CASE REPORT

Antiphospholipid syndrome presenting as acute mesenteric venous thrombosis involving a variant inferior mesenteric vein and successful treatment with rivaroxaban

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Accepted 10 March 2018

SUMMARY

Acute mesenteric venous thrombosis (MVT) is the rarest cause of acute mesenteric ischaemia, so thrombosis of a variant inferior mesenteric vein (IMV) is especially uncommon in the setting of antiphospholipid syndrome (APS). Here, we present such a case of seronegative APS initially manifesting as an anomalous IMV thrombosis in a 76-year-old woman. Although guidelines support anticoagulation with vitamin K antagonists in these patients, we anticoagulated with rivaroxaban (a direct oral anticoagulant (DOAC)) due to patient preference, which resulted in complete clinical and endoscopic resolution. IMV thrombosis is a rare form of MVT, only two case reports describe successful anticoagulation with DOACs in the setting of MVT and none report APS as an underlying aetiology. Therefore, this case provides the opportunity to review the pathophysiology of MVT, APS and their medical management including current trends in anticoagulation.

BACKGROUND

Acute mesenteric ischaemia (AMI) is a severe, life-threatening gastrointestinal emergency responsible for 1 in 1000 hospital admissions worldwide.¹ AMI has an estimated mortality rate of 40%.² All AMI is characterised by decreased mesenteric blood supply to the visceral organs of the gastrointestinal tract, but it can also be categorised into four groups based on the underlying mechanism: non-occlusive, arterial thrombosis, arterial embolic and venous thrombosis. Of these categories, venous thrombosis is the least common cause.

Most patients with acute mesenteric venous thrombosis (MVT) have predisposing risk factors such as underlying inflammatory processes, thrombophilia or malignancies.^{1 2} Rarely, autoimmune thrombophilias such as antiphospholipid syndrome (APS), a known cause of recurrent complications in pregnancy and arterial or venous thrombosis, can cause acute MVT. A diagnosis of APS can be made in the presence of typical clinical findings and laboratory evidence of antiphospholipid antibodies (aPL) such as the lupus anticoagulant or IgG or IgM antibodies to cardiolipin or beta2-glycoprotein according to the Sydney APS Classification Criteria (SCC).^{3 4} However, reports exist of 'seronegative' APS (SN-APS) patients with clinically significant

thrombosis in the absence of antibodies satisfying the SCC but with atypical aPL antibodies including isolated IgA antibodies to cardiolipin or beta2-glycoprotein. Therefore, the serological criteria for APS may be too restrictive, and non-criteria-related aPL antibodies should be tested for when there are clinical manifestations of APS and typical antibodies are absent.⁵⁻⁹

Given that APS can be lethal, current guidelines recommend lifelong anticoagulation with vitamin K antagonists (VKAs) such as warfarin to prevent thrombosis and future thrombotic events. Only ten cases of acute MVT caused by APS are described in the literature, all of which involved thrombosis of the superior mesenteric vein (SMV) and which were successfully treated with VKAs.¹⁰⁻¹⁹ There are no reports of APS causing MVT with involvement of a variant inferior mesenteric vein (IMV) or successful treatment with direct oral anticoagulants (DOACs) such as rivaroxaban, a direct inhibitor of activated factor X.

Here, we present a case of SN-APS presenting as acute MVT involving a variant IMV. Furthermore, the APS and consequent MVT were treated and subsequently improved after anticoagulation with rivaroxaban. Since this condition is rare and potentially lethal, this unusual case serves as a reminder of its unique presentation, pathogenesis and management so that front-line practitioners can promptly recognise and appropriately manage these cases.

CASE PRESENTATION

A 76-year-old woman with a medical history of hypertension and osteoarthritis presented with left lower quadrant abdominal pain and haematochezia for 4 days. She described the pain as 5/10 in severity (scale: 1, least painful; 10, most severe pain ever experienced). The pain was more intense on the left side and radiated to the right. She was passing at least three watery bowel movements per day that occasionally appeared bright red in colour without visible clots. There was no history of fever, chills, nausea, vomiting or joint pain, and she had not recently travelled abroad or been exposed to sick contacts.

