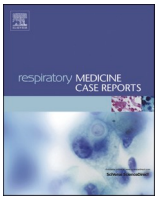
Respiratory Medicine Case Reports 55 (2025) 102213

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr



Case Report

Immune reconstitution inflammatory syndrome (IRIS): Case series and review of the literature

Sebastian Schregenberger [a], Vera Graup [b], Adrian Schibli [c], Benjamin Preiswerk [c], Irène Laube [d], Lars C. Huber [e], Melina Stüssi-Helbling [e,*]

[a] Department of Surgery, Division of Emergency Medicine, Hospital Buelach, Spitalstrasse 24, 8180, Buelach, Switzerland
[b] Department of Cardiology, University Hospital Zurich, Rämistrasse 100, 8091, Zurich, Switzerland
[c] Department of Internal Medicine, Division of Infectious Diseases and Infection Prevention, City Hospital Zurich, Birmensdorferstrasse 497, 8063, Zurich, Switzerland
[d] Department of Internal Medicine, Division of Pulmonology, City Hospital Zurich, Birmensdorferstrasse 497, 8063, Zurich, Switzerland
[e] Department of Internal Medicine, Clinic for Internal Medicine, City Hospital Zurich, Birmensdorferstrasse 497, 8063, Zurich, Switzerland

ARTICLE INFO

Keywords:
Immune reconstitution inflammatory syndrome
IRIS
Tuberculosis
TB
HIV
Lymphoma

ABSTRACT

Background: Immune-reconstitution inflammatory syndrome (IRIS) is a dysregulated host inflammatory response following the initiation of appropriate therapy targeting an infectious disease. It is most commonly reported in human immunodeficiency virus patients following the initiation of antiretroviral therapy; however, IRIS can also be seen in immunocompromised patients without HIV, when the immune system is recovering. The diagnosis is confirmed using clinical and laboratory data after excluding differential diagnoses and concomitant infections.
Case series: Here, we describe three cases of patients with IRIS that were treated at our tertiary care center. The first case involves a paradoxical IRIS in an HIV-positive patient with TB, where the re-initiation of ART led to an inflammatory response despite effective anti-tuberculous treatment (ATT). The second case highlights unmasking IRIS in an HIV-positive patient, where the initiation of ART revealed an underlying Epstein-Barr virus (EBV)-associated B-cell lymphoma. The third case describes paradoxical worsening of pulmonary TB in an HIV-negative patient, expanding the scope of IRIS beyond its conventional association with HIV infection.
Conclusion: These cases illustrate the various manifestations of IRIS and emphasize the need for timely diagnosis and appropriate management strategies to mitigate the potentially severe outcomes associated with this syndrome. Our report highlights the challenges faced in the diagnosis of IRIS which impede prompt onset of therapy.

1. Background

Immune reconstitution inflammatory syndrome (IRIS) is a dysregulated host inflammatory response that results from the rapid restoration of pathogen-specific immune responses following the initiation of an appropriate therapy [1,2]. It is mostly seen in patients infected with human immunodeficiency virus (HIV) receiving antiretroviral therapy (ART), with an estimated incidence of 18 % (4–54

* Corresponding author.
E-mail addresses: sebastian.schregenberger@spitalbuelach.ch (S. Schregenberger), vera.graup@usz.ch (V. Graup), adrian.schibli@stadtspital.ch (A. Schibli), benjamin.preiswerk@stadtspital.ch (B. Preiswerk), irene.laube@stadtspital.ch (I. Laube), lars.huber@stadtspital.ch (L.C. Huber), melina.stuessi-helbling@usz.ch (M. Stüssi-Helbling).

https://doi.org/10.1016/j.rmcr.2025.102213
Received 26 January 2025; Received in revised form 18 March 2025; Accepted 11 April 2025
Available online 12 April 2025

%) [3]. However, IRIS can also be found in HIV-negative patients in the setting of improving immunologic function, such as discontinuation of anti-TNF therapy, reduction of immunosuppression in transplant recipients, or following the initiation of antituberculosis therapy for tuberculosis (TB) [4,5].

