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Authors: Akashi, Yoriko, Park, Young Tae, Oetelaar, Garrett S, and Murakami, Masahiro

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Yoriko Akashi¹, Young Tae Park¹, Garrett S Oetelaar^{2,3} and Masahiro Murakami³

Abstract

Case summary A 2-year-old female spayed domestic shorthair cat was presented with a history of collapse, possible hypodipsia/adipsia, severe dehydration and hypernatremia. MRI of the brain revealed a failure of separation of the cerebral hemispheres as characterized by an absence of the rostral part of the corpus callosum, fornix and septum pellucidum and the presence of a single fused lateral ventricle. A diagnosis of hypodipsic/adipsic hypernatremia with lobar holoprosencephaly was made. Dietary management of the cat's condition was attempted by increasing oral water intake, but the cat's hypernatremia and azotemia persisted. Plasma arginine vasopressin (AVP) analysis revealed a low concentration of circulating AVP (2.3 pg/ml), prompting therapy with oral desmopressin in addition to the dietary management. This combined therapy decreased water consumption of the cat from 200 ml/day (85 ml/kg/day) to 100 ml/day (30 ml/kg/day), normalized plasma sodium concentration and resolved the azotemia.

Relevance and novel information To our knowledge, this is the second case report of an MRI diagnosis of lobar holoprosencephaly with hypodipsic/adipsic hypernatremia in a cat and the first case report of the successful management of this condition using oral desmopressin. This case report emphasizes that holoprosencephaly should be suspected in cats presented with hypodipsic/adipsic hypernatremia and highlights the utility of MRI in establishing the diagnosis. Measurements of plasma osmolality and AVP concentration corroborate the pathophysiology and support the use of oral desmopressin in addition to dietary management to resolve the hypernatremia.

Keywords: Holoprosencephaly; hypodipsic/adipsic hypernatremia; magnetic resonance imaging; oral desmopressin treatment

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Case description

A 2-year-old female spayed domestic shorthair cat was presented to the referral veterinary hospital with a 2-week history of weight loss, lethargy, collapse and severe dehydration. The cat was hospitalized for pancreatitis at another clinic 2 months prior. At that time, plasma biochemistry revealed hypernatremia (169 mmol/l; reference interval [RI] 155–165), elevated alanine aminotransferase (356 U/l) and hyperlipemia (>1000 U/l). Serologic tests for feline leukemia virus (FeLV) and feline immunodeficiency virus performed by ELISA (SNAP FIV/FeLV Combo Test; IDEXX Laboratories) were negative at the clinic. The cat was treated with supportive care, including intravenous (IV)

fluids, before being discharged. Additional history provided by the owner indicated that the cat had always displayed a lack of interest in spontaneous water intake.

¹Ve C Jiyugaoka Animal Medical Center, Meguro City, Tokyo, Japan

²VCA Canada CARE Centre, Calgary, AB, Canada

³Department of Veterinary Clinical Sciences, Purdue University, West Lafayette, IN, USA

Corresponding author:

Masahiro Murakami BVSc, MS, PhD, DACVR, Department of Veterinary Clinical Sciences, Purdue University, 625 Harrison St, West Lafayette, IN 47907, USA
Email: mmuraka@purdue.edu



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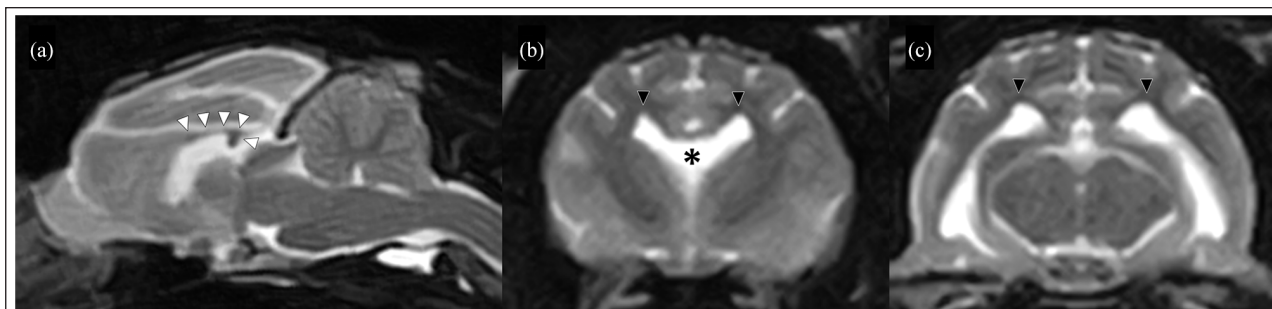


