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# Ulcerative Colitis-Associated Bronchiectasis, Pleural Effusion, and Bilateral Achilles Tendon Injury

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## ABSTRACT

*Ulcerative colitis is a chronic immune-mediated inflammatory bowel disease associated with diverse extraintestinal manifestations. Although the joints, skin, and eyes are the most frequently affected extraintestinal sites, involvement of the respiratory system and pleura is uncommon. The pathogenetic mechanisms potentially linked to dysregulated immune responses beyond the intestine remain poorly defined. Simultaneous development of bronchiectasis, pleural effusion, and bilateral Achilles tendon injury represents an exceptionally rare multisystem phenotype of ulcerative colitis, particularly when it occurs decades after colectomy in the absence of active intestinal disease.*

*A 76-year-old man with a 40 year-history history of ulcerative colitis and previous colectomy presented with productive cough, progressive exertional dyspnea, fatigue, acute bilateral posterior ankle pain, and swelling for two months. Laboratory investigations demonstrated elevated inflammatory markers including an erythrocyte sedimentation rate of 28 mm/h and C-reactive protein of 8.4 mg/dL, together with increased circulating levels of the proinflammatory mediators TNF-α, IL-1, and IL-6. Chest computed tomography revealed bilateral lower-lobe bronchiectasis and a left-sided pleural effusion. Magnetic resonance imaging showed partial rupture of the right tendon and the left Achilles tendons. Pleural fluid was a lymphocyte-predominant exudate, with negative microbiological and cytological studies. Comprehensive assessment excluded infectious, traumatic, drug-related, corticosteroid-associated, or other systemic causes of a lung disorder, pleural disease, and tendon injury. The patient was managed conservatively with physical treatment and splinting of both tendons. The pleural effusion resolved, whereas bronchiectasis persisted. Complete clinical and functional recovery of the bilateral Achilles tendon injuries was achieved within six months.*

*The simultaneous occurrence of these features, after exclusion of alternative etiologies and despite longstanding intestinal remission following colectomy, was considered compatible with an uncommon systemic phenotype of ulcerative colitis. The clinical profile may reflect persistent systemic immune dysregulation involving inflammatory signaling, connective-tissue injury, and impaired tissue repair. The elevated proinflammatory cytokine levels further support a potential shared immunopathogenic mechanism linking airway, pleural, and musculoskeletal involvement, although a direct causal relationship cannot be established on the basis of a single case. This case broadens the recognized spectrum of extraintestinal involvement in ulcerative colitis and emphasizes that multisystem complications may occur long after colectomy, even in the setting of sustained intestinal remission. The distinctive combination of airway, pleural, and musculoskeletal involvement may offer insights into the shared immunopathogenic pathways underlying extraintestinal disease in ulcerative colitis.*

## Keywords
Ulcerative colitis, Extraintestinal manifestations, Bronchiectasis, Pleural effusion, Immune dysregulation, Achilles tendon injury, Tendon tear.

## Introduction
Ulcerative colitis is a chronic disorder of the gastrointestinal tract characterized by dysregulated mucosal immune response. Extraintestinal manifestations occur in approximately 25–40% of patients that may involve multiple organ systems, most commonly the joints, skin, and eyes, underscoring the systemic nature of the disease [1-5]. Although uncommon, respiratory manifestations encompass a broad spectrum of disorders, including large- and small-airway disease, bronchiectasis, interstitial lung disease, and pleural inflammation or effusion [6,7]. Among these, bronchiectasis is the most frequently reported structural pulmonary abnormality and may occur independently of the activity or extent of intestinal disease. Pulmonary abnormalities may persist or arise after colectomy, suggesting that their pathogenesis is not necessarily dependent on ongoing colonic inflammation [8,9]. Pulmonary and pleural involvement, including bronchiectasis, interstitial lung disease, and pleural inflammation, have been reported in approximately 0.2–0.5% of clinically apparent cases [10]. Musculoskeletal manifestations of ulcerative colitis typically present as peripheral arthritis, axial arthropathy, or enthesitis [1-3]. In contrast, Achilles tendon disease is distinctly uncommon whereas spontaneous tendon rupture is exceptionally rare, with the available literature consisting primarily of isolated reports of tendinopathy or tendon injury [11,12]. To our knowledge, the concurrent occurrence of clinically significant pulmonary disease and bilateral Achilles tendon injury in a patient with ulcerative colitis has not been previously reported. The simultaneous occurrence of bronchiectasis, exudative pleural effusion, and bilateral Achilles tendon injury may therefore expand the recognized spectrum of extraintestinal sequelae associated with ulcerative colitis and underscores the importance of maintaining clinical vigilance for immune-mediated multisystem disease, even in patients with quiescent intestinal disease. The case in question may play a pivotal role in elucidating the pathogenesis of the primary disease along with its associated extraintestinal manifestations.

