Case Report

The Clot That Spoke Too Late: Chronic Pulmonary Thromboembolism Revealing Hidden Gastric Carcinoma

Vinothini S, Rajesh R, Mohammed Thaha S, Malarvizhi P, Anusha Devi T, Manikandan K*

Pulmonary thromboembolism is caused by obstruction of the pulmonary arterial circulation by thrombi, most often originating from deep veins of the lower limbs. Malignancy is a well-known risk factor for thromboembolism because it creates a procoagulant state. In this case, we report a 57-year-old male who presented with breathlessness, chest pain, and bilateral lower limb swelling. He had a prior history of pulmonary thromboembolism with no identifiable provoking factors and a negative thrombophilia workup, including normal homocysteine levels. Clinical examination showed tachycardia, elevated jugular venous pressure, and signs suggestive of right heart failure. Laboratory investigations revealed severe anemia with iron deficiency and positive stool occult blood. Imaging showed chronic pulmonary thromboembolic disease with pulmonary hypertension and right ventricular dysfunction. Further evaluation with abdominal imaging and endoscopy identified a gastric lesion, and biopsy confirmed moderately differentiated gastric adenocarcinoma with liver metastasis. The patient was managed with supportive care, anticoagulation, and chemotherapy. This case shows that unprovoked pulmonary thromboembolism can be an early presentation of an underlying malignancy and highlights the importance of systematic evaluation in such patients.

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Introduction

Pulmonary thromboembolism (PTE) is a life-threatening condition caused by obstruction of the pulmonary arterial system by thrombotic material, most commonly originating from deep vein thrombosis of the lower limbs. It leads to increased pulmonary vascular resistance, ventilation-perfusion mismatch, hypoxemia, and right ventricular hypertrophy. The clinical presentation may range from asymptomatic disease to severe respiratory compromise and hemodynamic instability. In some patients, persistent thrombotic obstruction can progress to chronic thromboembolic pulmonary hypertension, resulting in progressive cardiopulmonary dysfunction.[1] Among the important, acquired risk factors for thromboembolism, malignancy plays a major role. Cancer is a leading cause of morbidity and mortality worldwide and is characterized by uncontrolled cellular proliferation with the potential for invasion and metastasis. Malignancy is associated with a hypercoagulable state due to tumor-related factors, inflammatory mediators, endothelial dysfunction, and activation of the coagulation cascade. This prothrombotic tendency significantly increases the risk of venous thromboembolism, including pulmonary embolism, making thromboembolic events an important complication in cancer patients.[2]

Gastric cancer is one of the most common gastrointestinal malignancies and often presents late because of nonspecific symptoms. Patients may present with weight loss, anemia, occult gastrointestinal bleeding, or metastatic disease involving distant organs such as the liver. In addition to these manifestations, gastric malignancy is also recognized as an important cause of cancer-associated thrombosis and may predispose patients to thromboembolic events due to the associated hypercoagulable state.[3] In some cases, thromboembolism may even be the initial clinical presentation leading to the diagnosis of an underlying gastric carcinoma. The association between unprovoked thromboembolism and occult malignancy has been well documented, with a proportion of patients later found to have hidden cancers.[4] In our patient, there were no identifiable provoking factors for pulmonary thromboembolism at the initial presentation. Evaluation

Department of Internal Medicine, Madras Medical College, Chennai, Tamil Nadu, India.

Correspondence to: Vinothini S, Department of Internal Medicine, Madras Medical College, Chennai, Tamil Nadu, India. E-mail: vinothinisasi312@gmail.com

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for thrombophilia showed negative antinuclear antibody and antiphospholipid antibody profile, with normal serum homocysteine levels, making the thromboembolic event apparently unprovoked. This highlights the importance of considering an underlying occult malignancy in patients presenting with unexplained thromboembolism.

