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Anti-TNF Treatment and Tuberculosis

Kaya FO*
Assistant Professor, Department Internal Medicine, Medical School of University of Maltepe, Istanbul, Turkey

***Corresponding author:** Fatih Öner Kaya, Assistant Professor, Department Internal Medicine, Medical School of University of Maltepe, Istanbul, Turkey

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Introduction

Bacteria, lipopolysaccharide are largely by stimulating macrophages such as IL-2, to a lesser extent B and T lymphocytes, NK cells, mast cells and intestinal epithelial cells by production of TNF- α ; Induces acute phase reactions. Endotoxic shock, inflammation, regeneration, immunity, provides destruction apoptotic cell cytotoxicity. The development of tumours and inhibits viral replication [1-4].

TNF-α is secreted as a precursor. TNF-α converting enzyme is activated with. Three TNF-α jointly trimetric TNF-α becomes or Trimetric TNF-α TNFR-1 or by connecting to one of the TNFR-2 receptor IL-1β/6/8 increments. MCP-1/1α/2, RANTES, ICAM-1, VCAM-1 increases the cytokines. These molecules provides start and continuation of inflammation [2,3,5,6].

Creating TNF- α in the immune system against Mycobacterium granuloma tuberculosis allows the isolation of bacteria. Of activated macrophages in granulomas of epithelioid cells with TNF- α effect is transformed into cells combine and become giant cells. Suppression of the TNF- α enable the activation of tuberculosis by preventing the granulomatous inflammation [7,8,9].

Rheumatologic illnesses commonly used these drugs for the disease in countries deemed more active TBC biggest problem form [10,11].

Mycobacterium TBC infections of immune response that occurs TNF-α and p55 receptor signal is increased by raising the burden of Mycobacterium and Granuloma formation and demonstrates how to accelerate death [12]. Granuloma formation for chronic as TNF-α, IFN-γ, IL-12, IL-15 secretion and TNFR-1 and have the presence of TNFR-2 receptor. TNF-α makes the macrophages activated, dendrite cells, increasing migration of lymphocytes and bringing to maturity, inflammation causes the proliferation [13,9,14].

Anti- TNF- α drugs is 5 pcs. 3 of them infliximab, adalimumab, etanercept are older drugs and that's why we have more experience with developing TB cases .However new drugs such as Sertolizunab and Golimumab we haven't gotany experience and new experiences related to these drugs will be finalized in a few years Table 1.

In Spain around 2000 in the study increased 20 times the frequency, Infliximab and Etanercept TBC treated patients with Infliximab and Adalimumab were found to be lower than that. In 2000 and 2001, 100,000 100,000/1893/1113, 2003 de 17/100,000 cases were identified [15].

In a study published in 2004 in the USA between 1998 and 2002 was reported 25 cases and 100,000 connected to etarnecept/10 has been identified as. In this study, the doses of the drug for the first time between the detection of an active with TBC were determined as an average 11.5 months [16].

Askling and colleagues in 2005 Anti TNF-α in patients with rheumatoid arthritis drug to be kept according to 4 fold increase in Anti-TNF-α have enforced and implemented according to the generalized increase risk of patients identified TBC [17].

In Turkey the Association of Anti TNF-α drugs in Tuberculosis in patients; in 2005, in 2007, 100,000 100,000/26/25.2 has identified [18].

Hanta and colleagues in 2008, in their study of 192 patients diagnosed 3 active monitoring of TBC 3 years [19].

In 2009, James and his colleagues have shown that the patient was diagnosed with active 6 pieces TBC 702. [20].

In the UK in 2008 10.712 RA patients all Anti TNF-αlar for 100,000/95 (Adalimumab 144, infliksamib 136, etarnecept 39) has been identified as [21].

RATIO (French Research Axed onTolerance of Biotherapies) in 2003 for all Anti TNF-α s 100.000/116,7 (Adalimumab 215, infliksamib 187,5, etarnecept 9.3) identified as [22].

The reason of these differences; Adalimumab and

Table 1: Anti TNF-α drugs.

	İnfliksimab	Adalimumab	Etanersept	Sertolizumab	Golimumab
Class	Monoklonal Ac	Monoklonal Ac	Fc fuzyonprot	Monoklonal Ac fragman	Monoklonal Ac
Structure	Mouse / human chimeric IgG1κ	Human IgG1κ	Human sTNFR2-Fc	PEG-Human IgG1κ Fab	Human IgG1κ
Specificity	TNF	TNF	TNF/LTα	TNF	TNF
TNF ligantı	sTNF, tmTNF	sTNF, tmTNF	sTNF, tmTNF	sTNF, tmTNF	sTNF, tmTNF
LT ligantı			LTα2β1, LTα3		
sTNF	**medium**	**medium**	**strong**	**no data**	**no data**
tmTNF	**strong**	**strong**	**medium**	**no data**	**no data**

