Horseshoe Kidney and Nephrotic Syndrome due to Idiopathic Membranous Nephropathy

Key words: horseshoe kindney, nephrotic syndrome, membranous nephropathy

Urinary tract infection, hydronephrosis, calculi, and tumor of the renal pelvis are known as complications and multiple congenital anomalies can occur together with a horseshoe kidney (1). However, the occurrence of membranous nephropathy (MN) in a horseshoe kidney has been only reported in a few case reports (2, 3). Furthermore, horseshoe kidney and focal sclerosing glomerulonephritis were seen in another case (4). We would like to describe a case of MN occurring in horseshoe kidney.

An 18-year-old woman was admitted to our emergency department because of general edema, swelling. On the physical examination, arterial blood pressure was 120/70 mmHg, pulse was 90/min, and temperature was 37.2°C. On laboratory examination, the urine test rendered (++++) proteinuria and 24 hours urine protein excretion was 8-14 g/day, and creatinine clearance (Ccr) 80 ml/min. Urine microscopy showed granular casts, rare hyaline casts, 10-12 red cells, 8-10 leukocytes/mm³. Urine cultures were >10⁵/ml Escherichia coli. Proper antibiotic (cefuroxime) was started. After treatment, urine infection was alleviated. The blood tests showed the following results: hemoglobin: 13 g/dl, hematocrit 39%, white blood cell count: 8,800/mm³, platelets: 439, 000/mm³, sedimentation rate: 115 mm/h, total protein: 5.2 g/dl (γ-globulin: 0.2 g/dl), plasma albumin: 1.5 g/dl, blood urea nitrogen: 23 mg/dl, plasma creatinine: 0.8 mg/dl, total cholesterol: 493 mg/dl, triglycerides: 544 mg/dl, total bilirubin: 0.8 mg/dl, aspartate aminotransferase: 25 IU/l, alanine aminotransferase: 39 IU/l, LDH: 330 IU/l, gamma-glutamyl transpeptidase: 15 IU/l, alkaline phosphatase: 169 IU/l, sodium: 138 mEq/l, potassium: 4.5 mEq/l, chloride: 107 mEq/l. Serum immunoglobulin levels and complement levels (C3, C4) were within the normal range. Antinuclear antibody, anti double-stranded DNA, rheumatoid factor, hepatitis B surface antigen (HbsAg), anti-HBsg, hepatitis C virus antibody (anti-HCV), and human immunodeficiency virus antibody (anti-HIV) were negative. On the abdominal ultrasound and abdominal computerized tomography (Fig. 1) horseshoe kidney was revealed, as well as nephrolithiasis and minimal pelvicalcitasy. Furthermore, coelithiasis was seen. Kidney biopsy was taken for diagnostic purposes using a standard Trucut needle under computerized tomography. Biopsy material, examined by both light microscopy and electron microscopy, showed features diagnostic of membranous nephropathy. There was diffuse thickening of the glomerular capillary and tubulus basement membrane and subepithelial electron-dense deposits all along the glomerular capillary loops. Proteinuria (3.5 g/day) declined gradually over 5 weeks with methylprednisolone, and angiotensin converting enzyme (ACE) inhibitor treatment (10 mg/day, enalapril). Methylprednisolone was started with an 80 mg dose and then was gradually diminished. Finally, methylprednisolone was maintained with a 20 mg dose. Serum albumin (2.4 g/dl) and total protein (5.8 g/dl) were elevated and edema disappeared.

In summary, the coincidence of two renal pathologies in this patient may be a coincidence, but horseshoe kidney can predispose to immune deposit formation. The question is whether glomerulopathy is idiopathic or associated with the anatomic anomaly. More such cases will be necessary to determine whether MN is one of the complications of horseshoe kidney.

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Received for publication March 5, 2001; Accepted for publication August 10, 2001

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Figure 1. Computed tomography scan of the abdomen showing a case of horseshoe kidney (o).
References


