CLINICAL INVESTIGATION

Future pharmacogenomics aspects in rheumatoid arthritis

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Christiana Williams*

Editorial Office, Clinical Investigation, London, U.K

*Author for correspondence: clinicalinvestigation313@gmail.com

Introduction

Since its early clinical investigations in the 1980s, Methotrexate (MTX), a folate analogue, has been used as the first-line Disease-Modifying Anti-Rheumatic Medicine (DMARD). Although the actual mechanism of action of MTX, which was originally designed as a leukaemia treatment, has been proved to be beneficial in reducing inflammation in RA, the exact mechanism of action is still unknown. Other DMARDs have been developed, including azathioprine, leflunomide, chloroquine, sulfasalazine, and ciclosporin A. Despite the large number of treatments available, they all have significant toxicity and poor efficacy in common. Prior to the year 2000, remission from RA was not thought to be a possibility. The illness progressed to more severe versions in many cases, and the clinical specialist had to empirically determine the efficacy of each DMARD on each patient.

Following the success of anti-TNF therapy, considerable pharmacological research has led to the development of novel RA medications with great efficacy and safety profiles. In 2006, anti-CD20 antibodies (rituximab) were approved for the treatment of RA. Anti-IL-6 (tocilizumab) and anti-CTLA4 (abatacept) drugs have already been licensed for the treatment of RA, with many more expected in the coming years. The chances of any particular patient finding a highly effective treatment improve with each new therapy that develops. As a result, total disease remission, the most ambitious aim in RA treatment, is becoming a reality. We'll need technologies that can precisely predict which treatment is best for each patient now more than ever.

The discovery of genetic markers connected to the response to biologic therapies in RA is moving at a rapid pace. New sets of genetic markers will be discovered, comparable to anti-TNF response predictors that can predict reactions to a rising number of medications. Furthermore, as more patients achieve remission over time, biomarkers for therapy discontinuation will be required. Once we've reached low residual activity in RA, we should be able to tell the difference between people who don't need biological therapy anymore and those who are at danger of relapse. What steps must be taken on a daily basis to integrate genetic and genomic information into RA clinical practice?

First and foremost, we must find consistent and repeatable biomarker patterns. In terms of technology, we're making rapid progress, with more powerful high-throughput devices being introduced on a regular basis. However, we'll need to use more cost-effective technologies if we want to make genomics more accessible in clinical practice. Furthermore, the analytical capabilities of these technologies are limited by two main factors: the quality of the biological samples and the completeness of the clinical data associated with them.

Finally, for the application of pharmacogenomics in RA therapy, and for any difficult disease in general, the medical community's participation with this new type of medical knowledge will be important. As part of their undergraduate medical education, clinical practitioners will need to be educated and instructed on the use (and misuse) of genetic information for patient care. This will take some time, but it will become a necessity in the practice. New genetic and molecular markers for choosing the optimum treatment strategy for RA and many other common diseases will be available in the near future. When comparing the quality of life of a RA patient diagnosed before 2000 to one diagnosed today, there is a significant difference. Our goal is to maintain this level of progress for the next ten years of medicine in the future.

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