Figure 1 (a) Midsagittal and (b,c) transverse T2-weighted MRI of a 2-year-old female spayed domestic shorthair cat with primary hypodipsic/adipsic hypernatremia. White arrowheads outline the dorsal and caudal aspects of the corpus callosum. The rostral portion (genu and rostral body) of the corpus callosum is absent. The rostral portions of the lateral ventricles are fused resulting in a single ventricle (*). The dorsal corners of the fused lateral ventricle are upturned and pointed (black arrowheads)

On the initial presentation at the referral hospital (day 1), physical examination revealed depression, minimal responsiveness to noxious stimuli, hypothermia (37.7°C), tachycardia (180 beats/min), eupnea (24 breaths/min) and dehydration. The cat was cachectic, with a body weight of 2.36 kg and a body condition score of 1/9. Systolic blood pressure measured using a Doppler blood pressure device (PetMAP G3; Ramsey Medical) was within the RI (110 mmHg; systolic values).¹ Complete blood count (pocH-100i Automated Hematology Analyzer; Sysmex) showed a stress leukogram. Plasma biochemistry (Dri-Chem 4000v chemistry analyzer; Fujifilm) showed hypernatremia (217 mmol/l; RI 147–156), hyperchloremia (165 mmol/l; RI 107–120), normokalemia (3.5 mmol/l; RI 3.4–4.6), elevated creatinine (2.2 mg/dl; RI 0.8–1.8) and elevated blood urea nitrogen (BUN; 71.4 mg/dl [RI 17.6–32.8]). Plasma osmolality (calculated using the formula $2 \times [\text{Na}] + [\text{BUN}] / 2.8 + [\text{glucose}] / 18$) was increased at 466.9 mOsm/kg (RI 308–335), indicating hyperosmolar hypernatremia. IV fluid therapy was initiated using lactated Ringer's solution supplemented with potassium to manage the cat's hypernatremia and hyperosmolality. Fluid rate was modified to gradually decrease the plasma sodium concentration at a rate of 1 mEq/l/h. Survey radiographs of the thorax and abdomen were unremarkable. Abdominal ultrasonography showed no abnormalities and the maximum dorsoventral thicknesses of both adrenal glands were within normal limits (left: 3.3 mm; right: 3.8 mm).

Based on the history, physical examination, blood work and diagnostic imaging, differential diagnoses for the severe hypernatremia in this cat included primary hypodipsia/adipsia or possibly hypotonic fluid loss due to renal dysfunction.

Renal parameters returned to within the RIs in 24 h. There was mild improvement in the hypernatremia (193 mmol/l) as planned, as well as persistent hyperchloremia (148 mmol/l) and hypokalemia (3.2 mmol/l).

Plasma sodium, chloride and potassium concentrations were gradually improved over the next 72 h. Serum thyroxine level was evaluated (Dri-Chem Immuno AU10V analyzer; Fuji) and was within normal interval (1.07 µg/dl; RI 0.9–3.7).

Intermittent seizure activity developed on day 4 and anticonvulsants (2.5 mg/kg PO q12h [Convase Tablet 25 mg; DS Pharma Animal Health]), as well as prednisone (1 mg/kg PO q24h [Predonine Tablets 5 mg; Shionogi]), were added to the therapy on day 5. As the cat's appetite improved, fluid therapy was discontinued and the cat was discharged on day 6 with a continuous prescription of anticonvulsants and prednisone. From day 7, the IV fluid therapy was changed to 60 ml subcutaneous fluids (lactated Ringer's solution), initially administered once daily and then gradually weaned to twice weekly. Even though the cat was eating normally, there was a lack of interest in spontaneous water intake, and the owner reported that this behavior had been present since the cat was acquired. The owner was instructed to feed wet and dry foods and to add water to the food (200 ml/day in divided dose five times daily). The cat remained asymptomatic if additional water was provided.

