Thickened gastrointestinal wall findings on computed tomography: simplifying the diagnosis.

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Learning objectives

1. To demonstrate preferable CT protocols and way of MDCT interpretation of gastrointestinal (GI) wall thickening.
2. To separate bowel wall thickening into semiological categories based on CT findings to aid in narrowing the differential diagnosis.
Background

- With the development of multidetector computed tomography scanners (MDCT), computed tomography became an important imaging tool in the detection and characterization of bowel abnormalities.
- GI thickening is a nonspecific term that can refer to infectious, inflammatory, vascular, neoplastic and many other processes. The attenuation of the bowel wall and associated signs can be used in conjunction with clinical and endoscopic findings to provide a reasonable diagnostic approach.
- We illustrate through case, the different etiologies and CT signs of small bowel and colonic parietal damage.
Findings and procedure details

Normal MDCT anatomy of the Gastro-intestinal tract (fig.1)

Normal bowel wall is sharp outline, well differentiated from the peri-intestinal fat; in spontaneous contrast it is homogeneous density, tissue type, without identifiable layers.

Acceptable bowel wall thickness values on CT strongly depend on the degree of bowel distension and vary widely in the literature: the small bowel wall should not exceed 3 mm despite luminal distention, and the colonic wall can vary from 1 to 2 mm when the lumen is well distended to 5 mm when the wall is contracted or the lumen is collapsed. The bowel wall normally enhances after the administration of intravenous contrast material. The mucosa is the most intensely enhancing layer of the bowel wall and when enhanced may appear as a distinct layer. In contrast, the sub mucosa is less vascularized and is seldom seen as a separate structure on CT scans unless it is edematous, hemorrhagic or infiltrated by fat (fig.2 and fig.3).

CT acquisition protocol:

The protocol must be adapted to each situation and clinical question. The review protocol depends on the gut segment to review. In all cases, the distension of the digestive lumen provides a large difference in contrast between the light and the enhanced intestinal wall; which allows an accurate measurement of the thickness of the stretched walls. We usually proceed to filling colon with water-soluble contrast agent; and for small bowel enteroclysis (filling intestine with nasojejunal tube) the most recommended technique. The four phases of exploration (spiral acquisition mm slices) each have a role to clarify the nature of intestinal inflammatory lesions: - Phase without iodized contrast medium injection research spontaneous density anomalies (calcification, blood). The multiphase acquisition after injection (1.5 to 2 ml / kg body weight with an injection rate of 3 ml / sec) comprises: - Delayed arterial phase (~ 45 sec) studying vascular anomalies (hemorrhage, arterial thrombosis, early enhancement) - Portal phase (70-80 sec) corresponding to the digestive parenchymal time - Late-phase equilibrium (120- 300 sec) studying the enhancement of fibrous process. Analysis of the kinetic enhancement of parietal abnormalities can characterize tissue quotas encountered: Wall Thickening spontaneously high attenuation pattern = hematoma; No enhancement = ischemic necrosis; Late Enhancement = fibrous quota.

The transverse axial views are complemented by multiplanar reconstructions, 2D and 3D reconstructions.

Criteria thickenings analysis:
When thickening of the bowel wall is identified on CT, several imaging features must be assessed in order to narrow the differential diagnosis:

- Wall thickness (in mm).
- Character circumferential or not.
- Topography and number: single or multiple thickening.
- Extended length.
- Connection with the healthy zone.
- Spontaneous homogeneity and after contrast enhancement mode.

Extra-parietal signs:

- Fat infiltration peri-intestinal, peritoneum and mesentery.
- Locoregional lymph nodes and / or retroperitoneal.
- Achievement of adjacent structures.

This semiotic analysis often differentiates the CT appearance of tumor lesions and the number of inflammatory lesions.

**Approach to the thickened bowel wall:** When thickening of the small or large bowel wall is identified on CT, the first step to take is to access the extent of the involved bowel. Distinction should be made between:

- Focal (less than 5 cm of extension)
- Segmental (6-40 cm)
- Diffuse (>40 cm) involvement.

This is an important step in differentiating between benign and malignant causes of bowel wall thickening: while most bowel tumors present as a focal involvement, segmental and diffuse thickening of the bowel wall are usually caused by benign conditions. The exception is a small bowel lymphoma, which typically shows as a segmental distribution.

**Focal thickening of the bowel wall:** Focal thickening may be caused by tumors or by inflammatory conditions, and distinguishing between the two conditions should be attempted. In addition to the clinical presentation, analysis of the wall symmetry, degree of thickening and perienteric abnormalities provides additional information for the correct diagnosis. In the setting of focal wall thickening three main scenarios may occur:

1. Asymmetric focal thickening of the bowel wall Asymmetric thickening of the bowel wall corresponds to different degrees of eccentric thickening around the circumference of the involved segment and is typically caused by neoplasms.

