Switching biologic treatments in rheumatoid arthritis: does it increase the risk of serious infections?

Evaluation of serious infections, including *Mycobacterium tuberculosis*, during treatment with biologic disease-modifying anti-rheumatic drugs: does line of therapy matter?

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What was already known?

Rheumatoid arthritis (RA) is a chronic autoimmune condition that causes joint inflammation and can lead to damage and disability. When standard treatments fail to control symptoms, doctors may use advanced medications called biologic disease-modifying anti-rheumatic drugs – known as "biologics". These include various options like TNF inhibitors (etanercept, adalimumab, infliximab, certolizumab), IL-6 inhibitors (tocilizumab), B-cell targeted therapies (rituximab), and T-cell co-stimulation blockers (abatacept). However, these treatments can raise the risk of serious infections, including tuberculosis. This study aimed to find out if the number of different biologics a patient had tried affected their risk of infections.

What was discovered?

The study analysed data from over 33,000 treatment courses in the British Society for Rheumatology Biologics Register of Rheumatoid Arthritis (BSRBR-RA). It found that the overall risk of serious infections did not seem to increase depending on how many different biologics a patient had already tried. When it came to tuberculosis, cases were mostly seen with the first few lines of treatment, and infections were less common after 2009, likely due to improved screening methods.

Why is this important for patients?

These findings suggest that patients who need to switch from one biologic treatment to another can do so without significantly increasing their risk of serious infections. This supports the safe and flexible use of different biologic drugs in treating RA. However, special attention is still needed for tuberculosis risk, especially when starting biologics for the first time.

Should you wish to read this scientific paper in full, the text can be found online here: https://doi.org/10.1093/rheumatology/kead515

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