Case Report

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Intestinal obstruction induced by portal vein thrombosis in a female undergoing oral contraceptive therapy: a case report with comprehensive review

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Introduction: Portal vein thrombosis (PVT) is a rare medical condition that obstructs blood flow in the portal vein, with cirrhosis as a common predisposing factor. However, its association with oral contraceptive pills (OCPs), particularly with progestins, remains inadequately explored. This case report aims to contribute to this understanding, focusing on the rare presentation of PVT-induced intestinal obstruction in a female on prolonged OCP therapy.

Case presentation: A 45-year-old female presented with severe abdominal pain, vomiting, and constipation. Diagnosis revealed PVT-induced intestinal obstruction, an exceptionally rare occurrence in the context of prolonged OCP therapy. The patient's symptoms improved with conservative management, including rivaroxaban, highlighting the crucial role of early intervention.

Discussion: This case brings attention to the limited literature exploring the link between OCPs and PVT. Despite the generally safe reputation of OCPs, they can induce pro-thrombotic conditions, emphasizing the need for heightened clinical awareness. In this case, the rarity of intestinal obstruction in PVT, compounded by the absence of common risk factors, underscores the diagnostic challenges associated with such presentations.

Conclusion: PVT-induced intestinal obstruction in a patient on prolonged OCP therapy is exceptionally rare, emphasizing the necessity for multidisciplinary management. It provides crucial insights into suspecting, identifying, and treating this uncommon complication in non-cirrhotic individuals, contributing to the limited existing literature on the subject.

Keywords: intestinal obstruction, oral contraceptive pills, portal vein thrombosis, rivaroxaban, ultrasonography

Introduction

Portal vein thrombosis (PVT) is a medical condition marked by the partial or complete obstruction of blood flow in the portal vein, responsible for supplying 75% of the blood flow to the liver due to the presence of a thrombus[1,2]. While rare in the general population, its prevalence among cirrhotic patients is significantly higher, ranging from 4.4 to 15.8%, contributing to 5–10% of overall cases of portal hypertension in developed countries and up to one-third in developing countries, where a higher occurrence of infectious complications predisposes individuals to PVT[3–5]. The incidence of PVT in patients without cirrhosis remains uncertain. Autopsy studies indicate prevalence

HIGHLIGHTS

- This case explores a rare instance of portal vein thrombosis (PVT) causing intestinal obstruction, underscoring the importance of increased clinical awareness.
- Prolonged usage of oral contraceptives, particularly those containing ethinyl estradiol, is recognized as the primary contributor to PVT, while the influence of progestin compounds remains minimal, highlighting a less common but significant risk associated with contraceptive therapy.
- The effective management of PVT with rivaroxaban suggests its usefulness in treating such cases, prompting a closer look at newer oral anticoagulants.
- The case emphasizes the challenges in diagnosing PVT-induced intestinal obstruction, stressing the need for a comprehensive approach involving imaging modalities like ultrasonography and computed tomography (CT) scans.
- The study urges additional research on PVT prevalence in non-cirrhotic patients, the influence of oral contraceptives on PVT development, and the effectiveness of NOACs, particularly rivaroxaban, in preventing and treating PVT across various causes.

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rates ranging from 6 to 64%, while ultrasound-based diagnoses report prevalence of 5–24%[6]. Notably, a Swedish autopsy on 2400 studies found a 1% prevalence of PVT, with common predisposing factors including cirrhosis, hepatobiliary malignancy (primary or secondary), infectious or inflammatory abdominal diseases, and myeloproliferative disorders. However, in 14% of cases, no identifiable predisposing factors were found[7].

While OCPs are generally considered safe, they can induce side effects, including the development of pro-thrombotic conditions. This phenomenon is linked to the influence of oestrogen, a possible element in combined oral contraceptive pills, which raises fibrinogen levels and certain coagulation factors like II, VII, VIII, and X. The progestin component also contributes to this mechanism, though to a lesser extent which has been reported in the present case[8–10]. However, the connection between oral contraceptive medications and the occurrence of PVT remains an inadequately explored area in the existing literature, with very few reported cases[3,8]. This case report aims to contribute to the understanding of this relationship, specifically focusing on the rare presentation of PVT-induced intestinal obstruction in a female undergoing oral contraceptive therapy.

