

Oncology

Neobladder Obstruction: A Non-ischemic Cause for Hepatic Portal Venous Gas: Case Report

Haider K. Bangash^a, Steve P. McCombie^a, Omar K. Bangash^b, Dickon Hayne^{a,b,*}^a Department of Urology, Fiona Stanley Hospital, Perth, Australia^b School of Surgery, University of Western Australia, Perth, Australia

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ABSTRACT

Hepatic portal venous gas (HPVG) is a rare ominous radiological sign usually indicative of mesenteric ischemia. Increased detection of HPVG has been associated with a growing number of non-ischemic causes. A 64-year-old gentleman following radical cystectomy and neobladder formation developed clinical signs suggestive of bowel obstruction. HPVG was demonstrated on abdominal imaging. Urgent laparotomy revealed no evidence of ischemia. We hypothesize an obstructed neobladder permitted gas to enter the mesenteric circulation. The patient made a complete recovery with supportive management. © 2017 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Introduction

Presence of hepatic portal venous gas (HPVG) is an ominous sign usually indicative of mesenteric ischemia with an associated high mortality.¹ HPVG however has also been associated with several benign causes which can be treated non-operatively.

We report a 64 year old gentleman who underwent radical cystectomy and neobladder formation was found to have HPVG on post-operative day 6. To the best of our knowledge this is the first reported case of HPVG secondary to an obstructed neobladder.

Case presentation

A 64 year old Caucasian gentleman referred with visible hematuria was diagnosed with T1 high-grade urothelial carcinoma and carcinoma in-situ (CIS) of the bladder. He failed bladder sparing treatment with persistence of CIS. The decision was made to undergo elective radical cystectomy and orthotopic neobladder formation. His medical co-morbidities included type 2 diabetes mellitus, coronary artery bypass surgery and hypertension. Pre-operative laboratory tests were unremarkable and staging imaging was negative for metastasis.

A radical non-nerve sparing cystectomy, bilateral lymph node dissection and orthotopic ileal neobladder formation was performed using the Studer technique.² A 69 style Wallace uretero-uretero anastomosis over 7 Fr bander ureteral stents was performed, with the ureteral stents brought out from the neo-bladder through the mesoileum and skin. The urethral anastomosis was performed over 20 Fr indwelling urinary catheter (IDC) using 6 anastomotic sutures. Estimated blood loss was 600 ml with no intra-operative complications or concerns.

As per our centers cystectomy care pathway he was commenced on prophylaxis to prevent venous thromboembolism, gastrointestinal ulceration (pantoprazole), and ileus (metoclopramide and chewing gum). His IDC was flushed 6 hourly with 50 ml of 0.9% saline. He opened his bowels on day 4; he was upgraded to normal diet. On day 6 he had worsening nausea, vomiting and abdominal distension. The abdomen was distended though not peritonitic. He remained afebrile however his white cell count (WCC) increased to $14.70 \times 10^9/L$ with a mildly elevated serum lactate 2.5 mmol/L. His urine output remained at approximately 40 ml/h and his drain output was 400 ml over 24 h. Computed tomography (CT) of his abdomen and pelvis demonstrated a distended stomach with dilated proximal small bowel loops but no obvious transition point. Concerningly, it also demonstrated moderately extensive HPVG (Fig. 1), gas within the neo-bladder and likely within the neo-bladder wall (Fig. 2).

Colorectal team review advised urgent exploratory laparotomy to exclude ischemia of bowel and neobladder. During laparotomy, there was extensive small bowel dilatation without a transition point, the small bowel anastomosis was intact, and there was no

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* Corresponding author. Fiona Stanley Hospital, 11 Robin Warren Dr, Murdoch, WA 6150, Australia.

E-mail address: Dickon.Hayne@uwa.edu.au (D. Hayne).

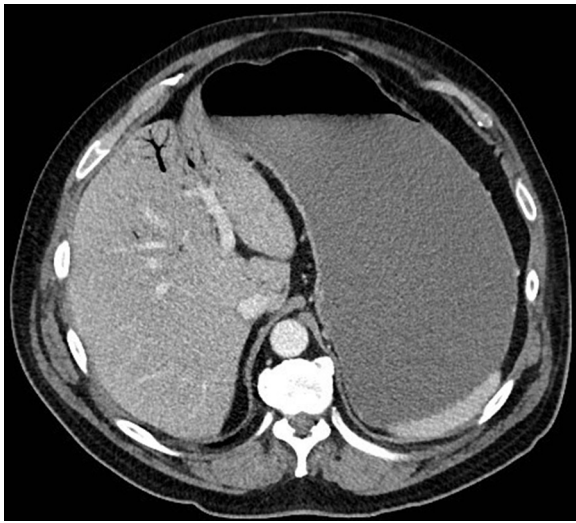


Figure 1. CT (portal venous phase) showing hepatic portal venous gas with a markedly distended stomach.

evidence of ischemic colitis. The neobladder was noted to be well perfused but significantly distended despite the IDC being in-situ. Intra-operative neobladder washout was performed and a large amount of mucous was evacuated. There was no evidence of neobladder leak.