IRIS is characterized by an exaggerated inflammatory response and can manifest as a paradoxical worsening of a previously diagnosed disease (infection or malignancy) or as the unmasking of a subclinical disease [6–9,13]. Tuberculosis-associated IRIS is one of the most frequent complications in patients receiving ART due to concomitant HIV infection. However, numerous opportunistic infections, including fungal (cryptococcosis, pneumocystis pneumonia, and histoplasmosis), viral (herpes simplex virus, herpes zoster virus, Epstein-Barr virus, hepatitis B and C), and parasitic infections (strongyloidiasis and toxoplasmosis), are associated with IRIS [12]. Importantly, malignancies such as Kaposi's sarcoma, Hodgkin's lymphoma, and non-Hodgkin 's lymphoma have also been associated with IRIS [9,13].

The incidence of IRIS in HIV patients is closely associated with the degree of immunosuppression at baseline (CD4 cell count <50c/ml), and the presence of a high pathogen burden at the time of ART initiation [7]. Other risk factors include starting ART close to the treatment of opportunistic infections, young age, male sex, and genetic predisposition [10]. IRIS usually presents within the first 4–8 weeks after ART initiation but has also been reported to occur many weeks later [11].

Although IRIS represents a common clinical entity, IRIS poses significant diagnostic and therapeutic challenges due to its broad clinical spectrum, which can mimic the progression of the underlying infection, antibiotic resistance to the detected pathogen, drug toxicity, or malcompliance. The clinical course of IRIS can range from mild, self-limiting symptoms to severe, life-threatening complications, depending on the organ involved and the underlying pathogen. The diagnostic process is further hampered by the lack of a universal definition or consensus on diagnostic criteria. However, IRIS should be considered if the following criteria are met: i) sudden deterioration of symptoms after an initial good response to therapy, ii) exclusion of conditions that reduce the efficacy of therapy (i.e., drug malcompliance or resistance), and iii) exclusion of other possible causes of clinical deterioration (i.e., opportunistic infections) [7,8,10].

Here, we describe a case series of three patients with IRIS. The first case involved paradoxical IRIS in an HIV-positive patient with TB, where the re-initiation of ART led to an inflammatory response despite effective anti-tuberculous treatment (ATT) [20]. The second case highlights unmasking IRIS in an HIV-positive patient, where ART initiation revealed an underlying Epstein-Barr virus (EBV)-associated B-cell lymphoma, presenting as a central nervous system (CNS) lesion [21]. The third case describes the paradoxical worsening of pulmonary TB in an HIV-negative patient, expanding the scope of IRIS beyond its conventional association with HIV infection.

These cases illustrate the various manifestations of IRIS and emphasize the need for timely diagnosis and appropriate management strategies to mitigate potentially severe outcomes associated with this syndrome. Our report highlights the challenges faced in the diagnosis of IRIS, which impede the prompt onset of therapy.

2. Case presentation

2.1. Case 1) Paradoxical IRIS in an HIV positive patient with M. Tuberculosis infection

A 31-year-old female refugee from the Ivory Coast was admitted to our hospital with a two-week history of fever, progressive cough, dyspnea, and hemoptysis.

Pulmonary TB and co-infection with HIV were diagnosed 8 months before (CD4-cell count 9/μL; HI-viral load 192′000/ml). Intensive phase anti-tuberculous treatment (ATT) with isoniazid, rifampicin, pyrazinamide, ethambutol, and ART (tenofovir, emtricitabine, dolutegravir) was initiated, resulting in sputum conversion and normalization of chest X-ray. Upon initiation of the

