease and EATL.\textsuperscript{9} Not only is EATL strongly associated with the same HLA types associated with celiac disease, but type I refractory celiac disease and some cases of EATL (approximately 16%) are both associated with trisomy 1q (an extra copy of the q arm on chromosome 1). The exact mechanisms behind those genetic associations are unclear, but noteworthy.

Celiac disease patients who do not comply with dietary restrictions are at risk for EATL because symptoms persist in this patient population. Studies have shown that adherence to a gluten-free diet for 5 years evens the risk of cancer in patients who have celiac disease with that of the general population.\textsuperscript{9}

**Other Malignancies**

People with celiac disease are at an increased risk of other types of malignancies besides EATL. In general, the risk of developing other types of non-Hodgkin lymphomas, including B-cell non-Hodgkin lymphomas, is 3 to 4 times that of the general population.\textsuperscript{9} In addition, there is a significant increased risk of adenocarcinoma originating in the small intestine (see Figure 4), as well as squamous cell carcinoma of the pharynx or esophagus.\textsuperscript{9,11}

**CT Imaging of Celiac Disease**

Imaging of the small intestine is a challenge because this organ cannot be completely visualized with an endoscope. The former gold standard, the small bowel series, yielded little information beyond the surface layer of the small intestine, and it did not allow simultaneous examination of extraenteric organs and structures. The ability to visualize structures outside the small intestine is especially important to clinicians because conditions originating in the small intestine often cause complications extending outside the organ. Such is the case, for example, with celiac disease–associated lymphoma or celiac disease–associated cavitating mesenteric lymph node syndrome.

Although capsule endoscopy yields excellent results, it has significant drawbacks:

- The camera can become trapped in areas of stricture.
Depending on its field of view and the rate at which it captures images, the camera may miss focal areas of disease or bleeding it passes by.

Capsule endoscopy acquires a significant amount of images, making for a time-consuming review and interpretation. The advent of multidetector CT has initiated a switch to CT imaging as the new standard for abdominal studies. The speed of acquisition, cross-sectional imaging (the ability to visualize wall, lumen, and structure outside the small intestine), accuracy of multiplanar reconstruction, and overall improved image quality have made CT imaging the first choice for imaging patients presenting with abdominal complaints. However, the relative newness of its use in enteric imaging and the scarcity of imaging studies in celiac disease make CT imaging in celiac disease a constantly evolving arena.

The general (not disease-specific) goals of a successful CT scan of the small intestine should be:

- Complete visualization of the organ and adjacent structures.
- Adequate distention of the intestinal loops to enable visualization of wall and lumen abnormalities.
- Elimination of peristalsis and respiratory artifacts. Abdominal imaging is not required for the diagnosis of celiac disease. However, as mentioned previously, the presenting signs and symptoms of the disease are often very nonspecific; therefore, a patient with celiac disease is likely to undergo a CT scan. In patients with an established diagnosis, a CT scan is required if symptoms recur (raising suspicion of complications) or if the disease is refractory to monitor and rule out complications, especially malignancies.

Two important procedures used in enteric imaging are CT enterography and CT enteroclysis. Both techniques specifically attempt to distend the loops of the small intestine fully using high-volume contrast administration so both lumen and wall features can be visualized in detail.

CT Enterography

Unlike conventional abdominal CT, CT enterography uses a large volume of contrast material, ingested orally, to distend the small intestine fully and enhance the definition of the wall and lumen. The technique can be used with neutral oral contrast, with additional iodinated intravenous (IV) contrast administered 50 to 70 seconds prior to scanning. Neutral oral contrast, which gives the intestine lumen an attenuation that is near that of water, leads to excellent differentiation of the mucosa against the hypodense lumen, allowing for detection of even small lesions and tumors on the intestinal wall. Choices of neutral oral contrast include water, water/methylcellulose solution, polyethylene glycol, and a low-concentration barium solution mixed with sorbitol. The latter has a low attenuation (20 HU). However, this attenuation is low enough that it does not interfere with bowel wall enhancement. This contrast agent is not absorbed as quickly as water because of the inclusion of sorbitol, but patients may experience diarrhea shortly after the procedure if it is used. Nevertheless, it is considered the best choice for both enterography and enteroclysis.

