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【 CASE REPORT 】

Central Diabetes Insipidus after Syndrome of Inappropriate Antidiuretic Hormone Secretion with Severe Hyponatremia in a Patient with Rathke's Cleft Cyst

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Abstract:

A 49-year-old man developed severe hyponatremia associated with transient headache and was diagnosed with syndrome of inappropriate antidiuretic hormone secretion (SIADH). Fluid restriction and sodium supplementation corrected the hyponatremia. However, several days later, the patient exhibited hypernatremia with thirst and polyuria. A detailed examination indicated central diabetes insipidus (CDI) with an intrasellar cystic lesion indicative of Rathke's cleft cyst (RCC). A case of RCC exhibiting headache, hyponatremia, and subsequent hypernatremia has been reported. Our case shows that CDI may appear after SIADH in patients with RCC, especially in those with serum sodium levels that unexpectedly increase rapidly beyond the reference range.

Key words: arginine vasopressin, central diabetes insipidus, desmopressin, Rathke's cleft cyst, syndrome of inappropriate antidiuretic hormone secretion, headache

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Introduction

Rathke's cleft cysts (RCCs) are benign sellar and/or suprasellar cystic lesions containing mucoid material originating from the remnants of Rathke's pouch (1, 2). RCCs are usually asymptomatic; however, they present with symptoms when they are large enough to compress surrounding structures or are associated with inflammation extending into adjacent tissues. Common symptoms include headache, visual disturbances, and endocrinopathies.

Arginine vasopressin (AVP) is an antidiuretic hormone produced in the hypothalamus and stored in the posterior lobe of the pituitary gland. AVP acts principally on the renal collecting tubules to increase water reabsorption. Impaired AVP secretion can cause water-electrolyte balance disorders, such as central diabetes insipidus (CDI) and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

CDI is characterized by a large amount of dilute urine due to a deficiency of AVP (3, 4). Clinical manifestations of CDI include thirst, polydipsia, hypotonic polyuria, and hypernatremia. In contrast, SIADH is characterized by water retention and dilutional hyponatremia due to excessive AVP secretion (5). SIADH can cause symptoms of hyponatremia, such as fatigue, appetite loss, nausea, and vomiting.

Both CDI and SIADH are relatively rare manifestations of RCC (6, 7). Furthermore, a case of RCC exhibiting headache, hyponatremia, and subsequent transient hypernatremia has been reported, with combined SIADH and CDI suggested as a possible etiology for the unusual manifestation of a rapid transition from hyponatremia to hypernatremia; however, AVP measurements were not provided (8).

We herein report an unusual case of a patient with RCC who developed headache and SIADH with severe hyponatremia followed by persistent CDI.

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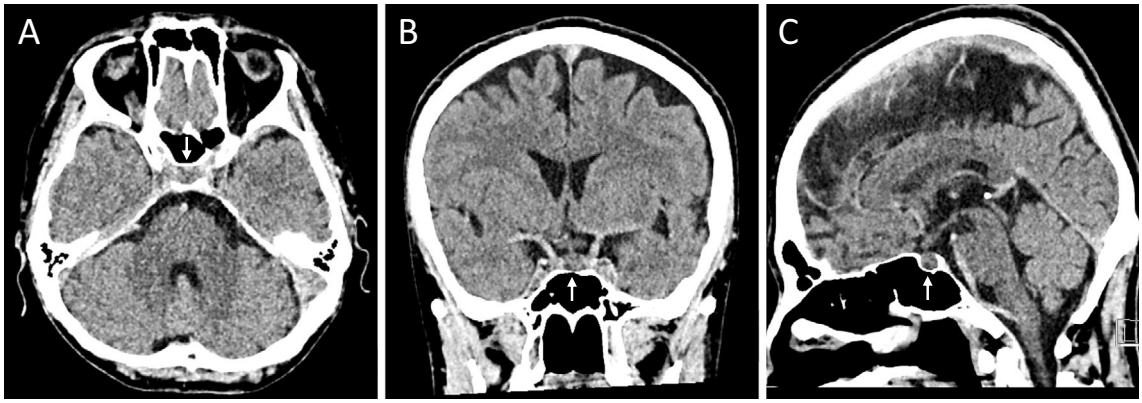


Figure 1. Computed tomography of the head (March 2019). Images in the transverse (A), coronal (B), and sagittal (C) planes.