## Case Presentation
A 76-year-old male presented with a 2-month history of progressive exertional dyspnea, left-sided pleuritic chest pain, and mucoid sputum production. Two weeks before admission, he developed sudden-onset, severe bilateral posterior ankle pain at rest, accompanied by localized swelling and marked tenderness. There was no preceding trauma, strenuous physical activity, or other mechanical stress. He denied recent fluoroquinolone exposure, treatment with systemic corticosteroids, or immunosuppressive agents. He was diagnosed with ulcerative colitis 40 years earlier, which had been refractory to medical therapy and ultimately required total colectomy and ileostomy 30 years before the current presentation. He had not received corticosteroid or systemic immunosuppressive therapy during the preceding 8 years. He was a lifelong nonsmoker and had no other relevant comorbidities. Family history included papillary thyroid carcinoma, hypertension, and type 2 diabetes mellitus in his father and hypertension in his mother. On admission, his temperature was 36.8°C, heart rate 92 beats/min, respiratory rate 14 breaths/min, and oxygen saturation 94% while breathing ambient air. Physical examination revealed percussion dullness and decreased breath sounds over the left lower posterior hemithorax, consistent with a pleural effusion. Bilateral swelling and marked tenderness were present over both Achilles tendon regions. Mild left lower-quadrant abdominal tenderness was noted without guarding or rebound tenderness.

Laboratory evaluation showed a white-cell count of 9,800 cells/μL, erythrocyte sedimentation rate (ESR) of 28 mm/h, and C-reactive protein (CRP) concentration of 8.4 mg/dL. Serum electrolytes, calcium, uric acid, renal function, and liver function tests were within normal limits. Autoimmune serologic testing, including antinuclear antibodies, rheumatoid factor, and anti–cyclic citrullinated peptide antibodies, was negative. Serum concentrations of tumor necrosis factor-α (TNF-α), interleukin (IL)-1β, and IL-6 were elevated at presentation, remained above the reference ranges during the first 3 months of follow-up, and subsequently declined into the reference range by 6 months (Table 1). Pulmonary function testing demonstrated a mild obstructive ventilatory defect, with an $FEV_1$/FVC ratio of 68%. Contrast-enhanced computed tomography (CT) of the chest revealed multifocal cystic and cylindrical bronchiectatic changes involving both lower lobes, accompanied by a left-sided pleural effusion (Figure 1). Sputum microscopy and cultures were negative for bacterial, mycobacterial, and fungal pathogens. Diagnostic thoracentesis yielded an exudative pleural effusion. Pleural fluid protein concentration was 3.8 g/dL, corresponding to a serum-to-pleural fluid protein ratio of 0.62; lactate dehydrogenase was 124 U/L, with a serum-to-pleural fluid ratio of 0.68. The total nucleated cell count was approximately 1,500 cells/μL, with lymphocytes accounting for 48% of nucleated cells. Cytologic examination showed no malignant cells, and Gram staining and bacterial, mycobacterial, and fungal cultures were negative. Both the tuberculin skin test and QuantiFERON-TB Gold assay were negative. A pleural fluid adenosine deaminase concentration of 24 U/L provided no supportive evidence for tuberculous pleuritis.

