CASE REPORT

Hepatic portal venous gas and portal venous thrombosis following colonoscopy in a patient with terminal ileal Crohn's disease

Adler Shing Chak Ma,¹ Iain Ewing,² Charles Daniel Murray,³ Mark Ian Hamilton³

¹Royal Free London NHS Foundation Trust, London, UK

²Department of Gastroenterology, Whittington Health NHS Trust, London, UK

³Department of Gastroenterology, Royal Free London NHS Foundation Trust, London, UK

Correspondence to
Dr Adler Shing Chak Ma,
adlerma@doctors.org.uk

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SUMMARY

A 27-year-old man developed extensive hepatic portal venous gas (HPVG) shortly after staging colonoscopy for active, ulcerating, terminal ileal Crohn's disease. Non-operative management was instigated with broad-spectrum antibiotics and thromboprophylaxis. Radiology at 72 h demonstrated resolution of HPVG but revealed fresh non-occlusive left portal vein thrombus. Anticoagulation with warfarin was continued for 1 year, during which the thrombus initially progressed and then organised with recanalisation of the portal vein. There were no long-term clinical consequences. HPVG has previously been documented as a rare complication of inflammatory bowel disease and endoscopic intervention. We hypothesise that the barotrauma sustained during endoscopy, in association with active ulceration and mucosal friability, predisposes to the influx of gas and bacteria into the portal system. We describe successful non-operative management of HPVG in this setting and draw attention to an additional complication of portal venous thrombosis, highlighting the importance of thromboprophylaxis and serial radiological examination.

BACKGROUND

Hepatic portal venous gas (HPVG) is hypothesised to result from entry into the portal circulation of intraluminal gas or gas produced by intestinal bacteria.^{1 2} It has been described as a 'diagnostic clue' to potentially life-threatening intra-abdominal pathology.³ HPVG was historically associated with acute mesenteric ischaemia and early studies suggested very high mortality rates of 75–80%.² Advances in cross-sectional imaging technology have led to increased detection of HPVG in more 'benign' conditions including inflammatory bowel disease (IBD), intra-abdominal abscess and, rarely, following instrumentation of the bowel.¹ Recent studies have therefore reported lower mortality rates of 25–35%.^{3 4}

It is likely that the barotrauma sustained during endoscopy represents an additional risk factor for HPVG in patients with Crohn's disease and active inflammation. As such, clinicians performing endoscopy should be alert to the risk of HPVG as a high level of clinical suspicion is required to instigate appropriate investigation and immediate treatment.

We report a further complication of associated portal venous thrombosis (PVT), which has not previously been widely described in connection with HPVG. This case illustrates the importance of

performing serial radiological examination following the diagnosis of HPVG to exclude evolving PVT.

CASE PRESENTATION

A 27-year-old man with suspected terminal ileal Crohn's disease underwent colonoscopy to establish a histological diagnosis and stage disease activity. He had first presented at the age of 16 with a 1-year history of weight loss, abdominal pain and diarrhoea. Barium small bowel follow-through at presentation revealed cobblestone mucosa and ulceration in the distal 30 cm of ileum, suggesting a diagnosis of Crohn's disease. Symptoms remitted after an initial 6-month course of prednisolone and mesalazine. Two subsequent flares with abdominal pain and vomiting were treated with empirical steroid therapy without further investigation. The patient had no other significant medical history, was employed as an events manager, smoked 10 cigarettes daily and had a family history of IBD in a distant cousin.

At the time of reassessment, aged 27, he experienced intermittent abdominal pain but reported no other symptoms. Small bowel barium follow-through was repeated at this time and demonstrated long segment luminal narrowing of the terminal ileum consistent with stricturing Crohn's disease. This was followed by colonoscopy with terminal ileal intubation, which was completed without technical difficulty. The colon was macroscopically normal with no inflammatory changes on histological examination. The terminal ileum was oedematous and active ulcerating disease was noted. The mucosa was friable and significant bleeding followed forceps biopsy but settled spontaneously without haemodynamic disturbance. Terminal ileal biopsies confirmed moderately to severely active Crohn's disease with features of crypt architectural distortion, villous flattening and ulceration.

