

A Case of Portal Venous Gas after Rectal Surgery without Anastomotic Leakage or Bowel Necrosis

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Portal venous gas has traditionally been considered an indicator of a poor prognosis due to bowel necrosis. Portal venous gas has recently been detected in patients with various clinical conditions, such as Crohn's disease, chemotherapy, and blunt abdominal injury without bowel necrosis. We herein report the first case of a patient with rectal cancer in whom portal venous gas developed after low anterior resection without anastomotic leakage or bowel necrosis. A 66-year-old man who had undergone low anterior resection started having severe diarrhea the day after the operation. A fever was present for 2 days after the operation but resolved on postoperative day 3. The patient complained of abdominal pain 5 days postoperatively. Computed tomography showed portal venous gas. Emergency open laparotomy was performed, but only limited ascites fluid without leakage or bowel necrosis was found. We irrigated the abdominal cavity and performed an ileostomy with insertion of a drainage tube in the rectovesical pouch. Only serous ascites was discharged through the drainage tube. The portal venous gas disappeared 3 days after the second operation. The patient was discharged in good condition 21 days after the first operation. Portal venous gas can develop after rectal surgery without anastomotic leakage or bowel necrosis. Conservative treatment is reasonable for patients without signs of bowel necrosis or peritonitis. However, patients with portal venous gas must be carefully observed because portal venous gas may be life threatening. (*J Nippon Med Sch* 2015; 82: 202–205)

Key words: portal venous gas, rectal cancer

Introduction

Portal venous gas has traditionally been considered an indicator of a poor prognosis due to bowel necrosis¹. However, the widespread use of computed tomography (CT) has led to an increased rate of detection of portal venous gas in patients with various conditions, such as Crohn's disease², chemotherapy³, and blunt abdominal injury⁴, without bowel necrosis. The rate of detection of a variety of benign and non-life-threatening conditions associated with the presence of portal venous gas is increasing^{5,6}.

We herein report the first case of a patient with rectal cancer in whom portal venous gas developed after low

anterior resection without anastomotic leakage or bowel necrosis.

Case

A 66-year-old man was admitted to our hospital for the treatment of rectal cancer. Low anterior resection was performed, and a well-differentiated adenocarcinoma, T3 N1M0, stage IIIa, was diagnosed. Severe diarrhea developed the day after surgery, and on the next 4 days 800, 1,930, 1,810, and 1,265 mL of diarrheal stool was produced. A fever was present for 2 days postoperatively but resolved on postoperative day 3. Five days after surgery, the patient complained of abdominal pain. Only a

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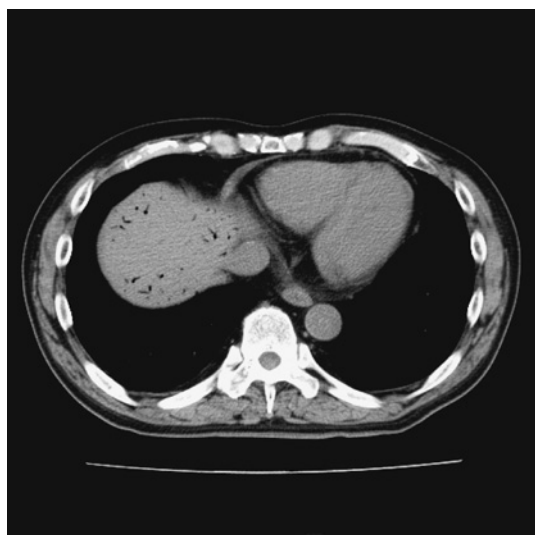


Fig. 1 CT 5 days after the first operation

Five days after the first operation CT showed portal venous gas mainly in the right hepatic lobe.

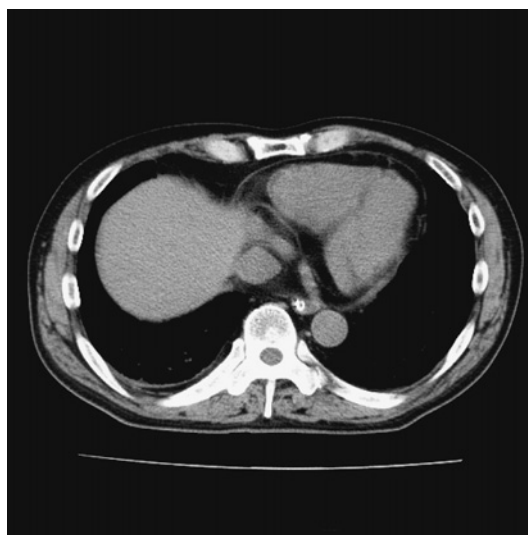


Fig. 2 CT 3 days after the second operation

8 days after the first operation (3 days after the second operation) CT showed that the portal venous gas had completely disappeared.

small amount of serous fluid, but no dirty fluid with feces, had been discharged from the drainage tube. The white blood cell count had not increased, and the C-reactive protein level was 13.97 mg/dL. An abdominal X-ray film revealed no free air, and CT showed limited ascites and portal venous gas (Fig. 1). A high CT value for adipose tissue was present around the rectum, but there were no signs of anastomotic leakage. *Clostridium difficile* toxin was not detected, but a fecal culture yielded *Pseudomonas aeruginosa*.

