Case report

Syndrome of inappropriate secretion of ADH in the course of chemotherapy for anaplastic large T-cell lymphoma of the lung

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ABSTRACT

The present study reports the case of a 40-year-old patient who presented SIADH syndrome due to anaplastic large T-cell lymphoma and antineoplastic drugs.

Key words: SIADH, hyponatremia, lymphoma, antineoplastic drugs

CASE REPORT

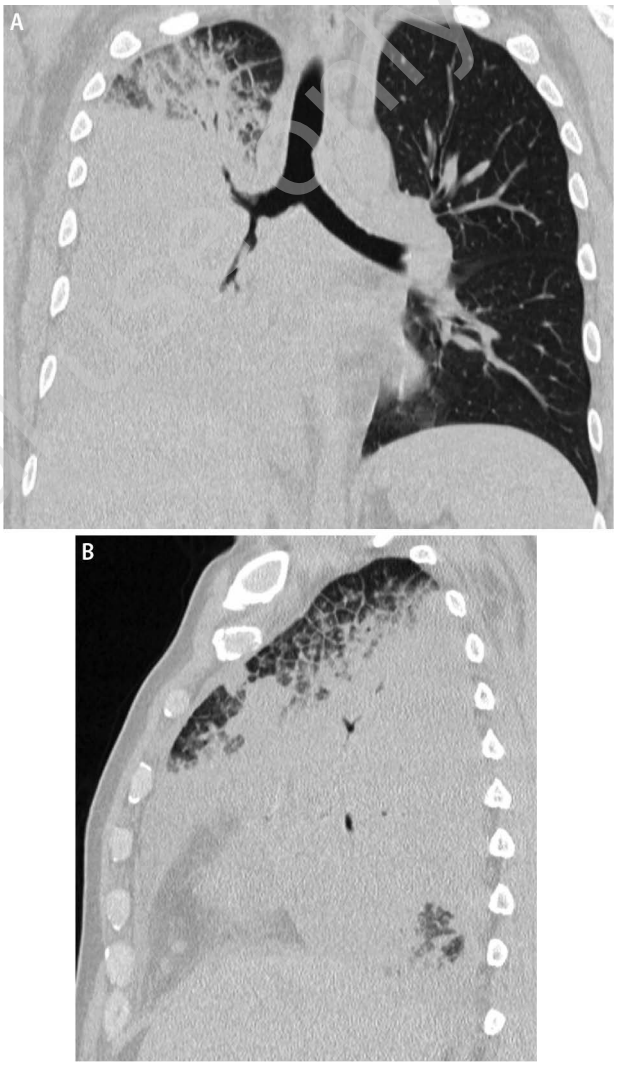
A 40-year-old patient with anaplastic large cell lymphoma (diagnosis based on a core biopsy of lung tumor) was admitted to the Hematology Department for the second course of chemotherapy. The first Hyper-CVAD chemotherapy course (cyclophosphamide, doxorubicin, dexamethasone, vincristine) with additional intrathecal methotrexate and cytosine arabinoside had been administered one month previously.

On history taking, the patient maintained full logical verbal contact. The patient was significantly weak and complained of chest wall pain. Therefore, additional tests were performed which showed (reference ranges in brackets): sodium – 116 mmol/l (135–145 mmol/l), potassium – 3.9 mmol/l (3.5–5.0 mmol/l), magnesium – 2.21 mg/dL (1.7–2.5 mg/dl), ionized calcium – 1.021 (1.12–1.32), phosphorus – 1.88 mg/dl (2.7–4.5 mg/dl), creatinine – 0.54 mg/dl (0.7–1.2 mg/dl), urea – 13 mg/dl (16.6–48.5 mg/dl), uric acid – 1.8 mg/dl (3.4–7.0 mg/dl), 25-(OHD) – 3 ng/dl (0–10 ng/dl means severe deficiency), serum osmolality 246 mOsm/kg H_2O (285–295 mOsm/kg H_2O), urine osmolality 663 mOsm/kg H_2O (700–1200 mOsm/kg H_2O). Urinalysis was normal. Sodium concentration in a urine sample was 35 mmol/l.

On physical examination, no features of overhydration or dehydration were found, and blood pressure values were normal. The diagnostics was expanded to include high-resolution computed tomography (HRCT) which revealed a unilateral extensive mass-like consolidation which invaded almost the entire right lung. The nodular thickening of the walls of the lobar bronchi and mild lymphadenopathy were observed. Pleural effusion was not observed (fig. 1A, B).

Further diagnosis of hyponatremia included the following additional laboratory tests (reference ranges in brackets): morning cortisol – 6.79 µg/dl (6.02–18.4 µg/dl), cortisol at 4 p.m. – 3.01 (2.68–10.5 µg/dl), ACTH in the morning – 31.4 pg/ml (10–60 pg/ml), TSH – 0.27 uIM/ml (0.27–4.2 uIM/ml), fT_3 – 3.1 pmol/l (3.1–6.8 pmol/l), fT_4 – 19.24 pmol/l (12–22 pmol/l). We excluded adrenocortical insufficiency, thyroid dysfunction and pseudohyponatremia in the course of hyperglycemia, paraproteinemia and hypertriglyceridemia [1]. Renal phosphate loss was ruled out due to concomitant hypophosphatemia. In order to expand the diagnosis of pulmonary lesions, a bronchoscopy was performed. It confirmed the progression of anaplastic T-cell lung lymphoma. Based on biochemical criteria and the clinical picture, Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) was diagnosed. Its etiology was complex including T-cell lymphoma and the use of antineoplastic drugs. The dominant etiological factor of SIADH syndrome was not indicated [1]. Based on the Naranjo algorithm, it was determined that SIADH was probably induced by drugs [2].

