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Case report

Association Between Ulcerative Colitis and Pulmonary Embolism: A Case Report

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Abstract

Introduction: Inflammatory bowel diseases (IBD) include Crohn's disease (CD) and ulcerative colitis (UC), conditions that primarily affect the intestines but can sometimes lead to extraintestinal complications such as venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE). **Case Report:** A 39-year-old woman was admitted with a 12-hour history of left-sided chest pain radiating to the ipsilateral scapular region, worsened by breathing and positional changes. She reported a prior episode of deep vein thrombosis in her right leg. Laboratory tests revealed microcytic hypochromic anemia and thrombocytosis. Imaging studies showed findings suggestive of pulmonary infarction, with a CT pulmonary angiogram confirming a thrombus at the segmental branch of the right lower lobe, accompanied by right-sided pleural effusion and atelectasis. Further evaluation for anemia, including a colonoscopy, revealed acute-phase pancolitis with minimal bleeding. An autoimmune panel was negative. Treatment was initiated with anticoagulation, mesalazine, and methylprednisolone. **Conclusions:** Thromboembolic complications occur in approximately 1% to 8% of patients presenting with active inflammatory bowel disease and are associated with increased morbidity and mortality. The underlying pathophysiological mechanisms likely involve a hypercoagulable state, platelet aggregation, impaired fibrinolysis, and genetic predisposition. Identifying risk factors and implementing appropriate therapeutic measures are crucial to preventing future thromboembolic events. The preferred prophylactic anticoagulation therapy is low-molecular-weight heparin, which is recommended for hospitalized patients at high risk during active disease phases.

Keywords

Ulcerative colitis, pulmonary embolism, inflammatory bowel disease, inflammation, coagulation.

INTRODUCTION

Inflammatory bowel diseases (IBD) include Crohn's disease (CD) and ulcerative colitis (UC), pathologies that mainly affect the intestine[(1)] and in some cases can generate extraintestinal complications[(1)]. Venous embolism includes pathologies such as deep vein thrombosis and pulmonary embolism, which are usually known complications of inflammatory bowel diseases[(1,2)].

It is estimated that the incidence of these complications is 0.26% annually in both CD and UC[(3)]; however, the actual risk remains unclear due to methodological designs and the heterogeneity of the studies conducted. Despite this, one of the main factors involved in this relationship is the inflammatory process, with key markers such as elevated C-reactive protein and thrombocytosis[(4,5)].

The objective of this article is to present the case of a patient with UC who presented pulmonary embolism as a complication.

CLINICAL CASE

This is a 39-year-old woman from Tunja, Colombia, who presented with a 12-hour history of left-sided chest pain

radiating to the ipsilateral scapular region. The pain worsened with breathing and positional changes. She reported a history of secondary anemia diagnosed five months earlier.

On physical examination, a heart rate (HR) of 115 beats per minute (bpm), blood pressure (BP) of 113/70 mm Hg, a temperature of 36.5 °C, and a weight of 53 kg were documented. Mucocutaneous pallor and tenderness to palpation were noted at the fourth intercostal space between the posterior axillary line and the left midclavicular line, with no other abnormalities observed. Admission laboratory tests revealed microcytic hypochromic anemia and thrombocytosis (**Table 1A**), while chest X-ray showed signs suggestive of pulmonary infarction (retrocardiac wedge-shaped opacity) (**Figure 1**).

Based on the findings, it was determined that the patient had a high probability of pulmonary thromboembolism, likely secondary to deep vein thrombosis and microcytic hypochromic anemia. Therefore, confirmatory studies were expanded, three units of red blood cells were transfused (admission hemoglobin: 5 mg/dL) (**Table 1B**), and anticoagulation with enoxaparin 80 mg daily (1.5 mg/kg/day) was initiated.

On the second day of hospitalization, a chest CT angiography (CT angio) report was received, showing a thrombus at the level of the right lower lobar segmental branch, right pleural effusion, and atelectasis (**Figure 2**).

