Restarting Biologics and Management of Patients with Flares of Inflammatory Rheumatic Disorders or Psoriasis During Active Tuberculosis Treatment

Fabrizio Cantini, Francesca Prignano, and Delia Goletti

ABSTRACT. Our aim was to review the evidence concerning optimal timing for restarting biologics in patients with active tuberculosis (TB), and the management of relapsing rheumatoid arthritis (RA), psoriatic arthritis (PsA), ankylosing spondylitis (AS), and psoriasis during treatment for TB. Few or no indications are available for 2 important challenges for clinicians: the timing for restarting biologics in patients with TB reactivation and the management of the underlying disorder. In the absence of clear evidence, guidelines and experts suggest restarting anti-tumor necrosis factor-α (TNF-α) agents after completion of an active TB therapy course, but no indications are available on the appropriate management of patients with flares of underlying rheumatic disease or psoriasis. Among anti-TNF-α agents, etanercept is associated with the lowest risk of TB reactivation, and non-anti-TNF-α biologics and several nonbiologic drugs are associated with low/no risk of TB reactivation. Therefore, for patients with relapsing RA, PsA, AS, or psoriasis during TB treatment we propose a therapeutic schedule modulated by disease activity and individual single drug-related TB risk. (J Rheumatol Suppl. 2014 May; 91:78–82; doi:10.3899/jrheum.140106)

Key Indexing Terms:
ANTI-TUMOR NECROSIS FACTOR ACTIVE TUBERCULOSIS BIOLOGICS
DISEASE FLARE DISEASE ACTIVITY

The incidence of tuberculosis (TB) varies greatly around the world, with the highest risk of infection in Southeast Asia (35%), Africa (30%), and the Western Pacific regions (21%)[1]. Among those infected by *Mycobacterium tuberculosis* (Mtb), only about 5–10% develop active disease during their lifetime. Because the host immune response is able to inhibit replication of Mtb, most patients carry latent TB infection (LTBI), remaining asymptomatic and non-infectious[2]. However, when the immune system is impaired, such as during treatment with anti-tumor necrosis factor-α (TNF-α) blockers, patients with LTBI are at increased risk of developing active TB, and investigations to detect LTBI have been recommended[3].

LTBI is defined as a positive tuberculin skin test or a positive interferon-γ release assay, without evidence of active TB based on symptoms (such as fever and cough), chest radiography, and microbiological isolation of Mtb[4,5,6]. Although these recommendations have been demonstrated to be effective in active TB prevention[7], some cases of TB still occur in patients receiving biologics, often due to defective adherence to the recommended LTBI screening procedures[8]. In the case of active TB, the appropriate management of the underlying rheumatic disease or psoriasis during the TB therapy, and when to restart the biologic are major concerns for clinicians. The aim of this report is to review the recommended interval for restarting biologics in patients with active TB complicating the underlying disease course and to propose a disease activity-adjusted therapeutic intervention for rheumatoid arthritis (RA), psoriatic arthritis (PsA), ankylosing spondylitis (AS), and psoriasis during the TB therapy.

Literature Review

LTBI detection and TB reactivation are important issues in patients exposed to biologic agents, particularly in those receiving anti-TNF-α agents, because of the pivotal role exerted by TNF in Mtb growth inhibition and granuloma formation[9,10,11]. Data from the literature confirm that the relative risk of TB reactivation in patients treated with anti-TNF-α is 1.6–25.1 times higher than in the general population, with the risk associated with monoclonal antibodies anti-TNF-α adalimumab (ADA) and infliximab (IFX) being higher than that associated with the soluble

From the Rheumatology Division, Hospital of Prato, Prato; Division of Clinical, Preventive, and Oncology Dermatology, Department of Surgery and Translational Medicine, Florence University, Florence; and Translational Research Unit, Department of Epidemiology and Preclinical Research, "L. Spallanzani" National Institute for Infectious Diseases (INMI), IRCCS, Rome, Italy.

F. Cantini, MD, PhD, Consultant in Rheumatology, Director, Rheumatology Division, Hospital of Prato; F. Prignano, MD, PhD, Division of Clinical, Preventive, and Oncology Dermatology, Department of Surgery and Translational Medicine, Florence University; D. Goletti, MD, PhD, Translational Research Unit, Department of Epidemiology and Preclinical Research, "L. Spallanzani" INMI, IRCCS.

