**Mycobacterium xenopi** pulmonary infection resulting in self-limited immune reconstitution inflammatory syndrome in an HIV-1 infected patient

Sebastiano Leone\(^1\), Sergio Giglio\(^12\), Patrizia Maio\(^1\), Piero Capasso\(^3\), Giuseppina dell’Aquila\(^1\), Mario Magliocca\(^1\), Francesco Saverio Nigro\(^1\), Piera Pacifico\(^1\), Nicola Acone\(^1\)

\(^1\)Infectious Diseases Division;  
\(^2\)AIDS service;  
\(^3\)Radiology Service, A.O.R.N. San G. Moscati, Avellino, Italy

The prognosis for patients infected with human immunodeficiency virus (HIV) type 1 has improved dramatically since the advent of Highly Active Antiretroviral Therapy (HAART), which allows sustained suppression of HIV replication and recovery of CD4 T cell counts (Lazzarin, 2004). In some patients receiving HAART, immune reconstitution is associated with a pathological inflammatory response leading to substantial short-term morbidity and even mortality (Shelburne et al., 2006). This report concerns a case of *M. xenopi* pulmonary infection resulting in self-limited immune reconstitution inflammatory syndrome in an HIV-1 infected patient.

**KEY WORDS:** Mycobacterium xenopi, HIV, HAART, Immune reconstitution inflammatory syndrome

A 39-year-old man with advanced HIV infection (CD4 lymphocyte count of 28 cells/mm\(^3\), HIV-RNA of 55200 copies/ml) was admitted to our clinic. There was no history of prior infection with mycobacteria, Mantoux test was negative, and the chest X-ray was normal. Treatment with tenofovir (TDF), emtricitabine (FTC) and efavirenz (EFV) was started. Co-trimoxazole was started for *Pneumocystis jiroveci* prophylaxis. One month later he presented with productive cough and a fever of 37.5°C. Blood investigations revealed C-reactive protein of 58 mg/L, erythrocyte sedimentation rate of 67 mm/h, lactate dehydrogenase of 600 U/L, white blood cell count of 4850 cells/mm\(^3\) with 65.4% neutrophils. The other examinations showed no abnormalities. Multiple blood cultures were sterile. At this time the CD4 lymphocyte count had increased to 65 cells/mm\(^3\) and the HIV plasma viral load was undetectable. A High Resolution Computer Tomography (HRCT) scan demonstrated ground-glass opacities (Fig. 1). *M. xenopi* isolate was obtained from multiple sputum sam-

---

**Corresponding author**  
Sebastiano Leone, MD  
Infectious Diseases Division  
A.O.R.N. San G. Moscati  
Contrada Amoretta - 83100 Avellino, Italy  
E-mail: sebastianoleone@yahoo.it
Testing for *Pneumocystis jiroveci* was negative. Serological tests for *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella pneumophila*, *Cryptococcus neoformans*, and Cytomegalovirus were negative. However, a spontaneous improvement in clinical symptoms was observed within two weeks. During follow-up, sputum cultures for *M. xenopi* were persistently negative. Two months later, the HRTC control was negative (Fig. 2).

IRIS has been reported in a limited number of patients with various and previous opportunistic infections and tumours associated with AIDS immunodeficiency despite adequate control of virologic and immunologic parameters. Paradoxically, patients may experience a progression of symptoms associated with worsening lymphadenopathy, pulmonary infiltrates and pleural effusions after HAART is started. The fall in blood HIV RNA levels and the return of immune function, including an increase in CD4 lymphocytes, restore the ability to mount an inflammatory reaction to infectious antigens (Lazzarin, 2004). Non-tuberculous Mycobacteria (NTM) disease along with its related mortality is a significant pathology as a cause of hospitalization among HIV-infected individuals (Miguez-Burbano et al., 2006). In the HIV population, NTM are a common cause of IRIS. However, only a few cases of *M. xenopi*-associated IRIS have been described (Field et al., 2006; Lawn et al., 2005). *M. xenopi* is usually a non-pathogenic colonizer of the airways. Most of the findings are fortuitous, which raises concerns about their clinical significance, especially for HIV-infected patients in whom concurrent pulmonary diseases are often present (Gazzola et al., 2004; Juffermans et al., 1998). A literature search via the MEDLINE database using the following MeSH terms ‘antiretroviral immune reconstitution syndrome’ or ‘immune paradoxical reaction’ or ‘immune reconstitution inflammatory syndrome’ or ‘immune reconstitution syndrome’ or ‘paradoxical worsening’ or ‘immune reconstitution’ or ‘immunorestitution’ and ‘*Mycobacterium xenopi*’ was performed.

Additional references were identified from citations in other published papers. Overall, only four references were identified (Bachmeyer et al., 2002; de Boer et al., 2003; Buckingham et al., 2004; Foudraine et al., 1999). The median baseline CD4 lymphocyte count and viral load (VL) were 19±16.7 cells/mm³ and 575114.5±60878.8 copies/mL, respectively. At the onset of IRIS the CD4 lymphocyte count and VL were 200±63.3 cells/mm³ and 557±626.5 copies/mL, respectively. All cases were treated with antimyobacterial drugs and were resolved without complications. However, the need for routine treatment of *M. xenopi* in HIV-infected individuals receiving HAART is doubtful. Recently, Kerbiriou et al. reported the outcomes of 20 HIV-infected patients.
receiving HAART who had respiratory symptoms and in whom *M. xenopi* was isolated. The median blood CD4+ lymphocyte count was 37/mm³. Fifteen of 20 patients received no antimycobacterial therapy and remained healthy after a median of ~4 years of follow-up, and two patients required treatment specifically for *M. xenopi* infection, both showed clinical improvement. The authors conclude that pulmonary *M. xenopi* isolation in HIV-1 patients receiving HAART does not usually require specific treatment (Kerbiriou et al., 2003). In conclusion, there is still very little evidence from controlled clinical trials. Future works are needed to clarify both the role of *M. xenopi* in pathogenesis of IRIS and the requirements for routine treatment.

**Conflict of interest:**
No conflict of interest to declare.

**REFERENCES**


ERRATA CORRIGE

The authors’ names of the article “Distribution of different carbapenem resistant clones of *Acinetobacter baumannii* in Tehran Hospitals” published in New Microbiol. 2009 July; 32 (3): 265-271

ARE

Morovat Taherikalani, Bahram Fatolahzadeh, Mohammad Emaneini, Setareh Soroush, Mohamad Mehdi Feizabadi

AND NOT

Taherikalani Morovat, Fatolahzadeh Bahram, Emaneini Mohammad, Soroush Setareh, Feizabadi Mohamad Mehdi

We apologize to those concerned for any inconvenience this error may have caused.