Once the diagnosis of pulmonary embolism was confirmed, further studies were conducted to evaluate the cause of the patient's chronic anemia. An esophagogastroduodenoscopy (EGD) was performed, which revealed a hiatal hernia and chronic antral gastritis. Meanwhile, colonoscopy reported active ulcerative pancolitis in the acute phase with scant bleeding (**Figure 3**), explaining the cause of the iron-deficiency anemia. Given these findings, treatment was initiated with mesalazine 1 g orally every 12 hours and methylprednisolone 70 mg intravenously every 24 hours for three days. Anticoagulant therapy was also maintained, as there was no evidence of active bleeding.

On the fourth day of hospitalization, given the diagnosis of active ulcerative pancolitis complicated by pulmonary embolism and microcytic anemia, the patient was evaluated by the gastroenterology department. The treatment was adjusted by increasing the mesalazine dose to 3 grams per day. Additionally, due to the risk of recurrent thrombotic events and potential cardiac complications, a transthoracic echocardiogram was ordered, which showed normal ventricular function without signs of pulmonary hypertension.

Once the treatment with intravenous corticosteroid was completed (sixth day of hospitalization) and upon evidencing an adequate clinical and paraclinical evolution (**Table 1B**), it was decided to discharge the patient with recommendations, follow-up appointments, outpatient examina-

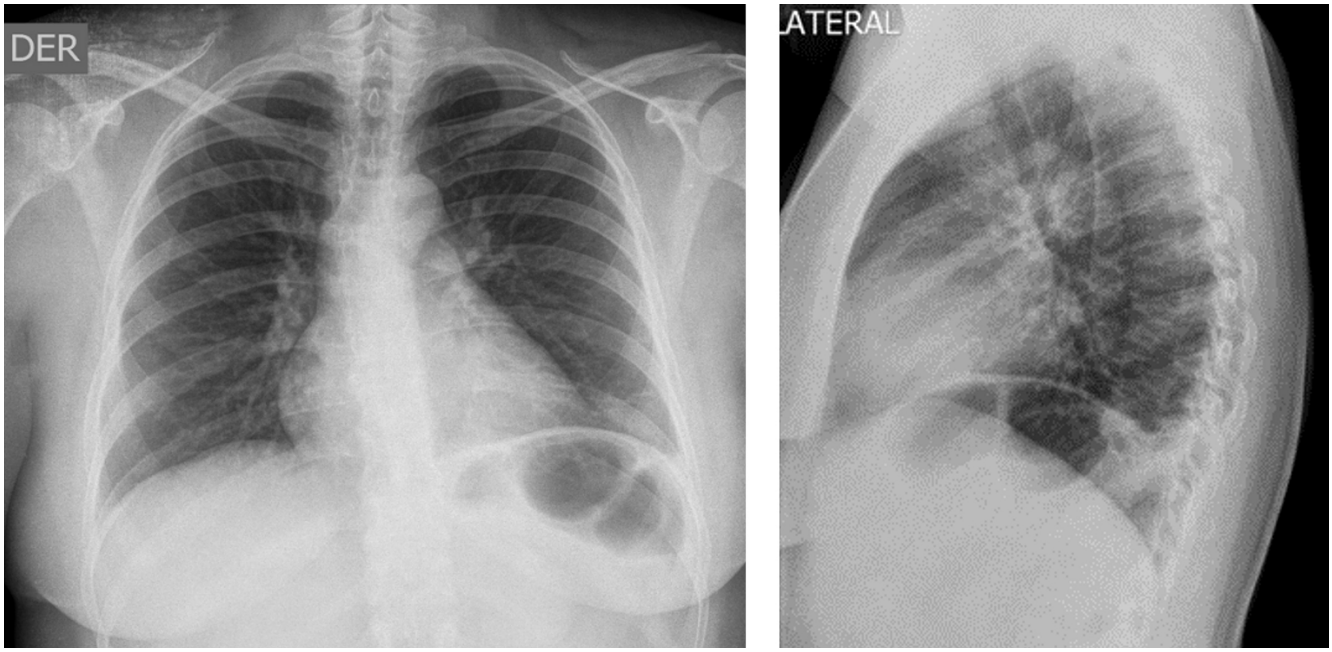

Figure 1. Chest X-ray. Findings suggestive of pulmonary infarction (retrocardiac wedge-shaped opacity) Images property of the authors.