Case Report

A 49-year-old Japanese man was admitted to our hospital in early April 2019 complaining of thirst, polydipsia, and polyuria. His personal and family medical history were unremarkable. The patient developed frontal headache (specifically, a gradually aggravating, throbbing pain in the forehead) in the morning in early March 2019. His headache worsened, and he developed nausea, appetite loss, and fatigue in the evening of the same day. The next morning, the patient visited the Neurosurgery Department of Uonuma Kikan Hospital. He was clear-headed. His height and weight were 169 cm and 56 kg, respectively. His body temperature was 36.7°C. No neurological abnormalities, such as paralysis or numbness, were found. Brain computed tomography (CT) detected no abnormalities in the cerebrum, cerebellum, or brain stem. No hemorrhaging, apoplexy, or calcification were observed in the intrasellar or suprasellar region. The pituitary gland was mildly enlarged (Fig. 1); however, this was not considered a cause of the patient's headache. His headache was treated with a non-steroidal anti-inflammatory drug (loxoprofen) for seven days. Although his headache improved, the nausea, appetite loss, and fatigue persisted and worsened. Furthermore, the patient experienced repetitive vomiting and revisited the same hospital in mid-March 2019. Because a blood test revealed severe hyponatremia, he was admitted the same day.

At admission, the patient's body weight, body temperature, blood pressure, and pulse rate were 56 kg, 36.6°C, 129/88 mmHg, and 77 beats/min, respectively. He did not present with symptoms or signs of dehydration, including thirst or dry mouth and skin. No edema was present in his extremities. The laboratory findings (Table 1) showed low levels of serum sodium (112 mEq/L) and plasma osmolality (Posm; 229 mOsm/kg) and relatively high plasma AVP levels (2.2 pg/mL) and urinary osmolality (Uosm; 514 mOsm/kg). Plasma cortisol levels (15.8 μg/dL) were normal. Chest and abdominal CT revealed abnormalities in the lungs, liver, pancreas, kidneys, and adrenal glands.

The patient received infusion therapy with normal saline; however, his serum sodium level measured 1 h later was reduced (110 mEq/L). He was diagnosed with SIADH. After receiving a 3% saline infusion (Fig. 2), his serum sodium levels increased to approximately 120 mEq/L, and he subsequently took oral sodium chloride (6 g/day). The patient was instructed to restrict his fluid intake to 800 mL/day. His fatigue, appetite loss, nausea, and vomiting resolved along with improved hyponatremia. His serum sodium level measured on day 4 of admission increased to 136 mEq/L. The dose of oral sodium chloride was decreased to 2 g/day, and he was discharged.

The day after discharge, the patient developed thirst, polydipsia, and polyuria, and the next day, he revisited the hospital. A blood test showed a high serum sodium level (147 mEq/L). Oral sodium chloride was discontinued but the hypernatremia persisted; the patient was admitted to the hospital's department of Endocrinology and Metabolism in early April 2019.

Upon a physical examination, his body weight, temperature, blood pressure, and pulse rate were 53 kg, 36.7°C, 129/96 mmHg, and 82 beats/min, respectively. His mouth was mildly dry. No thyromegaly, heart murmur, chest rale, or peripheral edema were detected. The 24-h urine volume was elevated (4,200 mL/day). Abnormal neurological findings, such as an impaired visual acuity or visual field defect, were not detected. The laboratory findings (Table 1) showed high levels of serum sodium (149 mEq/L) and Posm (298 mOsm/kg) and relatively low levels of plasma AVP (0.9 pg/mL) and Uosm (73 mOsm/kg).

A desmopressin stimulation test showed increased Uosm with reduced urine volume and increased urinary aquaporin-2 levels (Table 2A), indicating CDI.

Dynamic function tests for the anterior pituitary showed normal secretion of prolactin, thyroid-stimulating hormone, growth hormone, adrenocorticotropic hormone, luteinizing hormone, and follicle-stimulating hormone (Table 2B, C). A rapid adrenocorticotropic hormone stimulation test revealed a normal secretory reserve of cortisol (22.1 μg/dL) 60 min after the intravenous administration of tetracosactide acetate

Table 1. Laboratory Findings.