Intestinal obstruction from mesenteric venous thrombosis is exceptionally rare, with only 12 reported cases to date. PVT seldom involves intestinal obstruction, and our case is exceptionally rare, with no documented case reports to date in the context of prolonged oral contraceptive therapy, particularly with progestin-only pills[11]. However, a study by Gizem *et al.*[12] describes a case of a patient with undiagnosed chronic portal Vein thrombosis presented as intestinal obstruction. Some cases are asymptomatic, while others present as gastrointestinal bleeding or acute abdominal pain. Acute PVT leads to symptoms like abdominal pain, while chronic PVT may be asymptomatic or involve varices. Gastrointestinal bleeding, often linked to oesophageal varices, characterizes chronic cases. Timely diagnosis and management are crucial for such presentations with intestinal obstruction to prevent further complications[2,3].

Here, we present the case of a 45-year-old female with a chief complaint suggestive of bowel obstruction. The diagnosis revealed PVT-induced intestinal obstruction, presenting a rare occurrence in a patient undergoing prolonged oral contraceptive therapy. This case highlights the diagnostic challenges and management associated with identifying PVT-induced intestinal obstruction, particularly in the absence of common risk factors. The report sheds light on the complexities of recognizing and managing such cases, emphasizing the need for heightened clinical awareness and multidisciplinary collaboration. The work has been reported in line with SCARE guidelines 2023[13].

Case presentation

A 45-year-old female presented in an emergency department (ED) with severe diffuse abdominal pain for 10 days. The pain was radiating towards the back and was associated with multiple episodes of yellow-coloured vomiting that used to worsen with fluid and food intake. She also had constipation for 8 days. She had no fever, hematemesis, or hematochezia. For the last 6 months, she has been taking the oral contraceptive norethisterone 30 mg per day to treat heavy menstrual bleeding likely from uterine fibroids diagnosed 2 years ago. She denied smoking and alcohol use. She had no personal or family history of diabetes mellitus, hypertension, ischaemic heart disease, thrombosis, or thrombophilia.

On examination, her vitals were as follows: pulse 60 beats per minute, blood pressure 100/70 mmHg, and respirations 18 per minute. On abdominal examination, the abdomen was distended and diffusely tender with no rigidity or guarding. Bowel sounds were decreased, and viscera were not palpable. Abdominal ultrasound done in the ED showed dilated bowel loops with minimal interloop fluid and sluggish peristalsis. Computed Tomography (CT) of the abdomen and pelvis was done as part of further investigations. Meanwhile, the patient was admitted to a surgical ward and managed conservatively with intravenous analgesic, intravenous fluid, total parenteral nutrition, and gastric decompression via nasogastric tube. The next day, the CT report revealed PVT extending into the superior mesenteric and splenic veins with mild abdominopelvic ascites, mesenteric thickening, and congestion. Fluid-filled small bowel loops were seen in the mid-abdomen and left lumbar region, suggesting intestinal obstruction. Doppler Ultrasound was then done, which showed thrombosed and greater than 98% occlusion of the portal vein with much-reduced velocity, up to 5 cm/sec, and hepatofugal flow (Fig. 1). After 2 days of conservative treatment in the surgical ward, the patient was shifted in a medical ward. Considering liver cirrhosis as the most common cause of PVT, further investigations were done in the medical ward. An upper GI endoscopy was done to screen for variceal bleeding. The result was unremarkable. A triphasic CT abdomen was done to screen for hepatocellular carcinoma, which sometimes gets missed with a normal CT abdomen. But, no focal lesion was seen in the liver. However, by this time, triphasic CT showed a resolution of intestinal obstruction to somewhat with minimal free fluid (Fig. 2).