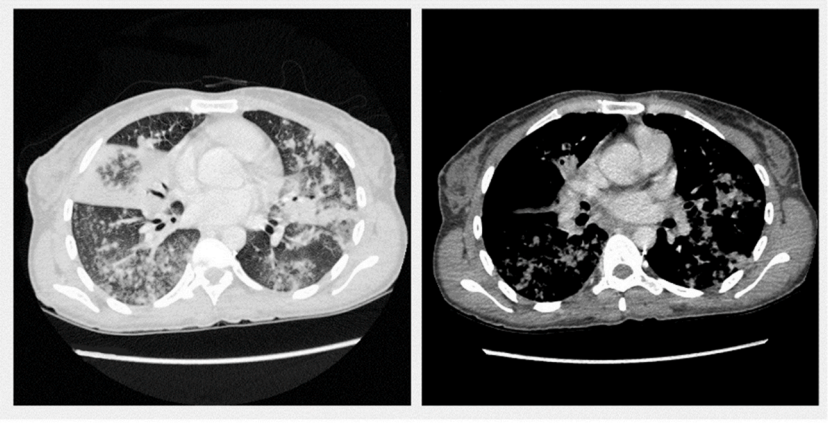


Fig. 1. ***case 1*:** CT of the chest before therapy (left) revealing bilateral consolidations, bilateral mediastinal and hilar lymphadenopathy, and middle lobe atelectasis. CT of the chest after therapy (right) with resolution of pulmonary infiltrates.

continuation phase ATT (isoniazid and rifampicin), the patient was lost to follow-up, and treatment was interrupted. Six months later, the patient was readmitted to our hospital. Sputum smears were positive for acid-fast bacilli (AFB), and in turn, both standard ATT and ART were re-initiated. Mycobacterial culture results were negative. The patient was discharged in good health with direct observed therapy (DOT).

Upon re-admission, the patient presented with fever, cough, hemoptysis, and papulopustular skin rash. Laboratory results showed a normal white blood cell count (Leukocytes 6.0 x 10^9/L), eosinophilia (0.9 x10^9/L,17.9 %), and elevated IgE (3141 kU/L). C-reactive protein (CRP) level was 48 mg/L (normal <5 mg/l), CD4 cell count was 134/μL, HI-viral load (HI-VL) < 40 copies/ml. Computed tomography (CT) of the lung showed bilateral consolidations, bilateral mediastinal and hilar lymphadenopathy, and middle lobe atelectasis (Fig. 1) due to a necrotic endobronchial mass (Fig. 2). Bronchoalveolar lavage (BAL) revealed a lymphocytic alveolitis (48 % lymphocytes, CD4+/CD8+ ratio 2.4) without eosinophilia. The microscopic evaluation of the specimen was negative for Pneumocystis jirovecii. Staining for AFB and PCR for M. tuberculosis were positive without detection of rifampicin resistance (Gene Xpert). The mycobacterial and bacterial cultures were negative. Endobronchial and lung biopsies by video-assisted thoracoscopy revealed granulomatous inflammation without AFB, parasites, or eosinophils. Histology (Fig. 2) was negative for lymphoma, Kaposi's sarcoma, and other malignancies. No evidence of vasculitis was found. Serological tests for parasites (Strongyloides, Toxocara, Schistosoma, Paragonimus, Filaria), Coxiella burnetii, Francisella tularensis, and Brucella were negative. Finally, the fungal cultures remained negative. Skin biopsy revealed eosinophilic folliculitis.

Paradoxical IRIS in a HIV-positive patient was hypothesized. Weight-adjusted treatment with methylprednisolone (0.5 mg/kgbw/d) was established and gradually tapered. Therapy resulted in immediate clinical improvement, with resolution of pulmonary infiltrates and a decrease of the endobronchial tumor (Fig. 1). Eosinophils normalized upon cessation of Cotrimoxazol.

2.2. Case 2) unmasking IRIS in an HIV positive Patient

A 63-year-old female from South Africa was admitted to our hospital with new-onset right-dominant tetraparesis. Two months prior, she had been diagnosed with HIV-Infection CDC Stadium C3 (CD4 cell count <22/μl; HI-VL of 1,740,000 c/ml) with HIV encephalopathy and wasting syndrome. Initial cMRI revealed mild central cerebral atrophy and microangiopathic alterations (Fig. 3). Cerebrospinal fluid (CSF) examination was remarkable for a slight barrier dysfunction and an intrathecal HI-VL of 121′000 c/ml, microbiological examination was normal without any signs of an acute infection. ART (bictegravir, emtricitabine, tenofovir-alafenamid) was started, and the patient was discharged in a stable condition.

