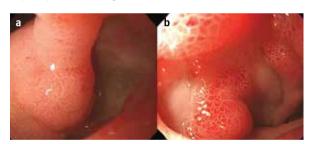
## Hepatic portal venous gas associated with hemorrhagic gastroduodenal ulcers

A 70-year-old man presented to the emergency department with a 7-day history of melena and epigastric soreness. His blood pressure was 130/80 mmHg and his pulse rate was 106/min. The physical examination was within normal limits except for anemic conjunctiva and mild epigastric tenderness. The laboratory tests were normal except for a hemoglobin level of 8.0 g/dL, white blood cell (WBC) count of 15400/mL with 77% neutrophils, and a C-reactive protein (CRP) of 2.6 mg/dL. Plain X-ray films of the chest and the abdomen were normal. Abdominal computer tomography (CT) showed duodenal bulb wall thickening and lumen narrowing along with tubular areas of decreased attenuation extending to within 2 cm of the liver capsule, which was consistent with HPVG in the liver (Figure 1). There were no gas densities in the mesenteric vessels any corresponding abnormalities in the large or small bowels. There is no evidence of intestinal inflammation. Sequentially performed upper gastrointestinal endoscopy showed two approximately 1.5-cm-sized, active, deep ulcers at the prepyloric antrum and the proximal portion of the duodenal bulb. Both ulcers showed erythematous and edematous surrounding mucosa, regular and sharp edges, and a flat and clean base without active bleeding (Figure 2). The rapid urease test was negative. The patient was treated with intravenous pantoprazole, ciprofloxacin and metronidazole. After 7 days, the air density in the liver disappeared on the follow-up abdominal CT (Figure 3) and the CRP returned to within normal limits at 0.2 mg/dL. After 2 months, upper gastrointestinal endoscopy showed two 0.5-cm-sized, linear ulcer scars at the prepyloric antrum and the duodenal bulb (Figure 4). HPVG is typically caused by severe diseases such as bowel necrosis or sepsis (1,2). It is a clinically important disease that has poor prognosis and distinguishing radiologic features such as CT (3,4). HPVG associated with only gastroduodenal ulcer and without the association of procedures such as dilatation, mucosal resection and foreign body removal is rare (5). In



**Figure 1.** In the abdominal CT, tubular areas of decreased attenuation extending to within 2 cm of the liver capsule suggesting of hepatic portal venous gas were noted (arrow).



**Figure 2 a, b.** Two about 1.5 cm sized, active, deep ulcers at the prepyloric antrum (a) and the proximal duodenal bulb (b) with erythematous and edematous surrounding mucosa, regular and sharp edge and flat and clean base without active bleeding were noted

this case, we hypothesized that HPVG was caused by air passing through the damaged mucosa of the ulcer. Moreover, the abdominal CT was obtained for evaluation of abdominal tenderness. We did not expect to find HPVG, which was an incidental finding. Therefore, this case shows that although the occurrence of HPVG associated with gastroduodenal ulcer is very infrequent, when the patient with a severe gastroduodenal ulcer has abdominal tenderness, leukocytosis and elevated CRP, HPVG should be differentiated.

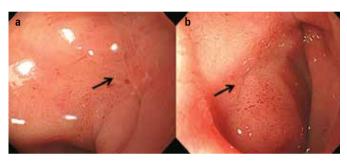
**Address for Correspondence:** Won Moon, Department of Internal Medicine, Kosin University Faculty of Medicine, Busan, Republic of Korea E-mail: moonone70@hanmail.net

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**Figure 3.** After 7 days with proton pump inhibitor and antibiotics, air density in the liver was disappeared on the follow-up abdominal CT.



**Figure 4. a, b.** After 2 months, two about 0.5 cm sized, whitish, linear ulcer scars were noted in the prepyloric antrum (a) (arrow) and the proximal duodenal bulb (b) (arrow).

**Conflict of Interest:** No conflict of interest was declared by the authors.

Youn Jung Choi, Won Moon, Seun Ja Park, Moo In Park, Sung Eun Kim Department of Internal Medicine, Kosin University Faculty of Medicine, Busan, Republic of Korea

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