Case Report
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Imaging Features of Gastric Invasive Aspergillosis: A Report of Two Cases
침습성위국균증의 영상소견: 2예 보고

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Invasive aspergillosis is an opportunistic infection that usually occurs in immunocompromised patients. Although there are a few rare reports of isolated invasive aspergillosis affecting the small intestine, isolated or disseminated gastric invasive aspergillosis is extremely rare. Herein, we report 2 cases of gastric invasive aspergillosis in a 72-year-old woman and a 43-year-old man; the woman had been recovering from ruptured left posterior communicating artery aneurysm, which presented as emphysematous gastritis and the man from acute subdural haemorrhage in the intensive care unit, which presented as a pseudoaneurysm on CT imaging.

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INTRODUCTION

Invasive aspergillosis is a rapidly progressive and highly lethal mycotic disease caused by *Aspergillus* species, usually *Aspergillus fumigatus*. The risk of this opportunistic infection is associated with the degree and duration of neutropenia. In particular, patients with severe neutropenia, a neutrophil count of less than $0.1 \times 10^3/\mu L$, that has had a duration of more than 3 weeks, are considered a high-risk group (1). Although the airway is the most common portal of entry, there are a few rare reports of isolated small bowel infection without pulmonary involvement (2-4). However, *Aspergillus* infection of the gastric wall is extremely rare. In this report, we describe the CT findings in 2 patients with gastric invasive aspergillosis.

CASE REPORT

Case 1

A 73-year-old woman presenting with a severe headache was admitted to our hospital. She had a past medical history of hypertension. The examining doctor was suspicious of hypertensive intracranial haemorrhage. The non-contrast-enhanced brain CT showed subarachnoid haemorrhage along the left sylvian fissure and tentorium. Emergency digital subtraction angiography confirmed an approximately 0.54-cm ruptured aneurysm in the left posterior communicating artery. An emergency open craniotomy and aneurysmal clipping were successfully performed. In the intensive care unit, one week after the operation, the patient complained of diffuse abdominal pain and progressive abdominal distension. Physical examination revealed epigastric tenderness and a tympanic sound in the upper abdomen. Blood tests revealed an elevated white blood cell (WBC) count ($24.29 \times 10^3/\mu L$, neutrophil 87%). Chest radiography showed patchy air-space consolidations in the right lower lung with costophrenic angle blunting. Our impression was pneumonia with parapneumonic effusion. Simple abdomen radiography was not reviewed at that time. Upon retrospective review of simple abdomen radiography,

emphysematous gastritis, which was missed by the examining doctor, was noted (Fig. 1A). However, the clinician thought that the cause of the abdominal pain and distension was unclear. The patient underwent a contrast-enhanced abdominal CT for further evaluation. The contrast-enhanced abdominal CT showed numerous intramural dirty air bubbles in the wall of the gastric fundus and body, and localised decreased wall enhancement was noted in those areas (Fig. 1B, C). There was no evidence of pneumoperitoneum. Contrast enhancement of the peripheral gastric and gastroepiploic arteries was not clearly visible. In addition, a focal wedge-shaped, low attenuating lesion was seen in the spleen (Fig. 1B). After considering all image features, our first impression was gastric gangrene due to non-specific infectious necrotising gastritis and splenic infarction. Because, the patient did not have any history of undergoing a medical procedure or aggressive vomiting, which can cause benign gastric emphysema, and the splenic infarction could not be explained by benign gastric emphysema alone, we considered ischemia and infarction of the stomach and spleen. However, gastric ischemia and infarction are extremely rare, because the blood supply in the stomach is well organized through a vessel network. The patient had no history of recent urgent hypotension or of a heart problem that would cause an embolism. The patient underwent emergency total gastrectomy with splenectomy and distal pancreatectomy. On gross pathological examination, the specimen showed diffuse gangrenous necrosis, without normal-looking tissue in the stomach (Fig. 1D). Microscopic examination revealed near-total, full-thickness necrosis, and numerous fungal, septate, tree-like branching hyphae consistent with invasive aspergillosis of the entire thickness of the gastric wall (Fig. 1E). In addition, fungal hyphae were seen in tail of the pancreas (Fig. 1F) and in the spleen. Despite treatment with antifungal agent medication, the patient died on day 20 after the gastric operation.