In this report, we describe a 57-year-old male who presented with complaints of breathlessness and chest pain for 6 months and swelling of both lower limbs for 5 days, in whom further evaluation revealed chronic pulmonary thromboembolism with an underlying gastric carcinoma with metastatic involvement, emphasizing the need for malignancy screening in such clinical scenarios

Case Presentation

A 57-year-old male presented with complaints of breathlessness and chest pain for 6 months and swelling of both lower limbs for 5 days. Breathlessness was insidious in onset and progressive in nature. It was aggravated on exertion and relieved with rest. It was associated with chest pain and palpitations. Chest pain was insidious in onset, progressive, dull aching in type, non radiating, and localized over the left chest. It was aggravated on exertion and relieved with rest. Palpitations were present during exertion, lasted for 5 to 10 minutes, and were described as fast and regular in nature. Swelling of both lower limbs was of acute onset and progressive. It was aggravated on prolonged standing and relieved on lying down. There was no history of orthopnea or paroxysmal nocturnal dyspnea, suggesting absence of left-sided cardiac failure. There was no history of cough, fever, or hemoptysis, indicating no infective or primary respiratory pathology. There was no history of abdominal distension, facial puffiness, or decreased urine output, suggesting no renal or hepatic cause for the edema.

Table 1: Baseline vital signs

Parameter	*Finding*
Blood pressure	100/70 mmHg
Pulse rate	107 per minute, regular rhythm, normal volume, no specific character, no radio radial or radio femoral delay
Respiratory rate	23 per minute, abdomino thoracic type
Jugular venous pressure	Elevated, 10 cm H_2O
Oxygen saturation	93% in room air

On examination, the patient was conscious, oriented, and afebrile, with a recorded blood pressure of 100/70 mmHg. Pallor and arcus senilis were present. Bilateral pitting pedal edema was present extending up to the mid calf region. There were no clinical signs suggestive of tuberculosis or infective endocarditis. No petechiae or purpura were present. Pulse rate was 107 per minute, regular in rhythm with normal volume and character. Respiratory rate was 23 per minute, indicating tachypnea, and was of abdominothoracic type. Jugular venous pressure was elevated at 10 cm of H_2O. Oxygen saturation was 93% on room air (Table 1). On cardiovascular examination, inspection and palpation revealed epigastric pulsations. The trachea was in the midline. Palpation showed the apical impulse in the left 5^{th} intercostal space in the midclavicular line. There were no palpable heart sounds, thrills, or parasternal heave. On percussion, the left cardiac border corresponded to the apical impulse and the right cardiac border corresponded to the right sternal border. On auscultation, a loud P2 was heard in the pulmonary area. Heart sounds in the mitral, tricuspid, and aortic areas were normal.

Laboratory investigations revealed hemoglobin of 6.7 g/dl, indicating severe anemia. Prothrombin time (PT) and international normalized ratio (INR) were 39 seconds and 2.7, respectively, showing prolonged clotting time likely secondary to anticoagulation therapy. Urine routine was normal with no albumin or sugar. Reticulocyte count was 0.4% and reticulocyte production index was 0.1%, indicating inadequate marrow response to anemia. Fasting blood sugar and postprandial blood sugar was 130 and 169 mg/dl, respectively. Viral markers including hepatitis B surface antigen, hepatitis C virus,

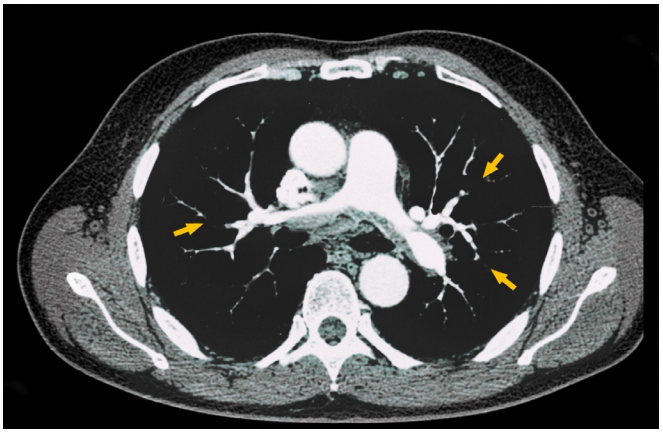

Figure 1: Computed tomography pulmonary angiography (CTPA) showing chronic thromboembolic disease involving bilateral subsegmental pulmonary arterial branches (yellow arrows), suggestive of chronic pulmonary thromboembolism.

