CASE REPORT

Transient extensive hepatic portal venous gas following hypotension in a dialysis patient

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SUMMARY

A 68-year-old woman developed abdominal pain during a haemodialysis session and was transferred to our hospital. Although initial CT scan at former hospital detected extensive hepatic portal venous gas (HPVG), repeated CT scan on admission performed 2 h after the initial scan demonstrated a marked decrease in HPVG. Although HPVG is associated in some cases with bowel necrosis and high mortality, HPVG completely resolved within 18 h with only conservative treatment in presented case. Because recent increased use of CT scan allows early and highly sensitive detection of HPVG, not only in bowel necrosis, but also in non-life-threatening cases, we usually face a clinical dilemma whether to perform exploratory laparotomy. No previous studies have evaluated the duration of HPVG in detail, although this case suggests the value of CT scan follow-up in relatively stable patient even when the course of HPVG is short.

BACKGROUND

Hepatic portal venous gas (HPVG), formerly considered an ominous radiologic sign, is associated in some cases with severe underlying abdominal disease, such as bowel necrosis.^{1 2} It usually requires urgent and mandatory operative intervention. The current increased use of CT scan allows early and highly sensitive detection of not only severe illness but also non-life-threatening causes of HPVG. Because the prognosis is related to the pathology itself and is not influenced by the presence of HPVG,¹ appropriate treatment depends on the underlying disease. However, it is often difficult to exclude bowel necrosis and we must decide whether to perform exploratory laparotomy. We report a case of extensive HPVG which resolved gradually and completely within 18 h with conservative treatment. This case suggests the value of short-term CT scan follow-up in relatively stable patients with HPVG.

CASE PRESENTATION

A 68-year-old woman developed diffuse abdominal pain during a haemodialysis session and was referred to the emergency room of another hospital. Her medical history included type 2 diabetes mellitus for over 20 years, and haemodialysis-dependent end-stage renal disease for 14 years. Haemodialysis was performed three times a week, and the patient often developed hypotension during the procedure. Hypotension during this particular haemodialysis produced a systolic blood pressure of approximately 60 mm Hg. Upon

presentation, the patient's blood pressure was 72/40 mm Hg, her pulse rate was 102 bpm and her body temperature was 37.1°C. Although physical examination revealed generalised abdominal tenderness without muscular guarding, abdominal CT scan demonstrated the presence of extensive HPVG (figure 1A). The gas ran into the main hepatic vein, splenic vein and mesenteric veins (figure 1B–D). With a strong suspicion of bowel necrosis, the patient was immediately transferred to our hospital. On admission, she was alert, and her blood pressure increased to 103/50 mm Hg with fluid resuscitation. The respiratory rate was 20 breaths/min, and her temperature was 36.0°C. Her abdominal pain gradually resolved and abdominal tenderness almost disappeared. The extremities were warm, there was no cold sweat and the capillary refill time was within 2 s.

INVESTIGATIONS

Laboratory data revealed the following: white blood cell count 9270/μl; haemoglobin, 11.8 g/dl; platelets $85 \times 10^4/\mu\text{l}$; blood urea nitrogen, 33 mg/dl; creatine, 2.23 mg/dl; lactate dehydrogenase, 307 IU/l; creatine phosphokinase, 30 IU/l; C reactive protein, 1.10 mg/dl; blood glucose, 242 mg/dl. Arterial blood gas showed no acidemia; pH of 7.440; pO₂, 77.0 mm Hg; pCO₂, 34.0 mm Hg; HCO₃⁻, 23.1 mmol/l; base excess, -0.6 mmol/l and lactate, 3.1 mmol/l. A second CT scan with contrast medium performed 2 h after the initial scan demonstrated a marked decrease in HPVG and the disappearance of gas in the mesenteric vein. Also, there was no apparent evidence of intestinal pneumatosis or occlusive bowel ischaemia (figure 2A–C). Because she was haemodynamically stable and the lactate level remained only slightly elevated, with her symptoms and the HPVG decreasing rapidly, we judged it was unlikely that the patient had bowel necrosis.

DIFFERENTIAL DIAGNOSIS

- ▶ Non-occlusive mesenteric ischaemia without bowel necrosis.
- ▶ Bowel infection.

TREATMENT

We started conservative therapy with close follow-up and administered intravenous antibiotics after blood sampling for culture.

OUTCOME AND FOLLOW-UP

Follow-up arterial blood gas analysis showed a decreased lactate to 2.1 mmol/l 1 h after admission. Therefore, we elected not to perform an emergent

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Figure 1 CT scan on admission. (A) A non-contrast CT scan showing peripherally distributed air in the intrahepatic portal venous system bilaterally within 2 cm of the liver surface. Gas was also detected in the main trunk of the portal vein (arrowhead). (B–D) Gas was also seen within the splenic vein and mesenteric veins (arrowhead). There were numerous areas of venous gas in the mesentery of the transverse colon (arrow). Severe calcification of the abdominal aorta is seen.

Figure 2 (A–C) Contrast-enhanced CT scan obtained on admission to our hospital performed approximately 2 h after the initial scan demonstrating diminished gas with no evidence of bowel necrosis, pneumatosis, or occlusion of the mesenteric arteries. (D) A non-contrast CT scan obtained the next day showing slight ascites but no evidence of remaining gas in the intrahepatic portal veins. Hepatic portal venous gas completely disappeared within 18 h.

laparotomy. Repeated CT scan performed on the second hospital day, 18 h after the initial examination, demonstrated slight ascites but the HPVG image had completely disappeared (figure 2D). Although the patient developed moderate melena, her condition gradually improved with only supportive treatment. Melena and ascites detected on the following day was consistent with transient bowel ischaemia. Owing to the clinical and CT scan findings and no occlusive bowel ischaemia, the patient was finally diagnosed as non-occlusive mesenteric ischaemia related to hemodialysis. She was transferred to the previous hospital with no further complications on the sixth hospital day.