An MRI was performed on day 53 using a 0.4 Tesla open-magnet MRI scanner (APERTO Lucent; Hitachi Medical Systems). The imaging protocol included T2-weighted (T2W) sequences in transverse, sagittal and dorsal planes; T2W fluid-attenuated inversion recovery sequence in a transverse plane; T2*W sequence in a transverse plane; and T1-weighted sequences in transverse, sagittal and dorsal planes before and after IV administration of gadoteridol (0.1 mmol/kg [ProHance; Eisai]). MRI revealed dysgenesis of the corpus callosum (CC) characterized by the absence of the genu and rostral body of the CC (Figure 1a). The fornix and septum pellucidum were also absent, resulting in a single fused lateral ventricle (Figure 1b). On transverse images, the corners of the fused lateral

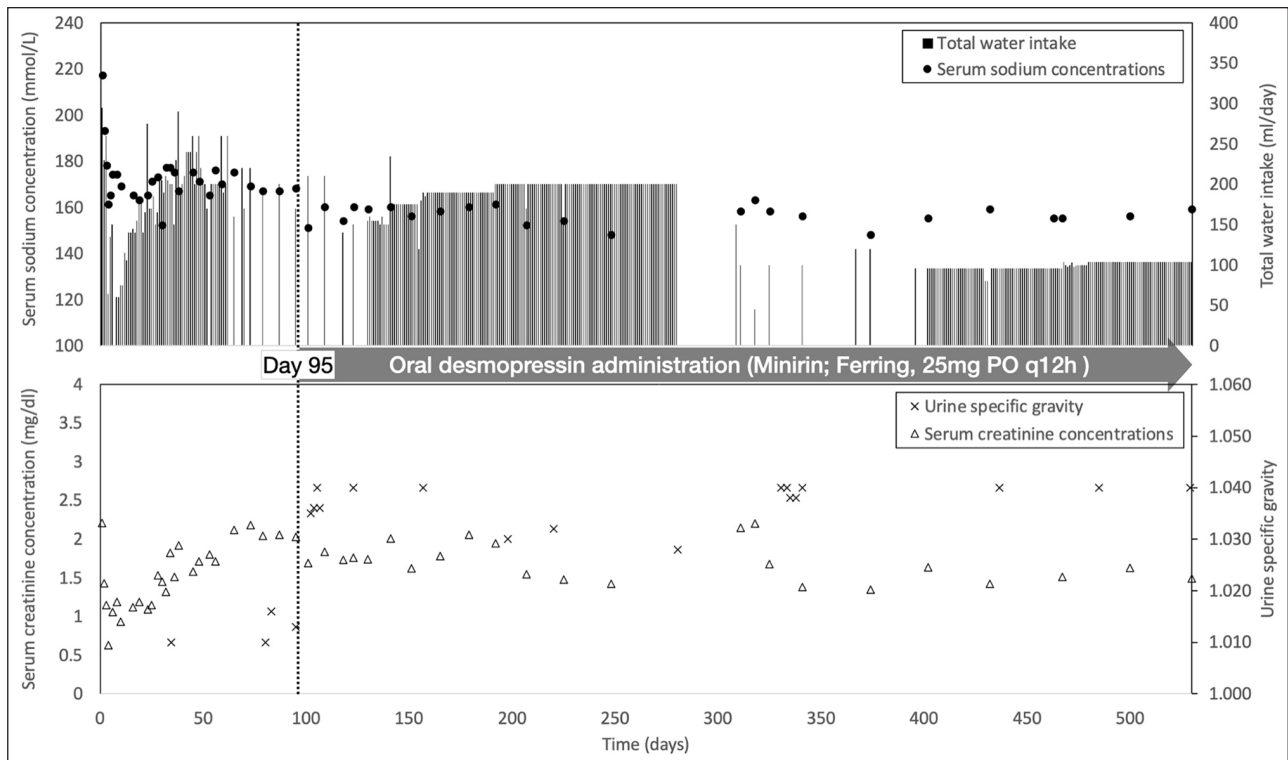


Figure 2 Changes in plasma sodium and creatinine concentrations, urine specific gravity (USG) and total water intake in a cat with primary hypodipsic/adipsic hypernatremia. Oral desmopressin administration (25 µg PO q12h) was started at day 95 (dotted line). After 1 week of oral desmopressin, plasma sodium and creatinine concentrations and USG improved, and the amount of water supplemented was gradually reduced

ventricle were upturned and pointed (Figure 1b,c). The olfactory bulbs were hypoplastic and the most rostroventral portions of the frontal neocortex were fused; the cingulate gyri, however, were well separated. Based on the MRI findings, a diagnosis of lobar holoprosencephaly (HPE) was made.