**Table 1:** Laboratory findings at admission and during follow-up.

| Laboratory variable | At admission | 3 months | 6 months | Reference range |
|---|---|---|---|---|
| ESR | 28 mm/hr | 18 mm/hr | 12 mm/hr | 0-15 mm/hr |
| CRP | 8.4 mg/dL | 6.2 mg/dl | 2.4 mg/dl | 0-0.5 mg/dL |
| TNF-α | 6.4 pg/mL | 3.8 pg/ml | 1.6 pg/ml | < 2 pg/mL |
| IL-1β | 5.8 pg/mL | 4.2 pg/ml | 2.4 pg/ml | < 3 pg/mL |
| IL-6 | 8.6 pg/mL | 6.8 pg/ml | 4.2 pg/ml | < 5 pg/mL |

Fiberoptic bronchoscopy demonstrated only small amounts of white-gray secretions within the lower-lobe bronchi. Bronchoalveolar lavage (BAL) fluid cultures were negative for bacterial, mycobacterial, and fungal pathogens, and cytologic examination revealed no malignant or other pathologic cellular abnormalities. Magnetic resonance imaging of both ankles was

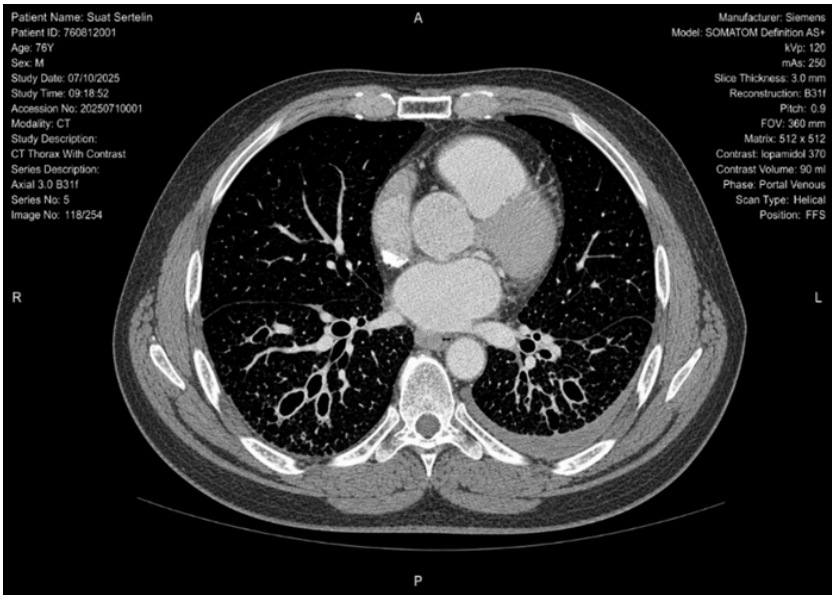



**Figure 1.** Contrast-enhanced axial chest CT demonstrates a left-sided pleural effusion and scattered cylindrical and cystic bronchiectatic changes involving both lower lobes.

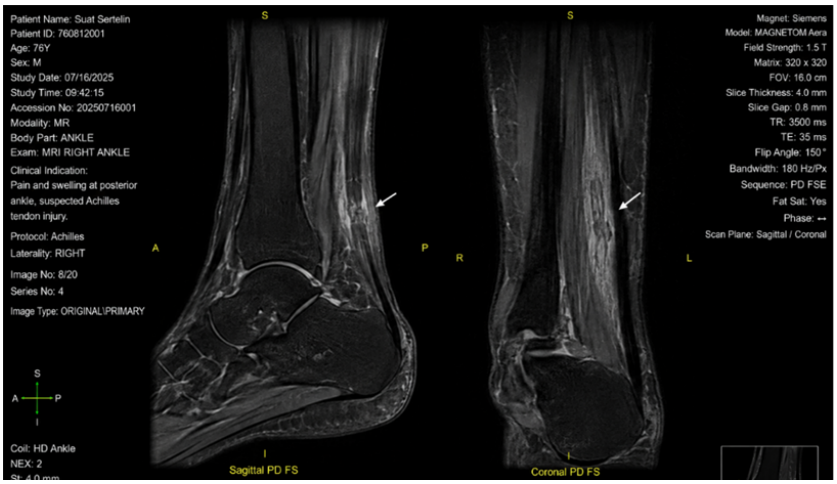



**Figure 2.** Sagittal and coronal T2-weighted fat-suppressed MR images revealing focal thickening and increased intratendinous signal within the midportion of the right Achilles tendon, with associated mild peritendinous edema, consistent with a partial-thickness tear. A Haglund deformity and small retrocalcaneal bursitis are present.

