MR enterography in Crohn disease

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Authors: S. Alfonso Cerdan¹, J. C. Pernas Canadell, D. Hernandez², C. Pérez Martínez¹, A. Lozano¹; ¹Barcelona/ES, ²08025/ES
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Learning objectives

Define the role of MR enterography in evaluation of small bowel in patients with Crohn disease and its correlation with findings on barium studies and CT.

Background

Crohn disease (CD) is a chronic inflammatory disease of the gastrointestinal tract with an unpredictable lifelong course that usually affects young people (with a peak age between 20 and 40 years), who are most vulnerable to the potential adverse effects of repeated exposure to ionizing radiation.

It is thought the etiology is multifactorial, including genetic, immunologic, infectious and microvascular factors, and smoking and lifestyle [1].

The prevalence is about 100-200 per 100,000 people in North America and Europe [2].

CD affects the entire digestive tract, but the bowel segment most frequently affected is the small intestine, but it is the least accessible with endoscopic techniques. Even more than 70% -80% of patients present the small bowel affected [3,1].

The pathological development of intestinal lesions occurs firstly by a submucosal lymphoid tissue hyperplasia, which causes lymphedema. Secondarily aphthoid ulcers are formed and evolve in deep ulcers, which might be complicated to abscesses, fistulas and strictures.

Enteric involvement usually is segmental and inflammation is transmural. The mucosal surface has aphtas and deep linear ulcers with healthy segments between them, so it is called "skip lesions". In advanced stages longitudinal and transverse ulcers coalesce leading on a cobblestone pattern of the intestinal mucosa.

Patients frequently are subjected to multiple imaging examinations in which they are exposed to ionizing radiation (intestinal series, CT), often beginning in adolescence or early adulthood, with consequent potential adverse effects (radio-induced tumors) [1,4,5]. The radiation dose in an abdominopelvic CT ranges from 6-28 mSv [2]. MR enterography is useful in monitoring CD with the advantage it does not expose the patient to ionizing radiation.

Imaging findings OR Procedure details
We describe and illustrate the technique of MR enterography reviewing semiology described in literature and its correlation with images of CT and barium studies.

We performed the study of patients with CD on a 1.5 T MR according to the MR enterography protocol: exploration begins with the patient placed in prone position 45 minutes after ingestion of hyperosmolar solution (manitol 5%). Prone position is chosen because this produces a degree of compression on the anterior abdominal wall; then this improves separation of the bowel loops and reduces the volume of the peritoneal cavity, reducing the number of sections required for each coronal acquisition [1,6], which reduces the length of breath hold required, resulting in improved patient compliance. The sequences performed were:

- Axial Single-shot spin-echo T2-weighted (HASTE) sequence on ileocecal area to verify the oral contrast has reached the right colon.
- Axial and coronal FISP images.
- To inject an antiperistaltic (10 mg intravenous) as hyoscine N-butyrbromide (Buscopan ®).
- Coronal and axial Single-shot spin-echo T2-weighted images. If they are repeated provide information on intestinal motility.
- Coronal Single-shot spin-echo T2-weighted fat-saturated images.
- To inject 10 mg of intravenous hyoscine N-butyrlbromide again.
- Coronal dynamic with gadolinium THRIVE 3D (basal, 30 sec, 70 sec, 240 sec) images for to enhance the bowel wall and other organs.

Typical changes in CD include ileal involvement, fat hypertrophy, mural thickening, aphthoid and linear ulcers, fistulas, patchy lesions, and cobblestone pattern [1,5].

Several MR findings indicate active disease, such as mural thickening greater than 4 mm [1], intramural and mesenteric edema, mucosal hyperemia, parietal enhancement and its pattern, ulcers and fistulas, vascular engorgement and inflammatory mesenteric lymph nodes (usually with increased uptake) [5,7].