Upon presentation, laboratory studies revealed leukopenia (2.18 10^9/l, range 3.9–10.2 10^9/l), CRP 20 mg/l (<5 mg/l), CD4 cell count 39/μl and HI-VL of 875/ml. cMRI showed a ring-enhancing lesion in the left frontal cortex with surrounding edema (Fig. 3). Examination of the cerebrospinal fluid revealed an elevated protein level of 1126 mg/l but a normal cell count (3 mononuclear cells/μL). PCR results for John Cunningham virus (JC), cytomegalovirus (CMV), Mycobacterium tuberculosis, and serologies for Toxoplasma (T. gondii IgG) were negative. The cultures were negative for Cryptococcus spp. Finally, the intrathecal PCR for Epstein-Barr virus (EBV) was positive. Brain biopsy and histological workup, including immunohistochemistry staining, showed prominent B-cell proliferation with expression of the oncogene latent membrane protein 1 (LMP-1) (Fig. 4), confirming the diagnosis of cerebral EBV-associated B-cell lymphoma. After a thorough review of the current literature, unmasking of IRIS was hypothesized. Treatment with methylprednisolone resulted in a slight improvement of symptoms. However, the patient refused further treatment and decided to follow a palliative care setting.

2.3. Case 3) Paradoxical worsening of M. tuberculosis infection in an HIV negative patient

A 51-year-old male construction worker from Gambia was admitted to our hospital with fever, progressive weight loss, cough, and

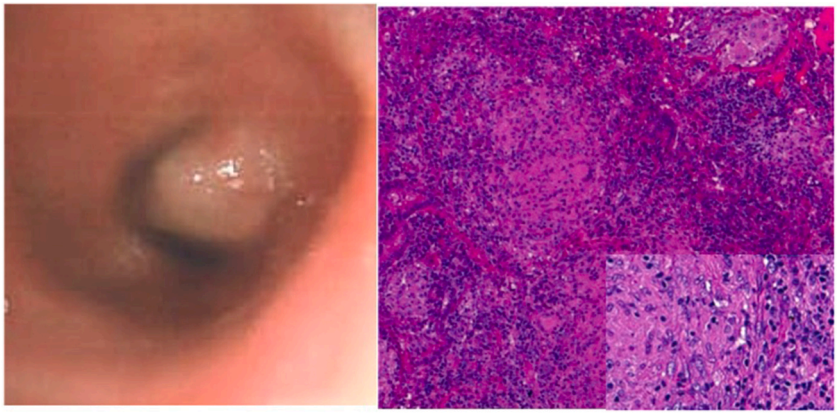


Fig. 2. ***case 1:*** Bronchoscopy (left) revealing an endobronchial mass in the middle lobe bronchus. Endobronchial biopsies (H&E) revealing granulomatous inflammation (right).

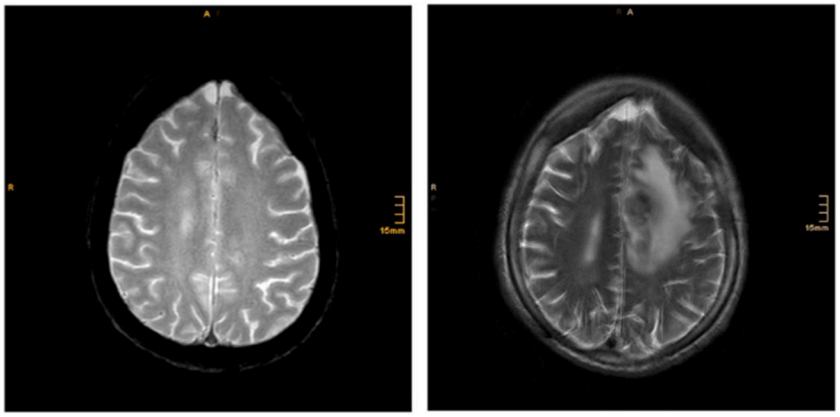


Fig. 3. ***case 2:*** T2-weighted MRI scan from August 2020 (left) and upon presentation in November 2020 (right), the latter showing a ring-enhancing lesion in the left frontal cortex.