Address correspondence to Dr. Cantini, Rheumatology Division, Hospital of Prato, Piazza Ospedale 1, 59100 Prato, Italy.
E-mail: fbrzcantini@gmail.com

receptor etanercept (ETN)[12,13,14]. ETN, ADA, and IFX have been licensed for the treatment of dermatologic and rheumatologic diseases including psoriasis, RA, psoriatic arthritis, and AS, while only IFX and ADA have obtained approval for treatment of inflammatory bowel diseases. Moreover, additional biologics are currently used in rheumatology including the 2 new anti-TNF-α drugs, certolizumab pegol (CTP) and golimumab (GOL), anti-IL-1 anakinra (ANK), anti-IL-6 tocilizumab (TCZ), anti-CD20 rituximab (RTX), and anti-CD28 abatacept (ABA); and in dermatology anti-interleukin (IL) 12/23 ustekinumab (UTK) has been licensed for the treatment of psoriasis over the past few years. Owing to their recent approval, the TB risk associated with CTP and GOL has not been fully investigated, whereas no increased TB reactivation risk has been observed in clinical trials and national registries of ANK, TCZ, RTX, ABA, and UTK[15,16,17,18,19,20]. It should be pointed out that psoriasis and RA, and concomitant nonbiologic therapies, contribute to increase the TB risk[21,22].

As regards the traditional nonbiologic therapy, in a recent report from Canada 50 cases of active TB were recorded in a large cohort of 24,282 patients with RA followed up for 23 years, demonstrating an adjusted relative risk associated with all nonbiologic immunosuppressive therapy significantly higher than in the general population[22]. The highest risk of 11.7 was recorded for leflunomide, followed by 3.8 for cyclosporine, 3.4 for methotrexate, and 2.5 for corticosteroids, while a very low risk was associated with hydroxychloroquine (HCQ), sulfasalazine (SSZ), and azathioprine. Confirming other studies[23,24], also in the Canadian study RA in itself was demonstrated to increase the risk of TB reactivation.

As recommended by all published guidelines/recommendations[4,5,6,7,25-36], if active TB disease occurs, TNF-α inhibitors should be discontinued, and anti-TB therapy should be promptly started. A 2-month induction period with isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a 4-month maintenance phase with isoniazid and rifampicin are recommended therapeutic strategies for active TB treatment[37].

However, whatever the underlying disease, it should be kept in mind that patients initiated anti-TNF-α therapy because of severe disease not responding to traditional therapy. Therefore, clinicians do have to face 2 crucial challenges: restarting biologic therapy at TB recovery and management of patients in the event of disease flare.

Restarting Biologics in Patients with Active TB

Guidelines from the settings of both rheumatology and dermatology agree almost unanimously on restarting anti-TNF-α therapy 1 month after beginning preventive TB chemotherapy in subjects with positive LTBI; however, indications on restarting biologics after active TB recovery are quite inconsistent. In the absence of specific clinical studies, experts suggest it is preferable to restart a biologic after completion of at least a 6-month course of active TB treatment[5,31,38,39]. Nevertheless, it is unclear what to do in the case of urgent need for reinitiation of the anti-TNF-α agent, dictated by the severity of the underlying disease flare; and it is unknown whether the duration of TB treatment should be modified.

How to Manage Patients Treated for Active TB with Severe Flares of the Underlying Disease

Severe disease flares in patients being treated for active TB may represent a difficult challenge for clinicians managing RA, PsA, AS, or psoriasis. Data in the literature are lacking, and currently available recommendations do not adequately consider this issue[4,5,6,7,25-36]. To the best of our knowledge, the only report is from the French RATIO registry, in which 6 patients restarted anti-TNF-α agents while receiving chemotherapy for active TB after 2 months (1 patient), 3 months (1 patient), 7.5 months (1 patient), and 12 months (3 patients)[37,38]. No patient had further infectious complications or worsening of active TB over a mean followup of 42.7 months.

