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Cryptococcal Immune Reconstitution Inflammatory Syndrome-Associated Bronchiolitis Obliterans Organizing Pneumonia

Kriptokokal İmmün Rekonstitüsyon Enflamatuvar Sendromu İlişkili Bronşiolitis Obliterans-Organize Pnömoni

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Abstract

Atypical presentations of cryptococcal infection have been described as manifestations of immune reconstitution inflammatory syndrome (IRIS) in patients with human immunodeficiency virus (HIV) infection following the introduction of combination antiretroviral therapy (cART). We describe a patient presenting with cryptococcal bronchiolitis obliterans organizing pneumonia (BOOP) as a cryptococcal-IRIS (C-IRIS), eight weeks after cART initiation. To our knowledge, this is the first patient with HIV disease reported to have cryptococcal IRIS-related BOOP.
Keywords: Human immunodeficiency virus, HIV, AIDS, immune reconstitution inflammatory syndrome, cryptococcosis, bronchiolitis obliterans organizing pneumonia, BOOP

Öz

İnsan immün yetmezlik virüsü (HIV) ile enfekte hastalarda kombinasyon antiretroviral tedavinin başlanmasını takiben immün rekonstitüsyon enflamatuvar sendromunun (IRIS) tezahürleri şeklinde kriptokok enfeksiyonunun atipik sunumları tariflenmiştir. Kombinasyon ART başlangıcından sekiz hafta sonra kriptokokal-IRIS (C-IRIS) kapsamında kriptokokal bronşiolitis obliterans organize pnömoni (BOOP) ile prezente olan bir hastayı tanımlıyoruz. Bildiğimiz kadarıyla, bu hasta HIV pozitif olan bir hastada bildirilen ilk C-IRIS ile ilişkili BOOP'tur.
Anahtar Kelimeler: İnsan immün yetmezlik virüsü, HIV, AIDS, immün rekonstitüsyon enflamatuvar sendromu, kriptokokoz, bronşiolit obliterans organize pnömoni, BOOP

Introduction

The introduction of combination antiretroviral therapy (cART) has lowered the incidence of opportunistic infections (OI), but a subset of these patients will experience exaggerated inflammatory responses to persistent foreign antigens after human immunodeficiency virus (HIV) treatment initiation, known as immune reconstitution inflammatory syndrome (IRIS) [1]. The precise immune mechanisms of cryptococcal-IRIS (C-IRIS) remain poorly understood. Cryptococcal-IRIS may present as a clinical worsening as the immune system is restored (paradoxical IRIS), which occur between 6% and 45% of patients with HIV-associated cryptococcosis 1-2 months post-ART initiation, or a new presentation of a previously subclinical cryptococcal infection (unmasked IRIS or "ART-associated" cryptococcosis)[2,3], within the first two months of ART, with clinical presentations similar to cryptococcosis that develop before ART[2,3]. IRIS has been described in association with multiple opportunistic pathogens, such as *Mycobacterium avium* complex,

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Mycobacterium tuberculosis, *Cryptococcus neoformans*, cytomegalovirus (CMV), and hepatitis viruses. Cryptococcal-IRIS most frequently presents as recurrent meningitis; however, it may manifest as intracranial space-occupying lesions and non-central nervous system (CNS) presentations (e.g., lymphadenitis, pneumonitis, and ophthalmologic complications)[3]. Risk factors for paradoxical C-IRIS include a low inflammatory response and CD4+ cell count at baseline, rapid immune restoration, and a high organism or antigen load at baseline during cART initiation[2]. We recently observed an unusual case of C-IRIS that occurs as bronchiolitis obliterans organizing pneumonia (BOOP) early after immune reconstitution in a patient with previously acquired immunodeficiency syndrome (AIDS)-presenting systemic CMV infection.

