Severe Lupus Cystitis with Obstructive Uropathy

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ual improvement in kidney function tests was achieved, where blood urea decreased to 60 mg/dl, and serum creatinine decreased to 1.5 mg/dl within 2 weeks. A significant improvement in the bladder trabiculations and capacity also occurred (fig. 2) with a conse-
Dear Sir,

A 29-year-old woman presented with a 4-week history of progressive urinary frequency and dysuria. Nine years earlier she had been diagnosed as having systemic lupus ery-thematosus (SLE) which subsequently was associated with multisystem complications including spontaneous abortions, pleuroperi-carditis, membranoproliferative glomerulonephritis, diarrhea, and involvement of the central nervous system with grand mal seizures. Up to this time she had been treated only with glucocorticoids for her SLE and its complications, and her kidney function tests were within the normal range until 2 months before admission. No cytotoxic agents including cyclophosphamide had been used previously.

Laboratory examinations revealed the following: serum creatinine 4.2 mg/dl; blood urea 119 mg/dl; erythrocyte sedimentation rate 100 mm in the first hour; ANA and anti-DNA antibodies were positive, and C3 and C4 levels were low. Urinalysis revealed a few erythrocytes and leukocytes in a high power field with no casts, proteinuria of 1.2 g/24 h, creatinine clearance of 23 ml/min, and negative urinary culture. Abdominal sonography showed severe bilateral hydroureteronephrosis and a trabicular urinary bladder with a very reduced capacity. Cystoscopy with cystography demonstrated that the bladder had a very reduced capacity, prominent trabiculations, thickening of the walls, and a very narrow ureteral orifice (fig. 1). Severe bilateral hydroureteronephrosis was also present. Bladder biopsy showed chronic interstitial cystitis, and no active vasculitis.

The patient was treated with a high dose of prednisone, and her lower urinary tract symptoms markedly improved. Also, a grad-

Fig. 1. Cystography shows the very reduced capacity of the urinary bladder with severe trabiculations of its walls.

quent improvement in the bilateral hydroureteronephrosis.

Involvement of the urinary system in SLE is usually limited to the kidneys and characterized by glomerulonephritis. Involvement of the urinary bladder in SLE is

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Fig. 2. Ultrasonography of the urinary bladder showing severe trabeculations of its walls and reduced capacity on admission (a), and the almost normal ultrasonographic appearance of the bladder after high-dose prednisone therapy 4 weeks later (b).

generally considered to be due to infections, neurogenic origins, or secondary to cyclophosphamide therapy which may cause hemorrhagic cystitis. Chronic interstitial cystitis as a major manifestation of SLE has rarely been reported [1,2]. The pathogenesis of interstitial cystitis in SLE is still unknown; however, immune complex-mediated vasculitis has been suggested [3].

In contrast to idiopathic interstitial cystitis, lupus cystitis is frequently associated with hydroureteronephrosis which is usually due to fibrosis of the ureterovesical junction [1], and to detrusor muscle spasm secondary to inflammation [2]. Patients with lupus cystitis generally respond to steroid therapy as happened with our patient, but some develop progressive bladder fibrosis with subsequent uropathy. As lupus cystitis may be a reversible cause of renal failure in patients with SLE, it is important to recognize this rare complication in order to prevent severe bladder damage and consequent obstructive uropathy and renal failure by early treatment with glucocorticoids or cytotoxic agents.

References

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494

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Odeh