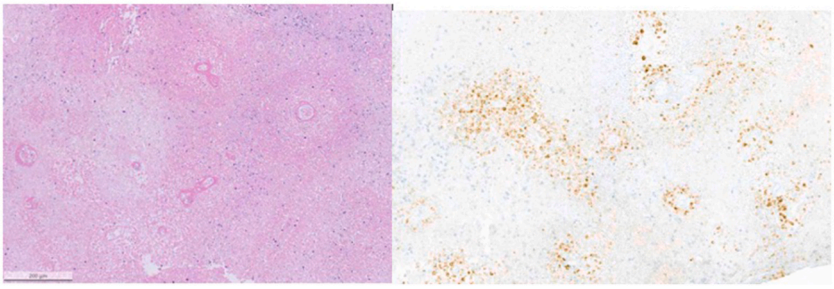


Fig. 4. ***case 2*****:** Brain biopsy with immune histochemistry staining in H&E on the left, and prominent B-cell proliferation and expression of the oncogene latent membrane protein 1 (LMP-1) on the right.

hemoptysis. Computed tomography (CT) of the chest revealed pathognomonic cavitary findings (Fig. 5) for pulmonary tuberculosis. Sputum smears were positive for AFB, and cultures grew Mycobacterium africanum, susceptible to standard ATT. CRP was elevated (187 mg/L; normal <5 mg/l), and leukocyte levels were normal (7.9 x10^9/L). The HIV screening results were negative. ATT was established (isoniazid, rifampicin, pyrazinamide, and ethambutol), and the patient was discharged in good condition with directly observed therapy. Three weeks later, the patient developed a sudden worsening of symptoms with fever and elevated inflammatory markers (CRP 294 mg/L, leucocytes 14.1 x 10^9/L). Computed tomography (CT) of the chest and abdomen revealed normal findings. Serology for EBV, CMV, Syphilis and Toxoplasmosis, as well as microscopy for Malaria, Screening test for HIV, and nasopharyngeal swabs for SARS-CoV-2 and Influenza were negative. Sputum and blood cultures were negative. The ATT drug levels were within the therapeutic range, and there was no evidence of adverse drug reaction/drug fever.

After the exclusion of other conditions explaining the clinical deterioration, a paradoxical upgrade reaction/IRIS was hypothesized. Weight-adjusted treatment with methylprednisolone resulted in the prompt resolution of the symptoms.

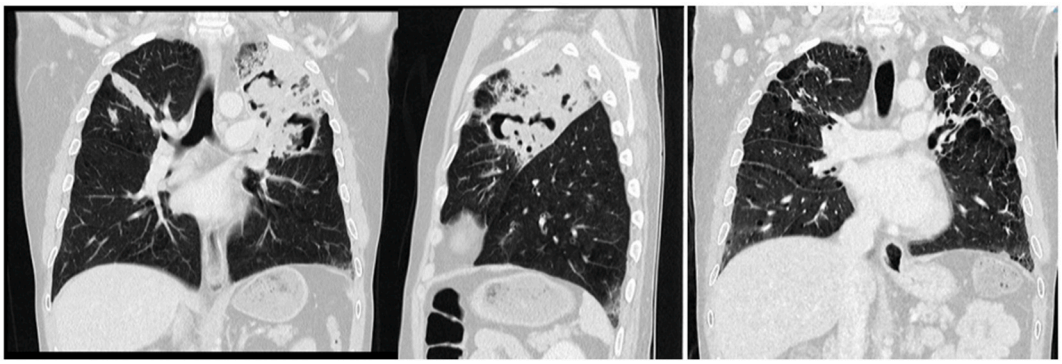


Fig. 5. ***case 3:*** CT scan of the chest with pathognomonic findings before (left and middle) and after (right) therapy.

