Successful treatment with tofacitinib for renal disorder due to amyloid A amyloidosis and immunoglobulin A nephropathy in a patient with rheumatoid arthritis

Sirs,

Amyloid A (AA) amyloidosis is a rare but serious complication of chronic inflammatory diseases like rheumatoid arthritis (RA), causing progressive organ dysfunction in the kidneys, neurons, or gastrointestinal tract (1).

Tofacitinib is an oral, small-molecule Janus kinase (JAK) inhibitor, used for the treatment of moderate and severe RA (2, 3).

Here, we report a case of a 76-year-old woman who developed renal disorder due to AA amyloidosis and immunoglobulin A nephropathy (IgAN). Tofacitinib therapy improved her kidney involvement. This patient developed RA at the age of 60. Anti-tumour necrosis factor α (anti-TNF-α) agents, infliximab and golimumab, tocilizumab (TCZ), and abatacept (ABA) were administered, in that order, to treat her active RA.

In December 2014, the patient, who was receiving ABA, was admitted to our department due to worsening kidney function. Signs of declining kidney function included a serum creatinine (s-Cr) level of 1.18 mg/dL (normal, 0.41-0.75), a proteinuria of 2.60 g/day (normal, <0.15), and over 100 red blood cells (RBCs) per high power field (/HPF) (normal, <5) with granular and erythrocytic casts.

On admission, her physical examination revealed active polyarthritis and pitting edema around the ankles bilaterally. Blood testing indicated C-reactive protein level of 7.34 mg/dL (normal, <0.30), a proteinuria of 2.60 g/day (normal, <0.15), and over 100 red blood cells (RBCs) per high power field (/HPF) (normal, <5) with granular and erythrocytic casts.

In this case, we could not exclude the possibility of spontaneous renal improvement. Further, we were unable to investigate the pathological findings of AA amyloidosis and IgAN after tofacitinib treatment. However, judging from the available clinical data, tofacitinib would probably improve renal abnormalities.

In conclusion, treatment with tofacitinib could provide a new immunosuppressive therapeutic strategy for AA amyloidosis and IgAN secondary to rheumatic diseases.

Acknowledgements

We thank Editage (www.editage.jp) for the English language editing.

T. WATANABE**, MD, PhD
T. HATTORI**, MD
Y. OGAWA*, MD, PhD
S. JODO**, MD, PhD

*These authors contributed equally.

1. Internal Medicine, Tomakomai City Hospital, Tomakomai, Japan;
2. Hokkaido Renal Pathology Center, Sapporo, Japan.

Please address correspondence and reprint requests to:
Dr Toshiyuki Watanabe,
Internal Medicine,
Tomakomai City Hospital,
Tomakomai, Japan;
E-mail: nabe0727@med.hokudai.ac.jp
Competing interests: none declared.

© Copyright CLINICAL AND EXPERIMENTAL RHEUMATOLOGY 2018.

References


Fig. 1. Findings of renal biopsy.
A: Periodic acid-Schiff stain: mesangial matrix expansion and cell proliferation with fibrous crescent (original magnification, x40).
B: Immunofluorescent stain for IgA: mild positive IgA within the glomeruli (original magnification, x20).
C: Immunostaining for amyloid A (AA) (brown); AA proteins were positive in the glomeruli (original magnification, x40).
D: Immunostaining for AA (brown); positivity of AA proteins in small vessel walls (original magnification, x40).