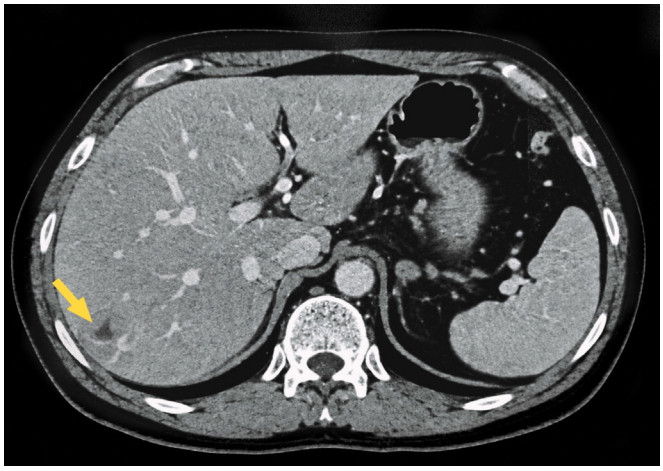

Figure 2: Contrast-enhanced computed tomography (CECT) abdomen showing a hypodense lesion in segment VI of the right lobe of liver (yellow arrow), suggestive of metastatic deposit.

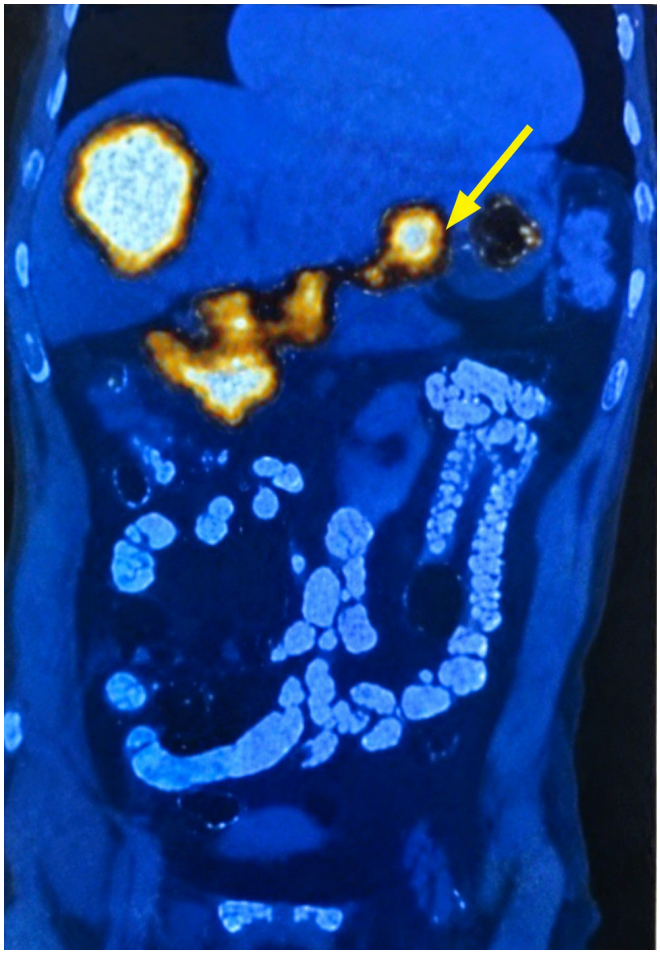

Figure 3: Positron emission tomography-computed tomography showing increased fluorodeoxyglucose uptake in the distal stomach with metabolically active hepatic lesion, suggestive of primary gastric malignancy with liver metastasis.