Case Report

A 49-year-old male patient, with stage C3 HIV/AIDS, recently admitted to our department from July 15 to August 20, 2013, with AIDS-related systemic CMV disease, was hospitalized in September 2014 due to persistent high fever for three days, associated with worsening dry cough. The patient did not present with headache, meningeal signs, and neurological symptoms, but presented sweats, poor appetite (one month), and 10-lbs weight loss. On admission, he was conscious, oriented, and febrile (39 °C). His pulse was 96 beats/min, respiratory rate was 28 breaths/min, and blood pressure was 140/90 mmHg. The leukocyte count was 7,130/µl with 79% neutrophils, 13.3% lymphocytes, and 5.8% monocytes. The hemoglobin level was 10.5 g/dl, hematocrit was 30.1%, and platelet count was 180,000/µl. The patient's blood sugar, creatinine, aspartate and alanine aminotransferase, alkaline phosphatase, and the total bilirubin were normal. A progressively increased lactate dehydrogenase and C-reactive protein were reported in the past two months. At baseline CD4+ 270/mmc (23%) and HIV-RNA 1,269,064 copies/ml were reported. Four and 12 weeks after starting HAART plasma HIV-RNA was 132 copies/ml and undetectable, respectively. At the same time points, CD4+ cells were 550/mm^3 and 790/mmc^3 respectively. The chest X-ray was negative, while a thoracic computerized tomography (CT) documented BOOP, with left lower lobe involvement, especially of the basal pyramid, the dorsal subpleural region of the right lower lobe and the iuxtascissural site, the anterior segment of the right upper lobe; and mediastinal adenopathies (short axis about 10 mm in the Barety and subcarinal) (Figure 1). Findings were more evident on the left lung, which had small spots of parenchymal consolidation. Abdominal ultrasonographic examination was negative. Bone marrow biopsy during hospitalization showed pathological findings that are consistent with HIV-related myelopathy. A careful ruled out occult meningitis. A histological study of a lung biopsy confirmed BOOP. The patient had started from eight weeks cART regimen containing darunavir 800 mg daily, ritonavir 100 mg daily, tenofovir 245 mg daily, and lamivudine 300 mg/daily and secondary prophylaxis for CMV disease with valganciclovir at 900 mg/daily. Active CMV infection was excluded by the absence of CMV-DNA from blood and urine and of CMVpp65 antigen in the peripheral blood leukocyte specimens, accompanied by a negative CMV-DNA and viral culture result from bronchoalveolar lavage fluid (BALF) and by a negative assay for CMV-DNA by a transbronchial lung biopsy specimen. Direct microscopic BALF examination and culture were negative for bacterial, mycobacterial, fungal, and viral pathogens, but it was positive for *Cryptococcus neoformans*. Blood cultures were negative for bacterial, mycobacterial, and fungal pathogens. The cryptococcal antigen titer for the serum was 1:1200. Fluconazole treatment (800 mg/day) was started. The patient's clinical condition improved, and after one week, the fluconazole dosage was reduced to 600 mg/d followed by fluconazole at 400 mg/d after two weeks. The patient was discharged from the hospital after three weeks of treatment, and he was treated with fluconazole maintenance therapy for 21 weeks. Viro-immunological and serum cryptococcal antigen findings are summarized in Table 1. After 10 weeks of therapy, the fluconazole dosage was reduced to 200 mg/d. The cryptococcal antigen titers remained negative during the maintenance treatment and three-year follow-up period with radiographic improvement (Figure 2).

Discussion

The incidence of OI is decreased in patients with HIV infection after cART introduction. Unfortunately, these agents have brought new problems, such as IRIS, determined by a restoration of vigorous immune response[4]. The exact incidence of C-IRIS is unknown. Chang et al.[5] reported that low cryptococcal mannoprotein (CMP) induced interferon-gamma-γ production pre-cART, but not high CMP-specific T-cell responses after cART, were risk factors for C-IRIS. ART-associated cryptococcosis has been documented even in the absence of cryptococcemia. A placebo-controlled study of fluconazole as primary prophylaxis in Uganda ART-associated cryptococcosis revealed

Table 1. Viro-immunological and serum cryptococcal antigen features

	CD4+	HIV-RNA	Serum cryptococcal antigen
Before BOOP	270	1.269.064	Negative
At the time of diagnosis of BOOP	380	10.180	1200
After 24 weeks follow-up period	850	1.200	Negative

BOOP: Bronchiolitis obliterans organizing pneumonia, HIV: Human immunodeficiency virus

1.0% of individuals receiving placebo and ART were serum Cryptococcus antigen-negative at a median of 11 weeks preART[6]. The characteristics presented by our case should fall within the definition of "unmasking C-IRIS," according to the proposed clinical criteria definitions on C-IRIS in HIV-1-infected individuals from the International Network for the Study of HIV-associated IRIS[3]. The diagnosis of cryptococcosis was made after the start of ART, in the presence of a good virological and immunological response with an increased number of CD4+ and a significant reduction of plasma viremia, in the absence of cryptococcosis recognition before the start of ART. The number of CD4+ T cells measured in peripheral blood does not necessarily reflect the function nor the number of CD4 cells at the site of an OI; however, it is noteworthy that in our patient, the cryptococcosis was diagnosed in the presence of a progressively increasing peripheral CD4+ cells count, suggesting the presence of immune reconstitution in response to ART. A previous prospective multicenter study of C-IRIS conducted on 101 patients with AIDS with cryptococcal meningitis who received HAART revealed that 13 experienced C-IRIS from two weeks to four months after HAART initiation[7]. Cryptococcal BOOP is an uncommon form of cryptococcosis. Most cryptococcal pulmonary infections present as pneumonia,