Although water is a cheap and readily available neutral oral contrast material, the small intestine absorbs...
it quickly, and it may not provide adequate distention across the entire length of the small intestine for the duration of the procedure. In addition, because full distention requires use of a large volume of oral contrast (1.5-2 L), water is contraindicated in patients with renal deficiencies or heart failure. There are drawbacks to the other neutral contrasts: methylcellulose has a taste many patients find unfavorable, and polyethylene glycol induces bowel movements, and the urge to defecate may make the patient acutely uncomfortable during the examination.

Positive oral contrast also can be used in CT enterography, but its use with IV contrast may obscure wall thickening, an important finding in inflammatory small intestine conditions. This happens because wall enhancement with IV contrast cannot be well differentiated from a lumen filled with positive oral contrast. In addition, positive oral contrast should not be used if CT angiography is required (eg, if bleeding in the small intestine is suspected). Positive oral contrast masks evidence of GI bleeding.

A significant disadvantage of CT enterography is that the loops of the small intestine may not always be distended fully. Partial or poor distention can be misinterpreted as hyperenhanced mucosa or wall thickening, thus mimicking several pathological conditions such as Crohn disease. Partial or poor distention also can obscure other pathologies. An additional disadvantage is that some patients may not tolerate the high volume of contrast they must ingest prior to scanning, even when it is divided into equal doses, as it normally is prior to the scan.

**CT Enteroclysis**

CT enteroclysis is, according to Schmidt, “small bowel distension by administration of a high volume of contrast medium via a nasojejunal (NJ) tube followed by axial CT acquisition.” The insertion of the NJ tube makes CT enteroclysis an invasive procedure, which is why it is used mostly with adults.

The inspiration for CT enteroclysis came from comparative studies in Crohn disease, where abdominal CT results were compared with results of enteroclysis in the same patients. Enteroclysis is an x-ray examination that demonstrates the movement of contrast through the small intestine. Although abdominal CT was superior in showing wall and extraluminal complications, the high fluid volume of enteroclysis proved better at revealing the effects of the disease in the lumen itself. These effects included narrowing and obstruction of the lumen. Not surprisingly, CT enteroclysis developed as a method combining the best of both worlds.

Like CT enterography, CT enteroclysis takes advantage of multidetector CT technology. The speed of scanning with multidetector CT allows for thin-slice images to be acquired over a breath-hold, reducing the effects of breathing and peristaltic motion on the images. As with all radiographic imaging of the small intestine, including CT enterography, proper bowel cleansing before the procedure is crucial. The cleansing procedure might include a residue-free diet and laxatives the day prior to scan. Fasting before scanning is required.

On the day of the scan, a thin NJ tube is inserted under x-ray guidance until its tip reaches the duodenum. The diameter of the tube depends, in part, on the viscosity of the contrast material. A balloon normally is inflated at the tip to prevent reflux of the contrast material, which can increase the danger of vomiting. Tube placement usually takes place under conscious sedation, although not all centers offer it and not all patients choose to take this option. In the case of conscious sedation, the patient’s breathing should be monitored throughout the procedure for signs of respiratory depression resulting from the sedatives.

A possible challenge to the performance of CT enteroclysis lies in the distance between the CT suite and the x-ray room where the tube is placed. When the patient is transferred from the x-ray room to the CT room with the tube in place, excessive movement may cause the tube to be displaced.

Contrast considerations and choices, as well as required volume, are identical to those seen in CT enterography. However, because the high volume of contrast material is infused through the NJ tube at a steady rate (up to 200 mL/min), an automatic infusion pump typically is used. For detection of inflammatory processes, iodinated IV contrast is required. Therefore, in the case of CT scans where celiac disease is a known or suspected diagnosis, CT enterography or enteroclysis always includes IV contrast. As is the case with CT enterography, medication to relax the intestine or slow peristaltic motion can be helpful. The choices are hyoscine butylbromide (Buscopan) or glucagon.
CT enteroclysis overcomes the poor distention problem that comes with CT enterography. Concerns about radiation exposure because of the additional imaging required to insert the NJ tube have been raised, though reports of overall radiation doses for the procedure vary widely.\textsuperscript{15,17} Concerns remain about the cost of the procedure and about adverse events (ie, aspiration of contrast material and respiratory depression). To date, however, CT enteroclysis seems to be the most sensitive technique in CT enteric imaging.

**Normal Appearance of the Small Intestine**

After administration of IV contrast, the small intestine normally demonstrates uniform wall enhancement. Abnormal attenuation is discovered by comparing an abnormal segment to adjacent normal areas.\textsuperscript{15} The average wall density during the portal phase, following a distention with 500 mL of water, varies between 107 HU and 111 HU (±4), increasing during the arterial phase to 118 HU to 120 HU (±5).\textsuperscript{15} A lumen diameter greater than 3 cm is abnormal and indicates dilatation is present, assuming the loops are distented adequately.

