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Dual Paraneoplastic Syndrome in Small Lung Cancer

Kamil Sardarli

Austin M. Morris

Jamal Abukhaled

in proximal and distal tubular epithelium with preference to the former, thus causing a net sodium excretion. These patients present with hypotonic hyponatremia, polyuria, and hypovolemia. In contrast, SIADH patients are euvolemic and normouric. A high FeNa despite volume depletion helps delineate RSWS further from SIADH. Treatment consists of salt and water replenishment, and recovery is the rule although recurrence is common. While carboplatin is less toxic, cisplatin has unequivocal superiority in most of the cancers in which it is used, making it difficult to replace it, especially when there is realistic curative intent.

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Syndrome of Inappropriate Antidiuresis (SIAD): A Clue to a Rare Diagnosis

Amara Sarwal, Sarah Gilligan, Nirupama Ramkumar, Josephine Abraham. *University of Utah Health, Salt Lake City, UT.*

Introduction: Syndrome of inappropriate antidiuresis (SIAD) was first described by Schwartz and Bartter in 1967. Small cell lung cancer is the malignancy most often associated with ectopic ADH release, however extrapulmonary small cell cancers, medications and other pulmonary disease can also be associated with SIAD. SIAD is rarely associated with prostate cancer. We report a case of SIAD with small cell carcinoma of the prostate.

Case Description: A 74-year-old man with history of cardiomyopathy, hypertension and left renal cancer with partial nephrectomy presented to our facility due to an irregular heartbeat. Upon further questioning, it was discovered that he had a 2 month history of gait unsteadiness, confusion and fatigue. He denied increased thirst or recent medication changes. He denied use of NSAIDs, PPIs, diuretics, SSRIs, or anti-epileptic medications. He is a former smoker with a 40 pack year history and is a social drinker. During initial workup in the emergency room, a sodium level of 125 mmol/L was discovered. He was initially treated with furosemide and 4 gram salt tabs daily however eventually transferred to the critical care unit for hypertonic saline when his sodium dropped to 115 mmol/L within 72 hours. Further workup revealed a TSH of 1.25 mU/L, morning cortisol of 19 ug/dL, serum osmolality of 236 mOsm/kg with urine osmolality of 500 mOsm/kg and urinary sodium level of 155 mmol/L. MRI brain and CT chest were unrevealing, however his CT abdomen revealed an enlarged prostate with a heterogeneously enhancing large nodule and an enlarged right pelvic lymph node, concerning for metastatic prostate cancer despite a normal prostate specific antigen (PSA) at 0.5 ng/mL. Biopsy revealed small cell carcinoma of the prostate. Radiotherapy as well as chemotherapy with cisplatin and etoposide was initiated. He continued treatment with salt tablets and furosemide with stabilization of his sodium at 130-136 mmol/L.

Discussion: Small cell carcinoma of the prostate is rare and accounts for <1% of all patients afflicted with prostate cancer. It is usually diagnosed at an advanced stage and PSA can be disproportionately low compared to conventional adenocarcinoma of the prostate. This case illustrates the need for diligent investigation when patients present with hyponatremia and SIAD.

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Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) Secondary to Untreated Parkinson Disease

Najjar Yaseen, Mina Awad, Geovani Faddoul. *Albany Medical Center, Albany, NY.*

Introduction: Hyponatremia is the most common electrolyte disorder associated with neurological conditions and is mediated by excess release of antidiuretic hormone. The etiology can range from a straightforward SIADH to the poorly understood mechanism of cerebral salt wasting. Whereas acute intracranial process such as acute stroke, intracranial bleeding or tumor are typical etiologies of SIADH, neurodegenerative diseases such as Parkinson's disease or ALS have not been known to be associated SIADH. However, SIADH has been reported as a side effect of anti-Parkinson medication e.g. Levodopa/Carpidopa. Parkinson's disease per se has not been known to be associated by SIADH and hyponatremia. We present a case of hyponatremia due of SIADH in the setting of untreated Parkinson.

Case Description: A 71-yo male patient with DM II and hypothyroidism presented to the hospital with progressive confusion, slow speech and severe fatigue over las few days accompanied with sluggish body movements for few months. On exam, he appeared to be euvolemic. Neurological exam revealed mild arm rigidity, bradykinesia, resting tremors, and stiff gait. Initial blood work showed hypo-osmolar hyponatremia (sodium 122 mEq/L, serum osmolarity 275 mOsm/Kg, uric acid 2.3 mg/L, TSH 3.5 UIU/mL, AM cortisol 24 UG/dL). Urine studies showed (urine sodium: 92 mEq/L, osmolarity 672 mOsm/Kg). A Brain MRI did not show any structural abnormality. CT chest showed localized infiltrate. Initially SIADH was thought to be due to pulmonary process. After starting him on fluid restriction of 1.5L/day and urea 15 mg BID, sodium improved gradually to 133 mEq/L on discharge. Urine osmolality continued to ranges 700-800 mOsm/Kg. Active pulmonary process was ruled out by pulmonologist. Parkinsonism was confirmed by outpatient neurology who started Carbidopa/Levodopa. As extrapyramidal symptoms improved with Carbidopa/Levodopa, urine osmolality improved to 400 mOsm/Kg. Successfully, sodium level was maintained between 135-137 while being off urea and fluid restriction. He was diagnosed with new-onset SIADH and extrapyramidal symptom.