According to the guidelines for TB treatment[39,40], the use of corticosteroids (CS) during the treatment of active TB disease is considered safe, and these drugs may constitute a valid therapeutic option to treat flares of RA and PsA. However, CS should be used at the lowest possible dosage, especially if we consider that in a large case-control British study increased risk of TB was found in CS users compared to nonusers, with adjusted OR for prednisone dose (or equivalent) < 15 mg/day or > 15 mg/day of 2.8 and 7.9, respectively[41].

Because in a recent report from the Northern California Kaiser database the adjusted OR was 2.0 for monoclonal anti-TNF IFX and ADA, and 0.6 for ETN[42], in the case of a severe disease flare, restarting anti-TNF treatment would represent a reasonable and prudent option, preferably with ETN, after completion of the 2-month TB induction therapy.

A good alternative would be to treat patients with low-risk biologics such as RTX, TCZ, or ABA for RA, and UTK for psoriasis. In particular, RTX was demonstrated to be safe in the treatment of 9 patients with a previous history of TB and concomitantly receiving CS 6–10 mg/day over a mean followup of 16 months[43].

Restarting TNF inhibitors in patients with AS should be postponed as long as possible, and in any case, only after a 2-month period of TB induction therapy.

The same schedule may be suggested for patients with severe relapses of PsA; otherwise, considering its efficacy either in PsA and psoriasis, UTK may be a valid alternative[44]. Although not yet approved, ABA has also been demonstrated to be effective in PsA[45], and in selected cases an off-label use may be proposed.

Proposal for the Management of Patients with Reactivated TB and Flares of Rheumatic Diseases or Psoriasis

In patients with RA, PsA, AS, and psoriasis who develop active TB, a full course of TB therapy should be promptly initiated and biologic therapy withdrawal is mandatory. In general terms, to control signs and symptoms of the underlying disease, patients with inflammatory rheumatic disorders may be treated with analgesics, nonsteroidal antiinflammatory drugs, low-dose CS, and in more severe cases, with low-risk disease-modifying antirheumatic drugs including HCQ and SSZ in RA, and only SSZ in AS and PsA, because of the inefficacy of HCQ in these patients. Patients with psoriasis should be treated with topical therapy and phototherapy. In collaboration with local infectious disease specialists or pulmonologists, patients should undergo monthly evaluation of toxicity of TB drugs and rigorously observe the international schemes for the followup of TB (months 2 and 6)[39].

In the absence of guidelines/recommendations, as summarized in Tables 1, 2, and 3, we suggest adjusting treatment as a function of disease activity. Based on the European League Against Rheumatism 28-joint Disease Activity Score (DAS28)[46], the severity of RA and PsA can be classified into low, moderate, and high. Subjects with low disease activity can be managed with symptomatic drugs, HCQ, and SSZ, and recommencement of a biologic agent after a full course of TB therapy; those with moderate activity can receive CS, methotrexate, cyclosporine, or azathioprine after 2 months of TB induction therapy, and in the case of high disease activity, a low-risk biologic such as ANK, RTX, TCZ, or ABA can be restarted after the first 2 months of TB therapy. Because the risk of TB reactivation is lower with ETN than with other anti-TNF-α agents, this biologic agent can be used in nonresponders to low-risk biologics.

According to the AS Disease Activity Score (ASDAS)[47], patients with inactive AS can be treated with nonsteroidal antiinflammatory drugs on demand, and those with moderate activity with full doses of these drugs. In patients with high disease activity, after 2 months of TB induction therapy, SSZ can be tried. Indeed, in a head-to-head randomized controlled trial of patients with AS, although the efficacy of SSZ was significantly lower than that of ETN, 52.9% of 187 patients achieved an Assessment of SpondyloArthritis International Society criteria 20 response[48]. In the case of non-response, anti-TNF therapy can be initiated, preferably with ETN because of its lower risk of TB compared to that of the monoclonal anti-TNF agents. Finally, patients with very high disease activity can

Table 1. Management of rheumatoid arthritis and psoriatic arthritis in patients with active tuberculosis complicating the disease course.

