PICTORIAL REVIEW

Perforated tumours in the gastrointestinal tract: CT findings and clinical implications

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ABSTRACT. Perforation usually requires emergency surgery and may affect the prognosis of patients with gastrointestinal tumours. Accurate pre-operative diagnosis of these conditions is important because proper management such as curative surgical treatment may be needed. The aims of this article are to illustrate CT appearances of perforated tumours of the gastrointestinal tract and to discuss their impact on clinical management.

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Perforation of the gastrointestinal (GI) tract can be caused by iatrogenic, traumatic and tumoral conditions. Perforation through tumours of the GI tract frequently leads to emergency conditions that require surgical management. In addition, most tumours in which perforation occurs are malignant, which may have a deleterious effect on the patient's prognosis.

We illustrate CT findings of GI tumours that can often present with perforation (Table 1), and also discuss the clinical impact of perforation.

Mechanism

Perforation of GI tumours can occur as a consequence of trauma and iatrogenic injuries [1–6]. Two fundamental factors responsible for development of spontaneous perforation are ischaemia and necrosis [1–6]. Neoplastic cells replace the bowel wall, and, subsequently, may undergo necrotic degeneration during rapid growth or under ischaemic conditions, both of which result in vulnerability to perforation. In addition, vascular occlusion by tumour cell infiltration, direct tumour embolisation and increased intraluminal pressure secondary to bowel obstruction may induce or accelerate bowel ischaemia [3, 5]. Bowel perforation occasionally occurs at a different location from the tumour site, as a result of increased intraluminal pressure caused by bowel obstruction or mucin-producing neoplasm.

Perforated gastric adenocarcinoma

Perforation of gastric adenocarcinoma is a rare occurrence, with a reported incidence of 0.4–6.0% [2]. It

usually affects the elderly (mean age 65 years) and presents at advanced tumour stages [2, 7, 8].

Diagnosis of gastric perforation can be made by CT findings, such as discontinuity of the gastric wall, the presence of a large amount of extraluminal air located mainly around the liver and stomach, and perigastric fat stranding with or without fluid (Figures 1 and 2) [9, 10]. Because the majority of cases of perforated gastric cancer are observed at advanced stages (tumour stage T3 or higher), underlying malignancy can be predicted by CT findings of gastric wall thickening with contrast enhancement and perigastric soft-tissue extension; omental cake formation and lymph node enlargement can be additional findings indicating malignant gastric perforation (Figure 1). However, perforation can occur in the early stages of gastric cancers when identification of the mucosal thickening may be limited on CT, and it usually occurs as a result of deep ulcerations (Figure 2) [2, 7, 8]. Therefore, underlying malignancy should be suspected in elderly patients with a gastric perforation in whom deep ulceration is assumed to be the cause on CT.

Perforated gastric cancer may be regarded as terminal disease because of the potential risk for peritoneal dissemination of tumour cells. Nevertheless, the most important factor predicting long-term survival has been reported to be tumour stage rather than tumour perforation [2, 7, 8].

Perforated colonic adenocarcinoma

The incidence of perforation in patients with colon cancer ranges from 1.2% to 10.0% [11]. Perforation occurs in one of two forms: it arises through the tumour itself, secondary to tumour necrosis, or it can occur at the colon proximal to the tumour as a result of “blow-out” of the proximal colon as a result of a closed-loop obstruction in which colonic pressure proximal to the obstructing cancer increases because of a competent ileocaecal valve

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Table 1. CT findings of perforated tumours in the gastrointestinal tract

Tumour type	CT appearances indicating malignancy	Common site	Other comments
Adenocarcinoma	Irregular wall thickening with contrast enhancement, omental cake formation (stomach), perigastric or pericolic mass invasion, lymph node enlargement	Stomach, colon	Early gastric cancer can perforate as a form of deep ulceration; colon cancer can show diastatic perforation of the caecum with closed-loop obstruction
Gastrointestinal stromal tumour	Large sized necrotic mass; presence of metastasis	Stomach, small bowel	Exophytic mass with internal haemorrhage or cystic degeneration or interval rapid growth has an increased risk of developing a spontaneous rupture
Metastasis	Focal bowel wall thickening with variable enhancement, coexisting primary malignancy at other sites	Small bowel	A small amount of free air is usually found around the perforated bowel
Lymphoma	Focal bowel wall thickening with homogeneous isoattenuation or hypoattenuation, multifocal bowel involvement, lymph node enlargement, hepatosplenomegaly	Small bowel	A small amount of free air is usually found around the perforated bowel, dilated bowel lumen (often)
Appendiceal mucinous neoplasm	Cystic dilated appendix (>1.3 cm) with enhancing wall nodularity, invasion of abdominal wall or retroperitoneum (may)	Appendix	The differential diagnosis from abscesses can be achieved by the presence of intratumoral/intramural calcification and a relatively paucity of surrounding fat stranding