**INTESTINAL INVOLVEMENT**

1. **Mural thickening**

Wall thickness exceeding 3 mm in a distended bowel loop is considered abnormal (Figure 1), and often reaches 5-10 mm in CD with bowel involvement.
When the thickened wall does not present edema shows low or intermediate signal intensity at HASTE and FISP sequences. Black edge artifact affects FISP sequence, so it difficults the wall thickness measurement, however does not affect HASTE sequence, which is optimal to measure.

2. Patterns of folds thickening

Three patterns of folds involvement in CD are:

- **Picket fence pattern**: diffusely thickened folds.

- **Reduction in or distortion of folds due to ulceration**.

- **Cobblestoning**: patchy areas of high or intermediate signal intensity within an affected bowel segment corresponding to ulcers (Figure 2 and 3a and b). The best sequence to detect it is on FISP images. However it is not comparable to the image provided by barium studies (Figure 3c).

The mesenteric border is usually affected in CD resulting in an asymmetrical image of the affected segment.

3. Ulcers

It is necessary an appropriate intestinal distension to depict them [1]. There are two types:

- **Moderate to deep ulceration**: hyperintense thin lines, longitudinal or transverse, within thickened bowel wall. HASTE is the best sequence to detect it (Figure 4 and 5).

- **Early and superficial ulceration**: difficult to diagnose by MR or CT images although a proper luminal distention is reached, being still better conventional fluoroscopy.

4. Stricture

This is divided into two types:

- **Functional**: proximal bowel loop is greater than 3 cm.

- **Nonfunctional**: when a narrowing in the bowel lumen greater than 10% compared with adjacent normal loop (Figure 6).

Stenotic segments show **wall thickening**, with the differential diagnosis of isolated stenosis infection, radiation enteritis, neutropenic enteritis in immunocompromised patients, carcinoid tumor / lymphoma / ADK (rare but more common in CD).

5. Acute wall edema

Affected bowel loops show higher signal intensity of the thickened wall in the HASTE sequence with fat saturation, which corresponds to mucosal or submucosal edema and
correlates with the activity of CD (Figure 7), although the absence of hyperintensity on the wall does not exclude activity. The presence of high signal intensity also may be due to intramural fat deposition occurring in chronic stenosis. We must do the differential diagnosis with a fibrous thickening of the wall, which has low signal intensity on T2 FISP and HASTE images.

6. Wall fat and chronicity

It occurs in chronic CD and is due to fatty infiltration of the intestinal wall that affects bowel and colon. We can differentiate fat from wall edema using HASTE sequences with and without fat saturation. If it is hyperintense on both sequences is edema and if the signal decreases in fat saturation corresponds to fat (Figure 8). FISP images show a chemical shift artifact if there is intramural fat, showing a thin black line. Fatty infiltration can also be observed in healthy obese people.

7. Patterns of wall enhancement

The evaluation should be done by comparing the enhancement of a pathologic bowel loop with a normal adjacent bowel loop or one at the same distance from the center of the field of view to avoid field inhomogeneity, which can influence the apparent enhancement (Figure 9).

Disease is considered active if there is a thickened wall greater than 4 mm with a ratio of the contrast enhancement of the pathological loop about a normal loop greater than 1.3:1 (Figure 10).

It has been described three patterns of contrast enhancement according to the activity of the disease:

- **Stratified or layered:** mucosal enhancement and less intense in the submucosa, which combined with submucosal edema corresponds to active disease (Figure 11a and b).

- **Diffuse:** corresponds to transmural inflammation, with an intense and homogeneous enhancement affects the entire wall thickness (Figure 11c and 12).

- **Low-level inhomogeneous:** in fibrosis.

8. Pseudosacculation-Pseudodiverticulum Formation

Their formation is due to the preservation of an antimesenteric border in an affected segment of bowel loop. Fibrosis and shortening of the affected mesenteric wall produce apparent dilatation of the opposing normal bowel wall. The sacculation is formed by the three layers of the wall (Figure 13).

