

Tuberculosis - Minimising the risk of Tuberculosis in patients starting Anti TNF Inhibitors

Summary Guideline to reduce the risk of Tuberculosis disease in patients starting anti-TNF Inhibitors

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TUBERCULOSIS – MINIMISING THE RISK OF TUBERCULOSIS IN PATIENTS STARTING ANTI TNF INHIBITORS

This Guideline is to be read in conjunction with the following Policy Directives:

PD2005_071 *Chemotherapy*

PD2005_072 *Preventive therapy*

PD2008_019 *Tuberculosis - Principles for management of people with Tuberculosis in NSW*

PD2008_016 *Tuberculin Skin Testing*

Introduction

Tumor necrosis factor (TNF) is a proinflammatory cytokine which has a pivotal role in the pathogenesis of several autoimmune diseases, including rheumatoid arthritis and other inflammatory joint disease, psoriasis, and inflammatory bowel disease.

Three anti-TNF α agents are now available in Australia (infliximab, etanercept, and adalimumab) to treat selected autoimmune diseases. However, TNF α is a significant component of the human immune response to infectionⁱ, and treatment with anti-TNF α agents is associated with an increased risk of infection. The development of active Tuberculosis (TB) disease has occurred in some patients who have received anti-TNF α therapy in countries with high TB prevalenceⁱⁱ.

The following guidelines have been developed to reduce the risk of active TB developing in patients receiving anti-TNF α therapy.

Before starting ANTI-TNF α inhibitors all patients should have:

1. A careful review of their history of exposure to TB, and an assessment to exclude active TB
2. A baseline Tuberculin Skin Test for evidence of TB infection^a
3. A Chest X ray to exclude active TB and assess evidence of past or current TB

Latent Tuberculosis

Patients with evidence of latent tuberculosis infection (LTBI) who have not previously received effective treatment for TB and in whom active TB is excluded should be treated with isoniazid (5mg/kg to maximum of 300 mg/day) and pyridoxine (25mg/day) for a period of 9 monthsⁱⁱⁱ. The first month of isoniazid treatment should be completed prior to starting an anti-TNF α inhibitor. Evidence of LTBI may include:

^a Footnote: While a positive IGRA is good evidence for Latent TB infection a negative IGRA may not exclude TB. Careful consideration must always be used when interpreting TB screening results.

1. TST \geq 5 mm
2. Radiological evidence of past TB

Patients with chest x-ray abnormalities, cough or other clinical features suggestive of active TB should have sputum examined for AFBs before commencing treatment with isoniazid. Those found to have active TB should have full treatment as outlined in Policy Directive PD2005_071 *TB Chemotherapy*.

Physicians should include the risk of potential adverse effects of isoniazid therapy in their assessment of the overall risk of commencing treatment with an anti-TNF α inhibitor.

Monitoring of isoniazid therapy, patients on isoniazid preventive therapy should have monthly assessment of:

- their hepatic function
- their compliance with the prescribed medication, and
- the development of TB.

Treatment of TB

Where active TB is diagnosed in a person receiving anti-TNF α therapy:

- cease anti-TNF α inhibitor
- reduce other immunosuppressants to lowest possible effective dose.

Patients with active TB should be referred to a NSW Chest Clinic for management and treatment of TB. Treatment should be in accordance with NSW Health TB treatment guidelines, see Policy Directive PD2005_071 *TB Chemotherapy*.

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References

1. Atkinson YH et al (1988) Recombinant human tumour necrosis factor-alpha. Regulation of N-formylmethionyl-leucylphenylalanine receptor affinity and function in human neutrophils. *J Clin Invest*, 81; 759 – 765
2. Gomez-Reino JJ et al (2003) Treatment of rheumatoid arthritis with tumour necrosis factors inhibitors may predispose to significant increase in tuberculosis risk. *Arthritis & Rheumatism*, 48; 2122 – 2127
3. Carmona L et al (2005) Effectiveness of recommendations to prevent reactivation of latent tuberculosis infection in patients treated with tumour necrosis factors antagonists. *Arthritis & Rheumatism*, 52; 1766 - 1772