CASE REPORT

Adipsic diabetes insipidus secondary to craniopharyngioma resection

María Alejandra Pérez,^{1,2} Henry Augusto Millán,¹ Julián Alberto Naranjo,² Andrés Flórez Romero³

¹Internal Medicine, Fundación Cardioinfantil, Bogotá, Colombia
²Internal medicine, Fundación Cardioinfantil, Bogotá, Colombia
³Fundación Cardioinfantil Instituto de Cardiología, Bogotá, Colombia

Correspondence to
 Dr María Alejandra Pérez,
 mariaperezardila@gmail.com

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SUMMARY

Adipsic diabetes insipidus is an infrequent disease which may be associated with craniopharyngioma. It may be secondary to the tumour's extension, as well as to resection of the mass. We present the case of a 24-year-old woman with a history of delayed puberty and hypothyroidism, but no prior study reports. She consulted due to a headache with warning signs associated with altered visual acuity. Brain MRI was performed which showed signs of a non-adenomatous lesion with suprasellar and hypothalamic extension. Following transcranial surgery, she developed diabetes insipidus criteria, with absence of thirst documented during the hospitalisation. The histopathological findings confirmed the diagnosis of craniopharyngioma. The patient was treated with desmopressin and received recommendations regarding rehydration according to the quantification of losses, with electrolyte stabilisation.

BACKGROUND

Central diabetes insipidus (DI) is characterised by decreased vasopressin secretion, with partial deficiency, or in more severe cases, complete deficiency, leading to a hyperosmolar state and increased serum sodium. However, patients with preserved thirst mechanisms have polydipsia, maintaining normal or high normal sodium levels.^{1,2}

The diagnosis of postoperative central DI is based on a diuresis greater than 2 mL/kg/hour or more than 2.5–3 L in 24 hours and a urinary density <1.005.³

Adipsic DI is a variant in which, following osmoreceptor damage in the circumventricular region, patients lose their thirst mechanism and have an increased risk of complications due to hypernatraemia.^{4,5} Approximately 100 case reports have been presented in the literature.⁵ Since it is rare, the publication of cases with this diagnosis is pertinent in order to allow a better characterisation and understanding of this disease. Following, we present the case of a patient with adipsic DI secondary to resection of a sellar lesion with suprasellar and hypothalamic extension, corresponding to an adamantinomatous craniopharyngioma.

CASE PRESENTATION

A 24-year-old female patient from Bogotá, Colombia consulted due to a 1-year history of oppressive, intermittent headaches, without an

identified trigger, of moderate intensity, which improved partially with analgesics, and were associated with decreased visual acuity. She consulted due to worsening of the headaches, which would wake her at night.

The patient had a history of primary hypothyroidism diagnosed 1 year prior to this consult, and was being treated with levothyroxine at 50 µg per day. On gynaecological history, she experienced menarche, thelarche and pubarche when she was 18 years old; and after she began treatment with oral contraceptives at age 21, her menstrual cycles normalised.

On physical examination, she had normal anthropometric measurements, Tanner 4 breasts and pubic hair, and bitemporal hemianopsia without altered visual acuity on the neurological examination. A 7 mm diameter nodule with a fibroelastic consistency was palpated in the left lobe of the thyroid.

On admission, the patient had a report of brain MRI (figure 1) performed the month prior to her emergency room consult, which described an infra-sellar and suprasellar mass with greater suprasellar involvement, extending posteriorly to the retro-clival region, with a non-homogeneous signal intensity with isointense, hypointense and hyperintense areas in T1 sequences, and hyper intensity and hypointensity in FLAIR (Fluid-attenuated inversion recovery) and T2 sequences. It had a predominantly peripheral irregular enhancement in the contrast phase and maximum dimensions of 43×44 mm, with a pressure effect on the hypothalamus.

The admission hormone profile (table 1) showed diminished ACTH (Adrenocorticotrophic hormone) 1.5 pg/mL, together with diminished cortisol: 1 µg/dL, suppressed TSH (Thyroid stimulating hormone): 0.07 mIU/mL and FT4 within the normal range: 1 ng/dL. Growth hormone, prolactin, follicle-stimulating hormone (FSH) and luteinising hormone (LH) levels were normal (table 1). Electrolyte levels were normal, with serum sodium at 139 mEq/L; urinalysis was not performed prior to the procedure.

The patient was diagnosed with panhypopituitarism and treatment was begun with hydrocortisone 50 mg every 8 hours intravenously prior to tumour resection.