**Imaging Uncomplicated Celiac Disease**

The appearance of jejunoileal fold pattern reversal was postulated as a specific celiac disease sign when barium studies were still the highest standard in small intestine imaging.\textsuperscript{18} Typically, the number of jejunal folds is greater than the number of ileal folds (see Figure 5). A jejunoileal fold pattern reversal is the occurrence of more ileal folds than jejunal ones (see Figure 6). In addition, imaging of patients with celiac disease sometimes can show a normal number of jejunal folds, but the number of ileal folds is increased to what would be a normal range in the jejunum.\textsuperscript{18}

When CT began to replace barium studies as the first-line imaging modality for nonspecific abdominal symptoms, Tomei et al sought to discover whether this pattern is equally apparent on CT and, if so, whether it could be equally useful.\textsuperscript{18}

Tomei and his team retrospectively reviewed CT scans from 22 consecutive patients with “clinically and histologically proven celiac disease.” All patients at the time of CT imaging were untreated, and all were diagnosed as adults. They ranged in age from 21 to 71 years. Their scans were compared to a group of

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\caption{CT enteroclysis in a 43-year-old man with uncomplicated celiac disease. Axial image shows reversed jejunal fold pattern with paucity of jejunal folds associated with greater number of ileal folds. Four jejunal folds per inch (arrows) and 6 ileal folds per inch (arrowheads) are visible. Reprinted with permission from the Radiological Society of North America and Dr Philippe Soyer, from Soyer P, Boudiaf M, Dray X, et al. CT enteroclysis features of uncomplicated celiac disease: retrospective analysis of 44 patients. Radiology. 2009;253(2):416-424.}
\end{figure}}
controls (n = 30; aged 21-78 years). The control group had abdominal CT scans, and intestinal disease was ruled out in all 30 cases.18 The radiologists reading the scans for Tomei et al were blinded to the participants’ diagnoses. Imaging was done following administration of diluted diatrizoate meglumine (Gastrografin) as oral contrast (1 participant in the celiac disease group refused and was imaged without contrast). Poor distribution of oral contrast prevented counting folds in 4 control participants. Jejunoileal fold pattern reversal was not seen in any participant in the control group.18

Tomei and his team counted the number of folds in 2.5 cm (1 in) at 3 different sites of the jejunum and 3 sites of the ileum for each participant (see Table 1). Folds could not be counted in 3 participants in the patient group because of gas, excessive fluid, or poor distribution of the contrast material. Complete jejunoileal fold pattern reversal was seen in 15 (68.2%) patient participants, and a normal number of jejunal folds but increased number of ileal folds was noted in 2 others. The difference between the jejunal fold numbers in celiac disease and control groups was statistically significant, as was the difference in ileal fold numbers (P < .001 in both cases).18 According to Tomei et al, jejunoileal fold pattern reversal “can be reliably seen at CT and can be used to suggest a diagnosis of celiac disease in clinically unsuspected adult patients.”18

Jejunoileal fold pattern reversal, as demonstrated on CT, now is considered a definitive diagnostic sign for celiac disease. In 2009 Soyer et al, using CT enteroclysis in 44 patients, found that jejunoileal fold pattern reversal had 100% specificity “and was the most discriminating independent variable for the diagnosis of uncomplicated [celiac disease] (odds ratio [OR], 39.9; P = .0001).”19

Following Tomei’s work, a combination of CT findings that includes jejunoileal fold pattern reversal, mesenteric lymphadenopathy (enlargement of the mesenteric lymph nodes), and intussusception was recognized as the most common CT presentation of celiac disease in adults. Tomei et al then sought to identify other CT findings that could trigger a suspicion of celiac disease in adults presenting with nonspecific abdominal symptoms.20 Their methodology was similar to that of their previous jejunoileal fold pattern reversal study, using 28 patients with confirmed celiac disease (aged 19-70 years) and 30 patients (aged 21-78 years) where intestinal disorders have been ruled out. They found that mild-to-moderate dilatation (3 or more segments of the bowel measuring more than 2.5 cm, with no stenosis upstream), combined with fluid excess in the lumen and dilution of oral contrast, was the most common combination after fold abnormalities.19 Fold abnormalities in that study included not only jejunoileal fold pattern reversal, but also total loss of jejunal folds or fold thickening — more than 2 mm thick.18 The study, as in their previous one, used abdominal CT with oral and IV contrast.