Figure 1. A 55-year-old female with a perforated gastric adenocarcinoma. (a, b) Axial CT scans show a focal wall defect at the anterior wall of the gastric antrum with a small amount of air around the perforation site (arrowhead). Enhanced wall thickening of the gastric antrum is seen (large arrows). Note infiltrating lesions of soft-tissue attenuation in the adjacent omentum, indicating peritoneal dissemination (small arrows).

Figure 2. A 75-year-old male with a perforated gastric adenocarcinoma. (a) Axial CT scan shows a large quantity of extraluminal air anterior to the liver and stomach and a focal wall defect at the gastric body (arrowhead). Findings of oedematous wall thickening of the stomach (arrow) with scant perigastric fat infiltration observed in this case are not distinguishable from those of benign gastric perforation. (b) After primary closure of the gastric perforation, a large, deep ulceration is seen at the gastric body on the coronal CT image (arrowhead). (c) Gastrosocopy revealed a large, deep ulceration at the body portion. Poorly differentiated adenocarcinoma without evidence of peritoneal metastasis was confirmed by pathology.

Figure 3. A 52-year-old male with a perforated adenocarcinoma in the sigmoid colon. (a, b) Axial CT scans show irregular enhanced wall thickening of the sigmoid colon (large arrows) and a focal wall defect at the medial aspect (arrowhead). A small abscess is seen above the left psoas muscle (small arrows).

(Figures 3 and 4) [11–13]. Perforation occurs most commonly in the sigmoid colon and caecum [11, 14].

On CT, perforated colon cancer is frequently accompanied by the formation of an abscess or fistula, and peritonitis. Identification of irregular, concentric wall thickening of the colon with contrast enhancement adjacent to the wall defect, as well as the findings described above, is needed for differentiation of a malignant perforation from a benign colon perforation on CT (Figure 3) [10]. In addition, metastases are more commonly found in cases of perforated colon cancer than in cases of non-perforated colon cancer [11]. In patients with obstructive colorectal malignancy, marked dilatation of the right-side colon, such as of the caecum without ileal dilatation, owing to failure of decompression through the ileocaecal valve may represent impending diastatic perforation of the caecum due to closed-loop obstruction (Figure 4) [12, 13]. In addition, a caecal diameter >9 cm can be associated with an increased risk of perforation, and pneumatosis may precede perforation [13]. A large amount of free air is usually observed in cases involving coexistence of colon cancer perforation with colonic obstruction [11, 12].

Similar to cases of perforated gastric cancer, short- and long-term survival in these patients are determined by the severity of peritonitis and tumour stage, respectively [11, 14].

Ruptured gastrointestinal stromal tumour

Gastrointestinal stromal tumours (GISTs), which derive from the interstitial cells of Cajal or their precursor cells, are noted for expression of *c-kit* (CD 117) [1, 4]. The majority of ruptured GISTs occur spontaneously, and are located in the stomach and small bowel [1, 3, 4]. Most ruptured GISTs are high-risk cases [4].

On CT, ruptured GISTs frequently appear as heterogeneous tumours of laminated or whirled pattern that may be related to areas of necrotic or haemorrhagic degeneration; in addition, if ascites, uncommon for GISTs, coexists with the tumour, a ruptured GIST should be considered (Figures 5 and 6) [1]. In terms of gross morphology, exophytically located GISTs with internal necrosis or cystic degeneration have an increased risk of developing spontaneous rupture (Figures 5 and 6). Many ruptured GISTs are large (mean size 10.2 cm) [1, 4]. Moreover, tumour size (>5 cm) and tumoral necrosis as well as mitotic rate have been regarded as predictors of malignancy [1]. Rapid growth of masses on follow-up images could also be a feature of GISTs at increased risk of spontaneous perforation (Figure 5) [4].