EXTRAINTESTINAL INVOLVEMENT

1. Comb sign
This sign is produced by an increase in mesenteric vasculature. FISP images show multiple hypointense short lines on the mesenteric border of the ileum, which are distributed in parallel (Figure 14). These lines lie in a perpendicular plane to the longitudinal axis of the affected intestinal wall, and are hyperintense after administration of paramagnetic contrast, due to the enhancement of the vessels (Figure 15). This finding is associated with disease activity. This finding is better depicted on FISP sequence.

2. Mesenteric edema

It is less common. Usually present in advanced active disease and we can spot it in adjacent mesentery to the affected intestinal loop. It usually associates with intestinal wall edema and contrast enhancement, which also indicate disease activity (Figure 16).

3. Fat hypertrophy

This consists in a hypertrophy of mesenteric fat with mass effect and displacement of the mesenteric vessels or abdominal viscera (Figure 17). Often it affects the intestinal mesenteric border. It occurs in longstanding CD with established transmural inflammation. It is a very specific sign for CD. The FISP sequence is useful for its detection.

4. Lymph nodes

In active CD lymph nodes are adjacent to affected bowel loop or use to lie along the vascular supply of an affected disease segment. They also enhance with contrast, are enlarged and edematous in HASTE with fat saturation images; however, they are better delimited on FISP sequence, where lymph nodes are hypointense (Figure 18). To differentiate edema from inflammatory changes is difficult without fat saturation.

5. Fistulas and sinus tracts

Fistulas are formed from deep ulcers which contact an adjacent epithelial surface. They are hyperintense on T2-weighted images and enhance gadolinium (Figure 19). Enteroenteric and enterocolic fistulas may form, or can contact other hollow viscera such as the bladder.

Enterocutaneous fistulas are difficult to see in prone position because of compression they distort, improving their detection in supine position.

Sinusal tract is a blind tube originated from the intestine, but it does not contact other epithelial surface. It is also hyperintense on T2-weight images and may be associated with abscesses.

6. Abscesses
These are encapsulated collections of pus with intensity as fluid, therefore are hyperintense on T2-weighted images and hypointense on T1-weighted images, with a rim enhancement (Figure 20). Abscesses often are heterogeneous due to the presence of solid material and gas. The latter is difficult to detect by MR imaging.

It should detect bulky abscesses as their presence is a contraindication to initiate anti-TNF therapy such as infliximab, therefore must be drained before starting this treatment [1].

Images for this section:

Fig 1: (a) axial HASTE image and (b) coronal HASTE fat-saturated image showing wall thickening of an ileal loop (arrows) in pelvis. (c) Axial CT image, less precise to detect the wall thickening. (d) Wall thickening of a jejunal loop of other patient on HASTE image.

Fig. 1: Wall thickening of an ileal loop in pelvis.
Fig 2: (a) coronal FISP image, (b) coronal HASTE image, (c) colangio-MR sequence applied in bowel and (d) coronal THRIVE with gadolinium enhancement image. Multiple intestinal ulcers alternating with normal mucosa in terminal ileum, forming a cobblestoning pattern (arrows) are shown.

Fig. 2: Cobblestoning pattern.
Fig. 3: Cobblestoning pattern.

**Fig 3:** (a) coronal HASTE image shows cobblestoning pattern in terminal ileum and (b) coronal CT image where pattern cannot be precised. (c) Barium study shows more precise image of the cobblestoning pattern.
Fig. 4: Deep ulcer in terminal ileum.
**Fig. 5:** Ulcers in terminal ileum.
Fig 6: (a) coronal HASTE image shows stricture and wall thickening of a distal ileum segment. (b) Coronal CT image, with poor definition of wall thickening and stricture.

Fig. 6: Stricture and wall thickening of distal ileum.
Fig. 7: Edema and wall thickening of terminal ileum.
Fig 8: longstanding DC. (a-b) Coronal HASTE and HASTE fat-saturated images and (c) coronal CT image. They show an important wall fatty infiltration of the right colon (arrows), sign of cronicity of CD.