In the medical ward, she was kept on complete bowel rest with nutrition given parenterally. rivaroxaban 15 mg orally twice a day was started. Meanwhile, samples were collected to test for antiphospholipid syndrome. The test result was negative. JAK2 mutation testing was not done due to no evidence of myeloproliferative disorder on complete blood count (CBC). Finally, it was concluded that prolonged oral contraception was the cause of PVT that led to intestinal obstruction in this patient. Her symptoms gradually improved over 7 days, and she started to pass flatus on the 9th day. Oral feeding was started then, which she was tolerating well. On the 12th day of hospitalization, a repeated Doppler ultrasound showed improved circulation in the portal vein with the thrombus in the resolving phase. Her heavy menstrual bleeding was planned to be addressed with a combination of mefenamic acid and tranexamic acid once the illness recovers, as the complaint was uneventful in the current scenario. The patient was discharged home on oral rivaroxaban 10 mg per day and called for follow-up after 3 weeks. Subsequent follow-ups were uneventful, and the patient was kept on oral rivaroxaban for three months with timely monitoring and evaluations.

Discussion

PVT is defined as the appearance of a thrombus in the portal vein or its branches, with or without an extension to the superior mesenteric vein or the splenic vein[14]. The pathophysiology of portal vein thrombosis involves elements of Virchow's triad,

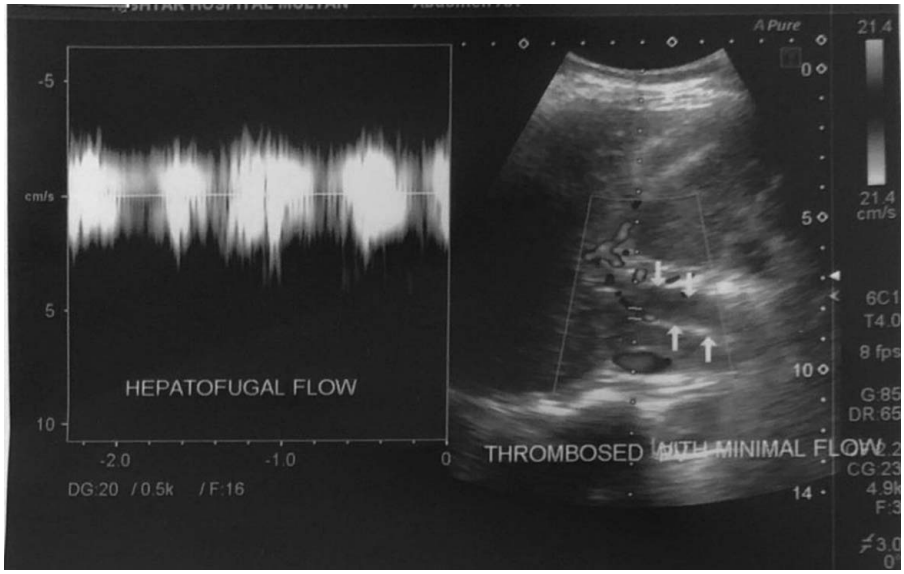

Figure 1. Doppler ultrasound showing thrombosed portal vein with hepatofugal flow.

comprising diminished portal blood flow, a hypercoagulable state, or injury to vascular endothelium[15]. It presents as a rare occurrence with limited general population data. A 2010 multicenter study suggested an incidence of 0.7 per 100 000 and a prevalence of 3.7 per 100 000 in developed countries, notably rising with cirrhosis or pro-thrombotic disorders. Cases involving individuals without cirrhosis are scarce, underscoring the significance of instances like the one presented[1,16].

Non-cirrhotic nontumoral portal vein thrombosis (NCPVT), the second leading cause of global portal hypertension, affects 70% of patients with identified risk factors. Both systemic and local factors contribute, with 12% of cases linked to oral contraceptives, highlighting their role in NCPVT aetiology and making this case noteworthy for reporting[17]. Contraception is prevalent among women, with 65.3% of those aged 15–49 reported to be using it and 14% using Oral Contraceptive Pills (OCPs) as per National Health Statistics Report (NHSR) published by the Center for Disease Control (CDC) in 2017–2019[18]. Despite being generally safe, OCPs are associated with an increased risk of coagulopathies, such as venous thromboembolism, myocardial infarction (MI), and thrombotic stroke. This risk, though more common in women over 35 who smoke, can affect any OCP user without any identifiable risk factors, as in the presented case[8]. Thrombosis risk in COCs primarily stems from the oestrogenic component, which induces a pro-thrombotic state by affecting platelets, clotting factors, and anticoagulant pathways. Although less influential, progestins also contribute to thrombosis, sharing similar mechanisms to oestrogen. Oestrogen also affects the hepatic synthesis and displays divergent inflammation responses. Collectively, these alterations shift the haemostatic balance towards prothrombosis in individuals on hormonal therapy, irrespective of the routes of administration.

