CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 6 (2015) 118–121

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Small bowel stricture as a late sequela of superior mesenteric vein thrombosis

Panoraia Paraskeva, Jacob A. Akoh*

Department of Surgery, Plymouth Hospitals NHS Trust, Derriford Hospital, Plymouth PL6 8DH, United Kingdom

ARTICLE INFO

Article history:
Received 1 October 2014
Received in revised form
21 November 2014
Accepted 21 November 2014
Available online 2 December 2014

Keywords:
Superior mesenteric vein thrombosis
Anticoagulation therapy
Bowel infarction
Bowel stricture

ABSTRACT

INTRODUCTION: The increasing frequency of use of CT in patients with acute abdomen is likely to improve the diagnosis of rarely occurring conditions/causes such as superior mesenteric vein thrombosis (MVT). Despite its severe consequences, MVT often presents with nonspecific clinical features.
PRESENTATION OF CASE: AD, a 64-year-old man was an emergency admission with vague abdominal discomfort of two weeks duration, acute upper abdominal pain, loose stools, fresh rectal bleeding and vomiting. A contrast enhanced abdominal CT showed thrombosis of the proximal portal vein and the entire length of the superior mesenteric vein (SMV) with small bowel ischaemia extending from the terminal ileum to the mid jejunal loops. Tests for paroxysmal nocturnal haemoglobinuria and Janus kinase 2 mutation yielded negative results. AD was readmitted seven months later with small bowel obstruction requiring segmental small bowel resection with end-to-end anastomosis. Abdominal CT had shown complete resolution of MVT but a small bowel stricture.
DISCUSSION: Thrombosis limited to mesenteric veins results in earlier and more frequent development of infarction compared to portal combined with mesenteric venous thrombosis. Most patients may be successfully treated with anti-coagulation therapy alone. However, surgery may be required to deal with intestinal infarction or late sequela of MVT.
CONCLUSION: This case demonstrates that MVT can be reversed by effective anticoagulation. However, the price paid for a mild to moderate effect on the bowel may be significant stricture later on. Patients escaping early bowel resection due to massive MVT leading to bowel infarction may still require resection later due to stricture.

1. Introduction

Acute mesenteric venous thrombosis (MVT) is an uncommon but established cause of intestinal ischaemia, distinct from mesenteric arterial occlusion. Diagnosis of early MVT is difficult due to lack of characteristic laboratory abnormalities or distinct physical features prior to the development of gut ischemia or autopsy.[1] Early identification of the condition allows for conservative treatment with anticoagulation and improved outcomes.[2] This has led to a change in the approach to the initial management with non-operative therapy being preferred to surgery. However, bowel stricture may develop in rare cases due to ischaemia, and a close follow-up of every patient after treatment for mesenteric vein thrombosis is necessary to ensure early diagnosis of this complication. We report a case of extensive thrombosis of the proximal portal vein and entire superior mesenteric vein successfully treated conservatively, but whom seven months later developed small bowel obstruction due to stricture.

2. Case report

AD, a 64-year-old man was an emergency admission with vague abdominal discomfort of two weeks duration, acute upper abdominal pain, loose stools, fresh rectal bleeding and vomiting. His past medical history included pulmonary embolism 14 months previously for which he had been under anticoagulation therapy with warfarin for six months and hypertension treated with lisinopril and amlodipine. He previously smoked a pipe (one ounce per day) for 12 years and stopped eight years prior to admission. He had no history of obesity.

On admission he exhibited tenderness in the mid-abdominal and right upper quadrant regions. Routine laboratory evaluation revealed leucocytosis [count of 12.7×10^9/L, with predominant

* Corresponding author at: Plymouth Hospitals NHS Trust, Level 04 Derriford Hospital, Plymouth PL6 8DH, United Kingdom. Tel.: +44 1752 432650; fax: +44 1752 517576.
E-mail address: Jacob.akoh@nhs.net (J.A. Akoh).

http://dx.doi.org/10.1016/j.ijscr.2014.11.071

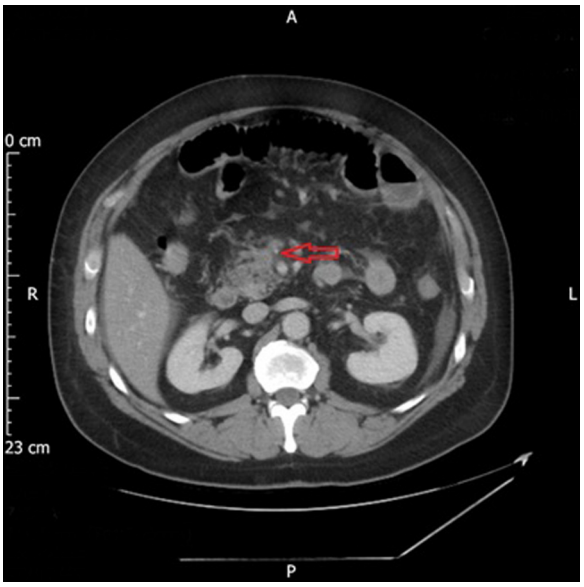

Fig. 1. Contrast enhanced abdominal CT showing superior mesenteric vein occluded by thrombosis (red arrow).