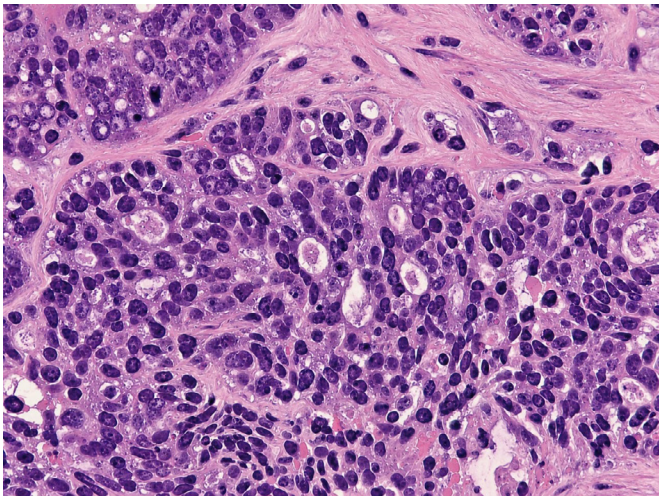

Figure 4: Histopathological examination (H&E stain, 40×) showing infiltrating malignant epithelial cells arranged in irregular glands and tubules within a desmoplastic stroma. The tumor cells exhibit pleomorphic hyperchromatic nuclei with focal signet ring cell morphology.

and human immunodeficiency virus were negative. Bilateral lower limb Doppler showed no evidence of deep vein thrombosis, with diffuse edema. Peripheral smear showed microcytic hypochromic anemia with normocytic normochromic cells, suggestive of dimorphic anemia. Iron studies showed ferritin 28 ng/mL, serum iron 50 μg/dl, total iron binding capacity 450 μg/dl, and transferrin saturation 18%, indicating iron deficiency anemia. Electrocardiogram showed normal sinus rhythm with a rate of 106 per minute, regular rhythm, right axis deviation, P pulmonale, and short PR interval. Echocardiography showed dilated right atrium and right ventricle, D-shaped left ventricle, dilated pulmonary artery and its branches, moderate tricuspid regurgitation, severe pulmonary hypertension, and right ventricular dysfunction with tricuspid annular plane systolic excursion of 10 mm.

Computed tomography pulmonary angiography showed chronic thromboembolic disease involving subsegmental pulmonary arterial branches without evidence of new clots or acute pulmonary embolism (Figure 1). With this a diagnosis of chronic pulmonary thromboembolism with pulmonary hypertension and right heart failure symptoms was made.

Ultrasound abdomen showed a hypoechoic lesion in the right lobe of liver measuring 15 x 10 mm with no organomegaly and no free fluid. Computed tomography abdomen showed a hypodense lesion in segment 6 of the right lobe of liver measuring 20 x 18 x 17 mm (Figure 2). Stool occult blood test was positive. In view of iron

Table 2: Summary of baseline laboratory, infectious work up, imaging, endoscopic, and histopathological findings

Parameter	*Finding*	*Reference value*
Total leukocyte count	7880 cells/mm³	4000–11000 cells/mm³
Hemoglobin	6.7 g/dl	13–17 g/dl
Platelet count	2.5 lakhs/mm³	1.5–4.5 lakhs/mm³
Blood urea	25 mg/dl	15–40 mg/dl
Serum creatinine	0.9 mg/dl	0.6–1.2 mg/dl
Total bilirubin	1.1 mg/dl	0.2–1.2 mg/dl
Direct bilirubin	0.3 mg/dl	0–0.3 mg/dl
Aspartate aminotransferase	56 U/L	10–40 U/L
Alanine aminotransferase	77 U/L	7–56 U/L
Alkaline phosphatase	140 U/L	44–147 U/L
Total protein	6.0 g/dl	6.0–8.3 g/dl
Serum albumin	3.2 g/dl	3.5–5.0 g/dl
Serum sodium	136 mmol/L	135–145 mmol/L
Serum potassium	3.9 mmol/L	3.5–5.0 mmol/L
Prothrombin time	39 sec	11–14 sec
International normalized ratio	2.7	0.8–1.2
Urine routine examination	Albumin nil, sugar nil	Nil
Reticulocyte count	0.4%	0.5–2.5%
Reticulocyte production index	0.1	More than 2
Fasting blood sugar	130 mg/dl	70–99 mg/dl
Postprandial blood sugar	169 mg/dl	Less than 140 mg/dl
Hepatitis B surface antigen	Negative	Negative
Hepatitis C virus	Negative	Negative
Human immunodeficiency virus	Non reactive	Non reactive
Antinuclear antibody	Negative	Negative
Antiphospholipid antibody profile	Negative	Negative
Serum homocysteine	Normal	5–15 μmol/L
Bilateral lower limb Doppler	No evidence of deep vein thrombosis, diffuse edema noted	No deep vein thrombosis
Peripheral smear	Microcytic hypochromic and normocytic normochromic anemia, dimorphic anemia	Normal red cell morphology
Serum ferritin	28 ng/mL	30–400 ng/mL
Serum iron	50 μg/dl	60–170 μg/dl
Total iron binding capacity	450 μg/dl	240–450 μg/dl
Transferrin saturation	18%	20–50%
Stool occult blood test	Positive	Negative
Carcinoembryonic antigen	8 ng/mL	0–3 ng/mL in non smokers
Cancer antigen 19-9	15 U/mL	0–37 U/mL
Alpha fetoprotein	8 ng/mL	0–10 ng/mL
Electrocardiogram	Normal sinus rhythm, heart rate 106/min, right axis deviation, P pulmonale, short PR interval	Normal sinus rhythm, no axis deviation, no P pulmonale, no conduction abnormality

