**SELF ASSESSMENT ANSWERS**

**Hyponatraemia in a lady with a pelvic mass**

Q1: What is the best explanation for her biochemical picture?

The biochemical results are compatible with the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). The patient displays the cardinal features of hyponatraemia, hypo-osmolality, and a urinary osmolality greater than that appropriate for the concomitant plasma hypo-osmoticity, in the absence of any clinical evidence of fluid volume depletion. Normal function of the kidneys, adrenal and thyroid glands is a part of the definition.

Q2: List the possible causes

Drug related SIADH as a complication of CHOP chemotherapy is the most likely etiology. Certain pharmacological agents can cause hyponatraemia and antidiuresis by releasing vasopressin, and others can cause it by potentiating ADH action. Cyclophosphamide and vincristine are the most likely causes of hyponatraemia in this setting. Given the clinical response to chemotherapy and the timing of the hyponatraemia, SIADH related to the neoplasm is considered less likely here. Furthermore, chemotherapy induced nausea, a potent stimulus of ADH release, was not present in the current case.

Q3: What is the management?

Regardless of the primary pathology, fluid restriction (to less than the sum of the urinary and insensible losses) remains the mainstay of the management strategy for SIADH.1 Once negative water balance is achieved, the serum sodium concentration will gradually increase. To restore a normal serum sodium level, sodium replacement is sometimes warranted. After fluid restriction and salt administration in this case, the biochemistry gradually improved (fig 1). No further recurrence of SIADH was noted with subsequent cycles of chemotherapy.

Any underlying disease responsible for the syndrome should be identified and targeted. Tumour removal, for instance, would be essential if there is evidence of SIADH in direct association with the malignancy.

It should be emphasised that the asymptomatic hyponatraemic state in this patient, as is usually the case, would not justify any rapid initial correction of the serum sodium concentration.

**Discussion**

The current diagnostic criteria for SIADH remain essentially as proposed by Bartter and Schwartz in 1967.2 As shown in this case, the cardinal requisite for the diagnosis of SIADH is fulfilled when the urine is more concentrated than appropriate for the given solute and water intake: a urine which is less than maximally dilute in the presence of plasma hypotonicity provides clear evidence that there is a defect in water excretion.

SIADH as a complication in an oncology scenario deserves emphasis. In fact, the syndrome of inappropriate secretion of antidiuretic hormone was first described in two patients with bronchial carcinoma and continued urinary sodium excretion.3 While tumour induced SIADH is more often encountered,4 autonomous release or action of ADH after treatment of the primary neoplasm may signify an alternative source for this disorder of excessive ADH.5 This is well illustrated in this case in which cyclophosphamide and vincristine are both putative causes of SIADH.

Vincristine increases neurohypophyseal ADH release by unknown mechanisms and the subsequent hyponatraemia is dose related.6 Furthermore, among a database involving 39 reports of SIADH associated with vincristine, 35 were Asians, suggestive of an Asian predisposition for a hyponatraemic complication from this vinca alkaloid drug.7

However, the timing of the hyponatraemia after chemotherapy in this case is more typical of a cyclophosphamide effect. It has been demonstrated that the antidiuretic effect of cyclophosphamide is delayed with a time course that parallels the excretion of the alkylating agent’s active metabolites.8 The time lag between the development of the hyponatraemia and the administration of the chemotherapy therefore favours the mechanism of cyclophosphamide mediated by its active metabolites.

Other cytotoxic drugs that have also been associated with SIADH include vinblastine, cisplatin, ifosfamide (synthetic analogue of cyclophosphamide), melphalan, and levocabastine.9

**Final diagnosis**

This is the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) secondary to chemotherapy, with the cyclophosphamide and vincristine being implicated.

**Learning point**

We should be alert to the diversity of the metabolic complications from any underlying malignancy, and more importantly, the sequelae of any subsequent chemotherapy. The role of vincristine and the delayed effect of cyclophosphamide may be the reasons for the SIADH.

References


Multiple pulmonary nodules: diagnosis in an young afebrile patient

Q1: What is the diagnosis?
Pulmonary hydatid disease.

Q2: What is the differential diagnosis?
Differential diagnosis of multiple pulmonary nodules includes neoplastic, infective, immunological, and vascular causes (see box 1).

Q3: How does this condition present?
The majority of intact pulmonary cysts are known to produce no symptoms or are occasionally responsible for a non-productive cough or minimal haemoptysis. However, when the cyst ruptures, an abrupt onset of cough, haemoptysis, sputum, fever, chest pain, expectoration, and life threatening anaphylactic reaction may develop. The expectations of membrane and/or hydatid sand is a clinically diagnostic indicator of ruptured hydatid cyst.

Other symptoms may arise such as Horner’s syndrome from superior sulcus cysts, bone pain from posterior mediastinal cysts, dyspnoea from tracheal compression, haemorrhage due to erosion of great vessels, and tension pneumothorax.

Q4: What other tests should be performed?
These include serological tests, sputum examination, and abdominal sonography. Serological studies in hydatid disease includes complement fixation tests, indirect haemagglutination test, and Casoni’s intradermal test. Positive serology is said to be lower in children and high in adults. Microscopic examination of the sputum may reveal hooklet from scolices.

Abdominal sonography is required to look for other possible sites of involvement—liver, spleen, pancreas, and kidneys.

Q5: What is the pathogenesis of the crescent sign?
The crescent or meniscus sign is produced when an enlarging cyst eventually erodes the bronchioles and with coughing and straining air may be introduced between the pericyst and endocyst, producing a radiolucent air shadow in the form of crescent meniscus.

Q6: What are other causes of the crescent sign?
This sign is highly reliable, but not pathognomonic of hydatid cyst because a similar appearance may be produced by intracavitary fungus ball, blood clot, pulmonary gangrene, and air cap within a tumour.

Final diagnosis
Pulmonary hydatid disease.

References

Box 1: Differential diagnosis of multiple pulmonary nodules
- Metastasis.
- Abscess.
- Hydatid cyst.
- Fungal infection.
- Wegener’s granulomatosis.
- Rheumatoid nodule.
- Septic embolus.
- Arteriovenous malformation.
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