A 50-year-old man with lupus nephritis WHO class V+IV-G(A), who had been treated with glucocorticoids and cyclophosphamide (2000 mg total dosage) for induction therapy, and glucocorticoids and mycophenolate (2000 mg/day) for maintenance therapy for approximately 2 years, presented with a 3-week history of abdominal distension. On admission, the patient had a body temperature of 38.4. Physical examination revealed a right hypochondriac mass without superficial lymphadenopathy. Laboratory tests showed a serum creatinine (Cr) level of 1.32 mg/dL, increased from 0.87 mg/dL measured 2 months before; lactate dehydrogenase of 459 U/L (normal, 115–230 U/L); and soluble interleukin-2 receptor of 21,000 U/mL (normal, 124–466 U/mL). Urine sediment was not active, with a few red blood cells/high-power field and stable proteinuria (2.0 g/gCr). Serum complements were not low, and anti-DNA antibodies were not increased. Abdominal computed tomography showed right kidney enlargement and a huge mass circumferentially invading the right pelvis and developing around the right ureter with hydronephrosis, which suggested aggressive tumor (Figure 1A). Pathological examination revealed diffuse large B-cell lymphoma, and he began chemotherapy including rituximab.

Reported by Bernatsky et al. in 2005, the risk of non-Hodgkin lymphoma (NHL) in lupus patients may be higher than that of the general population [1]. In addition, systemic lupus erythematosus itself is known to be a possible risk factor of NHL [2], along with immunosuppressive drugs and Epstein-Barr virus (EBV) infection [3]. The risk of developing hematological malignancies may increase after exposure to immunosuppressive drugs, particularly when the exposure duration exceeds 5 years [4]. EBV-infected human B cells are known to have increased expression of B cell-activating factor, thus contributing to monoclonality and lymphoma formation [5]. In this patient, the duration of...
exposure to immunosuppressive drugs was only 2 years, and the lymphoma tissue was negative for EBV-encoded RNA.

With treatment, the tumor reduced in size (Figure 1B), and renal function partially improved. However, the hydronephrosis still remained after general improvement in the patient’s condition (Figure 1B). Lymphoma-associated acute kidney injury occurs by a variety of mechanisms, which also differ widely in prevalence and clinical presentation. The possibility of NHL occurrence should thus be taken into account in the clinical course of lupus patients.

REFERENCES


