Lupus nephritis associated with Castleman's disease: A case report

Department of Internal Medicine1, Department of Pathology2, Research Institute of Clinical Medicine, Chonbuk National University Medical School, Jeonju, Chonbuk, South Korea

*Hyo Jin Han1, Tae Hwan Lee1, Kyung Pyo Kang1, Sik Lee1, Sung Kwang Park1, Jae Yong Kwak1, Kyu Yun Jang2, Myoung Jae Kang2, Won Kim1

Localized or generalized lymphadenopathy is a common finding in active systemic lupus erythematosus (SLE). Although lymphadenopathy associated with SLE demonstrates histological diversity, the incidence of lymphoid malignancy in SLE is very low. Castleman's disease (CD) is a rare atypical lymphoproliferative disorder characterized by enlarged hyperplastic lymph nodes. CD has been classified on clinical grounds (unicentric or multicentric) and by histological appearance (hyaline-vascular pattern, plasma cell predominance, or mixed). Whereas the unicentric form is usually benign and curative resection is possible, the multicentric form often has systemic symptoms and a clinically more malignant course. So, multicentric CD do not benefit from surgical treatment and should be candidate for aggressive systemic therapy (steroids, combination chemotherapy). To our best knowledge, there was no report about SLE associated with CD in our country, so we report a case of lupus nephritis associated with CD. A 26-year-old female patient was admitted because of general edema and dyspnea about 2 weeks ago. On physical examination, she had a malar rash, palpable cervical and inguinal lymph nodes, and both pretibial pitting edema, but do not have any oral ulcer, or arthritis. Laboratory tests revealed a severe nephrotic range proteinuria (24 hour-urine protein was 7,233 mg/day), hypoalbuminemia (serum albumin level was 2.1 g/dL) and hemolytic anemia (hemoglobin level was 8.9 g/dL, a schistocyte showed 3-5/HPF on peripheral blood smear, and a positive Coombs' test). A chest X-ray showed pulmonary edema combined with both pleural effusion. The diagnosis of SLE was made because she developed a positive antinuclear factor (1:640), with a high titer of anti-dsDNA antibodies (over 95 IU/mL) and a low complement level (serum C3 was 35.3 mg/dL and C4 was 4.7 mg/dL). We performed renal biopsy, specimens showed diffuse global proliferation and sclerosis compatible with lupus nephritis (Class IV-G). And an inguinal lymph node biopsy revealed morphologic features of CD. After aggressive chemotherapy (CHOP) and high-dose steroid therapy for 6 months, her symptoms with dyspnea, proteinuria and lymphadenopathy was near completely resolved.

Cerebral Salt wasting syndrome in a patient with tuberculous meningitis and basal ganglia infarction

Department of Internal Medicine1, Department of Neurology2, Gyeongsang National University Hospital, Jinju, Republic of South Korea

*Dong Wook Kim1, Hyun Seop Cho1, Jung Hee Jung2, Hyeon Jeong Lee1, Jong Woo Seo1, Kyusik Gang2, Hyun-Jung Kim1, Dong Jun Park1, Se-Ho Chang1

Hyponatremia is the most common electrolyte disorder in hospitalized patients with neurologic problems. These patients often diagnosed as the syndrome of inappropriate antidiuretic hormone (SIADH), but it is difficult to distinguish this from cerebral salt wasting syndrome (CSW). We present a case of a 50 year old man who developed cerebral salt wasting syndrome during treatment of tuberculous meningitis and basal ganglia infarction. A 50-year-old man was admitted to our hospital with drowsiness and fever. Physical examination showed temperature of 39 °C, a BP of 150/90 and drowsiness. His Glasgow coma scale (GCS) score was 9/15 (E2V2M5). Computerized tomography of the head revealed acute infarction of basal ganglia. Patient’s cerebrospinal fluid examination revealed WBC 99/mm3 (PMN 8%, lymphocyte 92%) with a protein of 280 mg/dl and a glucose of 36 mg/dl. Probable tuberculous meningitis was made as a diagnosis and the patient was started on antituberculosis drugs. On day 14 fever was subsided, but he developed hyponatremia. Laboratroy tests showed plasma sodium 126 mmol/L. At first the patient was managed as fluid restriction because we thought on syndrome of inappropriate antidiuretic hormone (SIADH). But hyponatremia was not corrected (plasma sodium 125 mmol/L). Intravenous 0.9% saline (4L/day) and fludrocortisone was applied to the patient and serum sodium was increased as 138.3 mmol/L and polyuria was decreased. He was diagnosed as cerebral salt wasting syndrome (CSW) based on his clinical signs and biochemistry. We describe a patient with tuberculous meningitis and basal ganglia infarction who developed hyponatremia secondary to CSW. CSW is rarer disease than SIADH. But it is important to differentiate CSW from other problems because of different management. We should consider CSW in a neurological patient with hyponatremia and differentiate from SIADH.