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Echocardiography	Dilated right atrium and right ventricle, D shaped left ventricle, dilated pulmonary artery and its branches, moderate tricuspid regurgitation, severe pulmonary hypertension, right ventricular dysfunction, TAPSE 10 mm	Normal chamber size and function, no pulmonary hypertension, TAPSE more than 17 mm
Computed tomography pulmonary angiography	Chronic thromboembolic disease involving subsegmental pulmonary arterial branches without evidence of acute pulmonary embolism	No thromboembolic disease
Ultrasound abdomen and pelvis	Hypoechoic lesion in right lobe of liver measuring 15 × 10 mm, no organomegaly, no free fluid	No focal lesion, no organomegaly, no free fluid
Computed tomography abdomen	Hypodense lesion in segment 6 of right lobe of liver measuring 20 × 18 × 17 mm	No focal lesion
Contrast enhanced computed tomography abdomen	Ill defined hypodense lesion in anterior segment of right lobe of liver with thin rim enhancement and central non enhancement in portal venous phase, suggestive of metastasis or abscess	No abnormal enhancing lesion
Upper gastrointestinal endoscopy	Pale mucosa with mosaic pattern in stomach, no fundal varices, mild portal hypertensive gastropathy	Normal mucosa
Colonoscopy	Grade II internal hemorrhoids, no evidence of growth in colon, single diverticulum near ileocecal junction	Normal colonoscopy
Oesophago gastro duodenoscopy biopsy, antrum and pyloric region	Infiltrating adenocarcinoma, moderately differentiated	No malignancy
Liver biopsy histopathology	Metastatic carcinomatous deposits	No malignancy
Positron emission tomography computed tomography	Increased metabolic activity in distal antrum and pylorus, suggestive of primary lesion	No abnormal metabolic activity

deficiency anemia with a positive stool occult blood test, upper gastrointestinal endoscopy followed by colonoscopy was done.

Upper gastrointestinal endoscopy showed pale mucosa with mosaic pattern in the stomach, no fundal varices, and features suggestive of mild portal hypertensive gastropathy. Colonoscopy showed grade II internal hemorrhoids, no evidence of growth in the colon, and a single diverticulum near the ileocecal junction. Following this, contrast-enhanced computed tomography abdomen showed an ill-defined hypodense lesion in the anterior segment of the right lobe of the liver with thin rim enhancement and central non-enhancement, suggestive of either metastasis or abscess. In view of elevated carcinoembryonic antigen and atypical imaging features, liver biopsy was done. Histopathology of the biopsy showed metastatic carcinomatous deposits. Further workup with positron emission tomography computed tomography showed increased metabolic activity in the distal antrum and pylorus, suggestive of a primary lesion (Figure 3).