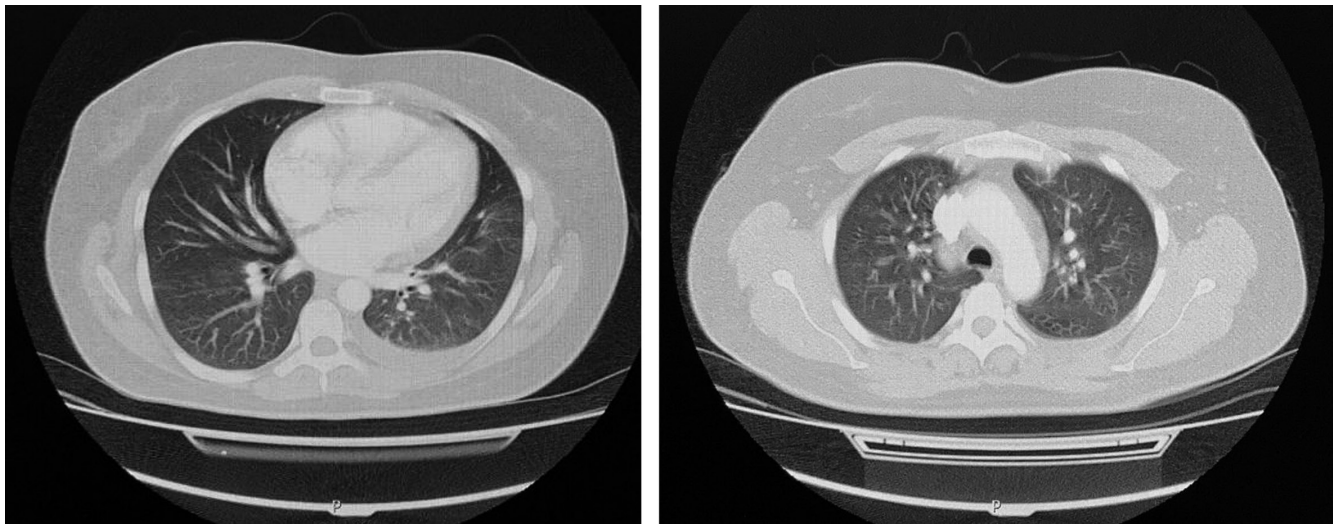

Figure 2. Chest CT angiography: thrombus at the level of the right lower lobar segmental branch, right pleural effusion, and atelectasis. Images property of the authors.