	2019 Mid-March	2019 Early April	
Hematology			
Red blood cells (×10⁴/μL)	516	426	(435-555)
Hemoglobin (g/dL)	16.6	14.0	(13.7-16.8)
Hematocrit (%)	44.7	41.3	(40.7-50.1)
White blood cells (/μL)	4,300	4,200	(3,300-8,600)
Platelets (×10⁴/μL)	12.6	17.4	(15.8-34.8)
Blood chemistry			
Total protein (g/dL)	7.0	7.4	(6.6-8.1)
Albumin (g/dL)	4.7	4.7	(4.1-5.1)
Uric acid (mg/dL)	3.6	7.1	(3.7-7.8)
Urea nitrogen (mg/dL)	10.7	9.5	(8.0-20.0)
Creatinine (mg/dL)	0.65	0.68	(0.65-1.07)
Sodium (mEq/L)	112	149	(135-145)
Potassium (mEq/L)	4.5	3.7	(3.5-4.8)
Chloride (mEq/L)	80	109	(98-108)
C-reactive protein (mg/dL)	0.36	0.02	(<0.30)
Triglycerides (mg/dL)	86	119	(50-149)
Casual plasma glucose (mg/dL)	107	96	(70-139)
Plasma osmolality (mOsm/kg)	229	298	(275-290)
Endocrinology			
Arginine vasopressin (pg/mL)	2.2	0.9	*
Thyroid-stimulating hormone (μIU/mL)	N.D.	1.87	(0.50-5.00)
Free thyroxine (ng/dL)	N.D.	1.10	(0.90-1.70)
Free triiodothyronine (pg/mL)	N.D.	2.96	(2.30-4.00)
Adrenocorticotropic hormone (pg/mL)	N.D.	17.7	(7.2-63.3)
Cortisol (μg/dL)	15.8	9.8	(7.1-19.6)
Dehydroepiandrosterone sulfate (ng/mL)	N.D.	2,369	(700-4,950)
Plasma renin activity (ng/mL/h)	0.5	1.4	(0.1-2.0)
Aldosterone (ng/dL)	11.4	8.2	(3.0-15.9)
Urinary chemistry			
Urinary osmolality (mOsm/kg)	514	73	(50-1,300)
Urinary sodium (mEq/L)	82	39	(88-285)
Urinalysis			
Specific gravity	N.D.	1.001	(1.005-1.030)
Glucose	N.D.	Negative	
Protein	N.D.	Negative	
Occult blood	N.D.	Negative	

Blood samples were taken in the morning with the patient in the supine position.

The reference range for each parameter is shown in parentheses.

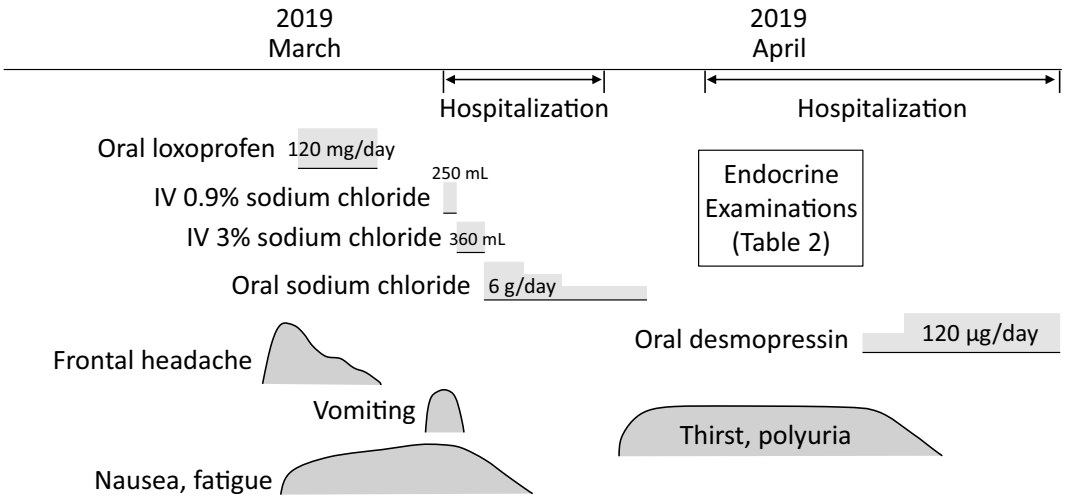
*The reference range for the plasma arginine vasopressin level is dependent on the plasma osmolality (5).