Soyer’s group — using CT enteroclysis in patients with uncomplicated celiac disease only — found that following fold abnormalities, splenic atrophy and vessel engorgement were other signs strongly correlating with uncomplicated celiac disease presentation in adults.19 They noted that splenic atrophy is present in up to 50% of adults with celiac disease.18

In 2011, Scholz et al published a comprehensive review correlating the physiology of celiac disease with corresponding CT findings.18 Pointing out that “pattern recognition for the diagnosis of small bowel diseases that create structural changes in the bowel wall is well accepted,” the authors argue that understanding the underlying celiac disease mechanisms at work in the small intestine and the mesentery would allow for a clearer interpretation of a collection of seemingly nonspecific findings on CT scans, all of which, when taken together, should clearly raise the index of suspicion toward celiac disease or its complications.20 The study, one of very few attempting to describe a complete CT pattern for the diagnosis of celiac disease, was based on reviews of patients’ charts and CT scans obtained between 1996 and 2009.

At the core of the disease process, according to Scholz et al, is “the small bowel malabsorption pattern.” The

### Table 1

**Comparison of Observed Folds**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. Jejunal Folds, range (mean)</th>
<th>No. Ileal Folds, range (mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4 to 6 (4.88 ± 0.78)</td>
<td>2 to 4 (2.84 ± 0.62)</td>
</tr>
<tr>
<td>Patients with celiac disease</td>
<td>0 to 6 (2.42 ± 1.61)</td>
<td>4 to 7 (5.11 ± 1.24)</td>
</tr>
</tbody>
</table>
process starts with the destruction of the villi, which normally absorb fluids, and enlargement of the crypts, which normally produce fluid. This process causes excess fluid to build up in the lumen, causing changes in the structure and tone of the small intestine’s wall, leading to the malabsorption pattern. On CT, the malabsorption pattern includes dilated, fluid-filled loops leading to dilution of contrast material. If barium-containing contrast is used, a phenomenon termed flocculation (flecks of hyperattenuating barium that precipitate in the loops) is seen. The peristaltic motion moves the fluid and contrast material in waves that create alternating low- and high-attenuation layers, similar to those seen in intussusception, but without the fat component present in intussusception. Additional pattern features include transient intussusception — the result of lost muscle tone in the small intestine’s wall — and jejunoileal fold pattern reversal.

In addition to the malabsorption pattern, Scholz et al points out that in celiac disease, lymphadenopathy is most pronounced in the upper mesentery because the bulk of the autoimmune attack takes place in the duodenum and proximal jejunum. The study considered lymph nodes to be prominent “when their cumulative axial area is greater than the axial area of adjacent blood vessels.” However, in fluid-filled loops with diluted contrast material, lymph nodes might not be observable.

Scholz et al’s review goes beyond other studies of CT imaging in celiac disease because it discusses a pattern extension into the colon as well. The authors argue that, when taken with the malabsorption pattern and prominent lymph nodes in the small intestine, the addition of the colonic malabsorption pattern strengthens the indications toward celiac disease.

The colonic malabsorption pattern on CT also is based, in part, on loss of muscle tone that affects the colon. As fluid pours in from the small intestine, it accumulates in the right colon, creating a “plume of fluid,” surrounded by air bubbles. In patients who eat a high-fat diet, fat adheres to the colon walls. In a sufficiently flaccid colon, large geodes of stool (> 4 mm) also could be noted, resulting either from lymphocyte infiltration or irritation from undigested food. The authors state that this colonic picture alone is not celiac-specific but becomes significantly more so if the small intestine malabsorption pattern and prominent lymph nodes also are present.

The wall of the duodenum and jejunum also can contain fat in celiac disease patients, a finding expanded by Scholz et al to the wall of the right colon, which connects to the ileum. These fat deposits, visible on CT images, might be the result of chronic inflammation that stimulates such a process.

A challenge in celiac disease imaging studies is the prevalence of other autoimmune conditions such as type 1 diabetes or systemic lupus erythematosus with celiac disease. These other conditions also can affect the small intestine and surrounding areas, changing CT findings in ways that may mask the typical signs pointing to celiac disease. In addition, the variety in the severity and presenting symptoms of the disease makes radiologic imaging difficult. No studies correlate disease severity with CT findings.

