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FULL PAPER

CT enteroclysis/enterography findings in drug-induced small-bowel damage

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Objective: To evaluate the CT enteroclysis (CTE)/enterography findings of patients with small-bowel mucosal damage induced by aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) and to compare these findings with the duration of drug use and endoscopic findings.

Methods: CTE findings of 11 patients (22 lesions) with drug-induced small-bowel damage were reviewed, including 8 NSAID users and 3 aspirin users. Three patients were short-term users (6 months or shorter) and eight were long-term users (3 years or longer). Nine patients also underwent videocapsule endoscopy (VCE) or double-balloon enteroscopy (DBE).

Results: Small-bowel abnormalities were visible in 8 of 11 patients (73%) on CTE. Multiple lesions were seen in five patients, including all short-term users. Lesions were classified into three types. Type 1 (mucosal patchy enhancement) was found in four of eight patients (50%,

12 lesions) all were short-term users. Small erosions with mild oedema/redness were shown by DBE. Type 2 (homogeneous hyperenhancement) was found in two of eight patients (25%, four lesions) who were long-term users. Large ulcers with marked oedema/redness were shown by DBE. Type 3 (stratification enhancement) was found in four of eight patients (50%, six lesions), both short-term and long-term users. Annular or large ulcers with strictures were shown by VCE or DBE.

Conclusion: On CTE, Type 1 lesions in patients with mostly short-term aspirin or NSAID use, Type 2 lesions in patients with long-term use and Type 3 lesions in both types of patients were detected. CTE may have usefulness for the detection of mild damage.

Advances in knowledge: Small-bowel abnormalities owing to aspirin or NSAID present with three different patterns on CTE.

CT enteroclysis (CTE)/enterography is a technique whereby enteral contrast medium is infused into the small bowel to obtain well-expanded loops and adequate endoluminal opacification for evaluation of lesions.^{1,2} In recent years, it has been widely recognized that low-dose aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) can cause damage to the mucosa of both the upper gastrointestinal tract and the small bowel.^{3–8} The usefulness of videocapsule endoscopy (VCE) or double-balloon enteroscopy (DBE) in the diagnosis of drug-induced small-bowel damage has been established, but few comprehensive reports have evaluated the CT findings of this condition.^{4,5} The CTE findings of drug-induced small-bowel damage were classified into three types, and the duration of drug use and endoscopic findings associated with each type were evaluated. The goal of this retrospective study was to describe the CTE findings in patients with small-bowel mucosal damage induced by NSAIDs, to compare CTE

findings with those observed on VCE or DBE and to correlate CTE findings with the duration of drug use.

METHODS AND MATERIALS

We reviewed the CTE findings of 11 (5 males and 6 females; mean age, 71 years; range, 56–85 years) patients diagnosed with drug-induced small-bowel damage, among 82 consecutive patients with obscure gastrointestinal bleeding who underwent CTE between July 2008 and November 2012 (Table 1). VCE ($n = 8$) and/or DBE ($n = 5$) was performed in 9/11 patients, and CTE was performed on the same or the following day. The CTE examinations were reviewed before the results of VCE or DBE were obtained. Although VCE or DBE was not performed in the remaining two patients because of bowel strictures, these patients were diagnosed with drug-induced small-bowel damage because their symptoms improved soon after discontinuation of the drug and the stricture resolved.

Table 1. Cases with abnormal findings on CT enteroclysis/enterography (CTE-C/CTE-G)

Case	Age (years)	Sex	Drug	Duration of drug use	CTE-C/CTE-G findings	CTE-C or CTE-G
1	56	M	NSAID	Over 10 years	Type 3 (1)	CTE-C
2	72	F	Aspirin	5 years	Type 3 (1)	CTE-C
3	78	F	NSAID	Over 10 years	Type 1 (1), Type 2 (1)	CTE-C
4	63	M	NSAID	3 years	Type 3 (1)	CTE-C
5	77	F	NSAID	3 years	Type 2 (3)	CTE-C
6	65	M	Aspirin	6 months	Type 1 (5)	CTE-C
7	81	F	NSAID	6 months	Type 1 (4), Type 3 (3)	CTE-C
8	80	M	NSAID	3 months	Type 1 (2)	CTE-G

F, female; M, male; NSAID, non-steroidal anti-inflammatory drug. Number in parenthesis represents the number of lesions.