N.D.: not determined

(0.25 mg). Serum levels of insulin-like growth factor-1 (172 ng/mL, reference range: 88-246 ng/mL) and free testosterone (14.6 pg/mL, reference range: 4.7-21.6 pg/mL) were normal.

Magnetic resonance imaging (MRI) of the brain (Fig. 3) showed no abnormalities in the cerebrum, cerebellum, or brain stem but revealed a 9-mm cystic lesion in the midline behind the anterior lobe of the pituitary, with the superior margin of the cyst lying behind the junction point of the infundibular stalk in the sagittal view, without a shift in the infundibular stalk on the coronal view, indicative of RCC. No mechanical compression of the infundibular stalk, hypothalamus, or optic nerve was observed. In terms of the posterior pituitary, the normal high-intensity physiological signal (9) was not detected on T1-weighted imaging.

The patient was started on oral desmopressin at 60 μg/day to treat CDI on day 6 of admission. The dose was adjusted to relieve his thirst, polydipsia, and dilute polyuria, and his symptoms resolved after taking 120 μg/day (twice daily) of desmopressin. Regarding the treatment plan for his RCC, surgical treatment was considered a potentially effective option to prevent future development of additional pituitary defects, but it may not necessarily resolve CDI (1, 2, 10). After the patient was informed of the treatment options, he



Time (day)*	1	5	10	16	17	18	19	20	21	28	31	34	37	40
Serum sodium (mEq/L)				112	118	129	136	147		149	146	146	141	142
Posm (mOsm/kg)				229				295		298	297	294	289	289
Uosm (mOsm/kg)				514				125		73	95	97	417	350
Plasma AVP (pg/mL)				2.2				1.3		0.9	0.8	0.9		
Serum creatinine (mg/dL)				0.65			0.85	0.75		0.68	0.71	0.72	0.69	0.66
Serum uric acid (mg/dL)				3.6				5.6		7.1	7.1	7.0	6.8	6.7

Figure 2. Clinical course of the patient. *The day on which the patient developed frontal headache is defined as day 1. Blank columns indicate that the laboratory parameters were not determined. AVP: arginine vasopressin, Posm: plasma osmolality, Uosm: urinary osmolality

chose conservative treatment. The patient was continued on oral desmopressin and discharged at 13 days after admission.

His RCC was closely followed up with MRI; the results obtained in July 2019, October 2019, and March 2020 indicated a decrease in the size of the RCC together with dramatic changes in the signal intensity from the RCC (high to low intensity on T2-weighted imaging at three months after the development of CDI) (Fig. 3).

Laboratory tests performed in October 2020 after a brief discontinuation of desmopressin revealed high levels of sodium (146 mEq/L) and Posm (294 mOsm/kg), and relatively low levels of plasma AVP (0.7 pg/mL) and Uosm (139 mOsm/kg). The patient experienced thirst and polyuria during the tests. He was considered to have persistent CDI, and oral desmopressin (120 µg/day) was continued.

The patient's clinical course during desmopressin therapy for CDI has been uneventful for more than 1.5 years, with no headache or anterior pituitary dysfunction.

Discussion

The differential diagnoses of hyponatremia associated with intracranial disorders include SIADH and cerebral salt-wasting syndrome (CSWS). CSWS is characterized by renal salt loss (natriuresis) leading to hyponatremia and extracellular fluid volume depletion; water and sodium replacement is the mainstay of treatment (11). In contrast, in SIADH, increased renal water reabsorption results in dilutional hyponatremia, and fluid restriction is the treatment of choice. Although it is often difficult to distinguish between SIADH and CSWS, given the clinical and laboratory similarities, the extracellular fluid volume status is the distinguishing feature. Our patient did not experience significant weight loss or symptoms of dehydration when he was severely hyponatremic. Fluid restriction effectively reversed the hyponatremia. Thus, SIADH was the most likely diagnosis. In addition, as non-steroidal anti-inflammatory drugs are known to induce hyponatremia by increasing renal water reabsorption via potentiated effects of AVP as a result of inhibition of renal prostaglandin synthesis (12), the drug the patient received to treat his headache may have exaggerated the hyponatremia.