Physical examination revealed abdominal tenderness in the left lower quadrant without rigidity or guarding. A rectal examination revealed

To cite: Singh K, Khan G. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223077

Figure 1 A non-contrast CT scan showing non-specific inflammatory changes in the distal sigmoid colon manifesting as bowel thickening (arrowhead).

non-prolapsed, thrombosed haemorrhoids and bright red blood in the stool.

INVESTIGATIONS

There was no laboratory evidence of anaemia, only elevated serum lactic acid (3.5 mmol/L; 0.5–2.2 mmol/L) and normal coagulation (activated partial thromboplastin time 28.8 s (20.4–33.4 s) and prothrombin time 12.1 s (9.5–11.8 s)). Stool studies were negative for faecal leukocytes, ova and parasites, and there was no growth of *Clostridium difficile*, *Salmonella* spp., *Shigella* spp. or *Aeromonas* spp. from stool cultures.

A non-contrast CT scan revealed non-specific inflammatory changes in the distal sigmoid colon manifesting as bowel wall thickening (figure 1). Flexible sigmoidoscopy revealed congested mucosa with multiple ulcers and exudates in the rectum and sigmoid colon (figure 2A). Proximally, the descending colon mucosa gradually normalised in appearance with a marginal

Figure 2 (A) Flexible sigmoidoscopy showing congested mucosa with exudates in the sigmoid colon. (B) Colonoscopy revealing disease resolution with a normal mucosa without congestion or ulceration.

Figure 3 H&E stain at ×10 magnification showing acute inflammation with fibrinous thrombi within blood vessels.

zone suspicious for ischaemia. Sigmoid colon biopsies revealed colonic mucosa with ulceration, acute inflammation, foci of mucosal necrosis and fibrinous thrombi without evidence of parasites or viral inclusions (figure 3). Given the high clinical suspicion for AMI, a CT scan of the abdomen with intravenous contrast was performed that revealed significant left colon wall thickening and multiple filling defects in a dilated IMV (figure 4). Furthermore, the IMV abnormally drained into the SMV rather than the splenic vein. A diagnosis of acute MVT was confirmed, and anticoagulation with intravenous unfractionated heparin (UFH) was initiated.

The hypercoagulation workup showed an increased titre of IgA antibodies directed against β 2-glycoprotein (table 1). The autoimmune disease panel was otherwise unremarkable (table 1), so a diagnosis of APS was made.

TREATMENT

On day 9, the patient's abdominal pain and haematochezia had significantly improved, and on day 11, the patient was

Figure 4 CT scan of the abdomen with intravenous contrast showing filling defects in a dilated IMV abnormally draining into the SMV (arrowhead). IMV, inferior mesenteric vein; SMV, superior mesenteric vein.

Table 1 The hypercoagulation workup confirming the diagnosis of antiphospholipid syndrome