Within hours of the procedure, the patient developed fever and rigours that were not associated with shock. Immediate treatment was started with cefotaxime while awaiting urgent cross-sectional imaging.

INVESTIGATIONS

CT of the abdomen revealed pronounced thickening and oedema of the terminal ileum in keeping with Crohn's disease, and extensive HPVG throughout the intrahepatic portal venous radicals

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Figure 1 Postcolonoscopy CT of the abdomen revealing extensive hepatic portal venous gas throughout the intrahepatic portal venous radicals.

(figure 1). The main portal vein was patent and there was no radiological evidence of intestinal perforation.

DIFFERENTIAL DIAGNOSIS

As intestinal perforation can also present with fevers and rigours following colonoscopy, cross-sectional imaging with CT must be performed urgently to clarify the diagnosis.

TREATMENT

Low-molecular-weight heparin was started at a prophylactic dose and antimicrobial cover was broadened with gentamicin, teicoplanin and ciprofloxacin. Following surgical review, a decision was made to proceed initially with non-operative management.

Repeat imaging at 72 h showed radiological resolution of HPVG, with no evidence of abscess or perforation. Non-occlusive thrombus was present within the left main portal vein radical. Anticoagulation was increased to treatment dose low-molecular-weight heparin and warfarin was started. Serial ultrasound scans performed over the following 72 h showed progression to occlusive left portal vein thrombus. Thrombophilia screening was undertaken and uncovered heterozygosity for factor V Leiden, but no other prothrombotic predisposition.

OUTCOME AND FOLLOW-UP

Symptomatic improvement and radiological resolution of HPVG were achieved with conservative management alone. Ultrasound performed after 6 months of warfarin therapy showed maturation of the portal venous thrombus with recanalisation, and no evidence of portal hypertension or atrophy. Warfarin was discontinued after 1 year, with no subsequent recurrence of portal vein thrombosis.

DISCUSSION

A young man with Crohn's disease developed fever and rigours in association with CT evidence of HPVG shortly after colonoscopy. In keeping with the existing literature on HPVG, we hypothesise that barotrauma sustained during endoscopy in the context of active mucosal inflammation predisposed to the insufflation of gas and bacteria into the portal system.

Crohn's disease, ulcerative colitis and endoscopic intervention are known to be risk factors for HPVG, although it is a rare finding in all three settings.^{3 5 6} HPVG is most commonly seen in the context of intestinal ischaemia and necrosis, where it carries a very high mortality rate of 75–80%.³ Early studies thus advocated emergency laparotomy following a diagnosis of

HPVG on plain imaging.⁷ Advances in imaging technology have resulted in increased sensitivity: CT scanning is the current gold standard for diagnosing HPVG. In a recent series of 182 cases, overall mortality was 39%.³ However, no deaths were reported among the seven cases of HPVG with ulcerative colitis, seven cases with Crohn's disease and seven cases following endoscopy.

As such, treatment of HPVG should be directed by the underlying condition and severity of illness.⁴ HPVG associated with IBD, in spontaneous as well as postcolonoscopy settings, frequently resolves with conservative management, although a low threshold for surgical review should be maintained. Alqahtani *et al*¹ described a case of HPVG following colonoscopy in a 26-year-old woman with Crohn's disease. This presented as right upper quadrant pain 30 min after colonoscopy and resolved with broad-spectrum antibiotics. In another case reported by Salyers and Mansour,⁸ HPVG developed following sequential colonoscopy and small barium follow-through in a 24-year-old man with Crohn's disease. Treatment was as for active Crohn's colitis and subsequent abdominal films demonstrated radiological resolution of HPVG within 24 h.

In the case presented here, HPVG similarly resolved with medical management. We describe an additional sequela of PVT detected at 72 h, following radiological resolution of HPVG. This was successfully managed with anticoagulation but highlights the importance of performing serial radiology following the diagnosis of HPVG to exclude developing thrombus.