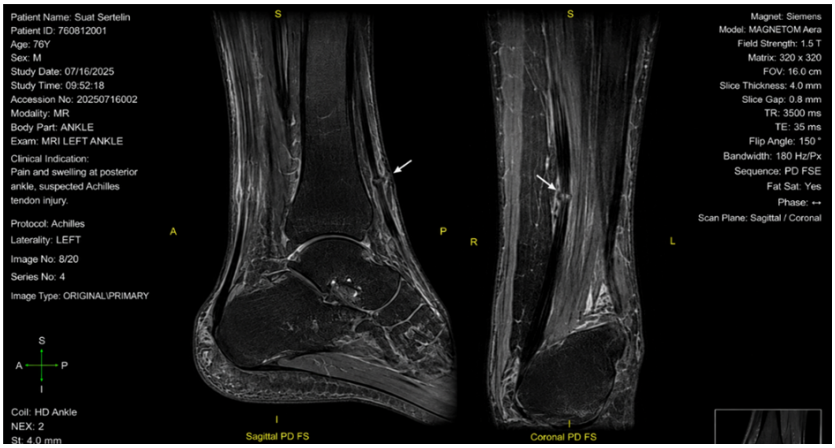



**Figure 3.** Sagittal and coronal T2-weighted fat-suppressed MR images demonstrating focal thickening and increased intratendinous signal within the midportion of the left Achilles tendon, with focal fiber disruption and mild peritendinous edema, consistent with a partial-thickness tear. A Haglund deformity and small retrocalcaneal bursitis also exist.

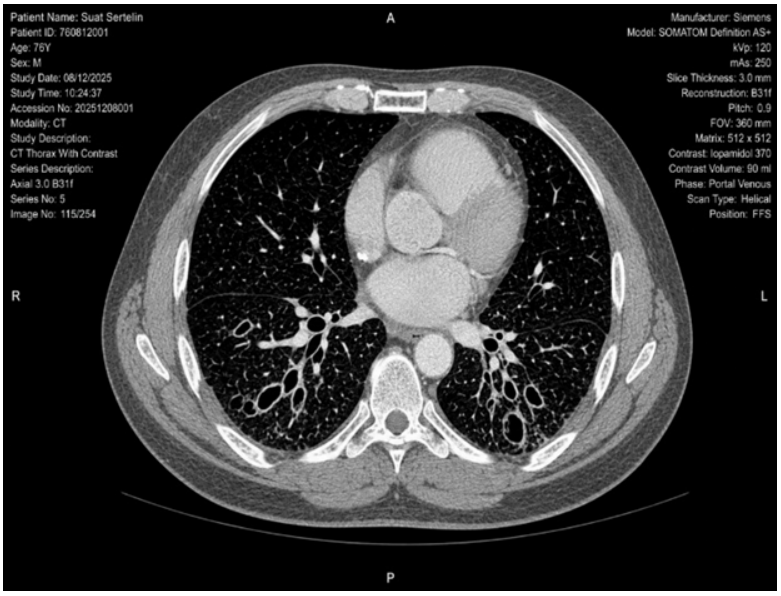



**Figure 4.** Follow-up contrast-enhanced axial chest CT showing complete resolution of the left-sided pleural effusion with persistent bilateral lower-lobe bronchiectatic changes.

performed using a 1.5-T scanner with a dedicated ankle coil. Sagittal and coronal fat-suppressed sequences demonstrated focal thickening and increased intratendinous signal within the midportion of both Achilles tendons, with focal fiber disruption and longitudinal fraying consistent with partial-thickness tears (Figures 2 and 3). Mild peritendinous edema was present on both sides. Haglund deformities and small retrocalcaneal bursae were also identified bilaterally.

A comprehensive diagnostic evaluation was undertaken to identify potential alternative causes of the concurrent pulmonary, pleural, and tendon abnormalities. Extensive microbiological testing of sputum, pleural fluid, and BAL specimens yielded no evidence of bacterial, mycobacterial, or fungal infection. Clinical, laboratory, and imaging findings remained negative for of malignancy, sarcoidosis, connective tissue disease, or drug-induced pulmonary toxicity. Established risk factors for Achilles tendon injury, including fluoroquinolone exposure, systemic corticosteroid therapy, trauma, and excessive physical activity, were excluded. Alternative infectious, malignant, inflammatory, pharmacological, or mechanical disorders that could adequately account for the simultaneous occurrence of the three organ-system abnormalities were not identified. In the context of longstanding ulcerative colitis, previous total colectomy, elevated circulating