Intraoperative findings during the transcranial resection were described as an extensive sellar, parasellar and suprasellar lesion which reached to the third ventricle and posterior fossa, with solid

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Figure 1 Image of intrasellar and suprasellar mass with greater suprasellar involvement, extending posteriorly to the retroclival region. Non-homogeneous signal intensity with isointense, hypointense and hyperintense areas on T1 sequences; hyperintensity and hypointensity on FLAIR and T2 sequences and predominantly peripheral irregular enhancement in the contrast phase. Approximate maximum dimensions of 43×44 mm. Adequate individualisation of the optic chiasm and optic nerves was not achieved. The lesion is associated with the interpeduncular region, with retroclival extension and a compressive effect on the pontomesencephalic brainstem.

and cystic components and calcifications. A total macroscopic resection of the lesion was performed. The microscopic study revealed that it was an adamantinomatous craniopharyngioma.

During the first 24 hours after surgery, the patient presented polyuria with a 7 cm³/kg/hour urine output associated with decreased urinary density (1.002) and hypernatraemia (152 mEq/L). She was in intensive care unit (ICU) and receiving nothing by mouth; therefore, intravenous rehydration was begun along with desmopressin 2 µg subcutaneously every 12 hours, achieving partial improvement of the urinary output (1 cm³/kg/hour) and hypernatraemia (144 mEq/L).

After being discharged from the ICU, she continued with 1:1 replacement of free water, depending on urinary losses. After 2 days of postoperative care, the patient was discharged with a sodium level of 144 mEq/L and a urinary output of 0.9 cm³/kg/hour, with an order for continued desmopressin 2 µg subcutaneously every 12 hours, levothyroxine 50 µg and prednisolone 5 mg per day.

The patient was readmitted 48 hours later due to a headache associated with fever; she had meningeal irritation signs along with signs of dehydration. A lumbar puncture was performed, and the cytochemical analysis of the cerebrospinal fluid (CSF) reported pleocytosis with neutrophil predominance, normal protein and hypoglycorrhachia; with a Gram stain showing no visible organisms. Antibiotic coverage was begun due to suspected meningitis; the CSF culture was negative. In addition, serum sodium was 164 mEq/L. A new brain MRI showed a frontal epidural haematoma with mass effect.

During her hospitalisation, the signs of meningeal irritation and inflammatory response markers progressively decreased

until the infectious process resolved. However, despite progressive adjustments to the desmopressin dose (table 2), the sodium levels could not be controlled. The doses were administered correctly by recording them in the hospital system. When asked about thirst, the patient denied having this symptom, in spite of having hypernatraemia (166 mEq/L) and a urine output of 900 cm³/hour. This was documented on the Analogue Thirst Scale (figure 2).

Adipsic DI was diagnosed and the patient and her family were educated regarding the necessary oral rehydration according to urinary losses, even in the absence of thirst. This intervention achieved progressive improvement in the sodium levels.

INVESTIGATIONS

Previously described.

DIFFERENTIAL DIAGNOSIS

The most common causes of adipsic DI are: craniopharyngioma (13%–30%),^{5 6} anterior communicating artery aneurysm rupture or clipping (14%–30%),^{5 6} and congenital causes such as encephalocoele, holoprosencephaly, agenesis of the septum pellucidum or dysgenesis of the corpus callosum. Other causes to keep in mind, which are less frequent, include neurosarcoidosis, macroprolactinoma, haemangioma and infectious causes.^{4 6}

In our case, in view of the craniopharyngioma diagnosis and its extension in the initial MRI, a broad tumour resection was performed, with DI presenting postoperatively.

Initially, it was assumed that the hypernatraemia with which the patient was readmitted was due to the infectious process and concomitant dehydration. However, after the infection was resolved, the patient continued to have inadequate electrolyte control associated with a lack of thirst.

TREATMENT

The patient was discharged with an order for orodispersible desmopressin 60 µg in the morning and 120 µg at night, levothyroxine 50 µg and prednisolone 5 mg per day. She was discharged once her serum sodium levels were normalised and her polyuria resolved.

In this patient, a complete resection was performed, which is why the neurosurgery team decided not to perform radiotherapy. In follow-up with images at 6 months, there were no lesions indicating recurrence.

OUTCOME AND FOLLOW-UP

The patient required several educational sessions regarding the importance of continuing desmopressin treatment. She was instructed on fluid intake aimed at titration according to urinary output, climate changes such as higher temperatures, exercise and increased intake in the event of an intercurrent infectious process.