Prednisone was discontinued on day 56. Urine specific gravity (USG) measured on day 59 was 1.016 and the plasma sodium concentration and plasma osmolality remained high (170 mmol/l [RI 147–156] and >340 mOsm/kg, respectively). Therefore, on the same day, the concentration of plasma arginine vasopressin (AVP) was analyzed in the same blood sample used for plasma sodium concentration analysis and was determined to be low at 2.3 pg/ml.

On day 79, plasma renin activity (<2.0 ng/ml/h [RI 0.3–3])² and serum aldosterone concentration (154 pg/ml [RI 40–195])² were measured using radioimmunoassay at an authorized clinical laboratory (SRL, Tokyo, Japan). The aldosterone level was within the RI at 154 pg/ml (RI 40–195), which ruled out hyperaldosteronism; this value, along with the low plasma AVP concentration and high plasma osmolality, supported a diagnosis of central diabetes insipidus.

Oral administration of a vasopressin analogue (25 µg PO q12h desmopressin [Minirin, Ferring Pharmaceuticals]) was started on day 95. On day 95, before oral vasopressin administration, the plasma sodium level was measured and remained high (168 mmol/l [RI 147–156]). After 1 week of oral desmopressin therapy, plasma sodium (150 mmol/l [RI 147–156]) and chloride concentrations (113 mmol/l [RI 107–120]), creatinine (1.69 mg/dl [RI 0.8–1.8]) and USG (1.040) normalized.

The amount of water added to each meal was gradually reduced to 100 ml/day on day 311, with no adverse effect on plasma electrolytes, renal values or USG (Figure 2). The cat remained adipsic but clinically stable with desmopressin administration and dietary management at the time of manuscript preparation, 18 months from the first visit to our clinic.

Discussion

Hypernatremia may result from hypotonic fluid losses (eg, vomiting/diarrhea and diabetes mellitus), or may occur secondarily to impermeant solute gain (eg, salt poisoning and hyperaldosteronism) or pure water deficit (eg, primary hypodipsia/adipsia, diabetes insipidus, fever and inadequate access to water).³ In

the current case, primary hypodipsia/adipsia or hypotonic fluid loss related to kidney disease were initially considered as a cause of the hypernatremia; however, kidney disease was considered less likely to be the main cause of the hypernatremia as the USG normalized with desmopressin therapy. Hyperaldosteronism was excluded based on normal blood pressure, low serum aldosterone levels and the ultrasound evaluation of the adrenal glands. The cat's lack of interest in drinking, coupled with the low plasma AVP concentration and the MRI diagnosis of HPE, lead to the diagnosis of holoprosencephaly with hypodipsic/adipsic hypernatremia and central diabetes insipidus.

Hypodipsic/adipsic hypernatremia is a rare disorder characterized by chronic or recurrent episodes of hypernatremia associated with a lack of thirst and dehydration. In humans, hypodipsic/adipsic hypernatremia has been reported as an uncommon complication of semi-lobar HPE.^{4,5} In cats, it has been reported to occur with HPE,⁶ intracranial neoplasia,^{7,8} hydrocephalus,⁹ head trauma¹⁰ and, in two cases, an undetermined etiology.^{11,12} HPE is a congenital brain malformation that results from failure of the prosencephalon to sufficiently divide into two cerebral hemispheres. HPE is rare in veterinary medicine but has been reported in a cat due to a teratogenic malformation,¹³ a pair of conjoined kittens,⁶ a cat with concurrent pure red cell aplasia and FeLV infection,¹⁴ and as an isolated disorder in another.⁷ The anatomic characteristics of lobar HPE in humans include non-separated rostroventral portions of the cerebral hemispheres, absence of the rostral portion of the CC (typically rostrum and genu), rudimentary formation of the frontal horns, a fully formed third ventricle and hypoplastic olfactory bulbs.¹⁵ The MRI findings of the cat in the present study showed an absence or reduction in the size of midline prosencephalic structures (CC, septum pellucidum and fornix) and incomplete separation of lateral ventricles. These imaging findings are in agreement with those previously reported in cats with HPE.