**Table 2:** Evidence supporting cytokine-mediated pathogenesis of extraintestinal manifestations in ulcerative colitis.

| Study (year) | Cytokine axis | Key finding | Relevance to present case |
|---|---|---|---|
| **Strober W et al. [13]** | IL-12, IL-23, IL-17 | Role of the IL-23/Th17 axis in sustaining chronic mucosal and systemic inflammation | Th17-mediated cytokine responses in arthritis, uveitis, and other extraintestinal manifestationss |
| **Guillo et al. [4]** | TNF-α, IL-6, IL-17 | Inflammatory cytokine networks govern disease expression | Cytokine-driven disease activity across organ systems beyond intestine |
| **Kim & Cheon [5]** | TNF-α, IL-1β, IL-6, IFN-γ | Cytokines induce endothelial activation, leukocyte recruitment, and aberrant immune-cell trafficking from the gut to extraintestinal organs | Evidence linking systemic cytokine release to extraintestinal manifestation development |
| **Rogler et al. [3]** | TNF-α, IL-23 | Systemic immune activation | Cytokine driven systemic, multi-organ inflammation including lung and musculoskeletal structures |
| **Verstockt et al. [25]** | IL-23, IL-12 | Regulator of immune activation and chronic inflammation in UC | IL-23 contribution to extraintestinal immune manifestations |
| **Aebisher D et al. [22]** | IL-23/IL-17,TNF-α | Chronic inflammation in joints, lungs, and skin | Link between Achilles tendon injury and pulmonary inflammation (bronchiectasis) |
| **Neurath MF et al. [23]** | TNF-α, IL-17 | EIM mediated by chronic immune activation | Support for bronchiectasis as UC-associated immune-mediated airway disease |
| **Fiorino et al. [26]** | TNF-α, IL-17, IL-23, IL-6 | Leukocyte trafficking and immune-cell activation driven by inflammatory cytokines | Cytokine involvement in musculoskeletal, ocular, dermatologic, and pulmonary manifestations. |
| **Tímár ÁE et al. [27]** | TNF-α, IL-6, IL-17 | Cytokine overproduction, immune-cell migration, and molecular mimicry between intestinal and extraintestinal tissues | Cytokine-mediated multisystem inflammation |

proinflammatory cytokine concentrations, and extensive exclusion of alternative etiologies, the overall clinical picture was considered potentially consistent with an ulcerative colitis–associated multisystem inflammatory phenotype despite apparent intestinal disease quiescence. The temporal association between the clinical abnormalities and the subsequent decline in circulating cytokine concentrations supported, but did not establish, a shared inflammatory mechanism.

The patient was managed conservatively with a structured physical therapy program and ankle splinting for bilateral partial-thickness Achilles tendon tears. Chest physiotherapy incorporating airway-clearance techniques was initiated for the bilateral lower-lobe bronchiectasis. The pleural effusion resolved completely within 6 weeks of presentation. Achilles tendon injuries improved progressively, with full functional recovery achieved after approximately 3 months of conservative treatment. Follow-up contrast-enhanced chest CT demonstrated complete resolution of the left pleural effusion, whereas the bilateral lower-lobe bronchiectatic changes persisted (Figure 4), consistent with established structural airway remodeling due to extraintestinal immune dysregulation caused by ulcerative colitis.

## Discussion

Ulcerative colitis is a chronic immune-mediated disorder that can extend beyond the gastrointestinal tract through a broad spectrum of extraintestinal manifestations that involve ocular, hepatobiliary, dermatologic, musculoskeletal, and, less commonly, pulmonary tract [1-7]. These systemic manifestations are thought to arise from genetic susceptibility, dysregulated immune signaling, aberrant trafficking of gut-primed lymphocytes, molecular mimicry, and persistent immune dysregulation [8,9]. Although respiratory involvement is uncommon, bronchiectasis is among the most frequently reported respiratory complication, whereas pleural disease is distinctly rare [6,7]. In a retrospective cohort of 563 ulcerative colitis patients, pulmonary involvement attributable to primary disease developed in 5 % during follow-up with airway disease predominating while pleuritis was identified in only 0.2 % of the patients [10]. This case is notable for the concurrent occurrence of bronchiectasis, an exudative pleural effusion, and bilateral Achilles tendon injury in a patient with longstanding ulcerative colitis despite apparent intestinal remission following colectomy. After comprehensive evaluation that excluded infectious, autoimmune, malignant, pharmacologic, and mechanical causes, this constellation was interpreted as a rare extraintestinal phenotype associated with ulcerative colitis. The temporal relevance between the multisystem abnormalities and elevations in circulating TNF-α, IL-1β, and IL-6 further raises the possibility of a shared systemic inflammatory process. Nevertheless, these observations remain associative and do not establish a causal relationship between ulcerative colitis and the observed pulmonary, pleural, and tendon abnormalities.