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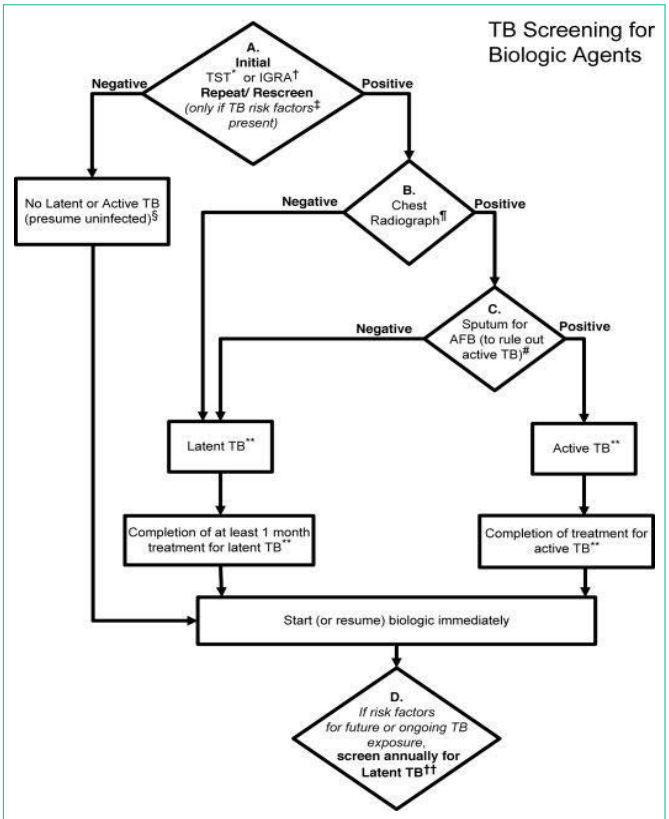

Figure 1: 2012 American College of Rheumatology recommendations update for Tuberculosis (TB) screening with biologic agent use. Depending on a patient's current therapy, the management may begin at an appropriate rectangle in the figure, rather than only at the top of the figure. The level of evidence supporting each recommendation for TB reactivation was "C" except for initiation of biologic agents in patients being treated for latent TB infection, where the level of evidence was "B."

*Anergy panel testing is not recommended.

†Interferon-γ–release assay (IGRA) is preferred if the patient has a history of BCG vaccination.

‡Risk factors for TB exposure are defined based on a publication from the US Centers for Disease Control and Prevention as: close contacts of persons known or suspected to have active TB; foreign-born persons from areas that have a high incidence of active TB (e.g., Africa, Asia, Eastern Europe, Latin America, and Russia); persons who visit areas with a high prevalence of active TB, especially if visits are frequent or prolonged; residents and employees of congregate settings whose clients are at an increased risk for active TB (e.g., correctional facilities, long-term care facilities, and homeless shelters); health care workers who serve clients who are at an increased risk for active TB; populations defined locally as having an increased incidence of latent Mycobacterium tuberculosis infection or active TB, possibly including medically underserved, low-income populations, or persons who abuse drugs or alcohol; and infants, children, and adolescents exposed to adults who are at an increased risk for latent M tuberculosis infection or active TB

§If the patient is immunosuppressed and false-negative results are more likely, consider repeating screening test(s) with Tuberculin Skin Test (TST) or IGRA.

¶Chest radiograph may also be considered when clinically indicated in patients with risk factors, even with a negative repeat TST or IGRA.

#Obtain respiratory (e.g., sputum, bronchoalveolar lavage fluid) or other samples as clinically appropriate for Acid-Fast Bacilli (AFB) smear and culture and consider referral to a TB specialist for further evaluation and treatment.

**In a patient diagnosed with latent or active TB, consider referral to a specialist for the recommended treatment.

††Patients who test positive for TST or IGRA at baseline often remain positive for these tests even after successful treatment of TB. These patients need monitoring for clinical signs and symptoms of recurrent TB disease, since repeating tests will not allow help in diagnosis of recurrent TB.

liksamibpatogenematurasyonfagosom in the inf inhibit the TBC, CD4 T-cell complement-dependent sitotoksiste and INF-γ inhibits the release [23].

All patients should be screened for latent infection is TBC. In The Scan; the medical history, physical examination, the Tuberculin Skin Test (TDT), chest radiography, socio-economic level, living environment and should be treated in the country where the frequency of TBC. The scans found as new TBC Delayed Gamma İnterferon, which measures the response against INF-γ Release Assay (IGRA) is available. Quentiferon TB-Gold test Myocabacterium TBC UN ESA-6, CFT-10 against antigens such as developing INF-γ level gauge. Quentiferon TB-Gold test, Non TBC does not cross react with Mycobacteria and BCG, booster effect is not visible, repeatable, as soon as there are advantages to be the answer [24].

Gardam and his friends if you have migrated in FROM AC chart in patients with active or if you have contact with TBC infected with TDT's 0-4 mm, latent TBC have proposed to start in terms of prophylactic therapy. Epidemiological risk factors in patients with 5-9 mm TDT agreed as positive. The rest of the Group of 10 mm and above are considered to be positive [7].

USA Breast Association guidelines in patients with positive 5 mm or above immunosuppressive agreed [25].

In Turkey, Rheumatologic research and Education Association, Turkish Thoracic Society has developed a guide with you. Accordingly, the TDT 0-4 mm resulting in 1-3 weeks after the booster dose in the question be repeated, with the repetition of 5 mm and above if there are Anti TNF-α drugs 1 months ago start and at least 9 months of INH 300 mg/day to proceed, it is suggested to use. Occurrences of 0-4 mm turns out to do the treatment but the physician's patient, according to the State risk set for deciding on an individual basis, provided that they can give the decision of prophylaxis. Active TBC is detected Anti TNF-α therapy should be immediately cut to begin treating TBC. Clinician Anti TNF therapy should consider also the possibility of extra pulmonary during TBC (Figure 1).

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