Case Reports

# SIADH associated with ovarian immature teratoma: a case report

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**Summary**

Immature teratoma in association with SIADH is rare. A 17-year-old patient presented with a pelvic mass and serum sodium concentration of 121 mmol/l. Subsequent investigation confirmed SIADH and grade 2 ovarian immature teratoma. No other causes of SIADH were found apart from the immature teratoma. There was no further recurrence of SIADH after the curative surgery and chemotherapy. We postulate that immature teratoma consists of neurohypophyseal structures which account for the ADH release.

*Key words:* SIADH; Immature teratoma; Sodium.

## Introduction

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is a common electrolyte disturbance encountered in malignant conditions, albeit rarely in ovarian immature teratoma. This condition should be suspected in any patient with the cardinal features of hyponatremia, hypoosmolality and a urinary osmolality greater than that appropriate for the concomitant plasma hypotonicity, in the absence of clinical evidence of fluid volume depletion. Normal function of kidneys, adrenal and thyroid glands is a part of the definition [1]. This article presents the second case of such a rare biochemical presentation in ovarian immature teratoma, and highlights the plausible pathogenesis.

## Case report

A 17-year-old student presented with lower abdominal pain and distension of one month's duration. She enjoyed good past health and had menarche two years before with normal menstruation. The patient reported no bowel or urinary symptoms.

After admission, a firm and immobile pelvic mass extending to the epigastrium was noted. An ultrasound examination confirmed a mass measuring over 20 cm in diameter, with multiple thick septa and solid areas. The right ovary could not be identified while the uterus and left ovary appeared normal.

Serum sodium concentration was 127 mmol/l (normal range 135-149 mmol/l) with low serum osmolality of 247 mOsm/kg. Her serum urea and creatinine were 3.0 mmol/l and 59 μmol/l respectively. Glucose and cholesterol levels were normal. Alpha-fetoprotein (AFP) was substantially elevated at 310 ng/ml (normal range 0-10 ng/ml) and CA125 was 795 U/ml (normal range < 35 U/ml). Radiographs of the chest were normal. Serum sodium level further decreased to 121 mmol/l with a concomitant urinary osmolality of 298 mOsm/kg (Figure 1). The patient remained awake and fully oriented, but complained of nausea and vomiting. Her mental status, cranial nerves and deep tendon reflexes were all normal. There were no signs of dehydration or ankle edema. On the third hospital day, fluid restriction and sodium supplement were commenced with a clinical picture of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

Laparotomy was performed eight days later and a 25-cm right ovarian tumor with intact capsule was found. There were two tumor deposits at the pouch of Douglas. Otherwise, the left ovary, uterus and omentum were normal. This was followed by a right salpingo-oophorectomy and frozen section examination revealed immature teratoma. Final histology confirmed the disease of Stage 2B, grade 2 immature teratoma. No sodium supplement was required after the operation (Figure 1).

She then received adjuvant combination chemotherapy (bleomycin, etoposide and cisplatin). After one year of follow-up, she had no tumour recurrence and experienced no further hyponatremic episodes. Her latest serum sodium concentration was 141 mmol/l.

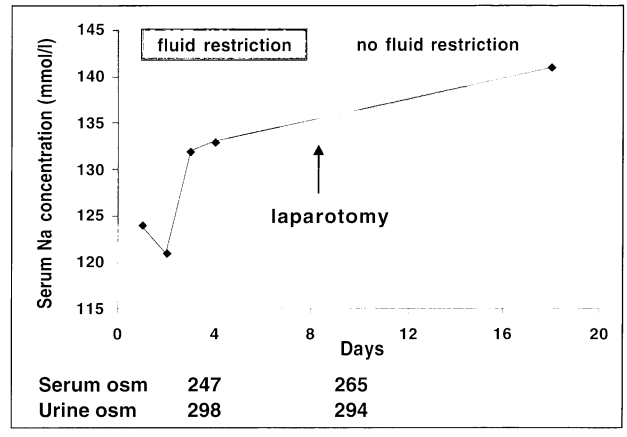


Figure 1. — Trend of serum and urine biochemistry.

## Discussion

The syndrome of inappropriate secretion of antidiuretic hormone was first described in two patients with lung malignancy and continued urinary sodium excretion [2]. Small cell carcinoma of the lung remains the most

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common and classical example of SIADH with neoplasm. It has also been described, albeit more rarely, in cancer of the pancreas, thymus, prostate, head and neck tumours and sarcomas [1, 3]. Evidence for ectopic (non-hypothalamic) tumour hormonal antidiuretic hormone (ADH) production has come from in vitro tissue analysis, in which neoplastic cells were demonstrated to synthesize, store and release ADH.

To the best of our knowledge, our case represents the second report of immature teratoma of the ovary in association with SIADH after the initial literature documentation in another Chinese patient [4]. Malignant ovarian germ cell tumours occur more commonly among Asians than Caucasians. Of the various germ cell tumours, immature teratomas account for less than one-fifth, and they occur usually in the first two decades of life as in our case [5]. Microscopic examination of immature teratoma consistently reveals a disorderly mixture of tissues with a neuroectodermal element. Although we did not test for the presence of ADH within the ovarian tumour tissue by bio- or radioimmunoassay, it would be tempting to hypothesize ectopic production of ADH within the teratoma neural tissue. The pluripotency of teratoma gives credence to such hypothesis [6]. Furthermore, the presence of pituitary components in other ovarian teratomas has been documented previously [7, 8]. Conceivably, most immature teratomas might have lost the ability to secrete hormones, which may explain the rarity of their presentation with SIADH.

Regardless of the primary pathology, fluid restriction remains the mainstay of treatment [9]. Unless the underlying cause of SIADH can be corrected, by surgery as in the present case and that reported previously [4], the fluid management strategy should be continued in order to maintain a normal serum sodium level. Resolution of SIADH after resection of the ovarian tumours, without further sodium requirement and fluid restriction measures, provided convincing evidence of the postulation of immature teratoma causing SIADH.

Autonomous release or action of ADH following treatment of primary neoplasm, on the other hand, may signify an alternative source for this disorder of excessive ADH. Indeed, an earlier report [10] documented another case of SIADH in a girl with malignant teratoma. The electrolyte disorder developed after vincristine chemotherapy, which was thought to be the causative agent of hyponatremia. Vincristine increases neurohypophyseal ADH release by unknown mechanisms, and subsequent hyponatremia is dose-related [11, 12].

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