Fig. 8: Wall fatty infiltration of the right colon.
Fig. 9: (a)-(d) coronal THRIVE 3D with gadolinium images, which allows evaluation of grade and pattern of enhancement of the inflammatory areas.

Fig. 9: Grade and pattern of enhancement of the inflammatory areas.
Fig 10: (a) and (b) coronal THRIVE 3D with gadolinium images show a diffuse pattern enhancement and a ratio of enhancement of the pathologic loop about a normal loop greater than 1,3:1, meaning active CD.

Fig. 10: Diffuse pattern enhancement.
Fig. 11: (a) coronal THRIVE 3D with gadolinium image shows stratified enhancement pattern in terminal ileum and (b) coronal CT image does not allow say type enhancement pattern. (c) Coronal THRIVE 3D with gadolinium image of other patient shows diffuse enhancement pattern in a thickened ileal loop.

Fig. 11: Different enhancement patterns.
**Fig 12:** (a) coronal HASTE image showing jejunal thickening (red arrows) and (b) coronal THRIVE 3D image after gadolinium administration showing a diffuse pattern of wall enhancement of jejunum (blue arrows).

**Fig. 12:** Diffuse enhancement pattern of jejunum.
**Fig. 13:** (a) coronal HASTE fat-saturated image and (b) coronal enhanced CT reconstruction, show a bowel pseudosacculation (arrows) and wall thickening. As an incidental finding is showed right kidney malrotation. (c) Pseudosacculations due to stricture on HASTE sequence of other patient.

**Fig. 13:** Bowel pseudosacculation and wall thickening.
Fig. 14: (a) y (b) coronal FISP images show mesenteric fat proliferation and comb sign (arrow).

Fig. 14: Mesenteric fat proliferation and comb sign.
Fig 15: (a) coronal FISP, (b) coronal THRIVE 3D with gadolinium and (c) coronal CT images show mesenteric vessel engorgement and the comb sign (arrows).

Fig. 15: Mesenteric vessel engorgement and comb sign.
**Fig 16:** (a-b) axial THRIVE with gadolinium and (c-d) axial enhanced CT images, showing edema and mesenteric fluid (arrows) near a thinned ileal loop with active CD.

**Fig. 16:** Edema and mesenteric fluid near an affected ileal loop.
Fig 17: (a) axial HASTE and (b) axial CT images show mesenteric fat hypertrophy (arrows). (c) Coronal FISP and (d) coronal THRIVE with gadolinium images of other patient show mesenteric fat hypertrophy (arrows) and mesenteric vessels engorgement.

Fig. 17: Mesenteric fat hypertrophy.
**Fig 18:** (a) coronal FISP and (b) THRIVE 3D with gadolinium images shows mesenteric hypointense lymph nodes on FISP sequence and high enhancement on THRIVE sequence (circles). (c) Coronal HASTE image with poor delimitation of lymph nodes (circle). (d) Coronal CT image shows lymph nodes (circle).

**Fig. 18:** Mesenteric lymph nodes.
**Fig. 19:** Fistula between right colon and psoas muscle.
Fig 20: (a-b) axial HASTE and CT images show an abscess in anterior abdominal wall within gas (arrows), near the ileostomy. (c-d) Axial THRIVE 3D with gadolinium and CT images show an abscess in right hemiabdomen, within gas too.

Fig. 20: Abscesses in anterior abdominal wall and in right hemiabdomen.
Conclusion

MR enterography is a radiation-free useful imaging technique in the assessment of inflammatory bowel disease activity and extramural complications.

Absence of ionizing radiation makes it particularly useful for repeated assessment in young patients, who are most vulnerable to the potential adverse effects of radiation, and who require multiple follow-up imaging due to the chronic course of illness.

References


Personal Information