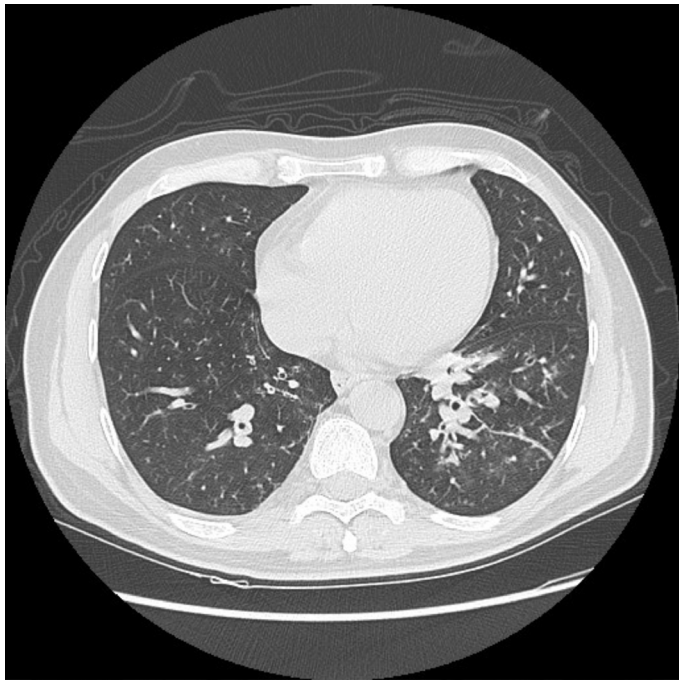

Figure 1. Chest computed tomography (CT) scans before antifungal therapy. A chest CT scan showed bronchial wall thickening and peribronchial alveolointerstitial hyperdensity of the lower left lobe, especially of the basal pyramid, blurred alveolar micronodules in the dorsal subpleural region of the right lower lobe and the iuxtascissural site, in the anterior segment of the right upper lobe; mediastinal adenopathies (short axis around the cm in Barety and subcarinal) were also documented **(Figure 1-4)**. 127×92 mm (300×300 DPI)

but pulmonary cryptococcomas have also been reported, and hematogenous spread may occur to other organs in 10% of patients, mainly to the CNS. A MEDLINE search documented seven case reports of pulmonary cryptococcosis with radiological and pathological findings of BOOP (Table 2)[8-14]. Two cases were diagnosed in immunocompromised patients, and two reports were described in immunocompetent individuals[8-13], while the immune status of two other patients was not specified. Carey et al.[8] reported the first case of BOOP associated with pulmonary cryptococcosis in a 56-year-old male patient who presented to the emergency department with a five-day history of non-exertional substernal stinging chest pain and a two-week history of malaise, non-productive cough, and occasional shortness of breath. An open lung biopsy was performed on day 11, which revealed BOOP in the lingula of the left lung. On the 18th hospitalization day, the culture of the open lung biopsy specimen yielded *Cryptococcus neoformans*. Amphotericin B therapy was initiated, and after five days of therapy, the patient was extubated and prednisone was added to the therapeutic regimen for BOOP. Tao et al.[9] reported three cases of pulmonary cryptococcosis. One of them was first suspected to have BOOP due to pulmonary cryptococcosis based on clinical and radiological findings. The patient was successfully treated with fluconazole. Tashiro et al.[10] reported an immunocompromised 66-year-old with BOOP diagnosed by clinical and BALF findings and treated with oral steroid therapy. After 6-7 months, multiple infiltrative shadows returned and were then joined by new multiple nodular shadows. The BALF