The PillCam SB capsule endoscopy system (Given Imaging Ltd, Yokneam, Israel) was used for VCE, and image interpretation was carried out on a Rapid Reader v. 6.5 (Given Imaging Ltd). DBE was performed with an EN-450T5/W (Fujifilm Corporation, Tokyo, Japan) or SIF-Q260 (Olympus Medical Systems, Tokyo, Japan) endoscope.

Standard of reference

The study co-ordinator, who had full access to the patients' records, including the pathological reports and the entire imaging history, established the standard of reference for determining the actual cause of obscure gastrointestinal bleeding.

Intestinal fluids were cultured and biopsy samples were examined to exclude inflammatory bowel disease, malignant tumours and infection.

During a final lesion-by-lesion analysis, the study co-ordinator, one radiologist and one gastroenterologist reviewed in consensus all cases of discrepant findings between CTE and VCE or DBE to find plausible explanations for discrepancies.

CT enteroclysis/enterography technique

Contrast medium was enterally infused into the small bowel via a duodenal tube ($n = 9$) or enteral contrast medium was orally administered ($n = 2$). Sedation was not carried out in any patient.

Infusion of an enteral contrast medium via a duodenal tube: CT enteroclysis ($n = 9$)

First, an intranasal endoscope (GIF type XP 260NS; Olympus Medical Systems) was inserted to the third portion of the duodenum; then, a guidewire (0.045 inch $\times$ 3500 mm; Create Medic Co., Ltd, Tokyo, Japan) was inserted into the jejunum through a hole for forceps; and a duodenum tube (16-Fr gastroenterography balloon catheter, 1500 mm; Create Medic Co., Ltd) with a balloon at the tip was inserted to the duodenojejunal flexure along the guiding wire under fluoroscopy after removing the intranasal endoscope. About 1800 ml of an oral solution for colonic cleansing [polyethylene glycol (PEG) electrolyte lavage solution, at a temperature of about 37°C] was then infused into the small intestine at a rate of about

150 ml min⁻¹ using a power injector (Master Flex® Easy-load 7518-10; Cole-Parmer Instrument, Inc., Vernon Hills, IL) under fluoroscopy after inflating the balloon at the tip of the tube.

Oral administration of an enteral contrast medium: CT enterography ($n = 2$)

Our regimen consists of oral administration of 1800 ml of PEG according to the following schedule: 400 ml, 60 min before scanning; 450 ml, 45 min before scanning; 450 ml, 30 min before scanning; and 500 ml, 15 min before scanning.

CT imaging

Immediately after the enteral contrast medium was completely infused and the intramuscular administration of the anticholinergic agent, patients were moved into the CT room. Scanning was performed using a 64-channel multidetector row CT scanner (Somatom® Definition dual-source CT; Siemens Healthcare, Erlangen, Germany) after intravenous injection of 100 ml of non-ionic contrast medium (iopamidol 370 mg iodine per millilitre, Oypalomin®; Fuji Pharma Co., Ltd, Japan) at a flow rate of 3 ml s⁻¹, with a delay of 40 s. Vascular malformation sometimes may cause bleeding from the small intestine, we selected a delay of 40 s close to the arterial phase.