Clinically, rupture in GISTs that occurs spontaneously or during surgery has been regarded as a poor prognostic factor because these patients have a very high risk of tumour recurrence [4].

Figure 4. A 99-year-old female with a colonic closed-loop obstruction secondary to rectal adenocarcinoma. (a) Coronal CT image shows an irregular enhancing mass in the rectum (arrows). (b) Marked luminal distension of the caecum (asterisk) and proximal ascending colon, and collapsed lumen of the terminal ileum (small arrow) are observed on a coronal CT image. Note free air bubbles (long arrows) in the left subhepatic space and ascites. Subsequently, an emergency operation revealed multiple perforations in the caecum.

Figure 5. A 79-year-old female with a ruptured gastrointestinal stromal tumour (GIST). (a) Coronal CT image demonstrates a large heterogeneous mass (large arrows) which shows an exophytic growth from the stomach (small arrows). Multifocal low-attenuation areas, corresponding to necrosis, with septa are seen within the tumour. The patient refused surgery. (b) Coronal CT image obtained 10 months later shows the interval growth of the mass (large arrows) abutting the stomach (small arrows). (c) A further 10 months later, the patient attended the emergency department of our hospital owing to development of acute abdominal pain. Internal high-attenuation haemorrhage was newly seen in the mass (large arrows) abutting the stomach (small arrows) on a coronal image. A large amount of high-attenuation ascites representing haemoperitoneum and clotted blood adjacent to the mass (asterisk) was also seen. A ruptured, high-risk GIST with haemorrhagic necrosis was confirmed pathologically after surgical resection of the tumour.

Perforated gastrointestinal metastasis

The most common malignancy responsible for perforation of GI metastases is lung cancer, which tends to involve the small bowel [6, 15]. The jejunum is more commonly affected by perforation than the ileum [3].

On CT, GI metastases of lung cancer appear as an intraluminal polypoid mass or wall thickening with variable patterns of contrast enhancement; in addition, they exhibit isoattenuation with a normal-appearing wall in many cases [15]. However, certain morphological patterns of bowel metastases are thought to be related to perforation; most cases of perforation occur in the

wall-thickening type, and usually accompany a small amount of free air around the pathological bowel (Figure 7) [15].

Perforated GI metastasis is an emergency situation requiring surgical intervention; it has a high operative mortality, and has been reported as a poor prognostic indicator [3, 6].

Perforated gastrointestinal lymphoma

Most GI lymphomas are non-Hodgkin's lymphomas of B-cell origin, whereas T-cell lymphoma does not

Figure 6. A 48-year-old female with a ruptured gastrointestinal stromal tumour (GIST). (a) Coronal reformatted enhanced CT image shows a large, irregular contoured mass with heterogeneous enhancement abutting the ileum. Note the slightly decreased enhancement in the lower lateral aspect of the mass (small arrows) compared with that of the upper medial portion (large arrows). (b) Pre-contrast scan shows high-attenuation fluid representing haemoperitoneum in the pelvic cavity. On pathological examination, this mass showed necrosis and rupture, corresponding to the lower portion of the mass. This mass was confirmed as a high-risk GIST.

Figure 7. A 79-year-old male with primary lung cancer (squamous cell carcinoma) presenting with acute abdominal pain. (a) Axial CT scan shows suspected focal wall thickening in the jejunum (wide arrow). Extraluminal air bubbles are seen around this jejunal loop (thin arrows). (b) The resected specimen shows three perforation sites induced by mucosal ulcerations (arrowheads). (c) Low-power photomicrograph (haematoxylin and eosin stain, $\times 4$) shows metastatic tumour infiltration mainly in the subepithelial layers, resulting in a bulging contour (large arrows). An irregular necrotic portion, connected to a mucosal ulceration, was found within the tumour (small arrows).

commonly involve the GI tract [5]. Peripheral T-cell lymphomas have a higher prevalence of bowel perforation (up to 50%) than B-cell lymphomas (<30%) [5]. The vast majority of bowel perforations in patients with GI lymphomas occur in the small bowel [3, 5, 16].

