

Pylephlebitis After Colonic Polypectomy Causing Fever and Abdominal Pain

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Abstract

Pylephlebitis is a rare condition with a high mortality risk if not recognized and treated early. The most common symptoms include fever and abdominal pain, with the majority of cases manifesting with a polymicrobial bacteremia. We report an elderly woman with pylephlebitis presenting with fever, abdominal pain, diarrhea, and vomiting, likely secondary to a polypectomy 6 weeks prior. Abdominal CT revealed portal vein thrombus and blood cultures grew *Streptococcus milleri* and *Haemophilus parainfluenza* type V. Pylephlebitis should be considered when symptoms and signs of infection develop following endoscopic procedures, particularly in patients with an underlying hypercoagulable disease.

Introduction

Pylephlebitis, a septic thrombus of the portal venous system, is a rare complication of intra-abdominal sepsis.¹ Appendicitis, diverticulitis, and cholecystitis are the most common inciting events.² It is believed that veins draining an area of infected abdominal viscera develop thrombophlebitis, after which there may be extension of the thrombophlebitis into the main portal venous system, resulting in an infected thrombus.

The diagnosis of pylephlebitis requires a thorough history and physical and a combination of laboratory and imaging investigations. Abdominal pain and fever are the most common findings at presentation.³ Of patients with pylephlebitis, 88% have bacteremia; of these, most have polymicrobial cultures, and *Bacteroides fragilis* and *Escherichia coli* are the most commonly isolated species.⁴ Computed tomography (CT) and ultrasound are most frequently used to demonstrate a thrombus in the portal vein.⁵ Pylephlebitis has also been associated with inflammatory bowel disease and pancreatitis, and should be considered in septic patients following any procedure that has the potential to increase exposure of the vascular system to bacteria.⁵ While pylephlebitis is rare, it occurs more often in individuals with hypercoagulable states such as malignancy and inflammatory conditions.²

Case Report

A 78-year-old woman presented with a 5-day history of diffuse abdominal pain with intermittent diarrhea and vomiting. On the morning of admission, she reported fever and weakness. The patient's medical history was significant for hypertension, colonic adenomas, and IgM monoclonal gammopathy of unknown significance

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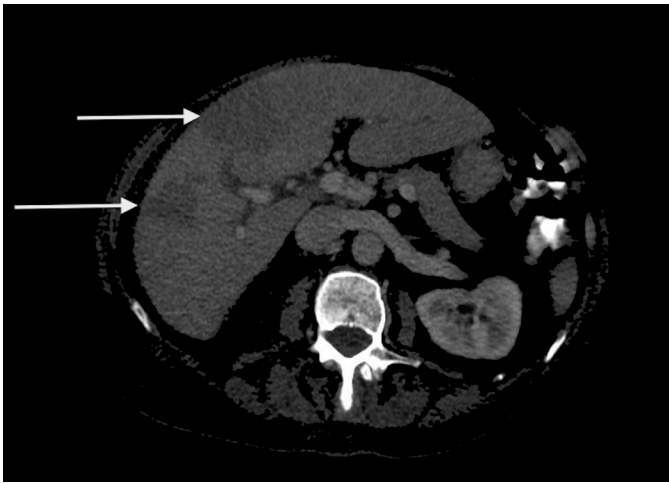


Figure 1. Abdominal CT showing hypoattenuating liver lesions.

(MGUS). Surgical history included bilateral hip arthroplasty secondary to osteoarthritis, L4-L5 spinal fusion, and bilateral cataract extraction. A snare polypectomy was performed for a large sessile polyp during colonoscopy 8 months earlier. A follow-up colonoscopy 3 months later revealed a residual polyp; 6 weeks later, colonoscopy with polypectomy was performed to remove residual polypoid tissue. During that third colonoscopy, submucosal fibrosis was seen and attributed to the first attempt to resect the polyp. The residual polyp measured 15 x 20 mm and was removed using a combination of hot avulsion and endoscopic mucosal resection. Pathology demonstrated a tubular adenoma.

On presentation, the patient appeared unwell with temperature 39.7°C, blood pressure 129/65, heart rate 109 bpm, and oxygen saturation 90% while breathing room air with a respiratory rate of 18. Physical examination revealed mild tenderness on deep palpation of all abdominal quadrants with no costovertebral angle tenderness. Serum lactate was elevated at 2.5 mmol/L, otherwise admission laboratory tests were normal (Table 1). Despite fluid resuscitation, on the second day of admission, the patient was tachycardic, hypotensive, and continued to experience diarrhea and rigors. Blood cultures from admission lab tests grew *Streptococcus milleri* and *Haemophilus parainfluenza* type V, and urine culture was positive for *Escherichia coli*. Her lactate continued to rise (5.2 mmol/L), and she developed a leukocytosis (12.34×10^9 cells/L). She was started on intravenous piperacillin-tazobactam. Transthoracic echocardiogram did not reveal any valvular lesion. Abdominal contrast CT revealed nonspecific hypoattenuating liver lesions (Figure 1), a non-occlusive thrombosis of the superior mesenteric and portal veins, and multiple gallstones with pericholecystic fluid without duct dilation (Figure 2). An abdominal ultrasound confirmed the presence of a thrombus in the ascending branch of the right portal vein. Following the diagnosis of portal vein