Laboratory study	Result	Reference range
Cardiolipin IgG (IgG Phospholipid Units; GPL)	<14 GPL	<14 GPL negative, 15–20 GPL intermediate, 21–80 GPL low to medium positive, >80 GPL high positive
Cardiolipin IgA (IgA Phospholipid Units; APL)	<11 APL	<14 APL negative, 15–20 APL intermediate, 21–80 APL low to medium positive, >80 APL high positive
Cardiolipin IgM (IgM Phospholipid Units; MPL)	<12 MPL	<14 MPL negative, 15–20 MPL intermediate, 21–80 MPL low to medium positive, >80 MPL high positive
Phosphatidyl serine antibody IgG (U/mL)	<10 U/mL	<10 U/mL negative, 10–20 U/mL equivocal, >20 U/mL positive
Phosphatidyl serine antibody IgA (U/mL)	<25 U/mL	<20 U/mL negative, 20–30 U/mL equivocal, >30 U/mL positive
Phosphatidyl serine antibody IgM (U/mL)	<20 U/mL	<25 U/mL negative, 25–35 U/mL equivocal, >35 U/mL positive
β 2-Glycoprotein I IgA (Standard IgA Units; SAU)	124 SAU/mL	$\leq$ 20 SAU/mL negative
β 2-Glycoprotein I IgM (Standard IgM Units; SMU)	<9 SMU/mL	$\leq$ 20 SMU/mL negative
β 2-Glycoprotein I IgG (Standard IgG Units; SGU)	<9 SGU/mL	$\leq$ 20 SGU/mL negative
Antinuclear antibodies	Negative	Negative
Activated partial thromboplastin time (s)	28.8 s	20.4–33.4 s
Prothrombin time (s)	12.1 s	9.5–11.8 s
Protein C activity (%)	59.50%	55%–123% activity
Protein S activity (%)	80.10%	55%–160% activity
Anti thrombin III activity (%)	62.30 %	75%–125 % activity
Factor V Leiden gene mutation	Negative for mutation	
Prothrombin G20210A gene mutation	Negative for mutation	
Homocysteine level	6.8 μ mol/L	5.0–20.0 μ mol/L

discharged with rivaroxaban. The decision to use rivaroxaban over other anticoagulants was made after thorough discussion with the patient, who was against taking warfarin or enoxaparin because she did not want injections or frequent phlebotomy for therapeutic monitoring.

OUTCOME AND FOLLOW-UP

Twelve weeks later, a hypercoagulation panel including aPL antibodies, antithrombin III and protein C and S activity was repeated and the diagnosis of SN-APS was confirmed after IgA antibodies to β 2-glycoprotein I serology remained detectable with a titre of 120 mU ($\leq$ 20 SAU/mL negative). Six months after presentation, the patient was asymptomatic, and a follow-up colonoscopy at that time showed complete resolution with normal colonic mucosa without ischaemic changes (figure 2B).

DISCUSSION

Acute MVT involves the SMV in approximately 95% of cases; the remaining cases involve the IMV.¹ As a result, the most common anatomical sites of involvement are the sites drained by the SMV: the ileum (63%–83%) and the jejunum (50%–81%), although the duodenum (4%–8%) can also be affected. In 14% of cases, the colon, drained by the IMV, is affected.^{20 21} It is not completely understood why thrombosis is less frequent in the IMV; however, the presence of collateral circulation between the internal iliac system, rectal venous plexus or systemic circulation via the left renal vein, splenic vein and hemiazygos veins may account for this difference in incidence.^{22 23} Generally, the IMV drains into the splenic vein before the splenic vein and SMV join to form the portal vein in approximately 40% of patients (figure 5A and B). In approximately 30% of patients, the IMV may drain directly into the portal vein and, even more infrequently (20% of the population and as in our patient), the IMV drains into the SMV instead of the splenic vein.^{24 25} This variant is usually discovered as an incidental finding on imaging and it is unknown whether these variants confer a higher risk of thrombosis due to a lack of evidence.

Clinically, when there is a variant IMV, venous thrombosis can be missed when colour-flow Doppler sonography is used, because the overall length of the confluence of the portal vein with variant venous drainage is generally shorter than the more prevalent variants such as the IMV draining into the splenic vein.^{24 25} Knowledge of IMV variant drainage is also crucial for surgeons so that appropriate anatomical landmarks can be identified when surgery is indicated.

Although tissue ischaemia by MVT represents the endpoint of occlusion by thrombus in the mesenteric circulation, many patients are predisposed to this sequence of events because of one or more risk factors described by Virchow's triad: endothelial injury, venous stasis or a coexisting hypercoagulable state.^{1 2 26 27} In APS, systemically circulating aPL antibodies induce a hypercoagulable state by promoting platelet aggregation and activation, synthesis of proinflammatory cytokines including interleukin 6 and tumour necrosis factor alpha and increased expression of platelet membrane glycoprotein IIB/IIIa and tissue factor by endothelial cells and monocytes.²⁸ During acute MVT, venous thrombosis leads to venous congestion, increased intravascular hydrostatic pressure within the mesenteric circulation and fluid leakage into the surrounding tissue with eventual bowel wall oedema and submucosal haemorrhage.²⁹ Ischaemia starts in the mucosal layer and progresses because of reduced arterial flow either due to arterial vasospasm or hypovolaemia, although these states can coexist.^{26 27 29} When collateral circulations such as the venous arcades or the vasa recta are involved, lethal complications can arise including infarction, necrosis, perforation and sepsis unless intervention is swift.