Imaging was performed from the level of the diaphragm to the pubic region. The scanning parameters were collimation 1 mm, table speed 8 mm s⁻¹. Axial and coronal images were obtained with a slice thickness of 2 mm and slice interval of 2 mm, and multiplanar views were created on an attached workstation. The images were interpreted by two independent radiologists experienced in abdominal CT interpretation. During the reading sessions, axial and multiplanar reformatted images were analysed with respect to the presence of the following three types. The reading criteria of CTE is finding the affected bowel mucosa without bowel wall thickening, strongly enhanced bowel mucosa, the whole affected bowel wall; homogeneously and strongly enhanced, the markedly thickened affected bowel wall with a stratification structure or target appearance.

Disagreements in interpretation were resolved by consensus after discussion. The CT findings were compared with the VCE or DBE findings.

Figure 1. Case 6—a 65-year-old male with aspirin-induced small-bowel injury (drug use of 6 months). (a, b) Multiple small hyperenhancements of the mucosa (arrows) were seen in the ileum with CT enteroclysis. These findings were classified as Type 1. (c, d) Small ulcers and erosions with redness and oedema in the ileum were seen with double-balloon enteroscopy (arrows).

RESULTS

Abnormal findings in the small bowel were detected by CTE in 8/11 patients (73%) diagnosed with drug-induced small-bowel damage (Table 1). Multiple lesions were found in 5/8 patients (63%). In addition, multiple lesions were shown in short-term users (6 months or shorter).

Three patients with ulcerative lesions malignancy were excluded by biopsy. Medication was stopped in 4 but continued in 5/11 patients. Change of the drug from NSAIDs to acetaminophen in 2/11 patients. Rebamipide was taken in all patients. Anaemia, erosions and ulcers were improved on follow-up VCE or DBE.

Figure 2. Case 3—a 78-year-old female with non-steroidal anti-inflammatory drug-induced small-bowel injury (drug use of over 10 years). (a) The homogeneous hyperenhancement of the whole bowel wall (arrow) was seen in the ileum with CT enteroclysis. Mesenteric hypervascularity and enlarged lymph nodes were also revealed (arrowheads). This finding was classified as Type 2. (b) Large ulcers with marked oedema and redness in the ileum were seen with double-balloon enteroscopy.

Figure 3. Case 5—a 77-year-old female with non-steroidal anti-inflammatory drug-induced small-bowel injury (drug use of 3 years). (a) The homogenous hyperenhancement of the bowel wall (arrow) was seen in the ileum with CT enteroclysis. Fat stranding and mesenteric hypervascularity were also revealed. This finding was classified as Type 2. (b) Large ulcers with marked redness and oedema in the ileum were seen with double-balloon enteroscopy.

Bowel damage was caused by NSAIDs in eight patients, and low-dose aspirin was the cause in the remaining three patients. 3/11 patients were short-term users of the drugs (6 months or shorter), and the other 8 patients were long-term users (3 years or longer).

The CTE findings of drug-induced small-bowel damage were classified into the following three types. In Type 1 (mucosal type), the affected bowel mucosa without bowel wall thickening was strongly enhanced. In Type 2 (homogeneous hyper-enhancement type), the whole affected bowel wall was homogeneously and strongly enhanced. In Type 3 (stratification enhancement type), the affected bowel wall was markedly thickened with a stratification structure or target appearance. Diaphragm disease was not seen in any patient.

Type 1 was found in 4/8 patients with abnormal CTE findings (12 of 22 lesions). The CTE lesions in Type 1 corresponded to the small erosions or ulcers with mild oedema, and redness was observed on endoscopic examination in all four patients (Figure 1). CTE findings of multiple lesions were seen in three of four patients (75%), all three were short-term users of drugs

(6 months or shorter). Type 2 was found in 2/8 patients (4 of 22 lesions), all with long-term drug use (3 years or longer). These CTE lesions corresponded to the large ulcers with marked redness or oedema on endoscopic examination (Figures 2 and 3). Fat stranding, enlarged lymph nodes (maximum short diameter of 5 mm or more) and hypervascularity around the bowel were seen in these two patients. Type 3 was found in 4/8 patients (6 of 22 lesions), all 4 with both short-term and long-term drug use.

