Repetitio mater studiorum est: Magnetic Resonance Enterography (MRE) in Crohn's Disease - a pictorial essay.

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Learning objectives

1. To emphasize the role of Magnetic Resonance Enterography (MRE) in diagnostic algorithm in Crohn's Disease (CD).
2. To illustrate imaging signs of active and chronic bowel disease as well as its complications, both mural and extramural, also in reference to structured reporting.
3. To briefly discuss MRE imaging protocol in reference to the one applied at our Department.

Background

Crohn's disease (CD) is an idiopathic chronic inflammatory disease of the gastrointestinal tract often encountered in young adults with tendency to frequent relapses. CD may affect any part of the digestive tract. However, the small intestine, in particular the terminal ileum, is most commonly affected. Disease is characterized by so called skip-lesions - areas of inflammation separated with regions of normal mucosa. Inflammation most commonly manifests as ulceration, varying from superficial lesions to deep linear ulcers. As it is often a transmural process complications such as bowel obstruction, fistulæ formation and abscesses are encountered. These findings allow to typically classify CD as primarily active inflammatory, penetrating of fibrostenotic disease. Treatment differs depending on the assessment of disease activity. Active inflammatory disease without penetrating component is generally treated with immnunosuppressants and steroids, penetrating disease is treated mainly with antibiotics and biologics and when an obstructing stricture is present it is usually treated surgically unless active inflammation coexists.

According to the joint consensus guidelines of European Crohn's and Colitis Organisation (ECCO) and European Society of Gastrointestinal and Abdominal Radiology (ESGAR) on the diagnosis and management of Inflammatory Bowel Disease (IBD) a single gold standard for the diagnosis of CD is not yet available. The diagnosis is established by clinical assessment combined with endoscopic, histological, radiological and biochemical findings. Computed tomography (CT) and Magnetic Resonance (MR) examinations are current standards for assessing CD, showing comparable diagnostic accuracy for the detection of small intestine inflammatory lesions. Both imaging techniques allow evaluation of the extension and activity of the disease based on wall thickness and increased enhancement after intravenous contrast media administration. Both are suitable to detect extraluminal complications. CT is less time-consuming and more easily available than MR. Advantages of MR include lack of ionizing radiation and improved...
soft tissue contrast resolution compared with CT as well as the ability to image the small
bowel dynamically while its limitations are higher cost, lower spatial resolution and greater
susceptibility to motion-related blurring and artifacts. However, in view of frequent follow-
up studies and lack of radiation exposure MR should be considered a method of choice
if accessible. A recent study by Hafeez et al. showed that MRE had a positive diagnostic
impact in patients under investigation for CD and influenced therapeutic strategy in 61%
of the patients. MRE shows an overall sensitivity of 85% for active inflammatory disease
(Lee et al. 2009) and correlates well with the surgical findings of stricture, abscess and
fistula with sensitivities of 95, 92, 72% and specificities of 72, 90, 76% respectively (Pozza
et al. 2011).

**Findings and procedure details**

We have retrospectively reviewed MRE examinations performed in years 2012-2014
at our Department and further selected those of 128 patients with CD confirmed with
histopathological examination. All examinations have been performed in a 1.5-T scanner,
using a phased array surface coil.

The recipe for optimal MRE includes adequate bowel distention, fast sequences, use of
a large field-of-view surface coil and administration of an intravenous contrast agent.

**Adequate bowel distention.** Several types of enteric contrast agents (CA) may be used
to study the small bowel with MR imaging - see Table 1. At our institution we administer
3% mannitol solution in 1-1.5 l. of water at regular intervals over a 40 to 60 minute period
prior to examination in adults patients. In paediatric population the volume of oral contrast
medium is adjusted according to the weight of the patient.

It may also be helpful to administer 10 - 20 mg of hyoscine butylbromide intravenously
prior to the examination or prior to contrast-enhanced sequences, depending on the
inclusion of cine sequences in the imaging protocol, if otherwise not contra-indicated (e.g.
history of cardiac arythmia, glaucoma) to decrease bowel motility. Alternatively you can
administer 1 mg glucagon intravenously or intramuscular if applicable. Contra-indications
to glucagon administration include a known hypersensitivity to glucagon or a history of
pheochromocytoma.

It is advised that patients fast for 4 to 6 hours prior the examination as it not only helps
to decrease the amount of food residue but also facilitates tolerance for the necessary
intake of large volume of enteric contrast.
### Table 1. Enteric contrast agents (CA) in MRE (after Allen et al. 2014 with alterations).