   **Malignant tumors** of the gastrointestinal tract Neoplasms have a chronic onset and may present as an eccentric focal mass or, more commonly, as a circumferential asymmetric thickening, usually greater than 3 cm in thickness. Contrast enhancement of malignant bowel tumors is frequently heterogeneous with areas of low attenuation due to ischemia
and necrosis. In addition, regional adenopathy and distant metastases, when present, support the diagnosis (fig.4).

_Gastrointestinal tuberculosis_ The inflammatory reaction usually produces eccentric wall thickening or a mass like lesion. Discontinuous areas of mural thickening with associated luminal narrowing in the small bowel are also common and in combination with ileocaecal involvement should suggest the diagnosis. Large perienteric lymph nodes of low attenuation due to caseous necrosis are also common and characteristic (fig.5). In addition, thoracic features of tuberculosis and other abdominal signs of involvement such as findings of peritonitis and hepatosplenic dissemination support the diagnosis.

_Crohn’s disease_ typically involves the right colon and the terminal ileum. Wall thickening in Crohn’s disease is usually eccentric or asymmetric because of preferential involvement along the mesenteric border of the bowel wall. Imaging features suggesting this diagnosis include the discontinuous involvement of the bowel wall ("skip areas"), signs of transmural inflammation such as fistulas and abscesses, and proliferation of the fat along the mesenteric border of the bowel (fig.6)

2. Symmetric focal thickening of the bowel wall

Circumferential and symmetric thickenings of the bowel wall are features usually attributed to benign conditions such as inflammatory, infections, bowel edema and ischemia. However, neoplasms such as well-differentiated or small adenocarcinomas may also display symmetric and homogeneous thickening of the bowel wall and should be considered especially when the thickened bowel has a focal extension and no significant perienteric fat stranding is seen.

Perienteric abnormalities (fat stranding) disproportionately greater than the degree of bowel wall thickening: This is a helpful clue in narrowing the differential diagnosis to mainly four conditions:

Diverticulitis: Diverticulitis occurs when the neck of a diverticulum becomes occluded, resulting in micro perforation and pericolonic inflammation. CT findings of acute diverticulitis include inflamed diverticula in combination with pericolonic fat stranding, which is more severe than the mild focal thickening of the adjacent bowel wall. Engorgement of the mesenteric vessels ("centipede" sign) and the presence of fluid at the base of the sigmoid mesentery ("comma sign") are two indicative signs of the inflammatory process (fig.7).

Epiploic appendagitis:

Acute epiploic appendagitis results from the torsion or venous occlusion of the epiploic appendage and is more frequent in the sigmoid colon. CT findings of epiploic appendagitis include the presence of a fat-density lesion corresponding to the inflamed appendix with surrounding inflammatory changes (fig.8).
Acute appendicitis:

Acute appendicitis occurs when the appendiceal lumen becomes occluded, resulting in inflammation, ischaemia and eventually perforation. CT findings of acute appendicitis include a fluid-filled dilated (>6 mm in diameter) appendix, thickness of the wall, and mild to moderate peri-appendicular fat stranding (fig.9).

Segmental or diffuse bowel wall thickening

It is typically secondary to benign conditions and usually does not exceed 10 mm in thickness from the luminal to the serosal surface.

The exception is the lymphoma, which despite being a malignant condition may present with a segmental distribution causing circumferential symmetric thickening of the bowel wall and homogeneous low attenuation after intravenous contrast administration (fig.10).

In the setting of segmental or diffuse bowel wall thickening, one of three attenuation patterns after intravenous contrast administration may occur:

Stratified pattern of attenuation

In this pattern, two (double halo sign) or three (the target sign) concentric and symmetric layers of alternating densities are recognized on the thickened bowel wall after intravenous contrast administration. This pattern indicates inflammation or ischaemia of the bowel where the inner and outer high-density layers correspond to the hyperemic mucosa and serosa, respectively, while the low-density layer presumably represents the oedematous submucosa.

Clinical presentation and adjacent findings such as perienteric findings help in narrowing the differential diagnosis:

Bowel ischemia

Although bowel wall thickening is a common finding in cases of bowel ischaemia (fig.11), the ischaemic bowel wall may also appear paper thin, particularly in cases of acute arterial occlusion (fig.12).

This results from oedema of the submucosa and hyperaemia or hyperperfusion of the mucosa and/or muscularis propria. This finding should be judged in the clinical context and associated imaging findings of bowel ischaemia, such as occlusion of the mesenteric artery or vein, bowel dilatation, engorgement of the mesenteric veins, and mesenteric oedema and ascites.

Intestinal pneumatosis and gas in the mesenteric or portal veins are indicative of severe ischaemia (fig.13) and are usually associated with the thinning rather than thickening of the small bowel wall due to bowel wall necrosis.
Idiopathic inflammatory bowel disease

Bowel wall thickening with a stratified pattern may be also seen in both ulcerative colitis (rectum in 95% of cases) (fig.14) and Crohn's disease (fig.15), indicating acute, active disease. Thus, wall thickening and pericolonic involvement are not as extensive in ulcerative colitis (fig.16) as they are in Crohn's disease.