CT of Celiac Disease Complications

Type II Refractory Celiac Disease

Because type II refractory celiac disease carries with it complications that include EATL and other malignancies, recognizing the condition in CT imaging of patients with celiac disease is necessary. Unfortunately, as with uncomplicated celiac disease, imaging studies often are nonspecific, and few studies illustrate unique characteristics of type II refractory celiac disease, especially since the condition is fairly rare.

In 2007, Mallant et al published a study attempting to characterize type II refractory celiac disease and EATL on CT imaging. The study enrolled 2 groups: group 1 was composed of 24 patients with uncomplicated celiac disease or type I, and group 2 had 22 patients (15 with type II refractory celiac disease and 7 with EATL). The 46 patients with proven celiac disease ranged in age from 18 to 88 years and were referred for abdominal CT because of recurrent symptoms with or without suspicion of EATL.

All patients fasted overnight before the scans, and 43 of the patients received 900 mL of diluted barium sulfate solutions divided into 2 doses (the night before and the morning of the scan). Patients received additional oral contrast (300-500 mL) 45 minutes before the scans. Forty-two of the patients also received nonionic IV contrast 70 seconds before acquisition. The researchers were
unable to find specific individual signs of type II refractory celiac disease or EATL, but they found evidence that raised suspicion of type II and prompted careful monitoring and possible intestinal biopsy (see Table 2).

For example, more patients in group 1 had an increased number of small mesenteric blood vessels compared to group 2 ($P = .02$). The increase is thought to be the result of an acute rather than chronic inflammatory process. In addition, more patients in group 2 had wall thickening and intussusception.

Because intestinal diseases often are diagnosed based on a clinical composite of findings, the results of this study suggest that further investigation or follow-up monitoring for EATL is warranted. It probably is useful to screen people with type II refractory celiac disease with fludeoxyglucose F18 (FDG) positron emission tomography (PET) and CT imaging.

**Intussusception**

Adult cases of intussusception account for less than 20% of total intussusceptions cases. The presence of intussusception on an abdominal CT scan of an adult should always raise suspicion of celiac disease. In celiac disease, because the condition is most often transient and nonobstructive, diagnosis of this complication is more likely to be an incidental finding rather than a scan performed specifically to diagnose intussusception. As a result, it is important to recognize the possible appearance of this condition on CT, especially in patients requiring imaging for unspecified abdominal symptoms.

Intussusception has 3 forms: enteroenteric, ileocolic, or colocolic. Patients with celiac disease generally present with the enteroenteric form. The condition is transient, so a follow-up CT scan may no longer show it. Although it is accepted that intussusceptions shorter than 3.5 cm are always “benign” in origin, Sandrasegaran et al found much longer intussusceptions that were still transient and had no significant cause.

On a CT scan, intussusception appears as “a soft tissue mass with well-defined enhancing rim.” The shape of the defect appears different — round, oval, or elongated like a sausage — depending on the orientation of the intussusciptum (see Figure 7). On cross-sectional imaging, intussusception has been described as a “target lesion” (ie, round, resembling a target).

Sandrasegaran et al refer to 3 different CT appearance patterns of intussusceptions described in the literature.

The first pattern is the target lesion. The mesentery is drawn into the intussusceptum and appears as a soft tissue bulge with a central fat density. This presentation usually is not accompanied by dilatation or obstruction of the intussusciptums.

A more advanced presentation is the “sausage-shaped” mass. This mass consists of alternating high- and low-attenuation layers corresponding to the wall and mesenteric fat layers in the intussusception itself, respectively. These layers are pushed into each other as the intussusception forms. Another advanced presentation is that of a kidney-shaped mass that shows evidence of ischemia (eg, air in the wall or peritoneum). In the advanced stages of intussusception, which typically would not be seen in celiac disease patients, the mesenteric fat might not be visible because of edema, and obstruction may be evident.