In most cases of acute MVT, medical management including bowel rest, intravenous fluids, analgesia and prompt anticoagulation are crucial steps to prevent progressive thrombus extension and to promote recanalisation of the affected circulation when the diagnosis is confirmed.^{1 2 26 27 30} When present, gastrointestinal bleeding is not a contraindication for anticoagulation because of the high mortality rate associated with bowel infarction.²⁴ The duration of anticoagulation varies depending on the underlying

Figure 5 (A) Schematic illustrating the most frequent variant of the IMV circulation, in which the IMV joins the splenic vein (left) and the anomalous circulation of the patient in which the IMV joins the SMV. (B) Three-dimensional reconstruction using CT to demonstrate the anomalous IMV draining into the SMV.

conditions associated with MVT such as inflammation, malignancy and thrombophilias such as APS, as in our patient. Local inflammation, for instance, is considered potentially treatable and temporary, so patients are anticoagulated for a minimum of 3 to 6 months.^{1 26 27 29} In contrast, an underlying malignancy, thrombophilia or the rare situation in which no identifiable aetiology is present requires lifelong anticoagulation.²⁷

Anticoagulation with parenteral heparin as a bridge to oral VKAs such as warfarin was historically the only treatment option for patients with acute MVT caused by APS. Low molecular weight heparin (LMWH) has been the preferred parenteral anticoagulant over UFH except in patients with renal failure or when urgent surgery is required, since the half-life of UFH is half that of LMWH and thus the risk of associated bleeding is lower.³¹ VKAs such as warfarin act by depleting the vitamin K supplies required for activation of the coagulation factors II, VII, IX and X and the regulatory proteins C and S. Anticoagulation with VKAs can be erratic, so the therapeutic range is defined as an international normalised ratio of 2.0–3.0 to prevent recurrent thrombosis or bleeding complications in these patients, although

a few cases of recurrent venous thromboembolism associated with APS have been reported in appropriately anticoagulated patients.³² In refractory APS or when there are contraindications to oral anticoagulation, LMWH is an option for long-term anticoagulation.³³

While VKAs have been the drugs of choice for long-term anticoagulation in patients with acute MVT, the use of these agents in other conditions such as deep vein thrombosis, pulmonary embolism and atrial fibrillation has decreased with the introduction of DOACs. DOACs include the direct thrombin inhibitor dabigatran or the direct factor Xa inhibitors rivaroxaban and apixaban. VKAs and DOACs share a similar bleeding risk, but the pharmacokinetics and pharmacodynamics of DOACs are more predictable, they have a quicker onset of action and broader therapeutic range and they do not have significant interactions with food, alcohol and other drugs that could precipitate thrombosis or bleeding.³¹ Thus, DOACs are ideal for patients taking multiple medications with the potential to interact with VKAs or in patients who do not want close therapeutic monitoring with frequent phlebotomy.