These patients had luminal strictures, and the lesions corresponded to annular or large ulcers associated with strictures observed on endoscopic examination (Figures 4 and 5). In one patient with Type 3 lesions and long-term use of NSAIDs, the capsule endoscope was retained for a week because of a stricture caused by a circular ulcer scar (Figure 6).

Normal findings were found on CTE in 3/11 patients diagnosed as drug-induced small-bowel damage. These three patients were all long-term users of drugs, and minimal mucosal damage, such as small erosions or small ulcers with mild redness and oedema, was shown by endoscopic examination (Figure 7).

Figure 4. Case 1—a 56-year-old male with non-steroidal anti-inflammatory drug-induced small-bowel injury (drug use of over 10 years). (a) Thickened wall with target appearance with hyperdensity of the mucosa and low attenuation of the submucosa was seen in the ileum with CT enteroclysis (arrow). This finding was classified as Type 3. (b) Large ulcer with marked oedema, and luminal stenosis in the ileum were seen with double-balloon enteroscopy.

Figure 5. Case 2—a 72-year-old male with aspirin-induced small-bowel injury (drug use of 5 years). (a) Thickened bowel wall with multilayer was seen in the ileum with CT enteroclysis (arrow). The lumen stricture was also revealed. This finding was classified as Type 3. (B) Circular ulcers and luminal stenosis in the ileum were seen with videocapsule endoscopy.

DISCUSSION

Recently, capsule endoscopy and balloon endoscopy findings have shown that use of NSAIDs and low-dose aspirin may induce a variety of mucosal abnormalities of the small bowel in a high proportion of patients.^{3–8} Drug-induced small-bowel damage starts with mild inflammation or small ulcers of the mucosa and progresses to include various lesions such as annular ulcers and strictures.^{3,4} However, the endoscopic findings of drug-induced small-bowel damage are non-specific, and the diagnosis requires evaluation of the history of drug use and elimination of other inflammatory disorders.

CTE can examine the small bowel less invasively, similar to capsule endoscopy.^{1,2} However, few studies have comprehensively evaluated the CTE findings of drug-induced damage. In this study, the CTE findings of drug-induced small-bowel mucosal damage were classified into three types: Type 1 (mucosal type) may correspond to multiple minor mucosal damage such as small erosions or ulcers; Type 2 (homogeneous hyper-enhancement type) may correspond to large ulcers with significant oedema and redness; Type 3 (stratification enhancement type) may correspond to annular or large ulcers with strictures, observed on endoscopic examination.

Type 1 was found in most patients with short-term use of drugs (6 months or shorter); Type 2 in patients with a history of drug use of 3 years or longer; and Type 3 in patients with both long-term and short-term use of drugs.

Subtle CTE findings in Type 1 were often overlooked. In Type 2, homogeneously enhanced whole affected bowel was suggested as chronic inflammation. In Type 3, the mucosal layer is enhanced reflecting hyperaemia, and the submucosal layer is not enhanced reflecting oedema.^{9,10} These intramural multilayered structures are called “target sign”. Therefore, the mucosal damage was more severe in long-term users (Type 2) than in short-term users (Type 1). In Type 3, hyperaemia and oedema of the mucosa can induce a small-bowel stricture.

In 1/4 patients with Type 3 (long-term user of NSAIDs), the capsule endoscope was retained for a week because of a small-bowel stricture caused by a circumferential ulcer scar, suggesting a risk factor for capsule retention. Bowel stricture in Type 3 may be induced by strong oedema, and even in Type 2 by chronic inflammation. Therefore, CTE is recommended as an initial choice of examination and may be better combined with double-balloon endoscope or capsule endoscope. Endoscopic technique

Figure 6. Case 4—a 63-year-old male with non-steroidal anti-inflammatory drug-induced small-bowel injury (drug use of 3 years). (a) Thickened bowel wall with multilayer appearance (Type 3) and lumen stricture (arrow) were seen in the ileum with CT enteroclysis. (b) Circular ulcers and a fibrous stenosis in the ileum were seen with capsule endoscopy. The capsule endoscope was blocked at this site for 1 week.