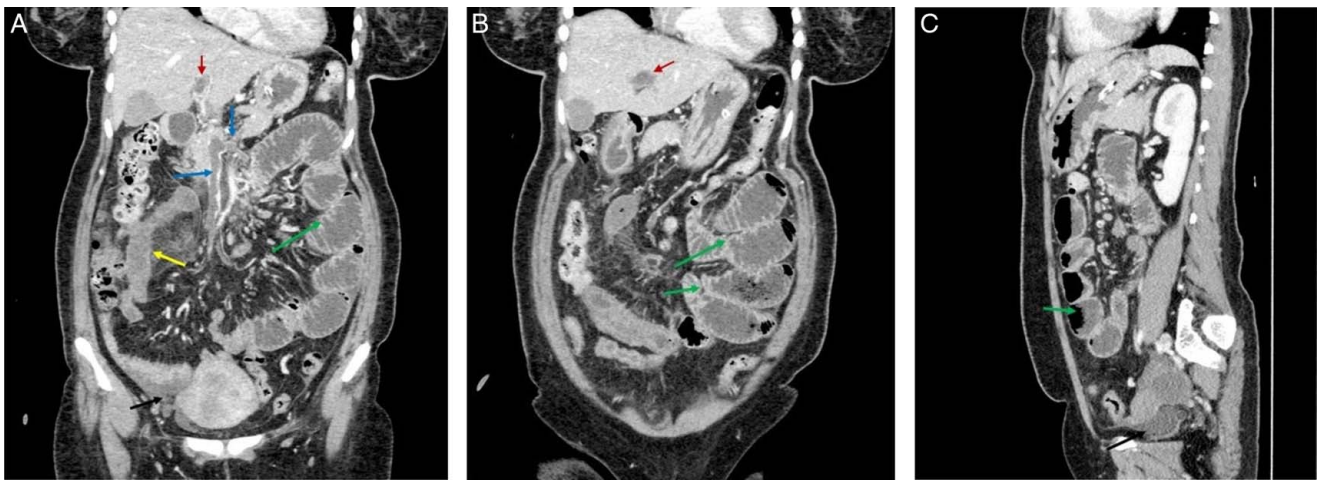

Figure 2. Post-contrast computed tomography images showing portal vein thrombosis (red arrows; (A, B) superior mesenteric vein and splenic vein thrombosis [blue arrows; (A)], dilated small bowel loops [green arrows; (A–C)], inflamed/oedematous gut loop [yellow arrow; (A)] and minimal free fluid [black arrows; (C)].

Progestin-only contraceptives are generally associated with a lower risk of thrombosis compared to combined oestrogen-progestin contraceptives, but they still necessitate careful consideration of individual risk factors and overall safety profiles[7,18,19]. Table 1 shows some of the risk factors contributing to PVT.

A case-control study by Bloemenkamp *et al.*[21] affirmed an elevated risk of venous thrombosis during the early stages of oral contraceptive (OC) use, particularly within the first 6 months and the initial year. Risk was threefold higher in the initial 6 months (95% CI, 0.6–14.8) and twofold higher in the first year (95% CI, 0.6–6.1) compared to prolonged use. Individuals with inherited clotting defects exhibit a higher and earlier onset of venous thrombosis during OC use, suggesting potential indicators of these defects in the early usage period. This underscores the potential long-term role of contraceptives in causing PVT. Combined oral contraceptives (COCs) are generally safe for most women, with exceptions such as age older than or equal to 35 years. Low-dose formulations (10–30 μg of oestrogen) offer reliable contraception and additional benefits like bleeding control and pelvic pain reduction. Despite a slight increase in thromboembolic risk, the overall benefits outweigh this concern, particularly compared to the risks associated with pregnancy or the postpartum period[20]. Additionally, first-generation progestogens like norethisterone pose a lower risk of venous thrombosis compared to third-generation progestogens, although the risk is slightly higher than second-generation progestogens[9]. In the presented case, the patient was under norethisterone 30 mg per day for six months for heavy menstrual bleeding due to uterine fibroids without a coagulation defect history. Table 2 underscores the scarcity of case reports documenting the contribution of contraceptives to PVT. Most patients with PVT had a history of long-term contraceptive use, suggesting an association between prolonged contraceptive therapy and PVT events.