However, their safety profiles are not fully established. DOACs are currently being investigated in clinical trials for use in APS and MVT,^{28 33} but there are concerns regarding the safety of these agents in emergent situations including severe coagulation-related bleeding, adverse events or when urgent surgery is needed. Since the specific factor Xa inhibitor antidote andexanet (against rivaroxaban and apixaban) and the monoclonal antibody idarucizumab (directed against dabigatran) are still being assessed in clinical trials, VKAs remain the recommended long-term anticoagulation, and the use of DOACs for these indications would therefore be considered off-label.^{34 35}

To the best of our knowledge, this is the first reported case of thrombosis of a variant IMV as the initial presentation of SN-APS and successful resolution with rivaroxaban treatment. IMV thrombosis has been reported in the setting of diverticulitis, pyelephlebitis, malignancy and thrombophilias.^{3 20 36–44} Our patient's MVT was uncomplicated given that she did not require thrombolysis, fibrinolysis or surgery. Although there are reports of successful anticoagulation with DOACs in the setting of acute MVT, these patients did not have APS; one patient developed MVT due to synthetic testosterone use and the other had diverticulitis.^{43 44} Also, because APS is rare—especially seronegative cases—clinical trials are limited and restricted to patients meeting the APS criteria, and there are reports of failed anticoagulation with DOACs in APS.⁴⁵ Our primary choice of long-term anticoagulation was, therefore, warfarin. However, because our patient did not want frequent injections or therapeutic monitoring, we offered rivaroxaban as an off-label option instead of foregoing anticoagulation altogether.⁴⁶ Fortunately, she improved clinically without evidence of recurrence or thrombosis at other sites.

In summary, the early detection of IMV thrombosis, diagnosis of SN-APS and initiation of anticoagulation in our patient was essential to preventing progression of thrombosis and complications requiring surgery. A high level of clinical suspicion is needed to accurately diagnose acute MVT when patients present with vague abdominal pain and haematochezia, because thrombosis of the mesenteric vessels is rare and potentially lethal if undiagnosed. Prior to discharge, a thorough discussion should be held with the patient to reinforce the importance of anticoagulation, therapeutic monitoring, side effect profiles and drug interactions of these agents. When patients do not want anticoagulation with VKAs, DOACs are likely to become a promising alternative when the current clinical trials report.

Learning points

- ▶ Venous thrombosis is the least common cause of acute mesenteric ischaemia; arterial causes are more common.
- ▶ A high level of clinical suspicion is needed to accurately diagnose acute mesenteric venous thrombosis (MVT) when patients present with vague abdominal pain and haematochezia.
- ▶ The inferior mesenteric vein is infrequently involved in MVT, and it can show anomalies.
- ▶ Antiphospholipid syndrome can predispose patients to venous thromboembolism, including in the mesenteric veins.
- ▶ Vitamin K antagonists remain the recommended choice for long-term anticoagulation, but the direct oral anticoagulants are showing promise and may be suitable alternatives under certain circumstances.

Acknowledgements We would like to thank Dr Ismail Zahir for help obtaining the pathology images and Dr Hasna Ansari for assisting with obtaining the radiographic images.

Contributors KS and GK both were involved in patient care of this patient. KS wrote this manuscript. GK proofread this manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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REFERENCES