Proximal small intestine intussusception is rare in adults but not in celiac disease patients. Sandrasegaran et al were among the first to describe CT findings of proximal small intestine intussusceptions, all in CT scans performed for other reasons. Of 24 adult patients (aged 24-65 years) enrolled

### Table 2

**Comparison of Patients With Classic Signs of Celiac Disease**

<table>
<thead>
<tr>
<th>Group</th>
<th>Smaller Spleen Volume</th>
<th>Lymphadenopathy</th>
<th>Intussusception</th>
<th>Increased Small Mesenteric Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (uncomplicated and type I, n = 24)</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>2 (type II and EATL, n = 22)</td>
<td>11</td>
<td>5</td>
<td>5</td>
<td>11</td>
</tr>
</tbody>
</table>

$^a < 122$ cm$^2$

Abbreviation: EATL, enteropathy-associated T-cell lymphoma
between 1997 and 2003, 2 patients had 2 intussusceptions each, bringing the total proximal small intestine intussusceptions examined on CT to 26. Of these cases, 22 were completely nonobstructive. A common CT finding in all 26 cases was central mesenteric fat within the lumen of the defect. In 4 cases, CT scans showed the mesenteric vessels also were drawn into the defect. Importantly, Sandrasegaran and his team showed that, unlike intussusception cases further down the digestive tract, proximal small intestine intussusception is a transient, nonobstructive defect.\textsuperscript{22} Their conclusion, although the study did not enroll patients with celiac disease, supports other observations of intussusception specific to patients with celiac disease, in which the findings of this defect on CT imaging often is incidental, and the condition does not persist. However, it is worth noting that intussusception could be triggered by a mass such as one caused by lymphoma — a particular concern in celiac disease patients. However, in such cases there should be other evidence on the CT scan to raise concern.\textsuperscript{22}

**Cavitating Mesentery Lymph Node Syndrome**

Cavitating mesentery lymph node syndrome is a rare but celiac disease–specific imaging finding. Prior to cross-sectional imaging, this condition usually was found during abdominal surgery or postmortem examinations.\textsuperscript{11} This condition has a high mortality rate (up to 50%)\textsuperscript{9} and often is associated with severe celiac disease.\textsuperscript{20}

The condition involves cystic changes in the mesentery lymph nodes. On CT scans, cavitating nodes usually appear with an atrophied spleen and severe villous atrophy. The lymph nodes are enlarged and vary in size, with a center attenuation of 0 HU or less. The nodes often have characteristic fat-fluid levels (see Figure 8).\textsuperscript{11} The fat-fluid levels help to distinguish these nodes from those seen in conditions such as tuberculosis or Whipple disease and are considered a specific celiac disease sign. It is unknown what causes this condition, although the constant exposure of the immune system to antigens because of the intestinal damage is thought to deplete the nodes.\textsuperscript{5}

**Malignancies**

Although the small intestine typically makes up 75% of the GI tract's length and 90% of its mucosal surface, malignancies originating there account for less than 2% of GI tract tumors.\textsuperscript{23} As mentioned previously, celiac disease patients are at an increased risk of specific small intestine cancers, most notably EATL.

CT enterography has proven efficient at finding up to 85% of both benign and malignant tumors of the
Computed Tomography of Celiac Disease

Telltale signs include wall thickening or enhancement, stricture, and a visible mass (see Figure 9). CT enteroclysis has proven to be as much as 100% sensitive when thin slices are used, although this number decreases to as low as 73% with larger slices in the 8 mm to 10 mm range.

Lymphadenopathy (lymph nodes measuring > 1 cm on the short axis) on a CT scan of a person with celiac disease should raise suspicion of lymphoma, and a biopsy could be warranted if other clinical signs are present. Ulceration of the small intestine wall, wall thickening, or the presence of a mass are additional signs of possible lymphoma seen on CT. As is the case with celiac disease in general and many of its complications in particular, the presenting CT findings in malignancies are not always very specific.

The difficulty of finding and diagnosing EATL in celiac disease patients led Hadithi et al to compare using FDG PET vs CT when evaluating patients with refractory celiac disease for the presence of EATL. The investigators enrolled 8 patients with EATL (aged 52-89 years) and 30 patients with refractory celiac disease (aged 44-71 years). This was a retrospective study between 2003 and 2005, predating the widespread use of the PET-CT. All enrolled participants received abdominal CT and whole-body FDG PET imaging. Patients fasted overnight before the CT scan and for 6 hours before the FDG PET scan.

For the abdominal CT examination, patients ingested 2 doses of 500 mL each diluted barium sulfate solution, administered the night before and the morning of the scan. They received an additional 200 mL of diluted barium sulfate and 100 mL IV iopromide (300 mg/mL) 15 minutes before scanning. The following were considered abnormal CT findings:

- Wall thickness greater than 3 mm.
- Lymph nodes greater than 1 cm on the short axis.
- Mesenteric fat infiltration.