Emergency open laparotomy was performed because we suspected anastomotic leakage. However, we found only limited ascites without anastomotic leakage or bowel necrosis. We irrigated the abdominal cavity with 5,000 mL of saline and performed ileostomy. A drainage tube was inserted into the rectovesical pouch.

After the second surgery, only serous ascites fluid was discharged through the drainage tube. The portal venous gas disappeared 3 days after the second surgery (Fig. 2). The patient was discharged in good condition 21 days after the first surgery. Colonoscopy 3 months postoperatively showed no evidence of anastomotic leakage.

Discussion

Two important points were illustrated by the present case. First, portal venous gas can appear after colorectal surgery without anastomotic leakage or bowel necrosis. Second, conservative treatment may be reasonable for patients with portal venous gas after rectal surgery without other findings indicative of bowel necrosis.

The most important finding is that portal venous gas

can appear after rectal surgery without anastomotic leakage or bowel necrosis. Portal venous gas has been reported in association with acute pancreatitis⁷, acute appendicitis⁸, gastrojejunal anastomotic leakage after laparoscopic gastric bypass⁹, and percutaneous endoscopic gastrostomy tube placement¹⁰. To the best of our knowledge, however, this is the first report of portal venous gas in association with laparoscopic rectal surgery.

Portal venous gas has traditionally been considered to be an indicator of a poor prognosis and to be associated with a high mortality rate. Two causes of portal venous gas have been considered. The first cause is increased pressure in the bowel lumen. Digestive tract dilatation, gastric ulceration, ulcerative colitis, Crohn's disease, or complications of endoscopic procedures can increase the pressure in the bowel lumen, thus causing gas to escape from the mesenteric circulation into the liver vasculature. The second cause is overproliferation of gas-forming bacteria. Mesenteric vascular occlusion, bowel ischemia, and subsequent bowel necrosis can induce overproliferation of gas-forming bacteria such as *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Clostridium* spp., and the gas that is overproduced by these bacteria can escape into the portal venous system through the mesenteric vasculature. Therefore, portal venous gas can be seen in relatively benign situations under these clinical conditions. Bowel ischemia can induce both causes. Bowel ischemia results in damage to the mucosal barrier, which, in association with overdistension of the bowel loops and proliferation of gas-forming bacteria, leads to gas moving

Table 1 biochemical examination of blood

| | | | |
|-----|---|-----|-------------|
| WBC | 8,100 / μ L | TB | 0.8 mg/dL |
| RBC | 360 / μ L | BUN | 8.0 mg/dL |
| Hb | 11.5 g/dL | Cre | 0.58 mg/dL |
| Ht | 33.4 / μ L | TP | 5.5 g/dL |
| Plt | 28.0 \times 10 ⁴ / μ L | Alb | 2.5 g/dL |
| AST | 24 IU/L | Na | 135 mEq/L |
| ALT | 30 IU/L | K | 3.2 mEq/L |
| LDH | 150 IU/L | Cl | 101 mEq/L |
| ALP | 299 IU/L | CRP | 13.97 mg dL |

WBC: White blood cell, RBC: Red blood cell, Hb: Hemoglobin, Ht: Hematocrit, Plt: Platelet, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, LDH: Lactate dehydrogenase, ALP: alkaline phosphatase, TB: Total Bilirubin, BUN: Blood urea nitrogen, Cre: Creatinine, TP: Total protein, Alb: Albumin CRP: C-reactive protein

from the intestinal lumen to the mesenteric veins and finally to the portal system.

The present case may have involved both of the above-described causes. The patient had severe diarrhea after surgery. The severe diarrhea might have induced bowel dilatation and increased the pressure in the bowel lumen. Although fecal culture yielded only *P. aeruginosa*, the overproliferation of gas-forming bacteria can induce severe diarrhea; moreover, severe diarrhea can induce the overproliferation of gas-forming bacteria. Although *C. difficile* toxin was not detected, we cannot rule out the possibility of *C. difficile* infection. This bacterium is the most common infectious cause of hospital-associated diarrhea¹¹. The diagnosis of *C. difficile* infection requires identification of *C. difficile* toxin in the diarrheal stool. The accuracy of current diagnostic tests remains inadequate, and the optimal diagnostic testing algorithm has not been defined¹².