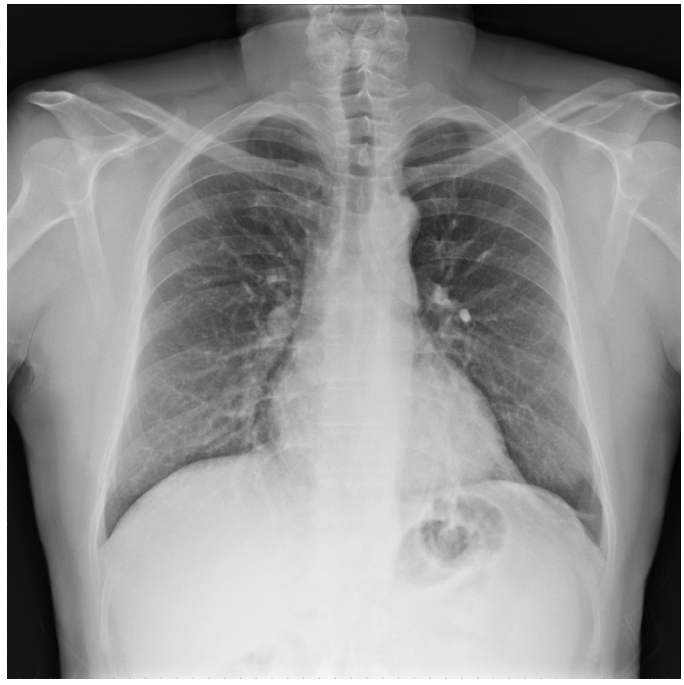

Figure 2. Chest X-ray film after antifungal therapy. No parenchymal lesions in place. Accentuation of the broncho-vascular texture, as a result of healing. 127×92 mm (300×300 DPI)

Table 2. Case series of bronchiolitis obliterans caused by *Cryptococcus neoformans*

Age/ sex	Background disease	Clinical features	Chest Radiographs	Chest CT scan	Pathological findings	Microbiologic findings	Therapy	Outcome	Authors
56 y/M	N.A.	Substernal stinging chest pain, malaise, nonproductive cough, and occasional shortness of breath	Diffuse interstitial infiltrates	N.A.	TBLN: BOOP in the lingula of the left lung	Culture of the OLB specimen yielded CN	L-AmB → keto + steroid	Improved	Carey et al.[8]
N.A.	N.A.	Respiratory symptoms	N.A.	Diffuse BOOP	TBLB: BOOP	CN on LB	Fluco	Improved	Tao et al.[9]
67 y/M	DM, hypertension, and two-vessel coronary artery disease	Back pain radiating to both legs weakness and muscle wasting of the left upper and lower extremities	N.D.	Bilateral, multiple lung nodules, in the right upper lobe and the lower lobes bilaterally	OLB: BOOP	A few budding yeasts, with narrow bases, were identified on OLB	Amph B, later changed to fluco, due to renal toxicity	Improved	Chantranuwat et al.[12]
31 y/M	N.A.	Cough	Bilateral patchy infiltrates	Patchy areas of air-space consolidation with air-bronchogram and adjacent ground-glass opacities	TBLB: epithelioid cell granulomas and organizing pneumonia.	Serum cryptococcal antigen was positive. Cryptococci were neither recognized in the BALF	Itra	Improved	Kishi et al.[11]
66 y/M	Immunocompromised host	Respiratory symptoms	Multiple infiltrative pulmonary shadows	Multiple infiltrative pulmonary shadows and multiple nodular shadows	N.D.	BALF revealed small bodies of Cryptococcus species. Serum cryptococcal antigen was positive	Steroids, amph B, flucy, and fluco	Improved	Tashiro et al.[10]
30 y/M	N.A.	Fever, non-productive cough	Bilateral upper and lower lobe consolidations and nodularity	Bilateral upper and lower lobe consolidations and nodularity	OLB: BOOP with Cryptococci in the alveoli and fibromucoid tissue. Serum cryptococcal antigen was negative	N.A.	Steroid, fluco	Improved	Kessler et al.[13]
63 y/F	Systemic lupus erythematosus	Widespread, painful erythema with profuse exudate of the left leg	N.A.	Multiple centrilobular nodules, bronchial wall thickening, and consolidation	TBLB: inflammatory infiltration with encapsulated yeast around respiratory bronchioles	C. neoformans serotype A was isolated from BALF	L-AmphB, flucy, followed by fluco	Improved	Abe et al.[14]