Table 1

Risk factors for PVT[12]

Local risk factors for PVT (70%)	Systemic risk factors for PVT (30%)
Cancer	Inherited
Any abdominal organ	- Factor V Leiden mutation
Focal inflammatory lesions	- Factor II (prothrombin) mutation
Neonatal omphalitis, umbilical vein catheterization	- Protein C deficiency
Diverticulitis, appendicitis	- Protein S deficiency
Pancreatitis	- Antithrombin deficiency
Duodenal ulcer	
Cholecystitis	Acquired
Tuberculous lymphadenitis	- Myeloproliferative disorder
Crohn's disease, ulcerative colitis	- Antiphospholipid syndrome
Cytomegalovirus hepatitis	- Paroxysmal nocturnal hemoglobinuria
Injury to the portal venous system	- Oral contraceptives
Splenectomy	- Pregnancy or puerperium
Colectomy, gastrectomy	- Hyperhomocysteinemia
Cholecystectomy	- Malignancy
Liver transplantation	
Abdominal trauma	
Surgical portosystemic shunting, TIPS	
Iatrogenic (fine needle aspiration of abdominal masses, etc.)	
Cirrhosis	
Preserved liver function with precipitating factors (splenectomy, surgical portosystemic shunting, TIPS dysfunction, thrombophilia)	
Advanced disease in the absence of obvious precipitating factors	

PVT, portal vein thrombosis.

PVT classification considers development time, localization, pathophysiology, and progression factors. It can be acute or chronic, extra- or intrahepatic, occlusive or non-occlusive, and progressive or self-resolving[2]. Clinical presentation and associated complications depend on thrombosis onset and extension. Patients may present with various clinical manifestations ranging from vague abdominal pain to sepsis due to ischaemic necrosis-associated perforation[22]. Acute PVT exhibits symptoms like nausea, vomiting, abdominal pain, diarrhoea, and sepsis, while chronic PVT may be asymptomatic or present with varices and ascites. Gastrointestinal bleeding is common in chronic cases[3]. In the presented case, diffuse abdominal pain, vomiting, and constipation were the acute symptoms prompting a diagnostic workup, which subsequently revealed the occurrence of intestinal obstruction. Notably, it is crucial to highlight that there is no prior published literature specifically addressing intestinal obstruction in PVT induced by contraceptives. This case report stands as the first documented instance of such a presentation. Notably, intestinal obstructions are generally rare manifestations of PVT, making this case particularly unique in its clinical presentation and emphasizing the importance of recognizing and documenting uncommon complications associated with PVT[12].

Complications of PVT include intestinal infarction with symptoms such as persistent pain, hematochezia, guarding, and multiorgan failure. However, the pain subsided with subsequent treatment, with no other significant findings related to complications in the presented case. Imaging studies, particularly CT scans, reveal crucial findings in cases of intestinal infarction[23]. Long-standing PVT can lead to complications related to portal hypertension, such as the development of portal cavernomas and portosystemic shunting, increasing the risk of bleeding from varices, especially in cirrhotic patients. Meanwhile, the upper GI endoscopy was unremarkable for any variceal bleeding in the presented case. Bleeding due to PVT is more common in cirrhotic individuals, and the presence of PVT exacerbates the bleeding risk in those with cirrhotic portal hypertension. Early diagnosis and management are essential to prevent severe complications associated with PVT[3].