Several biological mechanisms may plausibly connect ulcerative colitis with extraintestinal disease across anatomically distinct organs. Systemic immune activation, aberrant trafficking of gut-primed lymphocytes, circulating immune complexes, and cytokine-mediated tissue injury have all been proposed as mechanisms underlying extraintestinal manifestations. Cytokines including TNF-α, IL-1β, IL-6, IL-17, and IL-23 can promote endothelial activation, leukocyte recruitment, and amplification of inflammatory responses at sites distant from the intestine. In particular, dysregulation of the IL-23/Th17 axis has been implicated in several extraintestinal manifestations involving the musculoskeletal, ocular, dermatologic, and pulmonary systems [4,5,11-13]. Within the respiratory tract, shared mucosal immune pathways and sustained neutrophil-driven inflammation may contribute to airway injury and progressive bronchial wall remodeling, potentially resulting in bronchiectasis. In the present case, absence of recognized mechanical, pharmacologic, or other predisposing factors for tendon injury, together with concurrent elevations in TNF-α, IL-1β, and IL-6, raises the possibility that systemic inflammatory signaling contributed to Achilles tendon injury by promoting extracellular-matrix remodeling and increasing tissue susceptibility to structural damage and vulnerability [11-13]. Spontaneous tendinitis and tendon rupture have been reported only rarely in patients with ulcerative colitis, primarily as isolated case reports [12], whereas cytokine-mediated connective-tissue injury is well established in other systemic inflammatory disorders [14,15]. In the present case, elevated circulating concentrations of TNF-α, IL-1β, and IL-6 cytokines capable of inducing matrix metalloproteinase activity and promoting extracellular-matrix degradation—provide circumstantial support for a potential immune-mediated mechanism underlying tendon weakening and injury along with the absence of recognized mechanical stress, trauma, corticosteroid exposure, or fluoroquinolone use reduces the likelihood of several established causes of Achilles tendon injury [16-21]. Bilateral Achilles tendon injury provides an additional and particularly unusual component of the proposed multisystem phenotype.

The simultaneous occurrence of three anatomically distinct manifestations and their temporal relationship with systemic cytokine elevation are particularly noteworthy. One plausible model is that systemic immune activation created a permissive inflammatory environment in susceptible extraintestinal tissues, with cytokine-mediated endothelial activation and leukocyte recruitment contributing to pulmonary and pleural inflammation, while inflammatory signaling at the tendon or enthesis may have promoted extracellular-matrix remodeling and increased susceptibility to structural injury [22,23]. Although this proposed mechanism provides a plausible biological framework linking the pulmonary, pleural, and tendon abnormalities, a causal relationship cannot be established from the clinical and biochemical findings alone because circulating cytokine concentrations do not establish tissue-specific pathogenic mechanisms or causality. Table 2 summarizes key studies linking specific cytokine pathways to extraintestinal disease and their potential relevance to the present case. A hallmark feature of this case is the occurrence of these manifestations despite apparent intestinal remission after colectomy pointing out to the fact that extraintestinal disease does not necessarily parallel intestinal activity and may

emerge independently of active bowel inflammation. Systemic immunologic alterations associated with ulcerative colitis may remain clinically relevant even after definitive surgical treatment of the colon. The normalization of circulating TNF-α, IL-1β, and IL-6 concentrations during subsequent clinical improvement is compatible with this interpretation and provides temporal support for a relationship between systemic inflammatory activity and the clinical phenotype. However, cytokine normalization cannot distinguish a causal pathway from a nonspecific response to resolution of inflammation.