- Russell CE, Wadhwa RK, Piazza G, *et al.* Mesenteric venous thrombosis. *Circulation* 2015;131:1599–603.
- Choudhary AM, Grayer D, Nelson A, *et al.* Mesenteric venous thrombosis: a diagnosis not to be missed!. *J Clin Gastroenterol* 2000;31:179–82.
- Gómez-Puerta JA, Cervera R. Diagnosis and classification of the antiphospholipid syndrome. *J Autoimmun* 2014;48–49:20–5.
- Miyakis S, Lockshin MD, Atsumi T, *et al.* International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). *J Thromb Haemost* 2006;4:295–306.
- Hughes GR, Khamashta MA. Seronegative antiphospholipid syndrome. *Ann Rheum Dis* 2003;62:1127.
- Zohoury N, Bertolaccini ML, Rodriguez-Garcia JL, *et al.* Closing the serological gap in the antiphospholipid syndrome: the value of "non-criteria" antiphospholipid antibodies. *J Rheumatol* 2017;44:1597–602.
- Ruiz-García R, Serrano M, Martínez-Flores JA, *et al.* Isolated IgA anti- β 2 glycoprotein I antibodies in patients with clinical criteria for antiphospholipid syndrome. *J Immunol Res* 2014;2014:1–8.
- Tortosa C, Cabrera-Marante O, Serrano M, *et al.* Incidence of thromboembolic events in asymptomatic carriers of IgA anti β 2 glycoprotein-I antibodies. *PLoS One* 2017;12:e0178889–18.
- Murthy V, Willis R, Romay-Penabaz Z, *et al.* Value of isolated IgA anti- β 2-glycoprotein I positivity in the diagnosis of the antiphospholipid syndrome. *Arthritis Rheum* 2013;65:3186–93.
- Higa M, Kojima M, Ohnuma S, *et al.* Portal and mesenteric vein and inferior vena cava thrombosis associated with antiphospholipid syndrome. *Intern Med* 2001;40:1245–9.
- Yong Z, Wang L, Chen J, *et al.* Mesenteric venous thrombosis in a patient with antiphospholipid syndrome and systemic lupus erythematosus. *Asian Biomedicine* 2013;7:711–4.
- Donohue K, Minkove S, Weiler B, *et al.* Catastrophic antiphospholipid antibody syndrome presenting as intraabdominal abscess: an unusual case of thrombotic storm. *Chest* 2016;150:377A.
- Fried M, Van Ganse W, Van Avermaet S. Mesenteric vein thrombosis triggered by blunt abdominal trauma in a patient with the primary antiphospholipid syndrome. *Eur J Gastroenterol Hepatol* 2002;14:697–700.
- Martínez Vázquez C, Rivera A, Albo C, *et al.* [Superior mesenteric vein thrombosis and the primary antiphospholipid syndrome]. *Rev Clin Esp* 1993;192:153–3.
- Egwuonwu S. Acute superior mesenteric vein thrombosis in a patient with primary antiphospholipid syndrome: a case report and brief review of the literature. *J Gastrointest Dig Syst* 2015;05:1–3.
- Wasr WA, Shahrir MSM, Hussein H. Antiphospholipid syndrome presenting as mesenteric vein thrombosis and gangrenous small bowel. *The International Medical Journal of Malaysia* 2011;10:45–8.
- Kato N, Yukioka H, Yoshida G, *et al.* Superior mesenteric vein thrombosis presented transient false positivity for lupus anticoagulant under heparin treatment. *Eur J Emerg Med* 2002;9:67–70.
- Lee HJ, Park JW, Chang JC. Mesenteric and portal venous obstruction associated with primary antiphospholipid antibody syndrome. *J Gastroenterol Hepatol* 1997;12:822–6.
- Vacca A, Garau P, Cauli A, *et al.* Primary antiphospholipid syndrome with mesenteric venous thrombosis presenting with intestinal infarction: a case description. *Lupus* 2007;16:455–6.
- Rhee RY, Glociczki P, Mendonca CT, *et al.* Mesenteric venous thrombosis: still a lethal disease in the 1990s. *J Vasc Surg* 1994;20:688–97.
- Abu-Daff S, Abu-Daff N, Al-Shahed M. Mesenteric venous thrombosis and factors associated with mortality: a statistical analysis with five-year follow-up. *J Gastrointest Surg* 2009;13:1245–50.
- Johnson CC, Baggenstoss AH. Mesenteric vascular occlusion; study of 99 cases of occlusion of veins. *Proc Staff Meet Mayo Clin* 1949;24:628–38.