Figure 2. Abdominal CT showing a non-occlusive clot in the superior mesenteric vein (black arrow) with hypoattenuating perfusion abnormalities of the liver parenchyma.

thrombus, intravenous unfractionated heparin was started. The diarrhea resolved on day 5 of admission. The patient improved 1 week after starting piperacillin-tazobactam, and switched to oral amoxicillin-clavulanate for a total antibiotic course of 2 weeks. Imaging with abdominal magnetic resonance imaging and ultrasound showed persistent portal venous thrombus, so anticoagulation was continued for an additional 3 months, with total duration of anticoagulation to be determined at further follow-up.

Discussion

The diagnosis of pylephlebitis can be difficult and delayed when symptoms are not severe. While its course can be acute, multiple case reports indicate that symptoms can be mild and often do not initially warrant intensive investigations.^{1,6} Pylephlebitis should be considered in the differ-

Table 1. Trend of Laboratory Values

Laboratory Parameter	Admission	Day 1	Normal Range
Sodium, mmol/L	130	135	135–145
Potassium, mmol/L	3.1	3.3	3.5–5.0
Chloride, mmol/L	93	106	96–106
Urea nitrogen, mmol/L	6.0	N/A	3.0–7.0
Creatinine, μ mol/L	90	69	42–102
Glucose, mmol/L	8.5	N/A	4.0–7.8
Calcium, mmol/L	2.28	1.89	2.10–2.60
Albumin, g/L	42	32	35–50
Hemoglobin, g/L	138	116	115–155
White blood cells X 10 ⁹ /L	2.14	12.34	4.0–11.0
Platelets X 10 ⁹ /L	144	130	140–400
AST, U/L	46	134	7–40
ALT, U/L	24	65	10–45
ALP, U/L	77	70	35–125
Total bilirubin, μ mol/L	14	9	0–23
Venous lactate, mmol/L	2.5	5.2	0.5–2.3
PT, s	10.9	11.0	10.0–13.0
INR	1.03	1.04	0.90–1.20
aPTT, s	29.1	35.3	24.0–37.0
Troponin, μ g/L	2.48	N/A	<0.040

ALP=alkaline phosphatase; ALT=alanine transaminase; aPTT=activated partial thromboplastin time; AST=aspartate transaminase; INR=international normalized ratio; PT=prothrombin time.

ential diagnosis of elusive abdominal pain and pyrexia.² In our case, the cause for abdominal pain and pyrexia was not clear on presentation, and abdominal imaging was not initially performed. The first clue to suggest pylephlebitis was a polymicrobial bacteremia. We believe that the likely source in our patient was a colonoscopy with difficult polypectomy 6 weeks before presentation.

Routine colonoscopy with polypectomy is a low risk for bacteremia, with reported rates ranging from 1-4%. A prospective study found no increased risk of bacteremia when comparing colonoscopy with polypectomy to surveillance colonoscopy.⁷ However, higher risk procedures such as endoscopic retrograde cholangiopancreatography are more likely to cause transient bacteremia, with reported rates as high as 20%.⁷ The American Society of Gastrointestinal Endoscopy guidelines suggest that immunocompromised patients with neutropenia and those with hematological malignancy should receive antibiotic prophylaxis prior to endoscopic procedures.⁷ A previous case report discussed *Streptococcus milleri* bacteremia with pylephlebitis developing a few hours following endoscopic removal of a fishbone.⁸ Another report highlighted a pyogenic liver abscess and bacteremia 1 week after snare polypectomy of a 2 x 3-cm malignant polyp.⁹ In contrast, our patient presented many weeks after

her procedure. Thus, we believe that it is important to consider endoscopic procedures as a potential cause of sepsis, even weeks following the event.¹⁰

A complete past medical history is important to identify patients at risk for venous thromboembolism. In a review of case series, 40% of pylephlebitis patients had a concomitant hypercoagulable condition.¹¹ Our patient had a history of MGUS, which more than doubles the risk for venous thrombosis (95% confidence interval: 1.7–2.5) within 5 years of diagnosis.¹² In our patient, it is possible that the hypercoagulable state contributed to the slow development to the infected thrombus following the polypectomy.

Because no definitive evidence exists to support the routine use of anticoagulation in pylephlebitis, anticoagulation decisions should be made on a case-by-case basis. Patients with an incompletely resolved thrombus do not seem to have negative long-term outcomes.² If imaging performed after anticoagulation demonstrates collateralization, discontinuation of anticoagulation can be considered after 6 months.

In summary, our patient likely had an uncommon and delayed complication of polypectomy. The differential diagnosis of unexplained fever and gastrointestinal symptoms after endoscopic procedures should include pylephlebitis, particularly in patients with risk factors for thromboembolic disease.

Disclosures

Author contributions: ZR Gallinger wrote the manuscript, collected the data, searched the literature, and is the article guarantor. G. May and P. Kortan critically revised the manuscript. AM Bayoumi wrote the manuscript.

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Informed consent was obtained for this case report.

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