On CT, bowel wall thickening observed in T-cell lymphoma is not as severe as that in the B-cell type [5]. In addition, the bowel wall attenuation of T-cell lymphoma is homogeneous isoattenuation or hypoattenuation, which is similar to that of B-cell lymphoma [5]. Along with these morphological characteristics of

Figure 8. A 71-year-old female with a perforated peripheral T-cell lymphoma in the ileum. (a–c) Axial CT images demonstrate segmental, isoattenuated wall thickening of the ileum, which shows a mildly dilated lumen (arrows on c). Also note free air around the liver (thin arrows on a), enlarged mesenteric lymph nodes (small arrows on b) and mesenteric haziness (b). Perforation was found at surgery.

Figure 9. An 83-year-old female with perforated diffuse large B-cell lymphoma in the ileum. (a) Axial CT image shows mild wall thickening of the ileum in the right lower quadrant area (arrows). Multifocal free air (long arrows) and ascites (short arrows) are found in the peritoneal cavity. (b) The specimen shows a mass (arrows) with central perforation (arrowhead) in the ileum. This mass was confirmed pathologically as diffuse large B-cell lymphoma.

Figure 10. An 82-year-old male with a perforated mucinous adenocarcinoma of the appendix. (a) Axial and (b) coronal CT images show a large, low-attenuation mass with intratumoral calcifications (arrows) in the right lower quadrant. This mass extends to the right psoas muscle. Note the retroperitoneal spread of mucin through the discontinuity of the thin enhanced wall in the mass (small arrows on b).

the bowel wall, perforation can be presumed by observation of CT findings of pneumoperitoneum, peritoneal fat infiltration and ascites (Figures 8 and 9). However, because the small bowel is a preferential site for lymphomatous perforation, the amount of extraluminal air may be small, and the detection of focal wall defects on CT may be limited (Figures 8 and 9). Dilated lumen of the bowel as a result of lymphoma is often observed on CT (Figure 8). The presence of multifocal bowel involvement, lymphadenopathy and hepatosplenomegaly can be additional clues for the diagnosis of GI lymphoma as a cause of perforation.

Although bowel perforation has been associated with a worse prognosis in patients with GI lymphoma, it often does not adversely affect patients with lymphoma in remission [16].

Perforated appendiceal mucinous neoplasm

Appendiceal mucinous adenocarcinomas are associated with a higher incidence of perforation than appendiceal mucinous adenomas [17, 18]. Perforation can lead to diffuse peritoneal spread (so-called pseudomyxoma peritonei syndrome) or a localised sealing off around the appendix [17, 18].

On CT, cystic dilatation of the appendix with a luminal diameter >1.3 cm and mural calcification are the CT features indicating a coexisting mucocoele in patients with acute appendicitis [19]. In addition, while size, shape, wall thickness, septations and wall calcification are not helpful in differentiation of malignant from benign mucinous neoplasms, the finding of a cystic dilated appendix with enhancing wall nodularity, particularly in older patients, may indicate the presence of mucinous cystadenocarcinoma [17]. Perforated mucinous tumours appear as a dilated appendix with a focal wall defect, periappendiceal fat stranding and diffuse or localised low-attenuation mucinous fluid. Aggressive mucinous cystadenocarcinomas can invade the retroperitoneum or abdominal wall, and result in mucinous accumulation in these unusual sites (Figure 10) [18]. The presence of intratumoral or intramural calcification and a relative paucity of surrounding fat stranding can be clues for differentiation of a mucinous neoplasm from an abscess that presents with a low-attenuation lesion (Figure 10) [12].

Despite tumour spread to peritoneal surfaces, the outcome of treatments for perforated appendiceal mucinous tumours is not a bad one [18].

Conclusion

A variety of tumours arising in the GI tract can present with perforation. Although identification of the presence of a perforation by CT is important, diagnosis of the underlying cause of perforation will be more critical in administering proper management because the majority of tumour perforations are associated with malignant tumours.