DISCUSSION

We report a case of transient HPVG-related to non-occlusive mesenteric ischaemia in a dialysis patient. Interestingly, extensive HPVG diminished over time and completely disappeared within 18 h with conservative treatment alone. Non-occlusive mesenteric ischaemia includes all forms of mesenteric ischaemia without occlusion of the mesenteric arteries.³ It is seen most often in patients who are undergoing dialysis, with an estimated incidence of up to 1.9% per patient/year and approximately 20 times greater risk compared with that of the general population.⁴ The dialysis population is increasing and aging, and the potential risk of non-occlusive mesenteric ischaemia is increasing as a result. This condition should be considered and ruled out in every haemodialysis patient with unexplained abdominal pain, particularly if the pain is associated with hypovolaemia, especially hypotension with atherosclerosis, as in our case.⁴ Clinical manifestations of non-occlusive mesenteric ischaemia are usually non-specific, but the condition should be considered in patients with long-standing haemodialysis with pre-existing risk factors. Non-occlusive mesenteric ischaemia may cause mesenteric vascular infarction and bowel necrosis with high death rates. However, in mild cases spontaneous recovery has been reported.⁵ Mucosal damage is an inevitable occurrence in such cases and is a trigger for HPVG. When the CT scan was reviewed in our case, there were numerous areas of venous gas in the mesentery of the transverse colon. This suggests the existence of ischaemia with this lesion, allowing intraluminal gas to enter the mesenteric venous system. Although we did not undergo a colonoscopy, her moderate melena is consistent with mucosal damage due to ischaemic colitis after spontaneous recovery of non-occlusive mesenteric ischaemia.

HPVG is associated with numerous underlying abdominal diseases, ranging from benign causes to potentially lethal disease requiring prompt surgical intervention.^{1 2} Although the mechanism for the appearance of gas in the portal vein is not well understood, three major pathophysiological mechanisms for gas production are considered⁶: first, damage to the gastrointestinal mucosa, such as with bowel ischaemia; second, abnormal bowel distension with increased intraluminal pressure that permits luminal gas to pass into the portal venous system, such as with bowel obstruction and trauma; third, bacteraemia involving the portal venous system with direct production of gas.

Because previous studies reported that HPVG was associated most commonly with bowel necrosis and carried a high death rate, HPVG was formerly an absolute indication for surgery due to mesenteric ischaemia.^{1 2 7} However, with the development of highly advanced imaging techniques, particularly with the penetration of multidetector-row CT, the detection rate of HPVG has increased. Also, HPVG in non-life-threatening cases, such as diverticulitis, gastric dilation and inflammatory bowel disease, has now been frequently treated without surgical intervention,^{1 2} avoiding unnecessary exploratory laparotomy.

On the other hand, because early intervention is vital in cases of surgical illness such as bowel necrosis, HPVG should trigger a careful examination. With the aid of CT scan, the salvage rate has increased from 25% to 44% and supports aggressive surgery in ischaemic patients with HPVG.^{2 8} Because the prognosis is related to the pathology itself and not influenced by the presence of HPVG, it is critical to make an accurate diagnosis of bowel necrosis. CT findings of bowel necrosis or bowel ischaemia are non-specific and include bowel wall thickening, absent enhancement of the bowel wall, intramural pneumatosis and mesenteric oedema in addition to HPVG. The presence of massive HPVG carries a severe prognosis when found on plain radiographs versus with CT scan.^{2 9} But our case suggests that there is no direct correlation between the amount of HPVG and severity. The detection of HPVG by CT scan, alone, cannot determine clinical or surgical management. In practice, bowel necrosis is diagnosed on the basis of host risk factors, physical examination, laboratory findings, especially increased lactate level and radiological findings. Although there are published algorithms for management of patients with HPVG,^{2 10 11} they are neither perfect nor perfectible. Also, making a precise diagnosis of bowel necrosis without laparotomy is often difficult. Considering the findings in this case, short-term CT scan follow-up in relatively stable patients with HPVG is worth considering.¹²

In conclusion, in view of the increased rate of non-occlusive mesenteric ischaemia in haemodialysis patients, the use of CT scan increases the probability of detecting HPVG. Traditionally, HPVG has carried a bad prognosis with no choice other than urgent laparotomy. However, appropriate and accurate management of the underlying disease is the most important factor and the treatment of patients with HPVG should be individualised according to the clinical picture. No previous studies have evaluated the duration of HPVG in detail, although our case suggests the value of CT scan follow-up even when the course of HPVG is short. Stable symptoms without lactate elevation must be present to choose a 'watchful waiting' strategy.

Learning points

- ▶ With the development of multidetector-row CT, the detection rate of hepatic portal venous gas (HPVG) has increased even in non-life-threatening diseases.
- ▶ The prognosis is related to the pathology itself and there is no direct correlation between the amount of HPVG and severity.
- ▶ Appropriate and accurate management of the underlying disease is the most important factor and the treatment of patients with HPVG should be individualised according to the clinical picture.
- ▶ Short-term CT follow-up in relatively stable patients with HPVG is worth considering.

Competing interests None.

Patient consent Obtained.

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