Discussion: This case demonstrated an association of SIADH and untreated Parkinson's disease. In contrary to literature, SIADH resolved with anti-Parkinson treatment which supports Parkinson's disease as a causal mechanism. As dose increased of anti-Parkinson's agent, urine osmolality improved and extrapyramidal symptoms resolved.

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Dual Paraneoplastic Syndrome in Small Cell Lung Cancer

Kamil Sardarli, Austin M. Morris, Jamal Abu-Khaled. *BHSH Beaumont Health, Royal Oak, MI.*

Introduction: Small cell lung cancer (SCLC) is an aggressive neuroendocrine lung cancer associated with paraneoplastic syndromes – syndrome of inappropriate ADH (SIADH) in 7-16% of cases and Ectopic ACTH Syndrome (EAS) in 3.5-4.3% of cases. Both syndromes are associated with a poor prognosis in patients with SCLC. We describe a patient who presented with SIADH, leading to a diagnosis of SCLC who subsequently developed EAS.

Case Description: A 71-year-old female with a past medical history of hypertension, depression, and tobacco use presented with lethargy. She noted headache, but otherwise was unable to provide history due to altered mentation. Per her family, the patient recently had pneumonia treated with antibiotics and steroids and was complicated by hyponatremia. Upon evaluation, our patient was hemodynamically stable, with labs revealing Na 106 mmol/l, K 3.9 mmol/l, Cl 74 mmol/l, BUN 6 mg/dl, and creatinine 0.52 mg/dl. Serum osmolality was 232 mOsmol/kg, urine osmolality 543 mOsmol/kg, urine Na 130 mmol/l and urine Chloride 128 mmol/l. TSH was within normal limits and random cortisol level was 63.4 mcg/dl. She was managed in ICU with a DDAVP clamp and 3% saline. She was discharged on Urea therapy, which was later change to furosemide and sodium chloride tablets due to cost. At discharge, the etiology of her hyponatremia was most consistent with SIADH. CT scan of the chest followed by PET scan showed multiple enlarged mediastinal lymph nodes that were considered suspicious for malignancy. Lymph node biopsy confirmed metastatic SCLC. Our patient was started on concurrent chemotherapy and radiotherapy. Upon follow up, progression of her cancer was seen, prompting additional chemotherapy with carboplatin, etoposide, and atezolizumab. She was admitted 11 months after her first presentation due to hypernatremia and hypokalemia. She had metabolic alkalosis with a bicarbonate of 36 mmol/l, Na level 146 mmol/l, and K 2.7 mmol/l. Morning cortisol level was 54.2 mcg/dl and ACTH was 63 pg/ml. Cortisol level after low dose dexamethasone suppression test remained elevated at 66.6 mcg/dl. In the setting of active SCLC, findings were most suggestive of ectopic ACTH secretion.

Discussion: There are only 8 cases reported in literature with simultaneous or sequential EAS and SIADH. Cortisol and ADH have opposite effects on kidney sodium excretion. This may lead to masking of EAS by SIADH, leading to underdiagnosis.

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SIADH Escape or Tolvaptan Resistance in Progressive Small Cell Lung Cancer

Sunny Mandal,[1] Lakshmi Kannan.[2] [1]*University of Pikeville Kentucky College of Osteopathic Medicine, Pikeville, KY;* [2]*Pikeville Medical Center, Pikeville, KY.*

Introduction: Syndrome of inappropriate antidiuretic hormone(SIADH) occurs in 11-15% of patients with small-cell lung cancer. The pathophysiology is through direct tumor secretion, enhanced secretion of ADH from adrenal metastases, chemotherapy, opioids, NSAIDs, or side effects of treatment like nausea, vomiting, stress, and pain. We report a patient with small-cell lung cancer initially managed with tolvaptan but developed resistance to the aquaretic effects of the drug.

Case Description: A 61-year-old male with small cell lung cancer and chronic hyponatremia on tolvaptan presented with unsteady gait and weakness. Workup showed serum sodium of 111 mEq/L(baseline-122-125 mEq/L), serum osmolality of 236 mOsm/Kg H_2O, urine osmolality of 589 mOsm/Kg H_2O, and urine sodium of 62 mEq/L. The patient was admitted for 3% hypertonic saline for SIADH. His sodium improved to 120 mEq/L with the resolution of symptoms. Reviewing his records, the patient was diagnosed with T3N3 IIIC small cell lung cancer 11 months prior to this admission. He underwent two cycles of cisplatin and etoposide. In a span of 3 months, the patient had four hospitalizations for acute on chronic hyponatremia despite being on tolvaptan as his surveillance CT abdomen/pelvis revealed multiple new liver metastases. The timeline of events is shown in Figure 2.

Discussion: Syndrome of inappropriate antidiuretic hormone is characterized by euvolemic hypotonic hyponatremia where tolvaptan has been used since 2009. The first two cases of resistance to tolvaptan therapy were described in 2018 in patients with small-cell lung cancer and this is the third report of SIADH escape to tolvaptan. The possible cause of resistance to the aquaretic effects of the drug is due to extraordinarily high ADH levels from the progression of lung cancer. In these cases, successful treatment of the malignancy will eliminate or reduce the inappropriate ADH secretion. One key factor to consider is the search for new metastasis when patients present with recurrent acute or acute on chronic hyponatremia.