Figure 1A, B.
HRCT scans, the lung window reconstructed in the coronal and sagittal views. In the right lung, an extensive mass-like consolidation invading almost the entire lung is visible. Nodular thickening of the walls of the lobar bronchi.

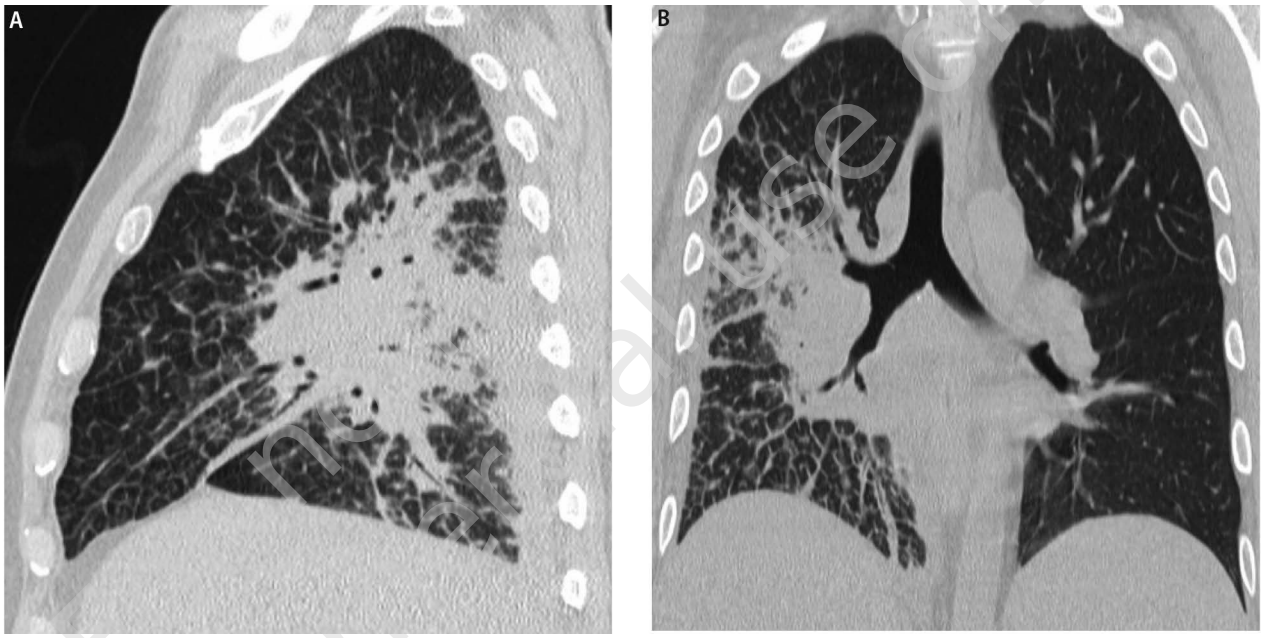


Due to mild symptoms of hyponatremia, fluid restriction and infusion of 150 ml of 3% NaCl were introduced in the patient, resulting in an increase in natremia to 132–135 mmol/l. Significant improvement in the clinical status was observed after natremia normalization, and a decision was made to administer the second course of chemotherapy. 12 days after the course, a follow-up HRCT was performed, showing the regression of pulmonary lesions. We observed a perihilar consolidative lesion distributed along the bronchovascular bundles with the inva-

sion of interlobular septal thickening and smooth outlines of the walls of the lobar bronchi. The size of the lymph nodes decreased (fig. 2A, B). Natremia remained within normal limits.

regardless of the underlying disease, might induce SIADH by increasing the activity of ADH in the renal tubules and by stimulating the secretion of ADH [3, 5].

Figure 2A, B.
Follow-up HRCT scans performed after 12 days. A reduced perihilar consolidative lesion distributed along the bronchovascular bundles with the invasion of interlobular fissures. Smooth outlines of the walls of the lobar bronchi.



DISCUSSION

Small cell lung cancer, characterized by the excessive production of ADH, is the most common neoplastic cause of SIADH [1]. Pulmonary lymphomas rarely contribute to the development of SIADH. SIADH is caused by drugs more commonly than by neoplasms [3, 4]. In our patient, cyclophosphamide and vincristine,

Notably, the symptoms of hyponatremia in the course of SIADH are often poorly expressed, and its occurrence in the course of a neoplastic disease is associated with an increased risk of mortality [1, 6, 7].

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Grzegorz Kade: 20%; Sebastian Spaleniak: 20%; Janusz Hałka: 20%; Dariusz Moczulski: 15%; Maciej Michalak: 20%; Aleksandra Straszyńska: 5%.

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The paper complies with the Helsinki Declaration, EU Directives and harmonized requirements for biomedical journals.