<table>
<thead>
<tr>
<th>Type of CA</th>
<th>Negative CA</th>
<th>Positive CA</th>
<th>Biphasic CA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signal intensity on T1-weighted images.</td>
<td>Low.</td>
<td>High.</td>
<td>Low.</td>
</tr>
<tr>
<td>Signal intensity on T2-weighted images.</td>
<td>Low.</td>
<td>High.</td>
<td>High.</td>
</tr>
<tr>
<td>Remarks</td>
<td>Provide ability to visualize higher signal intensity bowel wall against dark contents in the lumen.</td>
<td>Can mask bowel wall abnormalities.</td>
<td>Provide optimal contrast between the bowel lumen and its wall without obscuring bowel wall enhancement on T1-weighted images.</td>
</tr>
</tbody>
</table>

**Ferumoxil oral suspension.** Based on gadolinium chelates and manganese. Blueberry juice, milk.

Water, polyethylene glycol, diatrizoate meglumine, mannitol, locust bean gum solutions, methylcellulose, low-density barium suspensions.

### MRE imaging protocol.

Patients are imaged in the supine or prone position (if no stoma is present) using a phased array surface coil - see Fig. 1. Coronal sequences are faster to perform in the prone position due to thinner volume of tissue to imagine. Both positions, however, are equivalent in detection and characterization of bowel lesions. With the exception of diffusion weighted sequences (DWI) all scans are performed at breath-hold.

MRE protocol includes sequences post intravenous administration of 0.1 mmol/kg gadolinium-based contrast agent at 2ml/s followed by a 20 ml saline flush, both dynamic and delayed.

Table 2 displays the list of sequences used in our MRE protocol as well as their order and rationale. At times we extend our imaging protocol during the examination if we find it necessary for stating the correct diagnosis.
<table>
<thead>
<tr>
<th>Sequence</th>
<th>Remarks and Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 T1 FL2d Cor(^1) mbh(^2)</td>
<td>Encompasses the whole abdomen and pelvis. Provides anatomic overview and preliminary assessment of bowel distention.</td>
</tr>
<tr>
<td>2 T1 FL2d FS(^3) Cor mbh</td>
<td>Provides anatomic overview and preliminary assessment of bowels, incl. bowel walls.</td>
</tr>
<tr>
<td>3 T2 HASTE(^4) Cor mbh</td>
<td>Localization of findings. Susceptible to artifacts - bulk motions of intraluminal fluid may simulate filling defects. See: Fig. 2.</td>
</tr>
<tr>
<td>4 T2 HASTE STIR(^5) Cor mbh</td>
<td>Useful in evaluation of bowel wall oedema and intra-abdominal fluid collections. The use of fat saturation in one of T2-weighted sequences allows differentiation between submucosal fat and oedema, both bright on T2-weighted images.</td>
</tr>
<tr>
<td>5 T2 Trufi(^6) Cor bh(^7)</td>
<td>Chemical shift artifacts result in a &quot;black boundary&quot; effect around structures what makes mesenteric nodes and vessels more conspicuous but may simulate bowel wall thickening. See: Fig. 3b. Provides good delineation between the bowel and the mesentery.</td>
</tr>
<tr>
<td>6 T2 HASTE Tra(^8) mbh</td>
<td>Allows multiplanar correlation.</td>
</tr>
<tr>
<td>7 DWI(^9) Cor (b 0 50 500 800)</td>
<td>Displays areas of diffusion restriction in case of active inflammatory lesions and abscesses. Facilitates detection of lymph nodes.</td>
</tr>
<tr>
<td>8 T1 FL2d Tra mbh</td>
<td>Pre-contrast sequence.</td>
</tr>
<tr>
<td>9 T1 Vibe(^10) FS Cor dyn(^11) mbh (CM dynamic)</td>
<td>Performed in the arterial (30 s) and portal venous (60-70s) phases using a bolus triggering once contrast reaches descending aorta. Subtracted images are obtained as well. Slice thickness 1.6 mm.</td>
</tr>
</tbody>
</table>
T1 Vibe FS Cor mbh (CM delayed) Post-contrast delayed sequence. Facilitates to demonstrate extraluminal abnormalities such as fistulas. Slice thickness 1.6 mm.

T1 FL2d Tra mbh (CM) Allows multiplanar correlation and facilitates to demonstrate extraluminal abnormalities such as fistulas.

Examinations are performed in a 1.5-T scanner with the use of a phased array surface coil.