Figure 7. A 70-year-old male with aspirin-induced small-bowel injury (drug use of over 10 years). A small linear ulcer was seen with capsule endoscopy (arrows).

as the next choice may be performed depending on the presence or absence of bowel stricture revealed on CTE.

Differentiation diseases of Type 1 include acute enteritis. Intestinal fluids were cultured and clinical manifestations such as diarrhoea or the abdominal pain were excluded from it. Crohn's disease, Behcet's disease, ischaemia colitis and intestinal tuberculosis can be considered as a differential diagnosis of Types 2 and 3. Ulcers were biopsied, and intestinal fluids were cultured to diagnose drug-induced small-bowel damage.

Our study has several limitations. CT enterography provides lesser degrees of small-bowel distension than those achieved with CTE. The poor distention is a matter of concern because in a study 19% of the patients (20 of 106) had insufficient degrees of small-bowel distension using CT enterography.^{11,12}

The superiority of VCE over CTE for the detection of small-bowel abnormalities related to NSAIDs has been reported.¹³ Among the ten patients with discrepant findings between CTE and VCE, VCE showed linear ulcers in relation to the use of NSAID (in three of them, which were unseen at CTE). In one patient, small linear ulcer was seen with VCE but not seen with CTE. Linear ulcer may be overlooked in CTE.

The use of barium–air (CO₂) enteroclysis has been recommended in patients with obscure gastrointestinal bleeding and negative VCE.¹⁴ This examination can be considered as the most accurate imaging test for the diagnosis of mucosal

abnormalities and thus can be used to detect ulcers or erosion due to NSAIDs. As a consequence, this examination should be considered as the first-line examination when the pre-test probability suggests NSAID enteropathy as a cause of obscure gastrointestinal bleeding.¹⁴ On the other hand, the adequate distension of the stomach filled with water as a negative contrast agent has led to improved visualization of the gastric wall or the gastric tumour itself without overshooting artefacts derived from intraluminal air.^{15–19} The water-filling method at gastric CT allows clear depiction of the gastric wall and gastric tumour without overshooting artefacts by air in the lumen.^{15–19}

One limitation is the use of endoscopy as the standard of reference. In this regard, Haghighi *et al*²⁰ have reported interesting results involving 160 patients with obscure gastrointestinal bleeding in whom erosions and ulcers were found in 29% and 21% of patients at VCE, supporting the evidence that this test has a high diagnostic yield. However, the same authors have found similar or even greater proportions of small-bowel abnormalities in healthy patients who served as controls.²⁰

Comparison of precise lesions of VCE or DBE and CTE is difficult, therefore, it is considered as one of the limitations.

The findings were subtle in Type 1, but the affected bowel mucosa was strongly enhanced. Therefore, there were few interobserver variability and false positives, but there might be many false negatives. The weakly enhanced lesion and mild mucosal change were poorly visualized on CTE compared with VCE or DBE.

Our study was limited to a small number of subjects. Abnormal findings could not be revealed by CTE in three patients even with long-term use of drugs. Mild small-bowel damage such as small erosions or ulcers with mild oedema or hyperaemia in long-term users may not be detected by CTE. Therefore, further studies are required to establish the CTE findings of drug-induced small-bowel damage.

CONCLUSIONS

The CTE findings of drug-induced small-bowel damage were classified into three types: Type 1 was found in most patients with short-term drug use; Type 2 was found in patients with long-term use; and Type 3 was found in patients with both long-term and short-term use. Type 3 changes in patients with long-term use of drugs may be a risk factor for capsule retention.

Drug-induced small-bowel damage is more common than has previously been recognized and these lesions may be encountered in modern CTE or VCE and DBE.

CTE can be available regardless of small-bowel stenosis and can play a complementary role in diagnosis of the mucosal damage.

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