Oesophago gastro duodenoscopy with biopsy was done from the antrum and pyloric region. Endoscopy showed mucosal abnormality in the antropyloric region suggestive of a growth, for which biopsy was taken. Histopathological examination of the biopsy showed malignant epithelial cells arranged in glands and tubules with pleomorphic hyperchromatic nuclei. Occasional signet ring cells were seen (Figure 4). Tumor infiltration into lamina propria and muscularis mucosa was noted with surrounding desmoplasia and inflammatory infiltrate. Final impression was infiltrating adenocarcinoma, moderately differentiated, suggestive of primary gastric malignancy arising from the antropyloric region. Baseline laboratory, infectious work up, imaging, endoscopic, and histopathological findings are summarized in Table 2.

With this, a diagnosis of chronic pulmonary thromboembolism with severe pulmonary hypertension and right ventricular dysfunction, secondary to metastatic moderately differentiated gastric adenocarcinoma of the antropyloric region with liver metastasis, presenting with iron deficiency anemia was made.

The patient was managed with oxygen support because of breathlessness, pulmonary thromboembolism, pulmonary hypertension, and low oxygen saturation. Transfusion of 3 units of packed red blood cells was given to correct anemia and improve oxygen-carrying capacity, thereby reducing dyspnea and cardiac strain. Injection frusemide 20 mg intravenously twice

daily was administered to reduce fluid overload and relieve bilateral pedal edema and right heart failure symptoms. Injection pantoprazole 40 mg intravenously once daily was given to reduce gastric acid secretion and prevent gastrointestinal irritation and bleeding. Tablet albendazole 400 mg was given as a single dose for empirical deworming, as parasitic infestations can contribute to anemia. Injection vitamin B12 1000 microgram in 100 mL normal saline intravenously once daily and tablet folic acid 5 mg once daily were given to treat nutritional deficiency and support red blood cell production in dimorphic anemia. For metastatic gastric adenocarcinoma, palliative FOLFOX chemotherapy was initiated. Oxaliplatin was given as a platinum-based chemotherapeutic agent causing DNA damage in tumor cells. Leucovorin was administered to enhance the action of 5-fluorouracil. 5-fluorouracil was given as both intravenous bolus and continuous infusion to inhibit DNA synthesis and reduce tumor progression. The chemotherapy cycle was repeated once every 2 weeks as per standard protocol.

Discussion

Pulmonary thromboembolism results from obstruction of the pulmonary arterial circulation by thrombi, most commonly originating from the deep veins of the lower limbs.[1] It can lead to impaired pulmonary circulation, hypoxemia, pulmonary hypertension, and progressive right ventricular dysfunction. Malignancy is a well-established risk factor for venous thromboembolism because of the associated prothrombotic state.[2] Cancer-associated thrombosis occurs due to activation of coagulation pathways, release of procoagulant substances, inflammatory cytokines, and endothelial injury, thereby contributing to Virchow's triad. Gastric cancer, in particular, has been associated with an increased risk of thromboembolic events, and in some patients, thromboembolism may even be the first clinical manifestation of an occult malignancy.[3,4]

Several studies have shown that a proportion of patients presenting with unprovoked pulmonary thromboembolism are subsequently diagnosed with an underlying malignancy.[4] Similarly, previous reports have described pulmonary embolism as the initial or incidental presentation of gastric carcinoma, including clinically silent or atypical cases.[5] In contrast to patients with provoked thromboembolism who commonly have identifiable risk factors such as surgery, prolonged immobilization, trauma, or inherited thrombophilia, our patient had no obvious provoking factor at the initial presentation. Extensive evaluation, including antinuclear antibody profile, antiphospholipid antibody testing, and serum homocysteine levels, was negative. Previous studies have shown that patients with unprovoked venous thromboembolism may have negative thrombophilia workup despite the presence of an underlying occult malignancy.[6]

An important clue in the present case was the presence of anemia with positive stool occult blood testing. Iron deficiency anemia in elderly men should always raise suspicion for underlying gastrointestinal blood loss and malignancy. Current recommendations support gastrointestinal evaluation in patients with unexplained iron deficiency anemia, particularly when associated with thromboembolic disease. [7] In our patient, the coexistence of unprovoked pulmonary thromboembolism, dimorphic anemia, and occult gastrointestinal blood loss prompted further investigation.