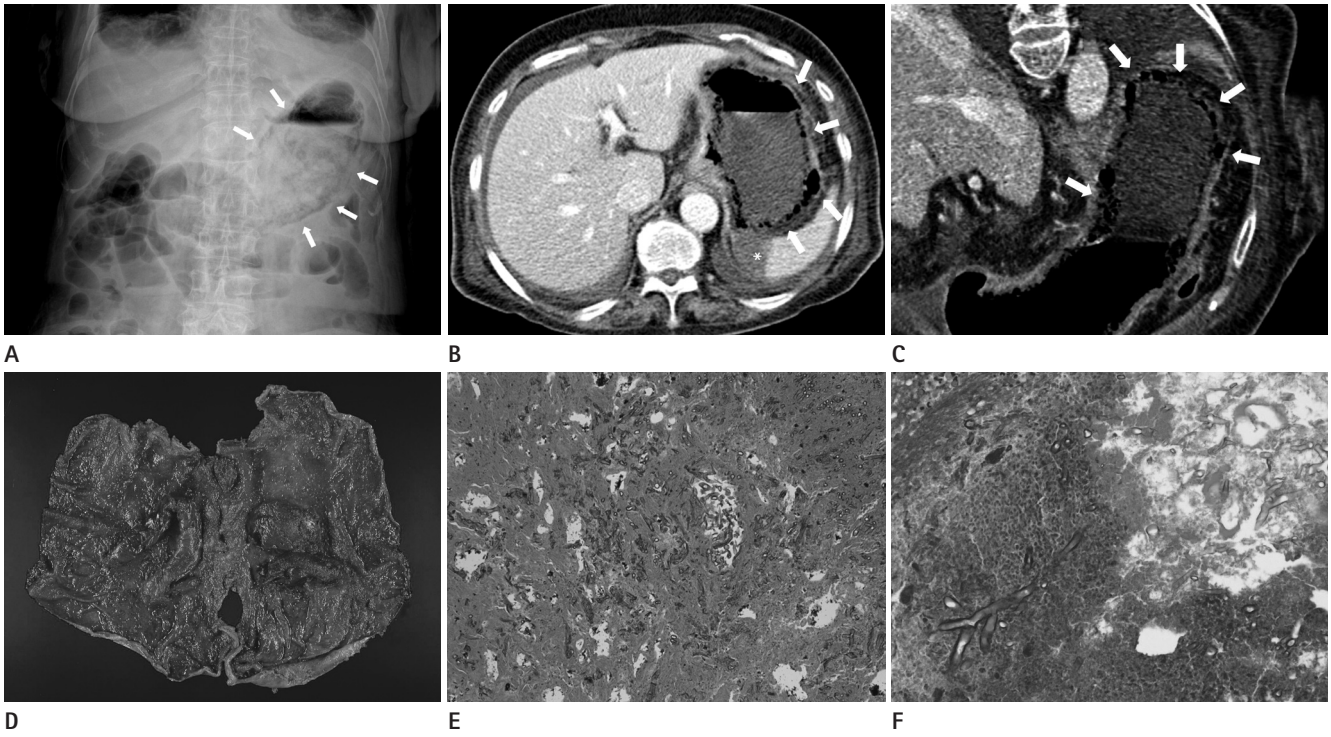


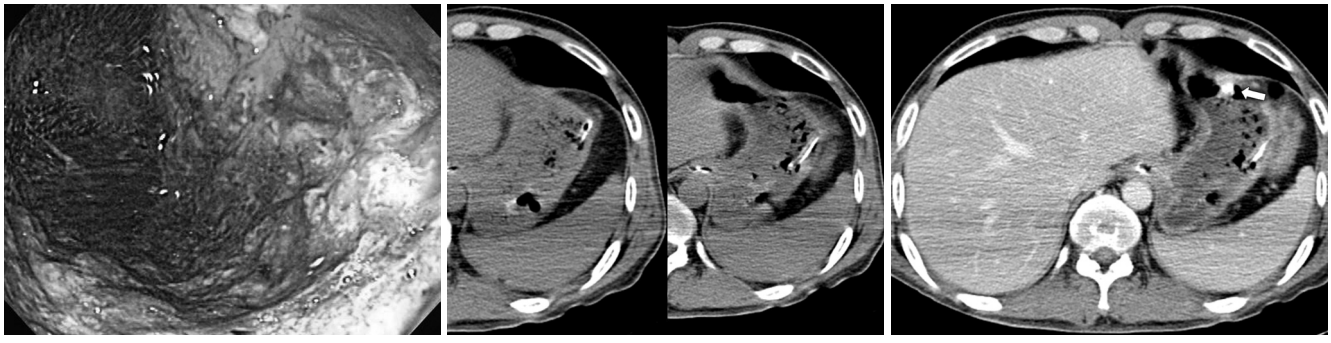
Fig. 1. A 73-year-old woman presenting with abdominal pain and distension, who was confirmed as having gastric invasive aspergillosis.
A. Simple abdomen radiography shows irregular dirty, elongated air bubbles (white arrows) along the gastric wall of the fundus and body.
B, C. Contrast-enhanced abdominal axial CT (**B,** portal phase) and a curved MPR image (**C,** portal phase) reveal numerous intramural dirty air bubbles (white arrows) in the wall of the gastric fundus and body. A localized decrease in wall enhancement is noted in those areas. A focal wedge-shaped low attenuating (asterisk) lesion is seen in the spleen.
D. The gross specimen displayed diffuse gangrenous necrosis in the stomach.
E, F. Microscopic examination reveals numerous fungal septate, tree-like branching hyphae consistent with invasive aspergillosis in the stomach (**E,** H&E, × 100) and tail of the pancreas (**F,** H&E, × 100).
Note.—MPR = multiplanar reformation