Four months after discharge, the patient continues receiving outpatient follow-up, being treated with desmopressin 120 µg every 8 hours, with urinary osmolarity of 275 mOsm/kg, serum sodium of 146 mEq/L and urine output of 2 L per day, reporting thirst beginning the second month after surgery. Her neurological examination still shows bitemporal hemianopsia, along with bradypsychia.

DISCUSSION

The main aetiology of central DI is idiopathic, followed in frequency by tumours and head trauma.⁷ In young

Table 1 Admission hormone profile

Hormone	Patient value	Normal value
Somatomedin C	103 ng/mL	107–367 ng/mL
Basal prolactin	9.82 ng/mL	4.79–23.3 ng/mL
ACTH	1.5 pg/mL	7.2–63.3 pg/mL
Growth hormone	0.07 ng/mL	Women up to 8 ng/mL
Follicle-stimulating hormone	4.12 mIU/mL	1.7–7.7 mIU/mL luteinising
Luteinising hormone	2.17 mIU/mL	1–11.4 mIU/mL luteinising
PM cortisol	1 µg/dL	2.9–17.3 µg/dL
TSH	0.07 mIU/mL	0.35–4.94 mIU/L
FT4	1 ng/dL	0.7–1.48 ng/dL

Table 2 Desmopressin dose used during hospitalisation and its relationship with sodium levels, urine density in isolated sample and urine output

Date (Surgery 1 November 2017)	Serum sodium levels (mEq/L)	Urine density	Desmopressin dose
4 November 2017	140	1.020	2 µg every 12 hours subcutaneous
6 November 2017	164	1.008	2 µg every 12 hours subcutaneous
8 November 2017	158	1.004	60 µg every 12 hours oral
10 November 2017	166	1.005	60 µg every 12 hours oral
12 November 2017	158	1.032	60 µg every 8 hours oral
14 November 2017	172	1.029	60–120 µg every 12 hours oral
16 November 2017	158	1.025	60–120 µg every 12 hours oral
18 November 2017	150	1.024	60–120 µg every 12 hours oral
20 November 2017	147	1.020	60–120 µg every 12 hours oral

adults, craniopharyngioma is the main tumour, followed by suprasellar germinoma. Suprasellar adenomas rarely cause central DI.¹

Vasopressin secretion may be altered following resection of tumours in the sellar or suprasellar region, due to hypoperfusion or direct cellular damage, most commonly developing transient DI (<6 months duration). The frequency depends on the type of tumour and the surgical approach.⁸ A triphasic pattern has been described in 1.1% of cases, in which the polyuric phase occurs in the first 24 hours to 5 days, due to the inhibition of vasopressin secretion secondary to hypothalamic dysfunction. The antidiuretic phase follows in the next 6–11 days, in which the posterior pituitary releases all residual hormones; after this, the neurohypophysis reserves are depleted and DI develops, generally permanently (>6 months duration).^{1,9,10} A biphasic pattern associated with DI and subsequent syndrome of inappropriate antidiuresis may present in 15.7% of cases.

The adipsia unlikely can occur with syndrome of inadequate antidiuresis instead DI or even without defect in vasopressin secretion.^{11,12} These cases are rare, but it is important to know, since the management is different to DI.

The main hormone deficit in craniopharyngioma is growth hormone (35%–95%), followed by FSH/LH (38%–82%) and corticotropin (21%–62%). Antidiuretic hormone deficit occurs in 6%–38% of cases^{13,14}; following resection, the prevalence may be 70%–90%.¹

Other sequelae after resection include hypothalamic dysfunction, which may manifest with an altered sleep cycle, sleep

respiratory disorders, autonomic dysfunction and thermoregulatory disorders.¹⁵

Although DI is usually a common complication in patients with craniopharyngioma presenting in 61% of patients, adipsic DI represents a more infrequent complication, in the review made by the authors, 20 patients were found who presented this pathology secondary to craniopharyngioma.^{16–24} No associations were found in the literature with a particular type of histopathological pattern.

