Most rheumatic diseases are commonly known as systemic chronic inflammatory diseases. Renal involvement in patients with these various rheumatic diseases is not a rare condition. Hill et al.\(^1\) reported that 18% of rheumatology clinic patients have a glomerular filtration rate (GFR) of 60 mL/min or less, which clearly is higher than the 5% reported within the general population. Because it can increase mortality and morbidity rate in rheumatic patients with renal dysfunction, renal manifestation or renal involvement of rheumatic diseases is clinically significant. The pathophysiological mechanism of the renal abnormality has been reported as a result of comorbidities, such as diabetes, hypertension, and cardiovascular complications, chronic inflammation, and/or long-term use of non-steroidal anti-inflammatory drugs or commonly used cytotoxic agents.

Biological disease-modifying anti-rheumatic drugs (bDMARDs), such as tumor necrosis factor (TNF) inhibitors, cytotoxic T lymphocyte antigen 4 (CTLA-4)-immunoglobulin, and interleukin-6 receptor antagonist, have been widely used in the treatment of various rheumatic diseases including rheumatoid arthritis (RA) and ankylosing spondylitis (AS).\(^2,3\) It is true that the biologic agents can result in membranous nephropathy or proliferative glomerulonephritis accompanied by proteinuria via direct invasion of glomerular visceral epithelial layer\(^4\) or development of autoimmune-mediated renal diseases. However, in general, it is plausible that reducing their inflammatory condition with biologic treatment could also have favorable effects on renal function with the potential to slow kidney disease progression and to reduce the subsequent risk of incident chronic kidney disease (CKD) and end-stage renal disease.\(^5\) However, only a little is known about the risk of renal function in patients with RA and AS after using bDMARDs.

In the current issue of *Journal of Korean Medical Science*, Kim et al.\(^6\) analyzed the relation between risk factors for the change in renal function and using bDMARDs to treat patients with RA and AS. They reported that the annual change in epidermal growth factor receptor
(eGFR) was significantly different between males and females ($P = 0.038$) in RA, but not in AS patients ($P = 0.126$). According to their conclusion, the difference occurred depending on gender, with males having higher possibility of annual decline in eGFR in RA and AS patients treated with bDMARDs. However, the fact that there are more women (89.1%) than men in RA and more men (82.4%) than women in AS requires attention to statistical interpretation.

On the other hand, Sumida et al.\textsuperscript{5} recently reported that a significant deceleration of eGFR decline was observed after biological administration in patients who were treated with biologics such as TNF inhibitors or CTLA-4 inhibitors ($-1.0$ vs. $-0.4$ [mL/min/1.73m$^2$/year] before and after biologic use). In this report, the patients were 20,757 US veterans diagnosed with RA and 89% of them were men while 4,617 started biologic therapy among them. The authors concluded that biologic agent administration was independently associated with lower risk of incident CKD and progressive eGFR decline.\textsuperscript{5} Therefore, further studies are required to determine whether the use of bDMARDs and eGFR decline is due to gender-specific characteristics, differences in ethnicity and diseases, or other possible factors.

### REFERENCES


