

Adipsia in a Diabetes Insipidus Patient

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Key Words

Diabetes insipidus · Adipsia · Cerebral tumor

Abstract

Central diabetes insipidus is a very common disorder after brain surgery or/trauma or even in the presence of brain inflammatory diseases. Polyuria and polydipsia are the clinical markers, but sometimes clinical situations are presenting with no thirst. These are not frequent but are life-treating conditions. Diagnosis is not easy, and for this reason some cases are treated late. We describe here a very infrequent oncological case of dangerous adipsic diabetes insipidus in a young girl who survived.

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Case Presentation

A hypothalamic lesion was diagnosed in a 13-year-old girl. She complained of headaches, and RNM revealed a tumor mass occupying the whole hypothalamic area. She underwent a biopsy, and the histological examination showed a germinal cell tumor (fig. 1, fig. 2). She was treated with radiotherapy above the skull and medullae with 30 and 24 Gy, respectively. Also, a 24-Gy boost was done above the tumor. She did not undergo chemotherapy.

At 13.58 years of age, the patient was examined at our endocrinology outpatient clinic. She was very weak, lethargic, and was neither hungry nor thirsty. She had a height of 137 cm and a weight of 26.4 kg, corresponding to -3.16 SDS and -4 SDS, respectively, and she did not have any pubertal signs. Bone age was delayed 2 years.

At first analyses we found the following: hypernatremia in several days (165/170/159, $n = 136$ – 145 mmol/l); hypercloremia (123/117/124, $n = 98$ – 107 mmol/l); normal urine osmolality (630/720, $n = 300$ – 900 mOsm/kg); low thyroid hormones (TSH 0.66 μ UI/ml, FT4

0.71, $n = 0.9$ – 1.7 ng/dl); high plasma osmolality (325/309, $n = 275$ – 295 mOsm/kg); low gonadotropins (LH 0.1 mUI/ml, FSH 0.51 mUI/ml); low ACTH (12.8 pg/ml) for plasma cortisol level (6.6 μ g/dl) and urine level (17.3 μ g/24 h, $n = 21$ – 85); undetectable somatomedin 1, and elevated prolactin (37.9 ng/ml). Potassium and calcium levels were normal. Panhypopituitarism was biochemically evident.

The patient did not listen to our claims for drinking and eating, so we had to insert an endogastric tube. She began eating, drinking water, and taking medication: levotiroxine (0.1 μ g/kg/day), hydrocortisone (0.05 mg/kg/day), and desmopressin with dosages slowly increasing up to 0.72 mg/day. She also started taking chlorpromazine 6.25 mg/day. After 1 month, the tube was removed and she began eating and drinking by herself. Water electrolytic balance was now completely normal as were thyroid hormones and cortisol levels.

At 16 years of age she started growth hormone treatment with 0.08 mg/kg/week and growth velocity improved considerably (from 0 cm to 5 cm/year).

After 18 months of treatment, she is quite well, with energy, a collaborative mood and is doing her scholar works with great strength. The tumor has disappeared.

After the attainment of a better stature, we hope to start estrogen therapy with the aim of getting sexual morphology and maturation.

Discussion

Central diabetes insipidus (CDI) is caused by a deficiency of arginine vasopressin, an antidiuretic hormone. Patients manifest polyuria, which usually is compensated with increasing water intake. However, some patients are not able to sense thirst due to hypothalamic osmoreceptor destruction and so they do not feel the need to drink. These patients develop a very dangerous dehydration which may cause their death.

Zantut-Wittmann et al. [1] described a case of very dangerous, severe rhabdomyolysis due to an adipsic hypernatremia, and Arima et al. [2] reviewed 149 patients with CDI and found 23 patients with adipsia. A total of 6 patients died during the follow-up, and 4 of them were adipsic, and infections were pointed out as a potential final aggression.

Hypernatremia is an electrolytic abnormality with very difficult understanding and above all without the occurrence of thirst. In a recent national survey in Denmark, Di Iorgi et al. [3] showed a prevalence rate of 23 CDI patients per 100,000 inhabitants in 5 years. Timely diagnosis for initiating specific treatment is very important because damage of the central nervous system is a very probable consequence. In the presence of adipsia, diagnosis is more challenging and many times difficult to achieve.

In a study by González Briceño et al. [4] of 159 patients, only 2 had hypernatremia due to adipsia.

Treatment includes forcing water intake because low vascular volume promotes a high sodium concentration and cellular lesion. Sometimes it is necessary recurring to a nasogastric tube or a vascular infusion. In our case, we tried chlorpromazine because it seems to improve osmoreceptor sensibility, as it was proposed by Malossi [5]. In fact, with a very low dosage we reached a better patient collaboration and with cumulative desmopressin treatment we were able to hydrate the girl. Rising osmotic reactivity by hypothalamic cells by chlorpromazine have been demonstrated several years ago by Allen et al. [6].

Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have nothing to declare.

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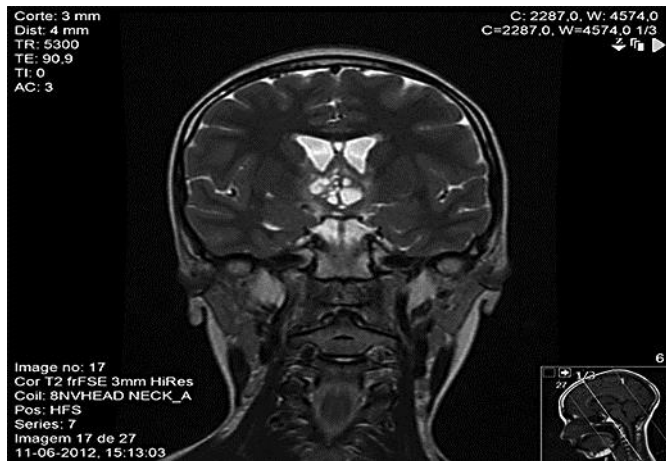


Fig. 1. Axial MRI (coronal view).



Fig. 2. Axial MRI (flair view).