3. Discussion

This case series highlights different clinical manifestations in patients with IRIS. Importantly, IRIS is a diagnosis of exclusion; there is no diagnostic test for IRIS, and the differential diagnosis is broad. The clinical spectrum depends on the underlying infection and host factors, particularly the degree of immunosuppression. There are two clinical scenarios to distinguish: the new presentation of a previously subclinical or occult disease (unmasking IRIS) and the deterioration of an already treated disease (paradoxical IRIS) [6–9]. Here, we discuss the similarities, differences, and clinical implications of the three cases of IRIS, emphasizing the diagnostic challenges that impede prompt onset of therapy.

The paradoxical IRIS in a 31-year-old HIV-positive patient with pulmonary TB (*Case 1*) illustrates a typical scenario in which re-initiation of ART led to an inflammatory response against residual Mycobacterium tuberculosis. The patient had a dramatic presentation that correlated with reconstitution of the immune system. Significant eosinophilia led to an extensive evaluation of possible differential diagnoses, thereby delaying the initiation of therapy. Finally, the diagnosis of IRIS was supported by the typical timeframe of clinical deterioration following ART initiation, exclusion of alternative diagnoses, and response to corticosteroids.

The unmasking IRIS in a 63-year-old HIV-positive patient presented with a neurological deficit and a new-onset brain lesion, which was later identified as EBV-associated cerebral B-cell lymphoma (*Case 2)*. Unlike paradoxical IRIS, unmasking IRIS reveals a previously undiagnosed infection or malignancy that emerges as the immune function improves. Delayed improvement in immune function unmasked the underlying CNS lymphoma, a rare but severe form of IRIS, highlighting the importance of considering malignancy in the differential diagnosis [9,13].

In contrast, the third case involved a 51-year-old HIV-negative patient with tuberculosis who developed paradoxical worsening of symptoms after the initiation of ATT. Although IRIS is commonly associated with HIV, this case underscores the fact that paradoxical reactions can occur even in the absence of HIV. Sudden clinical deterioration, along with the absence of alternative explanations, supported the diagnosis of TB-associated paradoxical IRIS, which responded well to corticosteroid therapy.

The pathophysiology of IRIS involves restoration of pathogen-specific immune responses after immune suppression, leading to an exaggerated inflammatory response. In HIV-positive patients, the rapid reduction in viral load and partial recovery of CD4 cell counts play a crucial role in this process. The common risk factors in these cases include severe immunosuppression at baseline (CD4counts $<50/\mu L$) and a high pathogen burden [11]. *Cases 1 and 2* had a very low baseline CD4 cell count ($<22/\mu L$) and high HI-VL, which predisposed them to IRIS after ART initiation. Although HIV-negative, the patient with tuberculosis in *Case 3* presented a paradoxical upgrade reaction/IRIS, demonstrating that severe infections, such as TB alone, can trigger similar immune dysregulation during therapy.

Although mild courses of paradoxical IRIS can be managed with supportive therapy and nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids remain the mainstay of treatment for moderate to severe IRIS, especially when the inflammatory response leads to significant morbidity [14–16]. TNF-α antagonists can be used as salvage or corticosteroid-sparing treatments in HIV-negative patients [18,19]. Management should further comprise specific therapy for the underlying infection. ART should generally not be interrupted, as it increases the risk of acquiring new opportunistic infections and recurrence of IRIS when therapy is later restarted [17]. Both paradoxical IRIS cases presented here (*Case 1 and 3)* responded well to corticosteroids, with significant clinical improvement and symptom resolution.

4. Conclusion

This case series emphasizes the importance of early recognition and diagnosis of IRIS, particularly in settings of severe immunosuppression and a high pathogen burden. In HIV-positive patients, close monitoring after ART initiation is essential to identify early signs of IRIS, especially in those with low CD4 cell counts. Differential diagnosis of IRIS should include infectious, inflammatory, and neoplastic processes, as illustrated by the diverse presentations in these cases. Notably, IRIS is not exclusive to HIV-positive patients. Management of IRIS requires a multidisciplinary approach that balances the control of inflammation with the treatment of underlying infections. Timely intervention with corticosteroids can significantly improve paradoxical IRIS outcomes. Efforts should be made to refine diagnostic criteria and therapeutic protocols to better address the diverse manifestations of IRIS across various clinical settings.