Although it is suspected that HPE is associated with a defect in hypothalamic osmoreceptors, the relationship between HPE and dysfunctional osmoregulation is not completely understood. Several structures associated with thirst regulation exist in the neuroparenchyma rostral to the CC, which may explain the commonality of hypodipsia/adipsia in dogs with lobar HPE,¹⁴ as well as the presence of hypodipsia/adipsia in the cat reported here. By contrast, hypodipsia/adipsia is uncommon in humans with HPE, although this may be a reflection of the fact that HPE in humans most frequently results in the absence of the caudal portion of the CC.^{16,17}

Hypodipsic/adipsic hypernatremia can be present either alone or together with impaired AVP release (ie, central diabetes insipidus).⁶ The deficiency in AVP secretion often accompanies a defect in the osmotic

regulation of thirst.¹⁸ This is likely because the osmoreceptors that regulate AVP secretion and those that regulate thirst are located very close to each other anatomically.¹⁸ Simultaneous hypodipsic/adipsic hypernatremia and central diabetes insipidus have been reported to have variable causes in humans such as vascular abnormalities,¹⁹ neoplasms,^{20,21} trauma,²² surgery,²³ hydrocephalus²⁰ or congenital malformations,⁵ or even in the absence of structural lesions.²⁴⁻²⁶ In cats, central diabetes insipidus with adipsic hypernatremia has been reported secondary to HPE⁶ and intracranial B-cell lymphoma.²⁷

The plasma AVP concentrations in normal cats and cats with central diabetes insipidus have been reported.²⁸ Mean AVP concentrations in water-restricted normal cats and a water-restricted cat with diabetes insipidus were 84.6 pg/ml (84.6 ± 56 pg/ml) and 1.3 pg/ml, respectively.²⁸ Moreover, the AVP concentrations in the aforementioned cats with HPE and intracranial B-cell lymphoma were similar to those in the water-restricted cat with central diabetes insipidus (1.1 pg/ml in both cases).^{6,27} Based on these studies, the plasma AVP level of the cat in the present report (2.3 pg/ml) was considered low which, in conjunction with the high plasma osmolality, was consistent with a diagnosis of central diabetes insipidus. However, there may be a partial suppression of vasopressin secretion contributed by steroid administration²⁹ in the cat.

One treatment for veterinary patients with hypodipsic/adipsic hypernatremia is to increase the amount of water available in the diet.^{6,14} This treatment was employed in the current case but the hypernatremia and azotemia did not improve even with a large amount of water (200 ml/day; 66 ml/kg/day) mixed into food.

Desmopressin is a vasopressin analogue and is used, along with forced hydration, in the treatment of hypodipsic/adipsic hypernatremia with low AVP concentrations in human medicine.^{5,20,26}

Oral desmopressin administration has also been reported as a treatment for central diabetes insipidus in cats.^{30,31} In these cases, excessive water intake (polydipsia) was reduced and the amount of water intake was normalized. In the cat in the present study, oral desmopressin administration corrected the low USG and normalized both plasma sodium concentration and plasma osmolality, even with only small amounts of water added to the food (100 ml/day; 30 ml/kg/day). Thus, for patients with hypodipsic/adipsic hypernatremia whose plasma sodium concentrations are difficult to control, plasma AVP concentration should be analyzed to determine the applicability of oral desmopressin as an adjunct therapy.

Conclusions

To our knowledge, this is the first case report to document the use of oral desmopressin to successfully

manage a cat with hypodipsic/adipsic hypernatremia and central diabetes insipidus secondary to HPE. HPE in cats is a rare congenital disorder that can be associated with hypodipsic/adipsic hypernatremia. When hypernatremia with osmotic baroreceptor dysfunction is suspected in cats, measurement of plasma AVP concentration and MRI of the brain are recommended. Oral desmopressin therapy may help the long-term management of hypernatremia and dehydration in cats with low plasma AVP.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned) animals. Established internationally recognized high standards ('best practice') of veterinary clinical care for the individual patient were always followed. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained it is stated in the manuscript.

Informed consent Informed consent was obtained from the owner of the animals described in this work for all procedures undertaken. No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

ORCID iD Masahiro Murakami  <https://orcid.org/0000-0001-7816-8311>

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