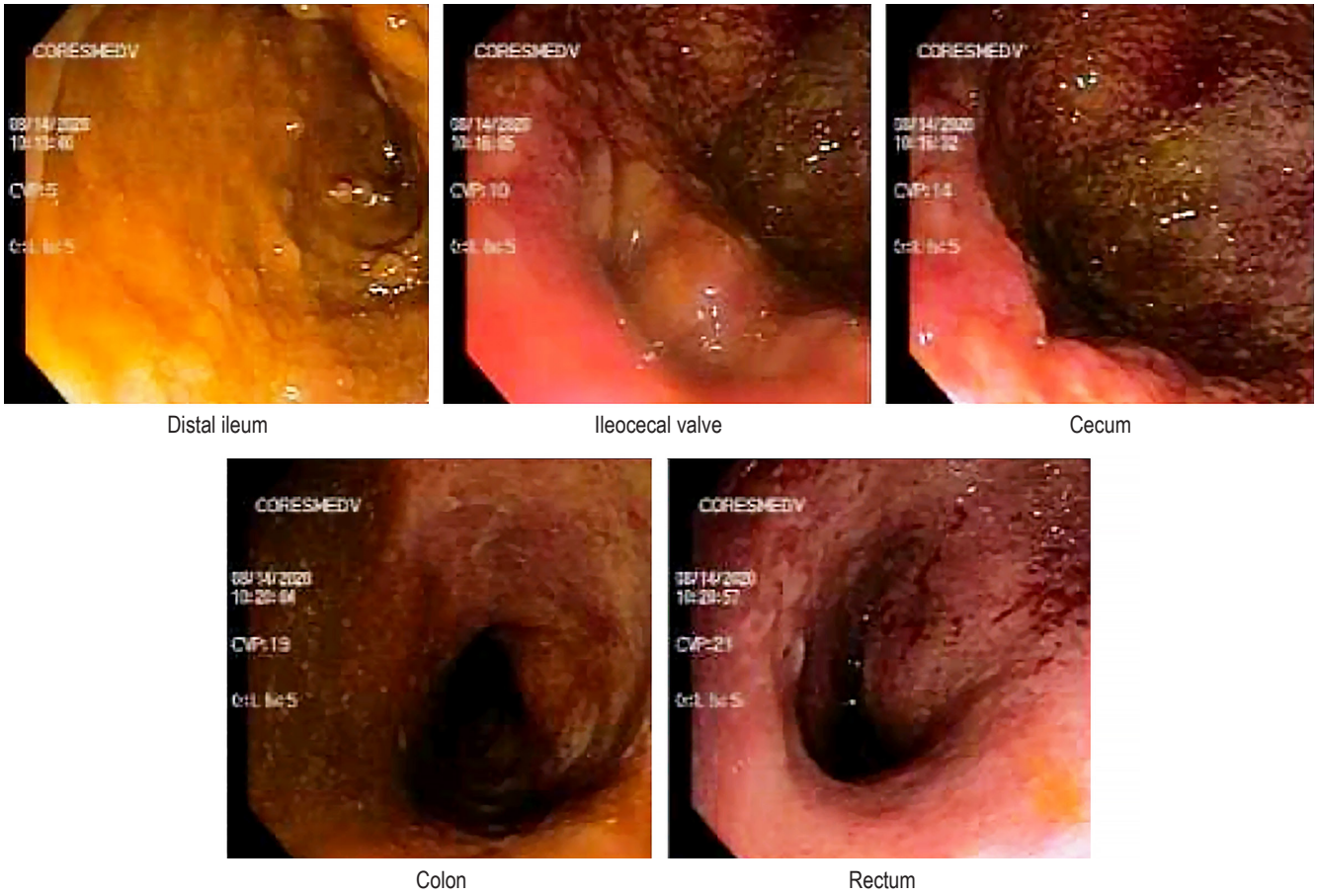

Figure 3. Colonoscopy. Findings revealed active ulcerative pancolitis in the acute phase with scant bleeding. Images property of the authors.

tions and alarm signs in the event of requiring assistance again in the emergency department.

One month later, the patient was reassessed in an outpatient consultation. Complementary studies ruled out other immunological diseases (**Table 1C**), and a colon biopsy revealed epithelial erosion, increased lymphoplasmacytic infiltrate, formation of lymphoid nodules, basal plasmacytosis, abundant neutrophils with crypt involvement, microabscesses, and depletion of goblet cells. These findings confirmed that the patient had active UC without dysplasia, and the established treatment was continued.

DISCUSSION

Thromboembolic complications of inflammatory bowel disease (IBD) have an estimated prevalence of 1% to 8%; however, postmortem studies report an incidence of 39%. Patients presenting with newly diagnosed IBD have a 3- to 4-fold increased risk of developing venous thromboembolism (VTE) compared to the general population[6-8]. Pulmonary embolism increases the risk of morbidity and mortality in this patient group, with reported mortality rates ranging from 18% to 25% in patients with IBD[9].

There are likely risk factors that contribute to the presentation of the condition, such as age 60 years or older with IBD, pregnancy, active and extensive IBD with complications (stenosis, fistulas, or abscesses)[10,11], obesity, smoking, family history of thromboembolic disease, use of oral contraceptives or hormone replacement therapy, immobility[8,12], dehydration, need for surgery, and being a carrier of central catheters[13]. Other hematological abnormalities have been found to be associated with thrombotic risk, such as anemia and hyperhomocysteinemia, as well as electrolyte imbalances or patients on corticosteroid treatment[13]. In our case, the patient had a history of deep vein thrombosis, along with findings of microcytic hypochromic anemia and thrombocytosis, which were considered attributable risk factors for the presentation of pulmonary embolism.

Among the pathophysiological events involved in the risk of thromboembolism, several hypotheses are described that point to the active state of IBD as an initial requirement. There is a state of hypercoagulability associated with an increase in procoagulant factors, such as factors V and VIII, and a decrease in anticoagulant factors like antithrombin III, protein C, and protein S[6,9]. The involvement of autoantibodies such as anticardiolipin (aCL) and lupus anticoagulant (LA) has also been suggested as precursors in the hypercoagulable state[7].

