

Lay Title: Starting anti-TNF α therapy does not significantly increase the risk of demyelinating events: results from the BSRBR-RA

Full Title: Demyelinating events following initiation of anti-TNF α therapy in the British Society for Rheumatology Biologics Registry in Rheumatoid Arthritis (BSRBR-RA)

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What was already known?

Anti-tumour necrosis factor alpha (anti-TNF α) antibodies are a type of biologic therapy used to treat a number of autoimmune diseases including rheumatoid arthritis (RA). They have however been previously linked to an increased risk of developing demyelination of the central nervous system – a disease process involving damage to the protective sheathes that surround nerves in the brain and spinal cord which affects the way these nerves transmit signals. The aim of this analysis was to look for evidence of demyelination having occurred in patients who had received anti-TNF α therapies and compare this to data on the incidence of demyelinating events in the general UK population in order to investigate whether the risk is higher in those on treatment.

What was discovered?

The British Society for Rheumatology Biologics Register for RA (BSRBR-RA) collects information on people with RA and other autoimmune diseases starting biologic therapies including anti-TNF α treatment. Information about patients' medical conditions is collected at the point at which they start treatment and also at regular periods of follow-up thereafter. Evidence of demyelination is one of several serious adverse effects that is evaluated at each follow-up. Any report of possible demyelination in a patient on anti-TNF α therapy was assessed based on the patient's symptoms and investigations in order to confidently say how likely it was that the patient had developed demyelination. 35 demyelinating events were reported in 13,489 patients included in the study who had been followed up for variable lengths of time. Of these, 71% developed demyelination in the first 5 years of starting the therapy. When comparing the incidence of definite or probable cases of demyelination in patients in the BSRBR-RA to the incidence of demyelination in the general UK population, no significant difference in the rate of events was seen.

Why is this important?

This information should be reassuring to both patients and clinicians considering using anti-TNF α therapies due to the evidence that these treatments do not significantly increase the risk of demyelination in this patient group. It should be noted however that patients who would be considered to have a high risk of developing demyelinating disease (such as those with a family history of demyelinating conditions including multiple sclerosis) are unlikely to have been prescribed anti-TNF α and would not have been included in this study. Therefore, this study can't comment on the safety of anti-TNF α in higher-risk patients.

Should you wish to read this scientific paper in full, the text can be found online here:

<https://nn.neurology.org/content/8/3/e992>