1 Cor - coronal plane, 2 mbh - multiple breath hold, 3 FS - fat saturation, 4 HASTE - half-Fourier acquisition single-shot turbo spin-echo, 5 STIR - short tau inversion recovery, 6 Trufi - true fast imaging with steady-state free precession, 7 bh - breath hold, 8 Tra - transverse plane, 9 DWI - diffusion weighted imaging, 10 Vibe - volumetric interpolated breath-hold examination, 11 dyn - dynamic imaging post intravenous contrast agent administration.

Interpretation of MRE images requires familiarity with the normal anatomy as well as imaging signs of active and chronic bowel inflammation.

Normal appearance of bowels in MRE. The small intestine is most commonly affected by CD. Work by Cronin et al., who retrospectively evaluated 65 subjects with no underlying small bowel disease followed up over a period of three years, established that the mean diameters of the duodenum, jejunum, proximal ileum, distal ileum and terminal ileum measure 25 mm, 24.5 mm, 19.5 mm, 19 mm and 19 mm respectively. The bowel wall thickness is similar throughout the length of the small bowel, measuring 1.5+/-.05mm. The number of folds per 2.5 cm decreases from 4.6 in the jejunum to 1.5 in the terminal ileum and the fold thickness varies from 2.1 - 1.8 mm. Due to increased surface area, the jejunum enhances more than the ileum - Fig. 4.

Normal large bowel wall thickness should not exceed 3 mm, when adequately distended.

The maximum enhancement of the bowel wall is observed between 70 and 85 seconds after intravenous contrast agent administration.

Imaging findings/pathology. Crohn's disease is classified as primarily active inflammatory, penetrating or fibrostenotic disease. Penetrating disease is characterized by presence of fistulas and/or abscesses while in fibrostenotic disease stricturing is seen. It is most crucial to remember, that multiple stages of the disease may coexist in the same patient or even in the same bowel segment.
Diagnostic criteria of active inflammation include:

1. Wall thickening-submucosal oedema. Wall thickening (between 3 to 12 mm) is usually associated with luminal stenosis. See: Fig. 3, Fig. 5 and Fig. 6. Remember: use of fat saturation in one of T2-weighted sequences allows differentiation between submucosal fat and oedema, both bright on T2-weighted images.

2. Mucosal hyperenhancement on T1-weighted post-contrast images in reference to adjacent bowel loops, with or without wall thickening. See: Fig. 3d, Fig. 5cg, Fig. 6e.

3. Stratified (layered) appearance of active inflammatory lesions on fat-suppressed T1-weighted post-contrast images due to mucosal and serosal hyperenhancement accompanied by submucosal oedema. According to some authors transmural enhancement without stratification is more common in early stages of CD and in paediatric population whereas the stratified appearance is associated with long-standing disease. See: Fig. 7.

4. Mural diffusion restriction. Persistent high signal on DWI with corresponding low signal on ADC map. See: Fig. 3ef, Fig. 5ef, Fig. 6bc.

5. Increased mesenteric vascularity adjacent to the inflamed bowel loop - "comb sign" - resulting from engorged vasa recta. See: Fig. 8.

6. Mesenteric lymphadenopathy. Prominent reactive enhancing mesenteric nodes up to 8 mm in short axis diameter may be present. See: Fig. 9.

7. Signs of penetrating disease:
   - Transmural inflammation. See: Fig. 7cf.
   - Formation of fistulas between an actively inflamed segment of the bowel and other loops of both small and large intestine, the urinary bladder and/or the skin. A stellate arrangement of small bowel loops is often seen in case of enteroenteric fistulas - "star sign" - Fig. 10. An enterovesical fistula is characterized by focal urinary bladder wall thickening at the site of the contact with the inflamed bowel loop - Fig. 11; gas may be present in the bladder. In up to 36% of patients with CD anal and perianal fistulas may be encountered - Fig. 12. Remember: in case of an obstructing stricture distal to a fistula, the latter is unlikely to close as long as the stricture persists.
   - Presence of abscesses. Abscesses are typically located adjacent to the inflamed bowel loop or are connected to it by a sinus tract. See: Fig. 10.
Restriction of diffusion is seen in DWI sequences and on Apparent Diffusion Coefficient (ADC) maps.

8. Mesenteric fibrofatty proliferation - "creeping fat". A mass effect caused by proliferation of mesenteric fat resulting in displacement of intra-abdominal viscerae or vascular structures, usually asymmetric. See: Fig. 7g.

9. Additional secondary findings: oedema, free fluid (Fig. 13) and enhancement in the surrounding soft tissues (Fig. 7c).