Similarly, thrombocytosis or platelet activation and aggregation occur due to the expression of markers such as GP53, CD-40 ligand, and P-selectin[13], which induce microvascular damage and cytokine activation. There are

Table 1. Laboratory tests

Diagnostic tests	Result	Reference
A. Admission tests		
Creatinine (mg/dL)	0.52	0.3-1.1
Plasma glucose (mg/dL)	89	74-106
Leukocytes (x 10^3/µL)	9.38	4.5-11.0
Neutrophils (%)	46	50-70
Lymphocytes (%)	45	20-40
Hemoglobin (g/dL)	5	11.0-16
MCV (fL)	9	80-99
MCHC (g/dL)	20	32-36
Platelets (x 10^3/µL)	848	150-450
Troponin # 1 (ng/mL)	0.004	0.0-0.3
B. Control tests		
Leukocytes (x 10^3/µL)	9.82	4.5-11.0
Neutrophils (%)	35	50-70
Lymphocytes (%)	47	20-40
Hemoglobin (g/dL)	8.2	11.0-16
MCV (fL)	9.4	80-99
MCHC (g/dL)	26	32-36
Platelets (x 10^3/µL)	767	150-450
Immunological tests	**Result**	**Reference**
C. Complementary tests		
Coagulation Protein C Activity (%)	93	70-130
Anti-dsDNA IgG Antibodies	Negative	>1:10
Anticardiolipin IgG Antibodies (GPL/mL)	2.06	>18
Anticardiolipin IgM Antibodies (MPL/mL)	1.36	>18
Antinuclear Antibodies (ANA)	Negative	>1:160
Extractable Nuclear Antigen, IgG Antibodies (EIA Units)	1.29	20-39
Anti-SSA (Ro) IgG Antibodies (EIA Units)	0.87	
Anti-RNP IgG Antibodies (EIA Units)	1.44	
Lupus Anticoagulant	1.3	
Citrullinated Peptide (Anti-citrulline) (U/mL)	<1	>5.0

MCHC: mean corpuscular hemoglobin concentration; dsDNA: double stranded DNA; GLP: glucagon-like peptide; IgG: Immunoglobulin G; IgM: Immunoglobulin M; MLP: mean levels of cardiolipin antibodies; RNP: ribonucleoprotein; SSA: anti-RO antibody; MCV: mean corpuscular volume.

decreased plasma concentrations of tissue plasminogen activator (tPA), the main activator of the fibrinolytic system, along with an increase in plasminogen activator inhibitor and thrombin-activatable fibrinolysis inhibitor, suggesting that fibrinolysis is also altered[7]. Additionally, genetic factors such as factor V Leiden, factor II, and mutations in the plasminogen activator inhibitor type 1 gene are involved in the development of thrombotic manifestations[6,9].

Therapeutic measures recommended for the prevention of thromboembolic events include hydration, the use of below-the-knee compression stockings (especially for venous thrombosis of the extremities), correction of vitamin deficiencies (particularly B6, B12, and folate), and an active lifestyle that involves constant mobilization. Prophylactic anticoagulant therapy is recommended for high-risk patients who are hospitalized during the active phase of the disease or those who have undergone surgical procedures. Recommendations are based on the use of low molecular weight heparin (LMWH) as the treatment of choice. Alternatively, unfractionated heparin or fondaparinux at low doses can be used. The risk-benefit of extending prophylaxis after hospital discharge for individuals with a higher risk of venous thromboembolism remains unknown; however, long-term treatment with vitamin K antagonists (warfarin, acenocoumarol, and fluindione) or non-vitamin K antagonists (dabigatran, apixaban, edoxaban, and rivaroxaban) is recommended for outpatient care [10,14-17].

CONCLUSIONS

Thromboembolic complications have an estimated prevalence of 1% to 8% in patients presenting with active-phase IBD. Pulmonary embolism, in turn, increases morbidity and mortality. The potential pathophysiological events involved include disruption of platelet aggregation, fibrinolysis failure, and genetic conditions that promote hypercoagulable states. It is crucial to identify risk factors and implement therapeutic measures to prevent future thromboembolic events. The prophylactic thromboembolic therapy of choice is LMWH, which is recommended for high-risk patients hospitalized during the active phase of the disease.

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Conflicts of interest

The authors state that they have no conflicts of interest.

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Asociación entre colitis ulcerativa y embolia pulmonar: caso clínico

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