The presence of adipsia implies damage to the osmoreceptors located in the circumventricular region of the hypothalamus.⁴ The risk factors for hypothalamic lesions are: presentation in children, hypothalamic disorder manifestations at diagnosis, hypothalamic invasion, height of the tumour greater than 3.5 cm from the medial line, multiple attempts to remove the tumour adhering to the hypothalamus and a hypothalamic radiation dose greater than 51 Gy.¹⁴

Adipsic DI is an infrequent but highly complex disease.⁵ Patients diagnosed with adipsic DI must show an inappropriate thirst response to osmotic stimulation (serum osmolarity >310 mOsm/kg) or hypernatraemia (sodium >150 mEq/L), which can be achieved by withholding water or administering a hypertonic solution.^{4,6} It may also be confirmed using a Visual Analogue Scale on which patients determine their thirst level on a scale from 0 to 10, with 0 being no thirst and 10 being severe thirst.⁵

The complications associated with adipsic DI include hypernatraemia, venous thromboembolism (13%), obstructive

Figure 2 Visual analogue scale on which the patient stated feeling no thirst. Taken with serum sodium: 166 mEq/L, urine density: 1.004 and urine output: 500 cm³/hour.

sleep apnoea (50%) and obesity (20%–46%).^{4–6} Behan *et al*'s study compared the percentage of hypernatraemia in patients with DI with preserved thirst versus that in patients with adipsic DI, finding ambulatory hypernatraemia in 18% of the patients with adipsic DI and in 1.8% of patients with DI, with a $p < 0.0001$.²⁵

Treatment includes desmopressin therapy; this treatment should be combined with patient education in order to maintain an adequate fluid intake. In our case, a progressive increase in the dose of desmopressin was required, possibly due to a complete absence of antidiuretic hormone, low absorption due to tissue hypoperfusion secondary to dehydration and adipsia. Daily weight checks during eunatremia allow the detection of patients who are non-compliant with the instructions. They should be trained to drink at least 2 L of water per day, but the amount should vary depending on the climate, insensible losses, exercise, infections or weight changes.⁴

It has been found that the pharmacodynamic and biological effects of the different administration routes of desmopressin are similar.^{26 27} In the study by Fjellestad-Paulsen *et al* in hydrated individuals, an increase in urinary density was found after 8 hours of administration in all routes, with the exception of sublingual and intrarectal routes.²⁷ The delay in the improvement of urinary density and hypernatraemia was due to an absence of precise correction of the losses presented by the patient, its state of volemia could be underestimated in the absence of the mechanism of thirst.

In Ogawa *et al*'s study, where 44 patients with a diagnosis of craniopharyngioma requiring surgical management with pituitary stalk resection were followed, all the patients required postoperative treatment with desmopressin, which was able to be discontinued in 23 of the 44 patients within a time period ranging from 1 to 136 weeks.²⁸ In adipsic DI, postoperative recovery of the thirst mechanism has been reported 4–9 months later, suggesting the existence of neuronal plasticity in vasopressin regulation.¹⁹

In the patient, no subsequent radiotherapy was performed, without evidence of recurrence at 6 months in follow-up with cerebral magnetic resonance, this because the resection was complete and the risk of inducing a hypopituitary lesion was considered. Regarding the possibilities of endocrine alterations induced by radiotherapy, Karavitaki *et al*¹⁴ reported no differences in the presentation of DI between subgroups of total resection or partial resection and radiotherapy, especially when the preoperative hormonal levels were normal. One of the complications associated with radiotherapy includes hypopituitarism, which is more common in paediatric patients and with radiation doses greater than 61 Gy.²⁹

One of the complications not associated with endocrine alteration in craniopharyngiomas is chemical meningitis. During the fever episode, the cultures were negative, so the diagnosis of aseptic meningitis was made, with the limitation of not having carried out studies for viral aetiologies. Cases of chemical meningitis have been reported due to rupture of the cystic content of the craniopharyngioma^{30 31}; however, they usually occur prior to the procedure. In our case, there was no reported rupture of the contents during the resection, nor did the patient present a cystic lesion after the procedure since the resection was complete, so the probability that the episode of meningitis is associated with the craniopharyngioma content is low.

Learning points

- Considering the prevalence of diabetes insipidus, as well as other endocrine disorders in patients with pituitary lesions such as adenomas and craniopharyngiomas, a complete pituitary hormone assessment should be performed prior to surgery. In the case of diabetes insipidus, altered serum sodium, urine density and osmolality should alert to the diagnosis.³² Postoperative management includes monitoring for surgical and endocrine complications, which, once again, include diabetes insipidus.³²
- Once this disease has been diagnosed and treatment began, the presence of thirst should be monitored, especially in patients with risk factors for hypothalamic lesions. This defence mechanism against osmolality and sodium concentration challenges constitutes one of the treatment lines, and therefore, its loss should direct patient education towards fluid control.

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