Diagnostic signs of chronic inflammation - fibrostenotic disease (Fig. 14), include:

1. Mural fibrosis. See: Fig 14 ab.

2. Stricture formation - fixed segments of narrowing that may be accompanied by mural thickening show low signal intensity on both T1- and T2-weighted images, lack of oedema and hyperaemia, enhance less than active inflammatory lesions. See: Fig. 15. Pre-stenotic bowel dilatation (small bowel diameter > 30 mm) may indicate pending obstruction. With asymmetric bowel wall involvement, pseudosacculation can occur.

Table 3 compares diagnostic features of active and chronic disease in MRE.

Table 3. Comparison of diagnostic signs of active and chronic disease in MRE
(adapted from Griffin et al. 2012 with alterations).

<table>
<thead>
<tr>
<th>Sign</th>
<th>Active inflammation</th>
<th>Fibrostenotic disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(including penetrating disease)</td>
<td></td>
</tr>
<tr>
<td>Increase in wall thickness.</td>
<td>Moderate to big.</td>
<td>Mild.</td>
</tr>
<tr>
<td>Wall enhancement.</td>
<td>Strong.</td>
<td>Mild.</td>
</tr>
<tr>
<td>Stratified mural enhancement.</td>
<td>May be present.</td>
<td>Variable.</td>
</tr>
<tr>
<td>Increased mesenteric vascularity - &quot;comb sign&quot;</td>
<td>Present.</td>
<td>Absent.</td>
</tr>
</tbody>
</table>


How to optimize your report?

Obtain relevant, vast clinical data from the referring physician - remember that not all thickened, hyperenhancing small bowel loops account for CD. Differential diagnosis for Crohn's Disease includes: normal collapsed bowel, which may appear thickened and hyperenhancing, infectious and ischemic enteritis, radiation-induced enteritis and vasculitis.

Include the following information in your report: length and location of involved bowel loops, type of encountered lesions - signs of active inflammation, penetrating or fibrostenotic disease with focus on presence/absence of bowel obstruction and extramural complications (fistulas/abscesses).

