useful as CDAI in terms of evaluating the efficacy of TCZ and other anti-rheumatic drugs that have strong inhibiting effects on inflammatory markers.

Figure 4 provides insight into the factors responsible for the difference in remission rates according to the CDAI and DAS28 scales. Many of our patients who satisfied the DAS28 remission criteria but not the CDAI remission criteria had a high SJC and high PGA score. By nature of the definition of CDAI, CDAI remission patients generally had low scores on all of the incorporated measures. The same results were obtained when the difference in the remission rates according to the SDAI and DAS28 scales was analyzed even though the formula for SDAI includes the CRP level as a component for assessing remission (Fig. 5).

A difference between DAS28-ESR and CDAI or SDAI remission among ACR response rates was also observed. All CDAI and SDAI remitters achieved ACR70, but among the 25 DAS28-ESR remitters, 17 (61%) achieved ACR70 while the others achieved no more than ACR50 or ACR20. Since all SDAI remitters achieved ACR70 even though the formula for SDAI includes an inflammatory marker (CRP), we concluded that the difference between the numbers of DAS28-ESR remitters and ACR70 responders was not caused by the inclusion of ESR in DAS28-ESR, but by the remission criterion used in the DAS28-ESR method. Sokka et al. [25] compared the performance of different definitions of remission, including DAS28, CDAI and ACR criteria. They concluded that the use of different definitions of RA remission leads to different results with regard to remission rate and that the rates of remission defined as "DAS28-ESR < 2.6" are higher than those defined by other means. Mäkinen et al. [26] suggested that a stricter criterion (DAS28-ESR < 2.32) is appropriate for defining DAS28-ESR remission. Using the stricter criterion suggested by Mäkinen et al., all patients with DAS28-ESR <2.32 in the SATORI study achieved ACR70.

In conclusion, our results confirm that DAS28-ESR has a validity comparable to that of other methods in terms of evaluating the RA treatment efficacy of TCZ, even though this drug strongly inhibits inflammatory markers.

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Conflict of interest statement NN, as a medical advisor, received a consulting fee and royalty for a systemic onset juvenile idiopathic arthritis patent from Chugai Pharmaceutical Co., Ltd., the product company of TCZ. He also works on the scientific advisory board of Hoffmann-La Roche who developed TCZ in collaboration with Chugai Pharmaceutical Co., Ltd. NT is an employee of Chugai Pharmaceutical Co., Ltd.

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Humanized anti-interleukin-6-receptor antibody (tocilizumab) monotherapy is more effective in slowing radiographic progression in patients with rheumatoid arthritis at high baseline risk for structural damage evaluated with levels of biomarkers, radiography, and BMI: data from the SAMURAI study.

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Abstract

Our aim was to assess the ability of tocilizumab monotherapy to reduce progressive structural joint damage in rheumatoid arthritis patients at high risk of progression. This study was a subanalysis from a prospective 1-year, multicenter, X-ray-reader-blinded, randomized controlled trial of tocilizumab [Study of Active Controlled Monotherapy Used for Rheumatoid Arthritis, an IL-6 Inhibitor (SAMURAI) trial]. All patients were categorized into two or three groups according to four independent predictive markers for progressive joint damage [urinary C-terminal crosslinking telopeptide (uCTX-II), urinary pyridinoline/deoxypyridinoline (uPYD/DPD) ratio, body mass index (BMI), and joint-space narrowing (JSN) score at baseline]. One-year progression of joint destruction was assessed in high-risk versus low-risk groups receiving tocilizumab monotherapy and compared with patients receiving conventional disease-modifying antirheumatic drugs (DMARDs) (n = 157 and 145, respectively). In patients at high risk of progression of erosion as estimated by high uCTX-II, uPYD/DPD, or low BMI, and at high risk of progression of JSN as estimated by low BMI or high JSN score, the 52-week changes in radiological erosion and JSN, respectively, were significantly less in patients treated with tocilizumab monotherapy compared with those receiving DMARDs for each type of risk factor. In patients at low risk, those receiving tocilizumab also progressed less than those on DMARDs, although the difference did not reach statistical significance. Tocilizumab monotherapy is more effective in reducing radiological progression in patients presenting with risk factors for rapid progression than in low-risk patients. Patients at high risk for progression may benefit more from tocilizumab treatment.

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