Disease Activity	Treatment
Low (DAS28 < 3.2)	NSAID, analgesics, HCQ, SSZ Restart biologics after 6 mo therapy for active TB
Moderate (DAS28 3.3–5.0)	After 2-mo induction therapy for active TB, CS (lowest possible dose) plus MTX, CsA, AZA Restart biologics after 6 mo therapy for active TB
High (DAS28 > 5.1)	RA: after 2-mo induction therapy for active TB restart a low-risk biologic: ANK, TCZ, RTX, ABA. ETN* in intolerant or nonresponder subjects PsA: UTK§ if severe PsA; ETN* in intolerant subjects or nonresponders ABA§ in selected cases

*ETN-associated TB risk is 3–4 times lower than that associated with monoclonal anti-tumor necrosis factor agents. §UTK and ABA are not yet licensed for the treatment of PsA. NSAID: nonsteroidal antiinflammatory drugs; HCQ: hydroxychloroquine; SSZ: sulfasalazine; CS: corticosteroids; MTX: methotrexate; CsA: cyclosporine; ANK: anakinra; TCZ: tocilizumab; RTX: rituximab; ABA: abatacept; ETN: etanercept; UTK: ustekinumab; RA: rheumatoid arthritis; PsA: psoriatic arthritis; TB: tuberculosis; DAS28: 28-joint Disease Activity Score; AZA: azathioprine.

Table 2. Management of ankylosing spondylitis in patients with active tuberculosis complicating the disease course.

Disease Activity	Treatment
Inactive (ASDAS < 1.3)	NSAID on demand
Moderate (ASDAS 1.4–2.0)	NSAID at full doses
High (ASDAS 2.1–3.4)	NSAID at full doses If no response, after 2-mos TB induction therapy: -Try SSZ -If no response, restart anti-TNF, preferably ETN*
Very high (ASDAS ≥ 3.5)	After 2-mo TB induction therapy: -Restart anti-TNF, preferably ETN*

*ETN-associated TB risk is 3–4 times lower than that associated with monoclonal anti-tumor necrosis factor. ASDAS: Ankylosing Spondylitis Disease Activity Score; NSAID: nonsteroidal antiinflammatory drugs; ETN: etanercept; TB: tuberculosis; SSZ: sulfasalazine; TNF: tumor necrosis factor agent.

Table 3. Management of psoriasis in patients with active tuberculosis complicating the disease course.

Disease Activity	Treatment
Low (PASI ≤ 5)	Topical therapy
Moderate (PASI 5.1–10.0)	Topical therapy and/or phototherapy (nb-UVB, excilite)
High (PASI > 10.0)	After 2-mos therapy for active TB restart UTK. ETN* in intolerant subjects or nonresponders. ABA§ in selected cases

*ETN-associated TB risk is 3-4 times lower than that associated with monoclonal anti-tumor necrosis factor; §ABA is not yet licensed for the treatment of psoriatic arthritis. nb-UVB: narrowband ultraviolet phototherapy; ETN: etanercept; ABA: abatacept; TB: tuberculosis; PASI: Psoriasis Area and Severity Index; UTK: ustekinumab.

receive anti-TNF, preferably ETN, after 2 months of TB induction therapy.

According to the Psoriasis Activity Index Score[49], patients with psoriasis with low disease activity can be treated with topical therapy only, those with moderate skin disease with topical therapy and/or phototherapy (narrowband-UVB, excilite), while patients with severe psoriasis, after a 2-month period of TB induction therapy, can restart UTK. ETN could be an alternative in non-responders to UTK, and in selected cases, an off-label use of ABA may be an option.

After completion of TB therapy course with recovery, it is recommended that patients be educated on the symptoms and signs of active TB through written instructions. In any case, patients should be referred to an infectious diseases specialist/pulmonologist twice a year in the first 2 years following interruption of the TB therapy and instructed to contact these specialists in the case of recurrence of TB signs or symptoms.

Several differently targeted biologics are currently available for treatment of RA, PsA, AS, and psoriasis. Among these, monoclonal anti-TNF-α agents increase the risk of TB reactivation although the risk is lower with ETN. In the case of active TB occurrence, prompt interruption of biologics is recommended. The optimal timing for reinitiation of anti-TNF-α blockers is unclear, but all experts suggest postponing the reintroduction until after a full course of TB therapy.

No indications are available from the published recommendations on the management of flares of the underlying rheumatic disease or psoriasis. In the present report, we suggest a therapeutic schedule for relapsing RA, PsA, AS, and psoriasis modulated on the disease activity and on the differing TB risk associated with nonbiologic and biologic therapies.

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