In this case presentation, Clinical symptoms, including abdominal pain, recurrent vomiting, constipation, and a distended, diffusely tender abdomen with decreased bowel sounds, triggered a diagnostic evaluation for acute intestinal obstruction. The absence of guarding and rigidity indicated the lack of intra-abdominal infection, intestinal infarction, and perforation—potential complications of PVT. In instances where PVT progresses to bowel ischaemia or multiorgan failure, the associated in-hospital mortality rate ranges from ~ 20–50%[3]. Subsequent ultrasound (USG) findings indicated features consistent with intestinal obstruction, managed conservatively in the surgical ward. The CT report unveiled portal vein thrombosis extending into the superior mesenteric and splenic veins, accompanied by mild abdominopelvic ascites. Notably, fluid-filled small bowel loops were observed in the mid-abdomen and left lumbar region, suggestive of intestinal obstruction. A Doppler ultrasound further

Table 2

Comparative analysis of the presented case with the relevant case studies

Aspects	Provided case	Katelynn *et al.*[10]	John *et al.*[29]	Tai-Lin *et al.*[30]	Jin-Wei *et al.*[31]	Gizem *et al.*[12]
Age	45 years	26 years	23 years	28 years	28 years	59 years/male
Presenting complaints	Severe abdominal pain, radiating to the back, yellow-coloured vomiting, constipation	Abdominal pain, nausea, back pain, headache, upper extremity numbness	Bloody diarrhoea, multiple syncopal events	Acute-onset upper abdominal pain	Continuous abdominal pain, distension, nausea, vomiting	Generalized abdominal pain, constipation, nausea, vomiting (1 day)
Medical history	Oral contraceptive (norethisterone) for last 6 months for heavy menstrual bleeding due to uterine fibroids.	Etonogestrel/ethinyl estradiol intravaginal ring	Oestrogen-containing contraceptive pill, regular tobacco use	Oral contraceptive use for several weeks	Oral contraceptives (ethinyl estradiol 0.03 mg, drospirenone 3 mg/d) for 13 months	No comorbid diseases, no history of previous surgery
Examination findings	Distended abdomen, diffuse tenderness, decreased bowel sounds	Moderate tenderness to palpation of the right upper quadrant and epigastric area	Acute intestinal ischaemia, syncopal events	Uncinate process pancreatic haematoma, superior mesenteric vein (SMV) and portal vein thrombosis	Tenderness in the epigastrium and periumbilical region	Tenderness in all quadrants, no defense or rebound, normal rectal examination
Imaging	Abdominal ultrasound (dilated bowel loops), CT abdomen (PVT, ascites, mesenteric thickening)	CT abdomen/pelvis with IV contrast (thrombus in superior mesenteric vein, main portal vein, intrahepatic portal veins, and distal splenic vein)	MR cholangiopancreatography (portal vein thrombosis), colonic biopsy (acute intestinal ischaemia)	CT revealed pancreatic haematoma, SMV, and portal vein thrombosis	CECT- revealed thrombus in SMV, right branch of PV, main vessels of PV and SV	Increased intestinal segment calibrations, air-fluid levels, thinned intestinal bowel walls on CT scans
Doppler ultrasound findings	Thrombosed and > 98% occlusion of portal vein, hepatofugal flow	Not specified	Not specified	Not specified	Not specified	Chronic portal vein thrombosis with no sign of mechanical obstruction
Management	Intravenous analgesics, fluids, TPN, gastric decompression	IV heparin 5800 units bolus, continuous IV heparin infusion of 18 units per kilogram per hour, later transitioned to rivaroxaban	Conservative management	Not specified	Gastrointestinal decompression; total parenteral nutrition; antibiotics; LMWH; laparotomy; bowel resection	Hospitalization, nasogastric tube, intravenous hydration, low weight molecular heparin for DVT prophylaxis
Anticoagulant therapy	rivaroxaban 15 mg orally twice a day	rivaroxaban 15 milligrams twice daily	Anticoagulant medication	Anticoagulant medication	LMWH sodium 5000 U (100 U/kg), then rivaroxaban	Anticoagulant medication
Additional risk factors	Not significant	Not significant	Regular tobacco use	Not significant	Not significant	
Outcome	Symptoms improved over 7 days, discharged on oral rivaroxaban, follow-up after 3 weeks	Discharged on hospital day 3 on rivaroxaban with haematology follow-up, discontinuation of hormonal contraception	Not specified	Discharged in stable condition, follow-up CT showed resolution of haematoma and partial resolution of thrombosis	Gradual improvement, complete resolution of PV and SV thrombi, collateral circulation well developed	Day 3: Regression of air-fluid levels, initiation of oral intake with liquids Day 5: Discharged with anticoagulant treatment