To our knowledge, PVT developing in association with HPVG as a postcolonoscopy complication has not been reported in the context of Crohn's disease. It is well known that IBD is a hypercoagulable state. Indeed, a postmortem detection rate of 40% in patients with IBD suggests that thromboembolic events are underdiagnosed in this population.⁹ Deep vein thrombosis and pulmonary embolism account for the majority of thromboembolic events in patients with IBD, as in the general population. The incidence of PVT is also higher in patients with IBD than in the general population.¹⁰ The mortality rate of untreated PVT in IBD has previously been reported to be as high as 50%, but treatment with low-molecular-weight heparin is usually effective.¹⁰ The European Crohn's and Colitis Organisation (ECCO) therefore recommends thromboprophylaxis in all hospitalised patients with IBD.⁹ As described above, low-molecular-weight heparin at a prophylactic dose was started early in the management of our patient, before an actual diagnosis of PVT was established.

It is currently understood that endothelial damage plays a key role in potentiating thromboembolism in IBD. Damage to the vascular wall activates the haemostatic system, triggering thrombus formation and inflammatory infiltration.⁹ Studies comparing patients with active IBD, inactive IBD and normal controls have revealed an upsurge in the levels of procoagulant factors, and decrease in anticoagulant factors in active disease.^{9 11} As anticoagulant mechanisms serve to dampen the inflammatory process, their suppression leads to a vicious circle of further inflammation and increased prothrombotic tendencies.

We found only two cases that linked HPVG with portal vein thrombosis. Both of these patients were significantly more unwell than the patient described in our case. Ng *et al*¹² reported on a 19-year-old Chinese man with Crohn's disease who was admitted with peritonitis and septic shock. Imaging revealed HPVG, PVT, gross ascites and pneumoperitoneum. Emergency laparotomy was performed and the patient eventually proceeded to total colectomy. Anticoagulation was delayed by severe coagulopathy, but partial recanalisation of the portal vein with collaterals was observed on follow-up CT. The patient

recovered following his fourth laparotomy and was discharged after an inpatient stay of 2 months. Mohammed *et al*¹³ described a 74-year-old man with chronic renal failure and *Klebsiella* bacteraemia. HPVG and PVT were diagnosed on imaging, in association with diverticular disease. The patient subsequently developed paralytic ileus, metabolic acidosis, and locules of gas and free fluid, in the abdomen and pelvis. He was too unwell for surgery and died on day 16 postadmission.

Although our understanding of the relationship between PVT and HPVG is limited, endothelial damage appears to be the underlying feature unifying the cases and studies described above. Given the very small number of cases, it is unclear whether HPVG represents an independent risk factor for PVT. However, the same features that predispose to HPVG—namely ulceration and mucosal friability in IBD, compounded by the trauma sustained during endoscopic intervention—are also likely to be implicated in the pathogenesis of PVT. As such, we believe that the association is important, and underscores the

necessity of starting thromboprophylaxis and screening for developing thrombus as part of the standard management of patients with HPVG and IBD.

Contributors IE and ASCM wrote the report. MIH and CDM were the consultants principally involved in the patient's care, and provided key data, made revisions and approved the article for submission.

Competing interests None declared.

Patient consent Obtained.

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Learning points

- ▶ Hepatic portal venous gas (HPVG) is a rare finding following colonoscopy in active Crohn's disease. Endoscopists should be aware of this clinical entity, and consider it in the differential diagnosis of patients who become acutely unwell following colonoscopy.
- ▶ HPVG alone is not an absolute indication for surgery: conservative management with aggressive antibiotic therapy and thromboprophylaxis is a reasonable initial approach, as evidenced by recent case series.
- ▶ Portal venous thrombosis may occur as a delayed complication.
- ▶ Thromboprophylaxis with low-molecular-weight heparin should be started early, and follow-up radiological investigation is indicated to exclude evolving thrombosis, even if not present at the time of discovery of HPVG.

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