Renal tubular acidosis and vasculitis associated with IgE deposits in the kidney small vessels

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RESUMEN

We report a woman with a history of allergies, polyuria, polydipsia, proteinuria, renal loss of electrolytes, renal tubular acidosis, nephrocalcinosis, and palpable purpura. A proximal defect was excluded by a normal bicarbonate reabsorption curve, and a distal tubular defect was shown because urine pH did not decrease to less than 6.4 despite ammonium chloride- induced systemic acidosis. Moreover, furosemide failed to improve urinary acidification. Urine-to-blood PCO2 gradient was less than 14 mm Hg, although the urine bicarbonate level reached values as high as 89 mEq/L. Combining bicarbonate and neutral phosphate infusions increased the urine-to-blood PCO2 gradient to only 20 mm Hg. These subnormal PCO2 gradient values point to proton-pump dysfunction in the collecting tubule. Histological evidence of tubulointerstitial disease accompanied the tubular defects. The striking histological feature was the presence of immunoglobulin E (IgE) deposits in glomeruli, tubuli, and vessels. Concurrent with these findings, she had high serum IgE titers and CD23 levels. IgE antibodies from her serum were reactive against human renal tubuli, with binding to two regions that matched two different proteins present in cortex and medulla. One of these proteins corresponded to carbonic anhydrase II (31 kd); the second, to an unidentified protein that seems attached to cell membranes. We suggest that these IgE antibodies could have had a pathogenic role in this patient's glomerular, tubular, and small-vessel disease.

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