References

1. Cegarra-Navarro MF, de la Calle MA, Girela-Baena E, García-Santos JM, Lloret-Estañ F, de Andrés EP. Ruptured gastrointestinal stromal tumors: radiologic findings in six cases. *Abdom Imaging* 2005;30:535–42.
2. Roviello F, Rossi S, Marrelli D, De Manzoni G, Pedrazzani C, Morgagni P, et al. Perforated gastric carcinoma: a report of 10 cases and review of the literature. *World J Surg Oncol* 2006;4:19.
3. Chao TC, Chao HH, Jan YY, Chen MF. Perforation through small bowel malignant tumors. *J Gastrointest Surg* 2005;9: 430–5.
4. Hohenberger P, Ronellenfitsch U, Oladeji O, Pink D, Ströbel P, Wardelmann E, et al. Pattern of recurrence in patients with ruptured primary gastrointestinal stromal tumour. *Br J Surg* 2010;97:1854–9.
5. Byun JH, Ha HK, Kim AY, Kim TK, Ko EY, Lee JK, et al. CT findings in peripheral T-cell lymphoma involving the gastrointestinal tract. *Radiology* 2003;227:59–67.
6. Yang CJ, Hwang JJ, Kang WY, Chong IW, Wang TH, Sheu CC, et al. Gastro-intestinal metastasis of primary lung carcinoma: clinical presentations and outcome. *Lung Cancer* 2006;54:319–23.
7. Shih CH, Yu MC, Chao TC, Huang TL, Jan YY, Chen MF. Outcome of perforated gastric cancer: twenty years experience of one institute. *Hepatogastroenterology* 2010;57: 1320–4.
8. Jwo SC, Chien RN, Chao TC, Chen HY, Lin CY. Clinicopathological features, surgical management, and disease outcome of perforated gastric cancer. *J Surg Oncol* 2005;91:219–25.
9. Furukawa A, Sakoda M, Yamasaki M, Kono N, Tanaka T, Nitta N, et al. Gastrointestinal tract perforation: CT diagnosis of presence, site, and cause. *Abdom Imaging* 2005;30:524–34.
10. Kim SH, Shin SS, Jeong YY, Heo SH, Kim JW, Kang HK. Gastrointestinal tract perforation: MDCT findings according to the perforation sites. *Korean J Radiol* 2009;10:63–70.
11. Abdelrazeq AS, Scott N, Thorn C, Verbeke CS, Ambrose NS, Botterill ID, et al. The impact of spontaneous tumour

- perforation on outcome following colon cancer surgery. *Colorectal Dis* 2008;10:775–80.
12. Kim SW, Shin HC, Kim IY, Kim YT, Kim CJ. CT findings of colonic complications associated with colon cancer. *Korean J Radiol* 2010;11:211–21.
 13. Gore RM, Szucs RA, Wolf EL, Scholz FJ, Eisenberg RL, Rubesin SE. Miscellaneous abnormalities of the colon. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*. 3rd edn. Philadelphia, PA: Saunders; 2008. pp. 1203–34.
 14. Tan KK, Hong CC, Zhang J, Liu JZ, Sim R. Surgery for perforated colorectal malignancy in an Asian population: an institution's experience over 5 years. *Int J Colorectal Dis* 2010;25:989–95.
 15. Kim SY, Ha HK, Park SW, Kang J, Kim KW, Lee SS, et al. Gastrointestinal metastasis from primary lung cancer: CT findings and clinicopathologic features. *AJR Am J Roentgenol* 2009;193:W197–201.
 16. Kako S, Oshima K, Sato M, Terasako K, Okuda S, Nakasone H, et al. Clinical outcome in patients with small-intestinal non-Hodgkin lymphoma. *Leuk Lymphoma* 2009;50:1618–24.
 17. Lim HK, Lee WJ, Kim SH, Kim B, Cho JM, Byun JY. Primary mucinous cystadenocarcinoma of the appendix: CT findings. *AJR Am J Roentgenol* 1999;173:1071–4.
 18. Sugarbaker PH. New standard of care for appendiceal epithelial neoplasms and pseudomyxoma peritonei syndrome? *Lancet Oncol* 2006;7:69–76.
 19. Bennett GL, Tanpitukpongse TP, Macari M, Cho KC, Babb JS. CT diagnosis of mucocele of the appendix in patients with acute appendicitis. *AJR Am J Roentgenol* 2009;192: W103–10.