L-AmB: Liposomal amphotericin B lipid complex, Keto: Ketoconazole, fluco: Fluconazole, CN: *Cryptococcus neoformans*, TBLB: Transbronchial lung biopsy, DM: Diabetes mellitus, OLB: Open lung biopsy, Itra: Itraconazole, BALF: Bronchoalveolar lavage fluid, Flucy: Flucytosine, CT: Computed tomography, BOOP: Bronchiolitis obliterans organizing pneumonia

revealed small bodies of Cryptococcus species and a positive result for anti-Cryptococcus antigen was also obtained from the serum, suggesting pulmonary cryptococcosis. Anti-mycotic therapy was started, including amphotericin B, flucytosine, and fluconazole[10]. A 31-year-old male patient was admitted to Toranomon Hospital because of cough and bilateral patchy infiltrates on the chest radiograph. Chest CT scan was suggestive of BOOP. Transbronchial lung biopsy specimen confirmed the presence of epithelioid cell granulomas without necrosis and the coexistence of organizing pneumonia. The titer of serum cryptococcal antigen was 1:256. Cryptococci were neither recognized in the specimen nor cultured from the bronchial lavage fluid. The patient was treated with itraconazole and the symptom disappeared. Chest radiograph showed spontaneous regression in a short period and the titer of serum cryptococcal antigen decreased to 1:16 after the therapy[11]. Authors from Chulalongkorn University, Bangkok, presented a 67-year-old male patient with diabetes who presented with back pain that radiate to both legs for six months, with weakness and muscle wasting of the left upper and lower extremities for two months, which suggested a progressive multifocal myeloradiculopathy, without pulmonary symptoms[12]. Chest X-ray and CT scan revealed bilateral multiple nodular infiltrate in the right upper lobe and the bilateral lower lobes mimicking metastases. A thoracoscopic lung biopsy demonstrated BOOP due to capsule-deficient Cryptococcus. The patient was treated with amphotericin B, which was later changed to fluconazole due to renal toxicity. Serum and cerebrospinal fluid for cryptococcal antigen were negative, and a CD4+ lymphocyte level was normal. The exact cause of myeloradiculopathy was unclear[12]. Kessler et al.[13] reported a case of an immunocompetent young adult without lung disease history, who presented with a prolonged course of significant lung involvement and BOOP. *Cryptococcus neoformans* was identified in the lung tissue obtained at the biopsy. He improved with steroids and high-dose fluconazole for three months and was completely asymptomatic after several months of follow-up[13]. A 63-year-old female patient with systemic lupus erythematosus, on maintenance therapy with tacrolimus and prednisolone, was diagnosed with cellulitis and respiratory bronchiolitis due to *C. neoformans*. She was treated with liposomal amphotericin B and flucytosine, which resulted in physical and radiographic improvement[14]. BOOP is a rare entity that should be assessed in a patient with HIV infection with unexplained radiologic findings and atypical clinical presentation. This very rare diagnosis should be added to the list of causes of alveolar pneumopathy with infiltration and fever that occur during an HIV infection. A careful analysis of the current literature has revealed the presence of few BOOP cases in HIV patients[15-25]. Usually, the initial diagnosis was very difficult and often confused with different opportunistic diseases, and after the histological examination of pulmonary biopsy, the patient frequently responded completely to corticosteroids. A retrospective review of open lung biopsy in patients with HIV and acute respiratory symptoms admitted to a specialist inpatient unit revealed that one patient had BOOP with emphysema, one had BOOP with pneumocystis pneumonia, and one had BOOP with *Pseudomonas aeruginosa* pneumonia[19]. The Zimbabwean study of HIV-infected adolescent patients with chronic lung disease (CLD) revealed that bronchiolitis obliterans was the most common cause of CLD, although many adolescents had co-existing radiological features of bronchiectasis. The authors speculate that bronchiolitis obliterans was caused by multiple bacterial or viral infections. HIV immunosuppression contributed by facilitating ongoing small airway inflammation. A further hypothesis is that progressive inflammatory bronchiolitis obliterans may be caused by HIV itself. Further research to delineate the cause and effective management of bronchiolitis obliterans in HIV-related CLD is needed[24]. The occurrence of BOOP in a 24-year-old Korean female patient diagnosed with HIV 10 years before and admitted because of a one-year history of cough and sputum production and a three-day history of fever has been recently documented. The case suggests that macrolides are a potential treatment option in patients with HIV infection having mild BOOP[25]. Based on our experience, pulmonary cryptococcosis should be considered in the differential diagnosis of patients with HIV having clinico-radiological features of BOOP. Early recognition and management of unmasking C-IRIS should help reduce its mortality[1-4].

Ethics

Informed Consent: Consent was obtained from all participants in this study.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: A.M., Concept: A.M., Design: A.M., Data Collection or Processing: F.U., Analysis or Interpretation: F.U., Literature Search: A.M., F.U., Writing: A.M., F.U.

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