Post-laparotomy he was kept nil-by-mouth with a nasogastric tube kept on free drainage. He was commenced on total parenteral nutrition (TPN). Mucous from the neobladder was sent for bacterial culture which demonstrated polymicrobial bacterial growth and he was continued on broad spectrum intravenous antibiotics. Regular neobladder flushes were performed which remained clear. He went on to make a full recovery however his inpatient stay was prolonged being discharged 20 days post-procedure.

Discussion

Hepatic portal venous gas (HPVG) was first reported in 1960 by Susman and Senturia in a patient with bowel ischemia secondary to superior mesenteric artery thrombosis.³ Since then HPVG has been considered an ominous sign necessitating urgent laparotomy.¹



Figure 2. Gas within the neobladder and likely within the neobladder wall. Banded stents arising from the neobladder are also noted.

Initial mortality rates were reported between 75 and 100%.⁴ There have now been over 100 cases of HPVG reported from various causes.⁵ The increased usage of ultrasound and CT in the inpatient setting has allowed for an increase in the early detection of HPVG, and better understanding of causative pathology. Contemporary studies have therefore reported lower overall mortality rates between 29 and 39%.⁵ HPVG is no longer considered pathognomonic for bowel ischemia and is considered a sign which requires diagnosis and should facilitate timely and targeted treatment.

The pathogenesis of HPVG is variable and still not fully understood, although three widely proposed theories exist.⁵ Firstly, an increased gastrointestinal intraluminal pressure from mechanical obstruction causes migration of swallowed gas via mural capillaries into the portal venous circulation. Secondly, that intestinal mucosal disruption may allow the passage of gas-forming bacteria into the bowel wall. Thirdly, due to the presence of a gas-forming abscess or porto-mesenteric pyelophlebitis.^{4,5}

Although ischemic causes of HPVG warrant emergency laparotomy, non-ischemic causes of HPVG can be safely managed conservatively by addressing the underlying cause. In most cases supportive treatment with intravenous fluids, close monitoring, intravenous antibiotics, NGT and bowel rest has been shown to have good outcomes.¹ It seems likely but remains unknown if the patient would have fully recovered with purely non-operative management.

HPVG is more likely to result from bowel ischemia in the post-operative patients than in the non-operative setting.¹ Our patient had worsening abdominal pain, tenderness, nausea, vomiting with rising inflammatory markers and a mildly elevated lactate. Given the concerning clinical, biochemical and radiological findings, an exploratory laparotomy was considered necessary due to the possibility of underlying bowel/neobladder ischemia. Reassuringly, the etiology was not intestinal ischemia.

We hypothesize a distended mucous filled neobladder undergoing IDC flushes resulted in high intraluminal pressure. Gas within the neobladder could have entered the mesenteric venous circulation due to mucosal disruption at the anastomosis. Bacterial culture of neobladder fluid demonstrated polymicrobial growth including *Klebsiella pneumoniae*, a gas forming species. As such infection may also have contributed toward gas formation and entrance into the hepatic-portal circulation via the neobladder.

The patient improved on subsequent management of bladder washout, TPN, bowel decompression and broad spectrum intravenous antibiotics.

Conclusion

Although ischemic bowel remains the most prevalent and perilous cause for HPVG, this case highlights a novel non-ischemic cause. The reported mortality rate for HPVG remains between 29 and 39%, however in patients with HPVG and no bowel ischemia this rate is 11.7%.¹ Most non-ischemic causes of HPVG can be managed non-operatively. Prognosis and treatment depend on the diagnosis and treatment of underlying cause.

Conflict of interest

Nothing to report.

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References

1. Abboud B, El Hachem J, Yazbeck T, et al. Hepatic portal venous gas: pathophysiology, etiology, prognosis and treatment. *World J Gastroenterol.* 2009;15:3585–3590.
2. Studer UE, Burkhard FC, Schumacher M, et al. Twenty years experience with an ileal orthotopic low pressure bladder substitute – lessons to be learned. *J Urol.* 2006;176:161–166.
3. Susman N, Senturia HR. Gas embolization of the portal venous system. *Am J Roentgenol Radium Ther Nucl Med.* 1960;83:847–850.
4. Chan SC, Wan YL, Cheung YC, et al. Computed tomography findings in fatal cases of enormous hepatic portal venous gas. *World J Gastroenterol.* 2005;11:2953–2955.
5. Nelson AL, Millington TM, Sahani D, et al. Hepatic portal venous gas: the ABCs of management. *Arch Surg.* 2009;144:575–581. discussion 581.