Several limitations should be acknowledged. First, the observations are derived from a single patient and therefore cannot establish a causal relationship between systemic cytokine elevation and the observed clinical phenotype. The possibility that the concurrence of cytokine elevation and multiple extraintestinal abnormalities occurred coincidentally cannot be excluded. Second, circulating cytokine concentrations may not accurately reflect tissue-specific inflammatory activity within the lungs, pleura, or Achilles tendons. Although TNF-α and IL-1β can induce matrix metalloproteinase expression and extracellular-matrix degradation, and IL-6 can sustain inflammatory signaling, these established biological effects do not demonstrate that the same pathways were responsible for the lesions seen in this patient [24-27]. Third, absence of tissue-level immunopathologic assessment further limits mechanistic inference that restricts the ability to determine whether the proposed cytokine pathways were active at the involved sites of disease. Finally, although alternative infectious, malignant, pharmacologic, traumatic, and mechanical causes were investigated, an unrecognized concurrent disorder cannot be completely excluded.

Similarly, the subsequent normalization of cytokine concentrations following clinical improvement supports a temporal relationship but should be regarded as supportive rather than definitive evidence of a shared pathogenic mechanism. Accordingly, the proposed unifying mechanism should be regarded as a hypothesis generated by the clinical phenotype rather than as a demonstrated pathogenic pathway. Multisystemic extraintestinal disease is exceedingly rare in ulcerative colitis and has been described only sporadically in individual case reports [28-30]. This case expands the recognized spectrum of extraintestinal disease. To our knowledge, the simultaneous occurrence of bronchiectasis, exudative pleural effusion, and bilateral Achilles tendon injury has not previously been described as a distinct clinical phenotype of ulcerative colitis. Its clinical significance lies not only in the rarity of the individual findings but also in their temporal concurrence despite apparent intestinal remission, suggesting that clinically important extraintestinal disease may occur independently of intestinal activity. Although the underlying mechanisms remain uncertain, this case supports a potential role for systemic immune pathways in the development of uncommon pulmonary and musculoskeletal complications of ulcerative colitis that may help elucidate the dysregulated processes underlying extraintestinal organ involvement in this disease. Further studies are needed to determine whether distinct cytokine profiles characterize patients with unusual pulmonary or musculoskeletal extraintestinal disease and whether targeted immunomodulatory therapy can modify the clinical course of these uncommon phenotypes.

## Conclusions

This case describes an exceptionally rare multisystem phenotype of ulcerative colitis characterized by bronchiectasis, exudative pleural effusion, and bilateral Achilles tendon injury despite prolonged apparent intestinal remission. The temporal association between elevated circulating TNF-α, IL-1β, and IL-6 concentrations and their subsequent normalization, together with the comprehensive exclusion of alternative etiologies, raises the possibility of a shared dysregulated systemic inflammatory process extending beyond the intestine. Cytokine-mediated endothelial activation, leukocyte recruitment, and altered extracellular-matrix homeostasis offer a biologically plausible framework that may link pulmonary, pleural, and tendinous involvement. However, the absence of tissue-level immunopathologic evidence precludes confirmation of a common pathogenic mechanism or a causal relationship. These observations instead highlight the heterogeneous and potentially systemic character of extraintestinal manifestations of ulcerative colitis, which may emerge even during prolonged periods of apparent intestinal quiescence.

Clinicians should recognize that uncommon extraintestinal manifestations of ulcerative colitis may occur even during prolonged apparent intestinal remission and may involve multiple organ systems concurrently. Awareness of this possibility may facilitate timely multidisciplinary evaluation and broaden the differential diagnosis of otherwise unexplained pulmonary, pleural, and musculoskeletal abnormalities in patients with a remote history of ulcerative colitis. The present case further underscores the diagnostic challenges and clinical complexity inherent in rare extraintestinal manifestations of the disease. Detailed phenotypic characterization of such patients may ultimately contribute to a deeper understanding of ulcerative colitis pathogenesis and its extraintestinal spectrum. Prospective studies incorporating tissue-level analyses and longitudinal assessment of systemic inflammatory and cytokine profiles are needed to determine whether distinct biological signatures characterize such multisystem phenotypes and whether these pathways may have diagnostic or therapeutic implications.

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