Dear Sir,

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease that may affect the skin, joints, kidneys, lungs, nervous system, and serous membranes and/or other organs. Lymphadenopathy is common in SLE. It has been reported that lymph node enlargement is observed in 25-67% of SLE patients [1-3]. But hilar and mediastinal lymph node enlargement due to SLE is rare [4].

We report a 15-year-old young girl with SLE, involving the cardiovascular, central nervous, pulmonary, renal and hematologic systems and the skin with cervical, axillary, inguinal and mediastinal lymph nodes. The patient was admitted to the nephrology ward with generalized edema, ascites, fever and lymphadenopathy and suspicion of lymphoma. Blood pressure was 140/90 mm Hg, temperature was 37.5 °C, multiple lymph nodes measuring from 0.5 cm to several centimeters were detected in the cervical, axillary and inguinal areas. Bilateral optic atrophy was detected. Laboratory findings on admission showed: hemoglobin 5.9 g/dl, hematocrit 19.6%, white blood count 3,700/mm³, platelets 186,000/mm³, sedimentation rate 80 mm/h, BUN 64 mg/dl, creatinin 2.2 mg/dl, total serum protein 5.4 g/dl, albumin 1.9 g/dl, cholesterol 335 mg/dl, total serum lipid 825 mg/dl, LDL cholesterol 216 mg/dl, triglyceride 439 mg/dl. Daily proteinuria was 9.6 g. Urine sediment contained 15-20 white blood cells, 20-25 red blood cells and granular casts. Antinuclear antibody and anti-smooth muscle antibody were positive and serum C3 level decreased. Thoracic and abdominal computerised tomography (CT) scan showed mediastinal, left axillary and retroperitoneal lymph node enlargement and pleural effusion. Pericardial effusion was detected by echocardiography. Cervical node biopsy and renal biopsy showed follicular hyperplasia and mesangiocapillary glomerulonephritis, respectively.

She received a 5-day pulse of methyl-prednisolone (25 mg/kg/day). And then 1 mg/kg/day methylprednisolone and dipyridamole (225 mg/day). One month later, full evaluation was negative for mediastinal, cervical and axillary lymph nodes. But 15 days later, she was admitted with generalized convulsion and supraventricular tachycardia. Blood pressure was 200/120 mm Hg. Uremia and electrolyte imbalance were not detected. Magnetic resonance imaging and CT of
the brain were negative. EEG revealed diffuse slow-wave activity. We suggested that convulsions and supraventricular tachycardia were related to SLE involvement in cerebrovascular system and myocardium, respectively, or high blood pressure due to the large amount of drugs given in a very short time.

She began to receive anticonvulsive drugs to stop the convulsions and β-blockers (atenolol 100 mg/day) for hypertension. One year later, she is symptom free with anti-epileptic drugs, atenolol (50 mg/day) and prednisolone (5 mg/day). Her renal function is normal and proteinuria negative.

We reported a patient who fulfilled the ARA criteria for SLE [5]. Mediastinal lymph node enlargement is known to be rare in SLE. In our case, lymphadenopathy was generalized and responded to corticosteroid treatment, so we were far away from suspecting malignant lymphoma. In the literature, we found only 1 SLE case with mediastinal lymphadenopathy who developed a superior vena cava syndrome [6]. Maybe this finding is rare due to the fact that thorax CT is usually not performed in SLE patients with lymphadenopathy.

References