

# CJASN

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- 162 Klotho in Clinical Nephrology: Diagnostic and Therapeutic Implications**  
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### On the Cover

#### *What is the diagnosis?*

A 74-year-old woman with past history of bronchial asthma (at the age of 50), diplopia, mononeuropathy multiplex, muscle weakness, paranasal sinus abnormalities, and positive for myeloperoxidase-ANCA (MPO-ANCA; at the age of 69) was admitted for the evaluation of kidney dysfunction because her serum creatinine levels had increased from 0.5 to 1.1 mg/dl (eGFR from 91 to 39 ml/min per 1.73 m<sup>2</sup>). Her body mass index was 22.1 kg/m<sup>2</sup>. Her white blood cell count was 25,500/ $\mu$ L, 59% of which were eosinophils. She had 3.3 mg/dl C-reactive protein, MPO-ANCA was <0.5 EU, proteinase 3-ANCA was <0.5 EU, and she was negative for anti-glomerular basement membrane antibody. The urine test showed mild proteinuria (0.49 g/d) and massive glomerular hematuria.

#### **Image Description:**

On light microscopy of a kidney biopsy specimen, nine out of 26 glomeruli showed cellular crescents, with rupture of the glomerular basement membrane, fibrinoid necrosis (left), and infiltration of eosinophils (right). Tubulointerstitial nephritis and infiltration of inflammatory cells and eosinophils were observed, particularly around the affected glomeruli (right). Immunofluorescence showed no immune deposits (pauci-immune pattern), and there were no electron-dense deposits on electron microscopy.

Left: A section stained with Periodic acid methenamine silver–Masson shows a cellular crescent (large black arrow), rupture of the glomerular basement membrane (small black arrow), and fibrin deposition (white arrow). Original magnification,  $\times$ 400.

Right: A section stained with hematoxylin and eosin shows fibrinoid necrosis (white arrow) with infiltration of eosinophils. Eosinophils were observed on tubulointerstitial nephritis (large black arrows) as well as on the affected glomeruli (small black arrow). Original magnification,  $\times$ 400.

#### **Teaching Points:**

Rapidly progressive GN on microscopic polyangiitis is characterized by a rapid loss of kidney function and the kidney biopsy specimen showing the formation of glomerular crescents, and eosinophilic infiltration is usually rare in the glomeruli. However, necrotizing crescentic GN with infiltration of eosinophils may suggest kidney disease related to eosinophilic granulomatosis with polyangiitis. Eosinophils may cause glomerular cytotoxicity by secreting fibrin-rich coagulant factors in eosinophilic granulomatosis with polyangiitis, in a mechanism involving IL-5. Eosinophilic granulomatosis with polyangiitis is a systemic small- and medium-vessel necrotizing vasculitis, characterized by tissue infiltration by eosinophils. It occurs in people with multiorgan lesions, including adult-onset asthma, mononeuropathy or polyneuropathy, and paranasal sinus abnormalities. These characteristics are different from those of microscopic polyangiitis. The frequency of positive MPO-ANCA is reported to be around 40%–50%. The relationship between disease activity and ANCA titer is not high as in microscopic polyangiitis, but the mechanism *via* IL-5 is reported to have a close association with the disease activity of eosinophilic granulomatosis with polyangiitis.

*Images and text provided by Naoya Toriu, Nephrology Center, Toranomon Hospital, Tokyo, Japan; Naoki Sawa and Junichi Hoshino, Nephrology Center, Toranomon Hospital, Tokyo, Japan and Okinaka Memorial Institute for Medical Research, Toranomon Hospital, Tokyo, Japan; Keiichi Kinowaki, Department of Pathology, Toranomon Hospital, Tokyo, Japan; and Yoshifumi Ubara, Nephrology Center, Toranomon Hospital, Tokyo, Japan and Okinaka Memorial Institute for Medical Research, Toranomon Hospital, Tokyo, Japan.*