Case 2

A 43-year-old man was transferred to the emergency room of our hospital for the evaluation of his comatose status. A non-contrast-enhanced brain CT showed acute subdural haemorrhage in the right fronto-parietal area. Emergency craniotomy with a haemorrhage evacuation was performed successfully. On day 11 after the operation, increased heart rate (120 beats/minute, blood pressure 130/70 mm Hg) was noted upon regular check of his vital signs. The haemoglobin level (8.9 g/dL) was decreased, and the WBC count (16.21 × 10^3/μL, neutrophil 80%) was elevated upon an emergency check of the complete blood count. The examining doctor was suspicious of gastric ulcer bleeding and confirmed bleeding by Levin-tube aspiration. The patient was then subjected to emergency esophago-gastro-duodenoscopy (EGD). EGD showed multiple variable-sized ulcers in the gastric body and angle of the stomach. The gastric fundus and body were filled with fresh blood clots. However, there was no evidence of exposed vessels or active bleeding. EGD performed on the following day revealed multiple ulcers covered with greenish exudate, but no active bleeding (Fig. 2A). A biopsy was performed on a large ulcer in the greater curvature side of the gastric lower body. A contrast-enhanced abdominal CT revealed a round, intense, enhancing nodular lesion of approximately 0.8 cm in size in the anterior wall of the gastric body (Fig. 2C). This lesion was not delineated on pre-contrast images (Fig. 2B), and there was no intraluminal haemorrhage to consider as the focal spot of contrast media leakage. Thus, we considered the possibility of gastric bleeding due to mycotic aneurysm. The examining doctor explained this possibility, as well as the need for endovascular treatment and the administration of an antifungal agent to his family. The patient's family then moved him to another hospital. Two days later, microscopic examination of the gastric tissue, which was acquired by EGD, confirmed the diagnosis of invasive aspergillosis.

DISCUSSION

Aspergillus species are saprophytic molds that can cause a broad pathogenic spectrum. Over the decades, invasive aspergillosis has substantially increased and become of greater concern (5, 6). Invasive aspergillosis is usually acquired by inhalation. The extrapulmonary involvement of invasive aspergillosis is rare, but can involve the brain, sinus, kidney, liver, heart and pancreas. It has been reported that almost 25% of the patients with invasive pulmonary aspergillosis developed disseminated infection, and approximately 41-47% of these patients displayed gastrointestinal tract involvement (2). Although the lung is the usual portal of entry into the human host, isolated small bowel infections without lung involvement have been reported (2-4). The most commonly involved sites in the gastrointestinal tract are, in descending order, the oesophagus, colon, and small intestine (6). However, primary or disseminated gastric invasive aspergillosis is extremely rare. In addition, there have been only 2 reports describing imaging findings associated with gastric invasive aspergillosis. Yeom et al. (5) reported disseminated gastric invasive aspergillosis in a kidney-transplanted patient. They described the CT findings of a diffuse,



A B C

Fig. 2. A 43-year-old man presenting with gastric bleeding, who was confirmed as having gastric invasive aspergillosis.
A. EGD shows multiple variable-sized ulcers with greenish exudate in the gastric body and angle.
B, C. Contrast-enhanced abdominal CT (**C,** portal phase) reveals an approximately 0.8-cm, round, intense, enhancing nodular lesion (white arrow), representing a presumed pseudoaneurysm in the anterior wall of the gastric body. This lesion is not delineated on serial pre-contrast images **(B)**.
Note.—EGD = esophago-gastro-duodenoscopy

low attenuating gastric wall thickening that was confirmed as a transmural infarction in pathological specimens. Trésallet et al. (7) reported a large defect of the posterior gastric wall potentially due to primary digestive aspergillosis.

