Severe Syndrome of Inappropriate Antidiuretic Hormone Secretion with Docetaxel Treatment in Metastatic Breast Cancer

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We report the case of a 76-year-old female with metastatic breast cancer who developed severe syndrome of inappropriate antidiuretic hormone secretion (SIADH) during treatment with docetaxel.

Our patient was first diagnosed with receptor-negative lobular breast cancer T2N1M0 in August 1996. Breast-conserving surgery was performed, the patient receiving standard irradiation and adjuvant chemotherapy with anthracyclines and cyclophosphamide. In October 1998, when bone metastases developed, the patient was started on tamoxifen and iv-bisphosphonates. By January 1999 the disease had progressed and treatment was therefore changed to a palliative regimen with weekly docetaxel 35 mg/m^2. This chemotherapy was well tolerated, apart from PNP (peripheral neuropathy) Grade II, onychosis and staining of the finger tips. The patient had a history of non-insulin-dependent diabetes, hypertension and malignant melanoma. Concomitant medication consisted of a β-blocker, thiazide diuretics, an oral antidiabetic, metamizole, metoclopramide and a benzodiazepine.

After administration of 8 courses of docetaxel the patient was admitted as an emergency case to our hospital in precomatose condition on 4 June 1999. Prior to admission she had suffered from increasing dyspnoea, physical weakness, fatigue and apathy for several days. Laboratory findings showed marked hyponatraemia with 106 mmol/1, hypochloridaemia with 68 mmol/1 and low plasma-osmolality with 225 mosmol/kg. Other laboratory evaluations revealed normal thyroid, renal and adrenal function. Initially, the patient received two normotonic saline infusions and was then treated with fluid restriction, potassium-substitution and loop-diuretics. Sodium levels started to rise slowly: from 106 (day 1) to 107 (day 2) to 111 (day 3) to 118 (day 4) to 121 (day 5), finally reaching 134 mmol/1 (day 10). Fortunately there were on central cerebral complications, such as pontine myelinolysis. A CT-scan of the brain showed on sign of metastases or cerebral oedema. The patient recovered with minimal neurological sequelae—such as slowing of speech and generally reduced alertness—and was discharged after 3 weeks. The antineoplastic regimen was changed to an oral treatment with an aromatase inhibitor and a bisphosphonate. Regarding electrolyte disturbance, the patient remains well up to the present time.

This patient developed a sever form of SIADH, which fulfilled the criteria, as described by Bartter & Schwartz in 1967 (1). SIADH is common in small cell lung cancer (SCLC), but only rarely has been described for breast cancer (2, 3). Apart from a paraneoplastic origin, SIADH can be caused by CNS disorders, benign lung disease and drugs. Some chemotherapeutic agents such as cyclophosphamide and Vinca-alkaloids have been associated with SIADH in isolated cases, thiazide diuretics and sulphonyl urea have also been reported in association with SIADH (4, 5). However, in this case the last-mentioned drugs are unlikely candidates for causing SIADH, as they had been taken by the patient for several years prior to this acute, serious event.

In this patient it is possible that treatment with docetaxel led to the development of SIADH. To our knowledge this is the first reported case of SIADH related to taxine chemotherapy in breast cancer. Clinicians should be aware of the possibility that docetaxel could cause SIADH, even in cases where the underlying cancer is not SCLC.

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