The second important finding in this case is that conservative treatment may be reasonable for patients with portal venous gas after rectal surgery but without signs of bowel necrosis. In the present case, the portal venous gas rapidly disappeared, as in other nonfatal cases. We suspected that the portal venous gas had been induced by anastomotic leakage and bowel necrosis and thus performed an ileostomy. However, the patient's condition after the second surgery suggests that neither laparotomy nor ileostomy was needed. When portal venous gas associated with ischemic bowel disease is encountered, other abdominal conditions should be considered both preoperatively and intraoperatively. However, careful observation of patients with portal venous gas after digestive surgery is needed because abdominal radiographs are not sensitive for early-stage bowel ischemia.

The prognosis of portal venous gas was historically considered to be poor. More recently, however, the prognosis has been considered to be more closely related to the cause of portal venous gas rather than to the presence of portal venous gas itself. Although bowel ischemia is the primary mechanism of portal venous gas formation, the presence of the portal venous gas itself does not provide any information about the extent of bowel necrosis. Bowel necrosis was reportedly detected in 14 of 33 (42.4%) patients with portal venous gas¹³. Additionally, portal venous gas that is associated with low systolic blood pressure, a high aspartate aminotransferase level, a high lactate dehydrogenase level, or intestinal pneumatosis reportedly indicates bowel necrosis¹³. However, intestinal pneumatosis by itself indicates bowel necrosis with or without portal venous gas.

Conclusion

Portal venous gas can appear after rectal surgery without anastomotic leakage or bowel necrosis. Portal venous gas by itself is not a surgical indication, and the most appropriate treatment depends mainly on the underlying disease. Conservative treatment is reasonable for patients without other findings indicative of bowel necrosis or panperitonitis. However, patients with portal venous gas must be carefully observed because the pathogenesis of portal venous gas is unclear, and portal venous gas can be life threatening.

Conflict of Interest: The authors declare no conflict of interest.

References

1. Kinoshita H, Shinozaki M, Tanimura H, Umemoto Y, Sakaguchi S, Takifuji K, Kawasaki S, Hayashi H, Yamaue H: Clinical features and management of hepatic portal venous gas: four case reports and cumulative review of the literature. *Arch Surg* 2001; 136: 1410-1414.
2. Pinto Pais T, Pinho R, Carvalho J: Hepatic portal venous gas and intestinal pneumatosis as initial presentation of Crohn's Disease: First case report. *J Crohns Colitis* 2014; 8: 1329-1330.
3. Zalinski S, Scatton O, Jacqmin S, Tacher V, Brezault C, Soubrane O: Portal venous gas following chemotherapy for colorectal cancer liver metastasis. *Eur J Surg Oncol* 2009; 35: 557-560.
4. Sen I, Samarasam I, Chandran S, Mathew G: Gastric intramural and portal venous gas following blunt abdominal injury. *Arch Trauma Res* 2013; 2: 95-96.
5. Inokuchi R, Fukuda T, Yahagi N, Nakamura K: Severe hepatic portal venous gas that spontaneously resolved within a day. *Intensive Care Med* 2014; 40: 1369.
6. Hou SK, Chern CH, How CK, Chen JD, Wang LM, Lee CH: Hepatic portal venous gas: clinical significance of

- computed tomography findings. *Am J Emerg Med* 2004; 22: 214–218.
7. Wu JM, Wang MY: Hepatic portal venous gas in necrotizing pancreatitis. *Dig Surg* 2009; 26: 119–120.
 8. Tuite DJ, Byrne A, Colhoun E, Torreggiani WC: Pneumatosis intestinalis and portal-venous gas: an unusual presentation of acute appendicitis. *Australas Radiol* 2007; 51: B137–139.
 9. Mognol P, Chosidow D, Marmuse JP: Hepatic portal gas due to gastro-jejunal anastomotic leak after laparoscopic gastric bypass. *Obes Surg* 2005; 15: 278–281.
 10. Bobba RK, Arsura EL: Hepatic portal and mesenteric vein gas as a late complication of percutaneous endoscopic gastrostomy tube placement in an elderly patient. *Dig Dis Sci* 2005; 50: 411–414.
 11. Antharam VC, Li EC, Ishmael A, Sharma A, Mai V, Rand KH, Wang GP: Intestinal dysbiosis and depletion of butyrogenic bacteria in *Clostridium difficile* infection and nosocomial diarrhea. *J Clin Microbiol* 2013; 51: 2884–2892.
 12. Ananthakrishnan AN: *Clostridium difficile* infection: epidemiology, risk factors and management. *Nat Rev Gastroenterol Hepatol* 2011; 8: 17–26.
 13. Koami H, Isa T, Ishimine T, Kameyama S, Matsumura T, Yamada KC, Sakamoto Y: Risk factors for bowel necrosis in patients with hepatic portal venous gas. *Surg Today* 2015; 45: 156–161.

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