Several days after resolution of the hyponatremia (Fig. 2), the patient unexpectedly developed hypernatremia with thirst and polyuria and was diagnosed with CDI accompanied by a small intrasellar cystic lesion.

The differential diagnoses of an intrasellar cystic lesion include neoplastic craniopharyngioma, arachnoid cyst, cystic pituitary adenoma, and RCC. Radiological investigation with MRI can be useful for differentiating among these disorders (1, 2, 13-15). Craniopharyngiomas, solid or mixed solid-cystic tumors arising from nests of epithelium along the infundibular stalk, generally occur in the suprasellar region and are often associated with calcium deposits. MRI shows the reticular enhancement of the solid portion. Arachnoid cysts are sacs filled with cerebrospinal fluid covered by arachnoidal cells and collagen, which are indicated by homogenous hyperintensity signals on T2-weighted MRI that

Table 2. Results of Endocrinological Investigations (April 2019).

A. Vasopressin stimulation test

	Time (min)				
	0	30	60	90	120
Urinary osmolality (mOsm/kg)	134	145	315	526	564
Urinary aquaporin-2 (ng/mL)	<0.35	<0.35	15.9	22.7	14.9
Urine volume (mL/30 min)	350	100	25	30	25
Plasma osmolality (mOsm/kg)	289	N.D.	291	N.D.	291

Vasopressin (5 units) was intravenously administered in the morning.
N.D.: not determined

B. Insulin tolerance test

	Time (min)			
	0	30	60	90
Plasma glucose (mg/dL)	93	28	67	85
Growth hormone (ng/mL)	0.48	0.96	4.70	2.42
Adrenocorticotropic hormone (pg/mL)	18.6	45.8	33.6	15.3
Cortisol (μg/dL)	11.6	14.0	18.4	14.4

Regular insulin (5 units) was intravenously administered in the morning.

C. TRH/LHRH stimulation test

	Basal	Peak (time at peak)	
Thyroid-stimulating hormone (μIU/mL)	1.99	14.26	(30 min)
Prolactin (ng/mL)	5.8	23.6	(15 min)
Luteinizing hormone (mIU/mL)	3.3	16.2	(60 min)
Follicle-stimulating hormone (mIU/mL)	5.4	9.9	(90 min)

Blood samples were taken before (basal) and at 15, 30, 60, 90, and 120 min after intravenous administration of thyrotropin-releasing hormone (TRH; 500 μg) and luteinizing hormone-releasing hormone (LHRH; 100 μg) in the morning.

are similar to those of cerebrospinal fluid. Cystic pituitary adenomas are often located off the midline and exhibit internal septations. T2-weighted imaging may show the fluid-fluid level or a hypointense rim. RCCs are usually found between the anterior and posterior lobes of the pituitary gland. MRI reveals well-demarcated homogenous lesions of variable intensity that are highly dependent on the cyst contents, which can range from clear, cerebrospinal fluid-like fluid to thick, mucoid material. In the present case (Fig. 3), the midline location behind the anterior lobe of the pituitary and a well-demarcated homogenous intensity signal suggested RCC to be the most likely diagnosis.

Table 3 summarizes the characteristics of a previously reported patient with RCC who exhibited headaches, hyponatremia, and subsequent hypernatremia and those of our patient. The time from the occurrence of headache to the development of hypernatremia varied between the cases. No anterior pituitary dysfunction or adrenal insufficiency was detected in either patient. In the previous case report, the authors speculated that the rapid transition from hyponatremia to hypernatremia was associated with enhanced release and subsequent loss of AVP due to mechanical stimulation and compression of AVP-producing cells from an enlarged intrasellar and suprasellar RCC; however, the AVP level was not determined (8). In the present case, a rise and rapid fall in plasma AVP levels were observed when the patient exhibited a rapid transition from hyponatremia to hypernatremia associated with a small intrasellar RCC leading to the diagnosis of combined SIADH and CDI.