CRediT authorship contribution statement

Sebastian Schregenberger: Writing – review & editing, Writing – original draft, Investigation, Conceptualization. **Vera Graup:** Writing – original draft. **Adrian Schibli:** Writing – review & editing. **Benjamin Preiswerk:** Writing – review & editing. **Irène Laube:** Writing – review & editing. **Lars C. Huber:** Writing – review & editing, Supervision. **Melina Stüssi-Helbling:** Supervision, Investigation, Conceptualization.

Statement of ethics/written informed consent for publication

The patients provided written informed consent for the publication of details of their medical cases and any accompanying images. This retrospective review of patient data did not require ethical approval in accordance with the local and national guidelines.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

Case 1 and Case 2 were previously presented as poster presentations at the Spring Congress of the Swiss Society of General Internal Medicine (SGAIM) in 2019 and 2021, respectively, and were published as abstracts in the congress' journal. They have been appropriately referenced in the text.

References

[1] H.H. Hirsch, G. Kaufmann, P. Sendi, M. Battegay, Immune reconstitution in HIV-infected patients, Clin. Infect. Dis. 38 (2004) 1159–1166, https://doi.org/10.1086/383034.
[2] S.A. Shelburne, R.J. Hamill, M.C. Rodriguez-Barradas, S.B. Greenberg, R.L. Atmar, D.W. Musher, et al., Immune reconstitution inflammatory syndrome: emergence of a unique syndrome during highly active antiretroviral therapy, Medicine 81 (2002) 213–227, https://doi.org/10.1097/00005792-200205000-00005.
[3] P.E. Namale, L.H. Abdullahi, S. Fine, M. Kamkuemah, R.J. Wilkinson, G. Meintjes, Paradoxical TB-IRIS in HIV-infected adults: a systematic review and meta-analysis, Future Microbiol. 10 (2015) 1077–1099, https://doi.org/10.2217/fmb.15.9.
[4] J. Cadena, G.R. Thompson, T.T. Ho, E. Medina, D.W. Hughes, T.F. Patterson, Immune reconstitution inflammatory syndrome after cessation of the tumor necrosis factor alpha blocker adalimumab in cryptococcal pneumonia, Diagn. Microbiol. Infect. Dis. 64 (3) (2009) 327–330, https://doi.org/10.1016/j.diagmicrobio.2009.03.019.
[5] H.Y. Sun, B.D. Alexander, S. Huprikar, G.N. Forrest, D. Bruno, G.M. Lyon, et al., Predictors of immune reconstitution syndrome in organ transplant recipients with cryptococcosis: implications for the management of immunosuppression, Clin. Infect. Dis. 60 (1) (2015) 36–44, https://doi.org/10.1093/cid/ciu711.
[6] S.L. Cheng, H.C. Wang, P.C. Yang, Paradoxical response during anti-tuberculosis treatment in HIV-negative patients with pulmonary tuberculosis, Int. J. Tubercul. Lung Dis. 11 (2007) 1290–1295.
[7] M.A. French, P. Price, S.F. Stone, Immune restoration disease after antiretroviral therapy, AIDS 18 (2004) 1615–1627, https://doi.org/10.1097/01.aids.0000131375.21070.06.
[8] G. Meintjes, S.D. Lawn, F. Scano, G. Maartens, M.A. French, W. Worodria, et al., Tuberculosis-associated immune reconstitution inflammatory syndrome: case definitions for use in resource-limited settings, Lancet Infect. Dis. 8 (2008) 516–523.