CT, computed tomography; LMWH, low molecular weight heparin; PVT, portal vein thrombosis, TPN, Total Parenteral Nutrition; DVT, Deep Vein Thrombosis.

revealed thrombosed and greater than 98% occlusion of the portal vein, characterized by a significantly reduced velocity (up to 5 cm/sec) and hepatofugal flow. PVT in our patient was detected promptly, and CT imaging confirmed the absence of intestinal infarction, suggesting a favourable overall prognosis. In individuals with PVT, liver function is usually preserved. Unless there is an underlying liver condition, routine laboratory tests tend to show normal or near-normal results. However, there might be a moderate decrease in prothrombin and other coagulation factors, while D-dimer levels are commonly elevated[3]. However, the patient had normal laboratory findings except for decreased haemoglobin levels due to heavy menstrual bleeding and increased blood urea nitrogen due to multiple episodes of vomiting. Comprehensive laboratory investigations ruled out common causes of portal vein thrombosis, including cirrhosis, antiphospholipid syndrome, and myeloproliferative disorders, among others. The patient's history of OCP use for the past 6 months emerged as the primary factor contributing to the intestinal obstruction, with no other identifiable possible risk factors.

According to the recommendations of the American Association for the Study of Liver Diseases (AASLD), acute PVT should be diagnosed in patients with abdominal pain lasting more than 24 h, regardless of the presence of fever or ileus. For prompt confirmation, a CT scan with a vascular contrast agent is recommended, and if not rapidly available, Doppler-sonography is a suitable alternative. For chronic PVT, consider it in those newly diagnosed with portal hypertension, diagnosed with Doppler-sonography followed by CT or MRI. In cases of acute PVT with high fever, the possibility of septic pylephlebitis should be considered, and blood cultures should be routinely obtained, which was not required in the above case. Diagnosis is based on the absence of a visible normal portal vein, and in acute PVT, vigilance for intestinal infarction is crucial, with features like ascites or thinning of the intestinal wall indicating potential surgical exploration[24]. USG is the preferred investigation, offering sensitivity and specificity of 60%-100%. Doppler imaging confirms absent flow or cavernomatous transformation. CT and MRI provide detailed information on thrombus extension, bowel impairment, and adjacent organs[3]. Diagnosing PVT-induced intestinal obstruction in the context of prolonged oral contraceptive use is challenging due to nonspecific symptoms, atypical presentations, and limitations in imaging modalities like ultrasonography. The condition's rarity and the potential for overlap with other abdominal issues further contribute to delays in diagnosis, emphasizing the importance of heightened clinical awareness and a comprehensive diagnostic approach[8,9].

The patient underwent conservative management, including intravenous analgesics, fluids, gastric decompression and nutritional support. OCP was stopped due to the risk of thrombosis, and rivaroxaban was initiated, and further investigations ruled out common causes. Doppler ultrasound revealed the resolving thrombus and improved circulation. The symptoms improved over seven days, and the patient was discharged on oral rivaroxaban with a follow-up scheduled after three weeks. Her heavy menstrual bleeding was planned to be addressed with a combination of mefenamic acid and tranexamic acid once the illness recovers.

The implementation of targeted therapeutic interventions is essential for addressing portal vein obstruction and preventing severe complications. PVT management involves correcting causal factors, preventing thrombosis extension, and achieving portal vein patency. Anticoagulant therapy is key in acute PVT, showing a 50% recanalization rate after six months. Resistance occurs in 10%, but benefits are seen in intestinal infarction. Early initiation within the first week yields a 69% recanalization rate[3,8,22,25]. In chronic PVT, anticoagulants, administered to 30%, prevent thrombotic events with low mortality. Variceal eradication before anticoagulation is considered. Thrombolytic therapy and surgical interventions are considered in select cases when anticoagulation fails[1,2,9].