AVP is synthesized by magnocellular neurosecretory cells in the hypothalamic supraoptic and paraventricular nuclei and then descends via axons that pass through the infundibular stalk and terminate in the posterior pituitary, where AVP is stored in vesicles until it is released into the blood circulation. The loss of magnocellular cells results in CDI. CDI associated with RCC is often accompanied by posterior pituitary inflammation (neurohypophysitis) (16). Furthermore, in a clinical and pathological study of patients with RCC and CDI, the onset of CDI with headache without anterior pituitary dysfunction was suggested to be strongly associated with rupture of the cyst wall adjacent to the posterior pituitary (17). In our case, the onset of headache was associated with an enlarged pituitary detected by CT (Fig. 1). Approximately three weeks later (following the development of CDI after SIADH), MRI revealed a small intrasellar RCC without compression of the infundibular stalk or hypothalamus (Fig. 3D); although the anterior pituitary gland was visible, no T1-weighted high-intensity signal from the posterior pituitary (9) was evident. Within three months, the MRI signal intensity in the RCC changed dramatically

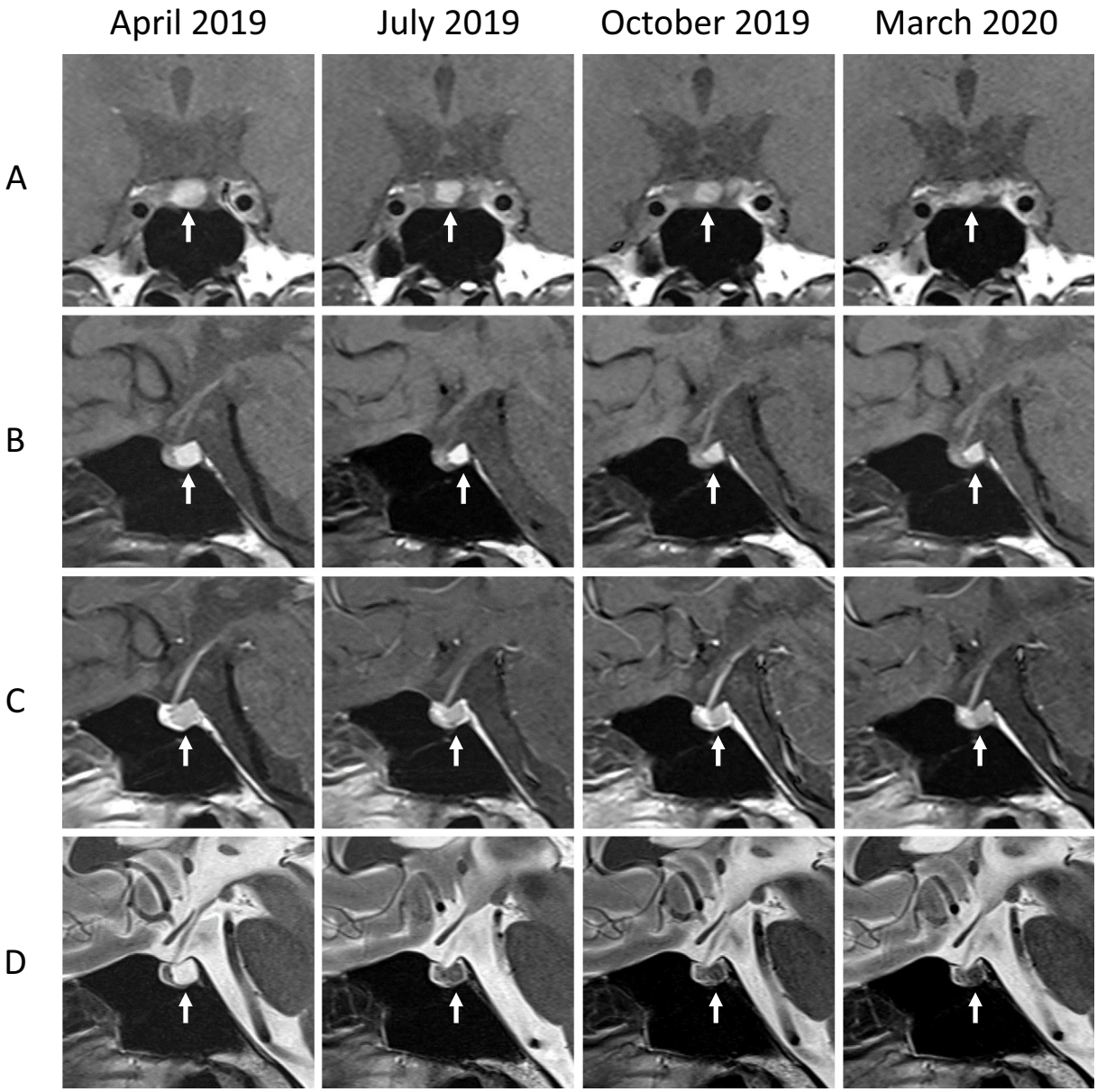


Figure 3. **Serial magnetic resonance imaging of the pituitary gland. A: T1-weighted sagittal, B: T1-weighted coronal, C: gadolinium-enhanced T1-weighted sagittal, and D: T2-weighted sagittal images. The greatest diameter of the patient's pituitary cystic lesion at different timepoints was 9 mm (April 2019), 7 mm (July 2019), 7 mm (October 2019), and 7 mm (March 2020).**

from high to low on T2-weighted imaging with the RCC shrinking, indicating the amelioration of RCC inflammation. These findings imply that RCC content first oozed into the posterior pituitary via a ruptured cyst wall, causing headache, neurohypophysitis, and SIADH with the acute release of pooled AVP into the circulation. As inflammation ascended through the magnocellular axons, SIADH persisted due to AVP released from the degenerating axons. Finally, CDI developed as a result of magnocellular cell loss due to inflammation.