[9] E. Letang, J.J. Lewis, M. Bower, A. Mosam, M. Borok, T.B. Campbell, et al., Immune reconstitution inflammatory syndrome associated with Kaposi sarcoma: higher incidence and mortality in Africa than in the UK, AIDS 27 (10) (2013) 1603–1613, https://doi.org/10.1097/QAD.0b013e328360a5a1.
[10] M. Müller, S. Wandel, R. Colebunders, S. Attia, H. Furrer, M. Egger, IeDEA Southern and Central Africa. Immune reconstitution inflammatory syndrome in patients starting antiretroviral therapy for HIV infection: a systematic review and meta-analysis, Lancet Infect. Dis. 10 (4) (2010) 251–261, https://doi.org/10.1016/S1473-3099(10)70026-8.
[11] W. Worodria, J. Menten, M. Massinga-Loembe, D. Mazakpwe, D. Bagenda, O. Koole, et al., Clinical spectrum, risk factors and outcome of immune reconstitution inflammatory syndrome in patients with tuberculosis-HIV coinfection, Antivir. Ther. 17 (2012) 841–848, https://doi.org/10.3851/IMP2108.
[12] M. Lanzafame, S. Vento, Tuberculosis-immune reconstitution inflammatory syndrome, J Clin Tuberc Other Mycobact Dis 3 (2016) 6–9, https://doi.org/10.1016/j.jctube.2016.03.002.
[13] S. Gopal, M.R. Patel, C.J. Achenbach, E.L. Yanik, S.R. Cole, S. Napravnik, et al., Lymphoma immune reconstitution inflammatory syndrome in the center for AIDS research network of integrated clinical systems cohort, Clin. Infect. Dis. 59 (2) (2014) 279–286, https://doi.org/10.1093/cid/ciu270.
[14] F.X. Blanc, T. Sok, D. Laureillard, L. Borand, C. Rekacewicz, E. Nerrienet, et al., Earlier versus later start of antiretroviral therapy in HIV-infected adults with tuberculosis, N. Engl. J. Med. 365 (2011) 1471–1481, https://doi.org/10.1056/NEJMoa1013911.
[15] L.C.K. Bell, R. Breen, R.F. Miller, M. Noursadeghi, M. Lipman, Paradoxical reactions and immune reconstitution inflammatory syndrome in tuberculosis, Int. J. Infect. Dis. 32 (2015) 39–45, https://doi.org/10.1016/j.ijid.2014.12.030.
[16] O.A. Uthman, C. Okwundu, K. Gbenga, J. Volmink, D. Dowdy, A. Zumla, et al., Optimal timing of antiretroviral therapy initiation for HIV-infected adults with newly diagnosed pulmonary tuberculosis, Ann. Intern. Med. 163 (2015) 32–39, https://doi.org/10.7326/M14-2979.
[17] World Health Organization, Guidelines: Updated Recommendations on HIV Prevention, Infant Diagnosis, Antiretroviral Initiation and Monitoring, World Health Organization, 2021. Available from: https://iris.who.int/handle/10665/340190.
[18] L. Armange, A. Lacroix, P. Petitgas, C. Arvieux, C. Piau-Couapel, P. Poubeau, M. Revest, P. Tattevin, The use of TNF-α antagonists in tuberculosis to control severe paradoxical reaction or immune reconstitution inflammatory syndrome: a case series and literature review, Eur. J. Clin. Microbiol. Infect. Dis. 42 (4) (2023) 413–422, https://doi.org/10.1007/s10096-023-04564-2.
[19] B.J. Marais, E. Cheong, S. Fernando, S. Daniel, M.R. Watts, L.J. Berglund, S.E. Barry, G. Kotsiou, A.P. Headley, R.A. Stapledon, Use of infliximab to treat paradoxical tuberculous meningitis reactions, Open Forum Infect. Dis. 8 (1) (2020) ofaa604, https://doi.org/10.1093/ofid/ofaa604.
[20] Swiss Society of General Internal Medicine, Abstracts (2019). Available from: https://www.sgaim.ch/fileadmin/user_upload/Adaptionen/Congress/BilderHighres/Bilder_FK_2019/Abstracts_2019.pdf.
[21] Swiss Society of General Internal Medicine, Abstracts (2021). Available from: https://www.sgaim.ch/fileadmin/user_upload/Ad.