Images for this section:
Fig. 1: Axial T2-weighted HASTE image. Patient is examined in the supine position due to presence of the stoma in the lower right quadrant of the abdomen.
**Fig. 2:** Coronal T2-weighted images. a. Normal bowel. b. T2-weighted HASTE image. Intraluminal flow voids (arrow) are visible as the sequence is susceptible to motion artifacts. Thickening of the bowel wall is seen in the lower left quadrant.
**Fig. 3:** Mural thickening in active Crohn's Disease - terminal ileum. Coronal T2-weighted HASTE image shows mural thickening (arrow) at the level of ileocecal valve. b. Coronal T2-weighted Trufi image. Thickening of the wall of the terminal ileum (arrow). Note the "black boundary" effect due to chemical shift artifacts. c. Axial T2-weighted HASTE image. Mural thickening (arrow). d. Coronal T1-weighted fat-saturated post-contrast image, oblique reconstruction. Hyperenhancement of the inflamed terminal ileum. e. Coronal DWI (b 800) image shows increased signal intensity consistent with mural diffusion restriction f. ADC map confirms restricted diffusion.
**Fig. 4:** Coronal T1-weighted fat-saturated post-contrast image. Normal bowel. Due to increased surface area the jejunum enhances more than the ileum.
Fig. 5: Mural thickening in active Crohn's Disease. a. Axial T2-weighted HASTE image shows mural thickening (arrow). Signs of mesenteric oedema are noticed adjacent to the dorsal aspect of the inflamed bowel loop (asterisk). b. Coronal T2-weighted fat-saturated HASTE image. Mural thickening. c. Axial T1-weighted post-contrast Fl2d image shows increased enhancement of the inflamed bowel wall. d. Coronal T2-weighted fat-saturated HASTE image, follow-up examination. Progression of active disease (increased mural thickening) is seen in 1 month observation period in reference to 4b. e. Coronal DWI (b 800) image shows increased signal intensity consistent with mural diffusion restriction. f. ADC map confirms restricted diffusion. g. Coronal T1-weighted fat-saturated post-contrast image. Hyperenhancement of the inflamed bowel loop.
Fig. 6: Mural thickening in active Crohn's Disease - ascending colon. a. Coronal T2-weighted fat-saturated HASTE image. Irregular mural thickening of the ascending colon (arrow). b. Coronal DWI (b 800) image shows increased signal intensity consistent with mural diffusion restriction (arrow). c. ADC map confirms restricted diffusion - note the low signal (arrow). d. Axial T2-weighted HASTE image shows mural thickening. e. Coronal T1-weighted fat-saturated post-contrast image. Avid enhancement of the inflamed part of the ascending colon.
Fig. 7: Example of stratified appearance of active inflammatory lesions. a. Coronal T1-weighted fat-saturated FL2d image. Irregular, severe thickening of the left colonic flexure and descending colon with adjacent mesenteric oedema (asterisks). b. Coronal T1-weighted fat-saturated post-contrast image shows avid stratified pattern of mural enhancement of the inflamed part of the colon (arrow). c. Coronal T1-weighted fat-saturated post-contrast image. Avid enhancement of the splenic flexure of the colon with wall thickening. Note the enhancement of the surrounding soft tissues. d. Coronal T1-weighted FL2d image shows mural thickening of the left colonic flexure and descending colon. e. Corresponding coronal T2-weighted Trufi image. f. Coronal T2-weighted HASTE image shows mural thickening of the colon with adjacent mesenteric oedema. g. Axial T2-weighted HASTE image shows severe mural thickening of the transverse colon associated with luminal stenosis as well as mesenteric oedema (asterisk) and fatty proliferation (arrow).
Fig. 8: Coronal T1-weighted fat-saturated post-contrast images (a,b). Increased mesenteric vascularity adjacent to the inflamed bowel loop - "comb sign".
Fig. 9: Lymph nodes. a. Coronal T1-weighted fat-saturated post-contrast image. Reactive lymph nodes adjacent to enteroenteric fistula. b. Coronal DWI image (b800) easily depicts lymph nodes in ileocecal area. c. Coronal T1-weighted fat-saturated post-contrast subtracted image. Same patient as in b.
**Fig. 10:** Case of an enteroenteric fistula. A stellate arrangement of small bowel loops is seen - "star sign". Note a small adjacent abscess (arrows). a. Axial T2-weighted HASTE image. b. Coronal T2-weighted HASTE image. c. Coronal T1-weighted fat-saturated post-contrast image. d. Coronal T2-weighted HASTE STIR image.
Fig. 11: Example of an enterovesical fistula accompanying an enteroenteric fistula. A stellate arrangement of small bowel loops is observed - sign of an enteroenteric fistula (white arrows). Focal urinary bladder wall thickening at the site of the contact with the inflamed bowel loop suggestive of an enterovesical fistula (red arrows). a. Sagittal T2-weighted image (examination protocol was extended with a sagittal sequence in order to facilitate diagnosis). b. Coronal T1-weighted fat-saturated post-contrast image.
Fig. 12: Case of a wide perianal fistula with communication to the skin. a. Axial T1 FL2d post-contast image. b. Axial T1-weighted fat-saturated post-contrast image (examination protocol was extended with a sagittal sequence in order to facilitate diagnosis). c. Coronal DWI (b800) image.
**Fig. 13:** Axial T2-weighted fat-saturated HASTE image (example of alteration in the imaging protocol). Mural thickening of a small bowel loop and free fluid in minor pelvis are seen.
**Fig. 14:** Example of fibrostenotic disease. A narrowed, strictured fragment of a small bowel loop with mural thickening and luminal stenosis is seen in the upper left quadrant of the abdomen. a. Coronal T1-weighted FL2d. b. Correlating axial T2-weighted HASTE image - note the low signal intensity of the thickened bowel wall. c. Coronal T1-weighted fat-saturated post-contrast image shows less intense enhancement that appreciated in active disease. d. Axial DWI (b800) image (example to alteration in the imaging protocol) and ADC map (e.) present no diffusion restriction.
**Fig. 15:** Fibrostenotic disease. a. Coronal T2-weighted HASTE image. Fibrotic stricture in the descending colon (arrow). b. Coronal T1-weighted fat-saturated post-contrast image shows corresponding mural enhancement in the skip.
Conclusion

Crohn's disease (CD) is an idiopathic chronic inflammatory disease of the gastrointestinal tract often encountered in young adults with tendency to frequent relapses. MRE is becoming the modality of choice in evaluation of Crohn's disease. It allows safe and noninvasive diagnostic tool without exposure to ionizing radiation as well as help in treatment planning and follow-up. Interpretation of MRE images requires familiarity with the imaging signs of active and chronic bowel inflammation. It can help assess the activity of the disease, demonstrate both mural and extramural complications as well as distinguish between inflammatory, structuring and penetrating disease.

Personal information

References

6. Fujii, T; Naganuma, M; Kitazunne, Y; Saito, E; Nagahori, M; Ohtsuka, K; Watanabe, M. Advancing Magnetic Resonance Imaging in Crohn's Disease. DIGESTION; 2014; 89; 1; p24-p30.