Various patterns of water-electrolyte balance disorders occur after neurosurgery. A typical pattern is a triple-phasic course comprising initial acute CDI, subsequent SIADH, and eventual chronic CDI (18). The initial phase of CDI is due to the inhibition of AVP secretion induced by damage to the infundibular stalk (magnocellular axons). In the second phase, (approximately seven days after surgery), SIADH develops as a result of AVP release from degenerating terminal axons in the posterior pituitary distal to the lesion and from axons proximal to the lesion that undergo ascending degeneration. In the third phase, (approximately 14 days after surgery), CDI reoccurs due to loss of magnocellular cells. This triple-phasic pattern has been observed after head trauma and in patients with RCC complicating hypophysitis (19). If we compare the clinical course of combined SIADH and CDI in our patient with the post-neurosurgery triple-phasic response, increased AVP release followed by decreased secretion of the hormone due to magnocellular cell loss can be seen to be a common underlying phenomenon. However, an important difference is that, in our patient, the initial insult

Table 3. Summary of Reported Cases of Rathke's Cleft Cyst Presenting with Headache, Hyponatremia, and Subsequent Hypernatremia.

Ref.	Age (years)	Sex	RCC			Hyponatremia			Period from headache to development of hypernatremia	Hypernatremia				Treatment
			Size (mm)	Location	Anterior pituitary dysfunction	Serum sodium (mEq/L)	Uosm (mOsm/kg)	Plasma AVP (pg/mL)		Serum sodium (mEq/L)	Uosm (mOsm/kg)	Plasma AVP (pg/mL)	Duration	
(8)	18	Female	12	Intra- and supra-sellar	[-]	129	346	N.D.	9 days	152	52	N.D.	Transient (several days)	Surgery for RCC
Present case	49	Male	9	Intrasellar	[-]	112	514	2.2	19 days	149	73	0.9	Persistent (>1.5 years)	Medication (desmopressin for CDI)

AVP: arginine vasopressin, CDI: central diabetes insipidus, N.D.: not determined, RCC: Rathke's cleft cyst, Uosm: urinary osmolality

probably resulted from chemical damage to the posterior pituitary caused by leaked RCC content, which may have induced the acute release of AVP, potentially explaining the absence of the initial phase of CDI. Furthermore, the time for the axons to degenerate from the posterior pituitary to the magnocellular cell bodies may explain the eventual development of CDI (19 days after the occurrence of headache) in our patient.

In conclusion, we encountered a patient with an intrasellar RCC who developed transient headache and SIADH with severe hyponatremia followed by persistent CDI. This case highlights the need for physicians to be aware that CDI may appear after SIADH in patients with RCC, especially in those with serum sodium levels that unexpectedly increase rapidly beyond the reference range.

The authors state that they have no Conflict of Interest (COI).

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