In our first case, contrast-enhanced CT imaging showed nodular and irregular collections of intramural gas bubbles in the gastric fundus and body, representing emphysematous gastritis, and revealed decreased wall enhancement in those areas. Unlike *Pseudomonas aeruginosa* or *Clostridium perfringens*, *Aspergillus* is not a gas-forming pathogen, and thus, this pathogen is not a direct cause of emphysematous gastritis. This finding on imaging can be explained by the typical characteristics of invasive aspergillosis. A disseminated form or a direct insult of *Aspergillus* hyphae invades medium- or small-sized blood vessels, which results in the sequence of disturbed blood flow, thrombosis, necrosis, and haemorrhagic infarction. Alternatively, it is possible that gastric wall necrosis caused by invasive aspergillosis, could be followed by a gas-forming bacteria superinfection (8). Our first case was disseminated invasive aspergillosis that involved the stomach, spleen, and pancreas. In contrast, the second case was manifested on images as a presumed pseudoaneurysm in the anterior wall of the gastric body. However, this imaging feature can also be explained by the same pathophysiology. The second case might be localised invasive aspergillosis limited to a single organ, which is why there was no evidence of any other organ involvement upon imaging and no clinical manifestation except for gastric bleeding.

From the radiological point of view, considering the imaging findings in our first case, it is essential to differentiate this condition from benign gastric emphysema caused by aggressive vomiting, instrumentation, drug use, etc. Images of benign gastric emphysema reveal a thin, linear distribution of air running parallel to the border of the stomach (9). Contrary to gangrenous emphysematous gastritis, gastric wall thickening is unusual in benign gastric emphysema (9). In addition, it is very helpful to understand the patient's clinical situation, such as that of an immunocompromised host or recent clinical history. Emphysematous gastritis can be caused by gas-forming microorganisms, such as *Streptococcus*, *E. coli*, *Pseudomonas aeruginosa*, *Clostridium perfringens*, and *Staphylococcus aureus* (*S. aureus*). There are also extremely rare reports of invasive gastric mucormycosis with emphysematous gastritis (8). Although invasive aspergillosis usually occurs in severely neutropenic patients, it is very difficult to differentiate this condition from emphysematous gastritis resulting from gas-forming organisms or other fungi by imaging alone. Considering the differential diagnosis of gastric aneurysm, pseudoaneurysm can be caused by infection, and the most common causative pathogens are *S. aureus* and *Streptococcus*. Sometimes, pseudoaneurysm can occur due to trauma, post-operative complications, and pancreatitis. However, these aetiologies cannot be differentiated by imaging alone. The patient history and clinical situation must be considered and correlated.

Because positive blood cultures are infrequent, a biopsy is essential to visualise fungal hyphae by microscopic examination for confirmation of a diagnosis (5). However, amphotericin B or a second-generation triazole antifungal agent should be used upon the suspicion of the diagnosis because it is a highly fatal, rapidly progressive disease in immunocompromised patients. Unfortunately, in spite of systemic therapy, the prognosis is poor in patients with intra-abdominal visceral organ involvement (10).

In conclusion, gastric invasive aspergillosis may present as emphysematous gastritis or pseudoaneurysm in the gastric wall, especially in an immunocompromised host. A prompt work-up and early, accurate diagnosis are very important because this is a rapidly progressing, life-threatening infection.

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침습성위국균증의 영상소견: 2예 보고

양동진 · 조승현 · 김성훈 · 신지열 · 이일기

침습성국균증은 주로 면역저하환자에서 발생할 수 있는 기회감염이다. 현재까지 국한성침습성국균증이 소장을 침범한 증례는 드물게 보고되어 있지만 국한성 혹은 파종성침습성국균증이 위를 침범한 경우는 매우 드물다. 저자들은 왼쪽뒤교통동맥류 파열 후 중환자실에서 회복단계에 있는 72세 여자 환자와 급성경막하출혈 후 회복단계에 있는 43세 남자 환자에서 각각 기종성위염과 거짓동맥류 형태로 발병한 두 증례를 보고하고자 한다.

대구파티마병원 영상의학과