In the group of EATL patients, FDG PET identified enhanced uptake in all patients and additional metastatic sites outside the GI tract in 2 patients. Abdominal CT showed abnormalities in 7 of the 8 subjects, with 1 false-negative finding. Abnormal CT findings included a thickened bowel wall in all 7 patients.

In the refractory celiac disease group, FDG PET showed increased uptake in 3 patients and equivocal readings in 3 additional patients. CT findings were
abnormal in 14 patients, 4 of whom were in concordance with the FDG PET positive or equivocal readings. None of the patients in the refractory celiac disease group had EATL. The sensitivity of FDG PET in Hadithi et al’s study was 100% to abdominal CT’s 87%. The specificities, with equivocal FDG PET readings counted as positive, were 80% for FDG PET and 53% for abdominal CT. Based on these results, the investigators recommended that patients with refractory celiac disease be evaluated with both modalities, and positive FDG PET results should be followed up with biopsies from the areas of increased uptake.

Lymphoma tumors arising in the small intestine have a variety of appearances on CT. The most common is an aneurysmal dilatation of the lumen with surrounding wall thickening, which appears in more than 50% of lymphoma tumors in the small intestine. Another characteristic appearance is a cavitating mass, resulting from ulceration of the mucosa extending into the tumor. A lymphoma tumor demonstrates poor contrast enhancement. Some lymphoma tumors manifest as nodules in the small intestine, but very small tumors may be missed during CT imaging. Similarly, some lymphoma tumors could be mistaken for benign polyps. However, the presence of lymphadenopathy with these polyps should raise suspicion of lymphoma.

Adenocarcinoma, another common cancer in celiac disease patients, may appear on CT as a soft tissue focal mass. Wall thickening, dilatation, and narrowing of the lumen also may be noted.

**Conclusion**

Celiac disease once was considered a pediatric condition, but it is increasingly recognized as a common autoimmune condition affecting people of all ages. Furthermore, the systemic effects of celiac disease and the possible severe ramifications — up to and including increased mortality rates per age group — are becoming better understood.

Adults with celiac disease may present for CT imaging with unspecified abdominal symptoms and without a definitive diagnosis. Although malabsorption symptoms of diarrhea and weight loss occur in adults with celiac disease, quite a few are constipated, and a considerable percentage is obese. While many CT findings are not celiac disease–specific, it is important to be aware of those that are and of telltale combinations — or patterns — of findings that may lead to an accurate diagnosis. Jejunal fold pattern reversals, or any small intestine fold abnormalities, are probably the most specific CT findings indicating celiac disease in adults. Intussusception in an adult patient is another strong indication that should significantly raise suspicion for celiac disease.

In patients with celiac disease, many of the effects of an immune system gone awry can be reversed with strict adherence to a gluten-free diet. Such a significant dietary change is not easy. It is still a challenge to find gluten-free products at food stores and restaurants, and the items are expensive. However, it is important that a professional dietician outline for the patient and family the possible risks of continuing to ingest gluten and the immediate and long-term benefits of strictly adhering to this diet. In particular, the significantly increased risk of malignancy that follows nonadherence should be emphasized.

Because reversing the damage from celiac disease can literally mean the difference between life and death, recognizing the disease and its complications early is crucial. Because the initial presentation of the disease (even in CT images) is often nonspecific, it is important to construct the entire clinical picture — imaging findings, physical complaints, and family and personal medical history — to reach the correct diagnosis. Increased awareness benefits the patient sooner.

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**References**


Computed Tomography of Celiac Disease

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*Your answer sheet for this Directed Reading must be received in the ASRT office on or before this date.

Read the preceding Directed Reading and choose the answer that is most correct based on the article.

1. Celiac disease is an autoimmune disease that causes inflammation and destruction in the small intestine, specifically the:
   a. colon.
   b. duodenum.
   c. jejunum.
   d. ileum.

2. The density of fat in the mesentery is:
   a. 100 HU to 160 HU.
   b. 30 HU to 45 HU.
   c. 0 HU.
   d. -100 HU to -160 HU.

3. Prolamines from rye and barley that trigger celiac disease are high in which amino acid?
   a. glutamine
   b. proline
   c. phenylalanine
   d. lysine

4. Young children with celiac disease present with which of the following?
   1. chronic diarrhea
   2. vomiting
   3. weight gain
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

5. Osteoporosis resulting from celiac disease is a condition that is:
   a. reversible in children with adherence to a gluten-free diet.
   b. reversible in adults with adherence to a gluten-free diet.
   c. irreversible.
   d. irreversible and indicates malabsorption.
6. In refractory celiac disease:
   a. abnormal intraepithelial lymphocytes infiltrate the wall of the small intestine.
   b. ulcerative jejunoileitis develops.
   c. symptoms persist and intussusception appears.
   d. signs and symptoms persist for 12 or more months despite dietary adherence.

