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On the Cover

What's the diagnosis?

A 56-year-old man with type 2 diabetes on insulin therapy suffered from anastomotic leakage and diffuse peritonitis after laparoscopic low anterior resection for rectal cancer. Stool culture grew methicillin-resistant *Staphylococcus aureus*. Peritonitis resolved after colostomy and 3 weeks of intravenous antibiotic treatment, but the patient developed nephrotic syndrome and hematuria 2 weeks after cessation of antibiotics. The urine protein/creatinine ratio increased from 0.1 mg/mg on postoperative day 24 to 12.4 mg/mg on postoperative day 62. Urine sediment analysis showed no erythrocyte initially but revealed more than 30 erythrocytes per high-power field on postoperative day 62. Serum creatinine level was elevated from 0.8 mg/dl to 1.1 mg/dl.

Image Descriptions:

Kidney biopsy revealed mesangial expansion (Image 1), nodular lesions, arteriolar hyalinosis, and linear IgG deposition along the glomerular basement membrane (Image 2), all of which suggested diabetic nephropathy. The biopsy also revealed endocapillary hypercellularity (Image 1) and granular IgA deposition in the mesangium on light microscopy (Image 3) and mesangial deposits on electron microscopy, which were compatible with postinfectious glomerulonephritis. 7-9 g/day of urinary protein excretion persisted for 2 months amid oral prednisolone and cyclosporin treatment, but then gradually decreased to 1-2 g/day in 9 months.

Teaching Points:

IgA-dominant acute postinfectious glomerulonephritis is observed in cases often after staphylococcal infection. Renal histology generally shows endocapillary hypercellularity. Diabetes is a major risk factor, possibly due to an increased susceptibility to staphylococcal infection. The elegant immunofluorescence images of this case demonstrated an IgA-dominant, acute postinfectious glomerulonephritis superimposed on diabetic nephropathy.

(Images and text provided by Yasuhiro Oda, MD, Nephrology Center, Toranomon Hospital, Tokyo, Japan; Masayuki Yamanouchi, MD, Nephrology Center, Toranomon Hospital, Tokyo, Japan; Hiroki Mizuno, MD, Nephrology Center, Toranomon Hospital, Tokyo, Japan; Junichi Hoshino, MD, MPH, PhD, Nephrology Center and Okinaka Memorial Institute for Medical Research, Toranomon Hospital, Tokyo, Japan; Naoki Sawa, MD, Nephrology Center, Toranomon Hospital, Tokyo, Japan; Takeshi Fujii, MD, PhD, Department of Pathology, Toranomon Hospital, Tokyo, Japan; Kenichi Ohashi, MD, PhD, Department of Pathology, Toranomon Hospital, Tokyo, Japan, and Department of Pathology, Graduate School of Medicine, Yokohama City University, Yokohama, Japan; and Yoshifumi Ubara, MD, PhD, Nephrology Center and Okinaka Memorial Institute for Medical Research, Toranomon Hospital, Tokyo, Japan)