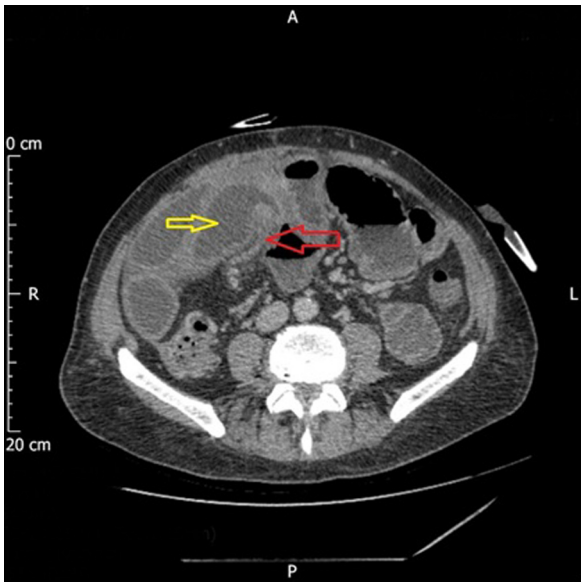

Fig. 2. Transition between dilated small bowel (yellow arrow) and strictured bowel (red arrow).

neutrophilia (neutrophils 11.4)], a raised C-reactive protein (CRP) of 111 (normal: 0.1–5 mg/L) and normal liver function tests. On suspicion of biliary pathology, an abdominal Ultrasound was performed, which revealed significant free fluid within the abdomen and a long segment of thickened small bowel thought to be due to inflammatory bowel disease. A contrast enhanced abdominal computed tomography (CT) showed extensive thrombosis of the proximal portal vein and the entire length of the superior mesenteric vein (SMV) with small bowel ischaemia extending from the terminal ileum to the mid jejunal loops (Fig. 1). An incidental finding of a 6 mm well defined, peripherally based nodule in the right lower pulmonary lobe was noted but this was discounted when a follow-up thorax CT performed three months later without any progression.

AD was started on anticoagulation with high dose of enoxaparin (low molecular weight heparin) 150 mg daily in addition to IV antibiotics (co-trimoxazole and metronidazole). Furthermore, he was commenced on IV fluids; a nasogastric tube was inserted to relieve bowel obstruction, and parenteral nutrition via a central venous catheter. AD responded to these conservative measures and resumed normal bowel movement. The fluorescently labelled inactive toxin aerolysin (FLAER) test for paroxysmal nocturnal haemoglobinuria (PNH) and DNA PCR V617F detection of Janus Kinase 2 (JAK2) mutation yielded negative results. Anticoagulation was changed to warfarin, aiming on an INR of 2.5, and he was discharged home 12 days after admission. AD was seen in the outpatient clinic at three and six months and finally discharged for care.

AD was readmitted as an emergency with a one day cramping abdominal pain, distention and biliary vomiting seven months after his initial presentation. Examination revealed abdominal distention with palpable bowel loops, diffuse abdominal tenderness and normal digital rectal examination. Routine laboratory evaluation revealed leucocytosis (11.8×10^9/L) and a CRP of 10 mg/L. CT imaging of the abdomen revealed features of mechanical small bowel obstruction (Fig. 2), most probably due to a post ischaemic small bowel stricture. The previously noted thrombus in the SMV had resolved. The symptoms resolved with conservative treatment, but he was electively re-admitted one week later for segmental resection of 30 cm of small bowel with end-to-end anastomosis. He did well and was discharged home five days later. Histological evaluation of the specimen revealed ulceration and active chronic inflammation with perforation (histopathological finding only) and surrounding granulation tissue formation without any evidence of neoplasia.

3. Discussion

MVT is a rare condition accounting for between 1 in 15,000 and 1 in 5000 admissions to hospital and 6–9% of all cases of acute mesenteric ischemia.[3,4] The prevalence of MVT has increased over the past two decades with the increased use of contrast-enhanced CT in patients presenting with abdominal pain and those with portal hypertension. CT is generally the imaging modality of choice due to its high sensitivity (90–100%), wide availability and relatively low cost. Ultrasound with Doppler analysis of flow in the mesenteric and portal venous systems has also been used with success, albeit with a lower sensitivity (73–80%) and higher level of technical difficulty.[1]

Different pathophysiologic mechanisms have been postulated for acute thrombus formation, local factors, such as recent splenectomy or pancreatitis associated with initial thrombosis in the large veins; and systemic hypercoagulable factors such as protein C deficiency or myeloproliferative disease that lead to thrombosis beginning in the intramural venules, vasa recta and venous arcades.[5] The main causes of MVT are abdominal malignant neoplasm, surgery, inflammatory conditions, inherited disorders (protein C or S deficiency, factor V Leiden deficiency, antithrombin deficiency), or acquired (hematologic diseases: polycythaemia vera, myelofibrosis, thrombocythaemia, JAK2 gene sequence variation, antiphosholipid antibodies, paroxysmal nocturnal haemoglobinuria; non hematologic diseases: malignancy, oral contraceptive pills, pregnancy, nephrotic syndrome, hyperhomocysteinaemia).[6–8] Detection of the JAK2 sequence variation has replaced bone marrow examination as the first test for screening myeloproliferative diseases because it can differentiate reactive thrombocytosis from essential thrombocythaemia and primary from secondary polycythaemia.[9] With the use of more extensive investigation, the frequency of patients with primary MVT has decreased, varying from 0% to 49% in the literature.[10] Investigations did not yield any underlying aetiology in our case and it can therefore be assumed he had idiopathic or primary MVT.