Initially, CTPA demonstrated chronic thromboembolic disease involving the subsegmental pulmonary arterial branches. Further evaluation with abdominal ultrasonography and CT abdomen revealed a suspicious liver lesion, following which contrast-enhanced CT abdomen showed features suggestive of metastasis. Elevated tumor markers and atypical imaging findings prompted liver biopsy, which revealed metastatic carcinomatous deposits. Subsequently, PET-CT demonstrated metabolically active circumferential wall thickening in the distal antrum and pylorus, and upper gastrointestinal endoscopy with biopsy confirmed gastric adenocarcinoma. Similar reports in literature have emphasized the importance of systematic evaluation with imaging, endoscopy, and histopathological examination for identifying occult gastrointestinal malignancy in patients presenting with unexplained thromboembolism and anemia.[8]

The management of pulmonary embolism primarily focuses on preventing further thrombus formation and treating associated symptoms. Anticoagulation remains the cornerstone of therapy, while thrombolytic therapy is reserved for selected high-risk cases.[9] However, in malignancy-associated thrombosis, treatment of the underlying cancer is equally important because persistent tumor activity contributes to continued hypercoagulability and recurrent thromboembolic events.[10] In the present case, the patient initially received supportive management and anticoagulation, followed by palliative chemotherapy after confirmation of metastatic gastric carcinoma.

The relationship between malignancy and venous thromboembolism has been extensively described in literature. Lee and Levine reported that both cancer and anticancer therapy significantly increase the risk of thromboembolic disease, with variations depending on tumor type and disease burden.[1] Khorana et al. further demonstrated that patients receiving chemotherapy have an increased incidence of venous thromboembolism, emphasizing the persistent thrombotic risk associated with active malignancy and treatment.[2] Heit et al. also highlighted the increasing incidence and recurrence of venous thromboembolism in older individuals, along with its associated morbidity and mortality.[11] The prevalence of occult cancer was low among patients with a first unprovoked venous thromboembolism.[12] Unlike many reported cases where malignancy is identified early because of constitutional or gastrointestinal symptoms, our patient initially presented predominantly with cardiopulmonary manifestations related to chronic thromboembolic disease and pulmonary hypertension, which delayed recognition of the underlying gastric carcinoma.

Future advances in the management of pulmonary thromboembolism and malignancy-associated thrombosis are focused on early diagnosis, improved risk stratification, and targeted therapy. Advanced imaging techniques, biomarker-based screening, and molecular profiling may help in earlier identification of occult malignancy in patients presenting with unprovoked thromboembolism. Development of personalized anticoagulation strategies and targeted therapies may further improve outcomes in patients with cancer-associated thrombosis.

Overall, this case emphasizes that unprovoked pulmonary thromboembolism should prompt careful evaluation for occult malignancy, especially in older patients and in those with anemia or evidence of gastrointestinal blood loss. Early recognition and systematic evaluation can aid in timely diagnosis of underlying malignancy and appropriate multidisciplinary management.

Conclusion

Pulmonary thromboembolism is a common condition with multiple risk factors, among which malignancy is important. Cancer-related thrombosis occurs due to activation of coagulation pathways, inflammation, and endothelial injury. In some patients, thromboembolism may be the first clinical sign of an underlying malignancy. When no clear provoking factor is identified, further evaluation is required to identify hidden causes. Early diagnosis helps in proper management and prevention of complications.

This case reported a 57-year-old male who presented with breathlessness, chest pain, and lower limb swelling with a history of prior unprovoked pulmonary thromboembolism. Initial evaluation showed no thrombophilia, but further workup revealed iron deficiency anemia with positive stool occult blood. Imaging and endoscopic evaluation identified gastric adenocarcinoma with liver metastasis along with chronic pulmonary thromboembolic disease and pulmonary hypertension. This case shows the need for a stepwise clinical approach in patients with unexplained thromboembolism and supports screening for gastrointestinal malignancy when anemia is present.

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