7. There may be a genetic association between ______ refractory celiac disease and ______ .
   a. type I; enteropathy-associated T-cell lymphoma (EATL)
   b. type II; EATL
   c. type I; ulcerative jejunoileitis
   d. type II; ulcerative jejunoileitis

8. A major challenge in endoscopy of the small intestine is the:
   a. difficulty visualizing the entire organ.
   b. cost.
   c. taste of barium.
   d. expertise required.

9. ______ is the first choice for imaging patients presenting with abdominal complaints.
   a. Endoscopy
   b. A small bowel series
   c. Computed tomography (CT) imaging
   d. Positron emission tomography (PET)

10. Which is not a primary general goal of a successful CT scan of the small intestine?
    a. complete visualization of the organ and adjacent structures
    b. adequate distention of the intestinal loops to enable visualization of wall and lumen abnormalities
    c. elimination of peristalsis and respiratory artifacts
    d. identification of abnormal wall thickening

11. For detection of inflammatory processes, an intravenous (IV) contrast is needed in CT scans of suspected or known celiac disease.
    a. true
    b. false

12. Imaging of patients with celiac disease sometimes can show a ______ number of jejunal folds, but the number of ileal folds is ______ to what would be a normal range in the jejunum.
    a. normal; decreased
    b. decreased; increased
    c. normal; increased
    d. increased; decreased

13. Which of the following CT findings are strongly associated with uncomplicated celiac disease?
    1. fold abnormalities
    2. splenic atrophy
    3. vessel engorgement
    a. 1 and 2
    b. 1 and 3
    c. 2 and 3
    d. 1, 2, and 3
14. The malabsorption pattern in the small intestine has what effect on contrast material?
   a. Contrast material is diluted.
   b. Contrast material is concentrated.
   c. Contrast media is absorbed.
   d. There is no effect.

15. Flocculation is the concentration of barium solution in the small intestine loops.
   a. true
   b. false

16. ________ could cause changes to or masking of typical CT findings in celiac disease.
   a. Adenocarcinoma
   b. Lymphoma
   c. The presence of other autoimmune diseases
   d. Liver cirrhosis

17. In the 2007 Mallant study, a pattern of CT findings that may raise suspicion of type II refractory disease includes:
   a. lymphadenopathy, wall thickening, and intussusception.
   b. lymphadenopathy, wall thickening, and ulcerative jejunoileitis.
   c. lymphadenopathy, ulcerative jejunoileitis, and atrophied spleen.
   d. atrophied spleen, ulcerative jejunoileitis, and intussusception.

18. In which form does intussusception in patients with celiac disease usually present?
   a. enteroenteric
   b. ileocolic
   c. colocolic
   d. nontransient

19. According to Sandrasegaran, a first presentation of intussusception on CT has what appearance?
   a. reniform
   b. sausage-shaped
   c. target lesion
   d. oval

20. Cavitating mesentery lymph node syndrome is associated with which of the following conditions?
   a. uncomplicated celiac disease
   b. type I refractory celiac disease
   c. adenocarcinoma
   d. severe celiac disease

21. What is the center attenuation of affected lymph nodes in cavitating mesentery lymph node syndrome?
   a. 0 HU or less
   b. 45 HU
   c. 100 HU
   d. 115 HU or more

22. Which of the following are a sign of a malignancy in the small intestine on CT images?
   1. transient intussusception
   2. wall enhancement
   3. wall thickening
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3
23. In lymphadenopathy as visible on CT, the lymph node measurements are ______ than 1 cm on the ______ axis.
   a. less; short
   b. greater; short
   c. less; long
   d. greater; long

24. The most common CT appearance of a lymphoma tumor in the small intestine is:
   a. ulceration and cavitation.
   b. aneurysmal dilatation of the lumen with surrounding wall thickening.
   c. diffuse ulceration across the wall.
   d. soft tissue mass extending into the mesentery.

25. On CT, ______ is a possible appearance of adenocarcinoma.
   a. soft tissue focal mass
   b. aneurysmal dilatation of the lumen with surrounding wall thickening
   c. focal mass with lymphadenopathy
   d. wall thickening with ulceration