Newer oral anticoagulants (NOACs)like rivaroxaban are cost-effective, convenient alternatives for thromboembolic events, eliminating daily injections and offering predictable pharmacokinetics. Effective in preventing and treating PVT, they show low recurrence rates, especially in cirrhosis and malignancy cases. Studies reveal the efficacy of rivaroxaban in preventing thrombus progression and promoting recanalization. Rivaroxaban works by inhibiting Factor Xa with fast action, rare drug interactions, easy administration and reliable clearance upon discontinuation. NOACs enhance patient compliance compared to traditional anticoagulants like low molecular weight heparin (LMWH), warfarin. Long-term rivaroxaban therapy may lead to increased bleeding risk, gastrointestinal symptoms, transient liver enzyme elevation, and, rarely, severe bleeding events. Monitoring and prompt reporting of symptoms are essential for safe use[26,27]. Rivaroxaban, specifically, demonstrates efficacy in PVT associated with long-term oral contraceptive use. AASLD recommends three months of rivaroxaban for PVT, regardless of symptoms, and long-term use for permanent risk factors. American College of Clinical Pharmacy (ACCP) suggests three months for symptomatic patients, none for asymptomatic, and indefinite for those with permanent risk factors[26].

The management goals in PVT, particularly concerning bowel obstruction, are focused on preventing bowel infarction, perforation peritonitis, and disease recurrence. Early initiation of anticoagulation therapy is crucial, especially in the acute phase. In cases of acute PVT, emergency surgery is indicated if there is evidence of bowel gangrene and perforation peritonitis. Surgery is considered for patients experiencing intestinal stricture and obstruction in the chronic phase. The timing of presentation is critical, emphasizing the need for prompt intervention to avoid severe complications as supported by the favourable outcome in the above case[11,28].

This case report is constrained by several limitations. Firstly, its reliance on a single case restricts the generalizability of findings. Moreover, the subjective interpretation of gastrointestinal symptoms introduces potential bias in both diagnosis and management. Additionally, the absence of a control group impedes the establishment of a direct link between oral contraceptive use and PVT-induced intestinal obstruction. Our findings emphasize the importance of implementing standardized assessment protocols and seeking multidisciplinary consultations to mitigate such biases. Future research endeavours should prioritize the integration of objective measures to ensure more reliable clinical evaluations. Furthermore, while this report focuses on rivaroxaban, it may not fully encompass the diverse landscape of anticoagulant treatments. Thus, further research is warranted to gain comprehensive insights into this aspect.

Further research should explore the prevalence of PVT in non-cirrhotic patients and investigate the impact of oral contraceptives on PVT development, identifying potential risk factors.

Studying the effectiveness of NOACs, especially rivaroxaban, in preventing and treating PVT induced by various causes, including oral contraceptives, is crucial. Larger-scale studies are needed to establish guidelines for diagnosing and managing PVT-induced intestinal obstruction, considering its uncommon presentation and potential complications. Through prospective cohort studies, randomized controlled trials, and systematic reviews, we can enhance our understanding of this complex medical intersection and improve patient outcomes through evidence-based interventions.

Conclusion

In this case report, we explore the rare occurrence of PVT-induced intestinal obstruction in a female on prolonged oral contraceptive therapy without any identifiable predisposing or precipitating factors. PVT, often linked to common causes like cirrhosis, takes an unexpected turn in this case, revealing complexities in diagnosis and management. Despite the rarity of such incidents, the report sheds light on the potential association between oral contraceptives and PVT, raising increased clinical awareness. The patient's favourable outcome with rivaroxaban prompts a closer look at NOACs in PVT treatment. However, limitations, such as a single-case focus and lack of a control group, highlight the need for broader research in this intriguing medical intersection.

Ethical approval

The study is exempt/waived from ethical approval in our institution as it poses minimal risk to the patient and the study is for educational purpose/activities.

Consent

Written informed consent was obtained from the patient for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

M.W.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript. T.N.Y.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript. A.B.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript. M.H.: follow-up the patient , final approval of the manuscript. A.A.R.: follow-up the patient , final approval of the manuscript. W.A.: follow-up the patient , final approval of the manuscript. A.K.Y.: final approval of the manuscript. O.K.R.: final approval of the manuscript. P.P.: final approval of the manuscript.

Conflicts of interest disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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