- Ookura R, Masuko H, Ishizu H, *et al.* Inferior mesenteric venous thrombosis that required operations: report of two cases. *Hepatogastroenterology* 2009;56:687–91.
- Falkowski AL, Cathomas G, Zerz A, *et al.* Pylephlebitis of a variant mesenteric vein complicating sigmoid diverticulitis. *J Radiol Case Rep* 2014;8:37–45.
- Krumm P, Schraml C, Bretschneider C, *et al.* Depiction of variants of the portal confluence venous system using multidetector row CT: analysis of 916 cases. *Rofo* 2011;183:1123–9.
- Harnik IG, Brandt LJ. Mesenteric venous thrombosis. *Vasc Med* 2010;15:407–18.
- Singal AK, Kamath PS, Tefferi A. Mesenteric venous thrombosis. *Mayo Clin Proc* 2013;88:285–94.
- Chighizola CB, Ubiali T, Meroni PL. Treatment of thrombotic antiphospholipid syndrome: the rationale of current management: an insight into future approaches. *J Immunol Res* 2015;2015:1–20.
- McKinsey JF, Gewertz BL. Acute mesenteric ischemia. *Surg Clin North Am* 1997;77:307–18.
- Hmoud B, Singal AK, Kamath PS, *et al.* Mesenteric venous thrombosis. *J Clin Exp Hepatol* 2014;4:257–63.
- Bounameaux H. Unfractionated versus low-molecular-weight heparin in the treatment of venous thromboembolism. *Vasc Med* 1998;3:41–6.
- Sciascia S, Lopez-Pedraza C, Cecchi I, *et al.* Non-vitamin K antagonist oral anticoagulants and antiphospholipid syndrome. *Rheumatology* 2016;55:1726–35.
- Vargas-Hitos JA, Ateka-Barrutia O, Sangle S, *et al.* Efficacy and safety of long-term low molecular weight heparin in patients with antiphospholipid syndrome. *Ann Rheum Dis* 2011;70:1652–4.
- Lu G, DeGuzman FR, Hollenbach SJ, *et al.* A specific antidote for reversal of anticoagulation by direct and indirect inhibitors of coagulation factor Xa. *Nat Med* 2013;19:446–51.
- Schiele F, van Ryn J, Canada K, *et al.* A specific antidote for dabigatran: functional and structural characterization. *Blood* 2013;121:3554–62.
- Yazgan C, Akkas M, Ozmen MM. Inferior mesenteric vein pylephlebitis due to sigmoid diverticulitis. *BMJ Case Rep* 2015;2015:bcr201512599.
- Aggarwal A, Garg S. Image diagnosis: inferior mesenteric vein thrombosis. *Perm J* 2015;19:120–1.
- Vakil A, Guru P, Reddy DR, *et al.* Diffuse cholangiocarcinoma presenting with hepatic failure and extensive portal and mesenteric vein thrombosis. *BMJ Case Rep* 2015;2015:bcr2014209171.
- Otsuka R, Maruyama T, Tanaka H, *et al.* [A case of adenocarcinoma of the sigmoid colon with inferior mesenteric vein thrombosis]. *Gan To Kagaku Ryoho* 2014;41:1663–4.
- Matsumura A, Hatakeyama T, Ogino S, *et al.* [Two cases of colorectal cancer with tumor thrombus in the inferior mesenteric vein]. *Gan To Kagaku Ryoho* 2015;42:2224–6.
- Hwang ET, Kang WS, Park JW, *et al.* [Portal-splenic-mesenteric venous thrombosis in a patient with protein S deficiency due to novel PROS1 gene mutation]. *Korean J Gastroenterol* 2014;64:110–4.
- Cazalas G, Mattei S, Martin B, *et al.* [Inferior mesenteric vein gas and septic thrombophlebitis secondary to sigmoid diverticulitis]. *J Radiol* 2010;91:75–7.
- Parthvi R, Mehra S. Use of rivaroxaban for mesenteric vein thrombosis secondary to diverticulitis: a case report. *Am J Ther* 2017;24:e629–30.
- Katz H, Popov E, Bray N, *et al.* Mesenteric vein thrombosis caused by secondary polycythemia from AndroGel. *BMJ Case Rep* 2014;2014:bcr2014206023.
- Schaefer JK, McBane RD, Black DF, *et al.* Failure of dabigatran and rivaroxaban to prevent thromboembolism in antiphospholipid syndrome: a case series of three patients. *Thromb Haemost* 2014;112:947–50.
- Agno W, Beyer-Westendorf J, García DA, *et al.* Guidance for the management of venous thrombosis in unusual sites. *J Thromb Thrombolysis* 2016;41:129–43.

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