Infectious enteritis or colitis and pseudomembranous colitis

In most cases of infectious enteritis the small bowel wall appears normal or mildly thickened.

By contrast, infectious colitis typically manifests with significant wall thickening, which may demonstrate either homogeneous enhancement or a striated pattern due to intramural oedema.

Stranding of the pericolic fat and ascites are also commonly seen (fig.17).

White pattern of attenuation

The white pattern is caused by intense enhancement of the bowel wall when its density is equal to or greater than that of venous vessels in the same scan.

This pattern can be seen mainly in two clinical entities: ischaemia and inflammatory bowel disease (fig.18, 19 and 20).

Grey pattern of attenuation

The grey pattern of attenuation indicates mild to diminished enhancement of the bowel wall and is considered when the attenuation of the bowel wall is similar to that of the muscle on contrast-enhanced scans. Intestinal ischemia: This pattern is particularly common in cases of mesenteric venous occlusion and bowel obstruction, where the bowel oedema is more pronounced due to venous congestion. In patients with chronic Crohn's disease or chronic radiation enteritis (fig.21), involved bowel loops may show diminished enhancement due to the development of transmural fibrosis.
**Fig. 1:** Normal pattern of the digestive wall: small bowel wall should not exceed 3mm with intense enhancing mucosa (arrow). The colonic wall can vary (1 to 5mm) but should be regular with homogeneous enhancement (astriks).

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Fig. 2: less vascularized submucosa on CT scan (arrows) due to oedema.

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Fig. 3: Enteroclysis, axial CT: before and after contrast injection, there is a significant difference in contrast between light and enhanced bowel wall.

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Fig. 4: Malignant tumors: axial and coronal enhanced CT scans shows a asymmetric thickening of the cecum (large arrow) and a mesenteric lymph nodes (arrow)

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Fig. 5: Intestinal tuberculosis: axials enhanced CT scans shows an eccentric wall thickening in cecum (large arrow) with large mesenteric lymph nodes with one presented a low attenuation due to caseous necrosis (arrows)

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Fig. 6: Crohn's disease: two axial and two coronal enhanced CT scans shows the discontinuous involvement of the bowel (colon and ileum bowel; arrows) wall adjacent lymphadenopathy and proliferation of the fat along the mesenteric border of the bowel "Comb aspect of the mesentery".
**Fig. 7:** Diverticulitis: axial and coronal enhanced CT scan show Symmetric focal thickening of the bowel wall with homogeneous enhancement and a mesenteric signs of inflammation.

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**Fig. 8:** Para coeccal appendagitis: presence of a fat-density lesion corresponding to the inflamed appendix with surrounding inflammatory changes (arrow).

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**Fig. 9:** Retro cecal acute appendicitis: dilated appendix, thickness of the wall, and per-appendicular fat stranding.

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Fig. 10: Lymphoma of the ileum: circumferential symmetric thickening of the bowel wall and homogeneous low attenuation after intravenous contrast administration (arrow). Note the associated ascites (asterisk).

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**Fig. 11:** Bowel ischaemia: bowel wall thickening (arrows). Mesenteric artery occlusion (large arrow).

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**Fig. 12:** Bowel ischaemia: thin bowel wall in acute arterial ischemia

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Fig. 13: Severe ischemia: Intestinal pneumatosis and gas in the mesenteric veins

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**Fig. 14:** Acute ulcerative sigmoiditis in HRC.

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Fig. 15: Stratified bowel wall thickening (arrow) indicating acute, active Crohn’s disease

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Fig. 16: Thickened and stratified bowel wall causing obstruction in ulcerative colitis.

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**Fig. 17:** Crohn's disease.

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Fig. 18: Severe enteritis in immunodeficiency disorder.

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**Fig. 19:** Inflammation of small bowel due to vasculitis: Rheumatoid Purpura

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**Fig. 20:** Hypertrophic gastropathy and enlarged mucosal folds

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**Fig. 22:** Chronic Crohn’s disease: diminished enhancement of the bowel wall (arrows) due to the development of transmural fibrosis. Note inter enteric fistula (large arrow).

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Conclusion

Most acute gastritis and enteritis have similar CT presentation. The discovery of a scanner wall thickening of the stomach and/or small bowel, facing an array of acute abdomen, should suggest a wide range of causes. Systematic analysis of the characteristics of this thickening, including its topography and overall assessment of the abdominal cavity addition discriminating evidence to confirm the etiologic diagnosis or restrict diagnostic hypotheses.
Fig. 21: Algorithm approach to the bowel wall thickening. Adapted from the electronic poster "Bowel wall thickening-a complex subject made simple" DOI: 10.5444/esgar2011/EE-063

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