

# Pituitary Teratoma Presenting as Central Diabetes Insipidus with a Normal MRI Finding

Young Soo Kim,<sup>1</sup> Seok Gu Kang,<sup>2</sup> and Young OK Kim<sup>1</sup>

<sup>1</sup>Division of Nephrology, Department of Internal Medicine and <sup>2</sup>Department of Neurosurgery, Uijeongbu St. Mary's Hospital, The Catholic University of Korea, Seoul, Korea.

Received: November 26, 2009

Revised: December 11, 2009

Accepted: December 11, 2009

Corresponding author: Dr. Young OK Kim,  
Division of Nephrology, Department of Internal  
Medicine, Uijeongbu St. Mary's Hospital,  
The Catholic University of Korea,  
Uijeongbu 480-717, Korea.

Tel: 82-31-820-3347, Fax: 82-31-847-2719

E-mail: cmckyo@catholic.ac.kr

· The authors have no financial conflicts of interest.

Dear Sir,

Central diabetes insipidus (DI) is a diverse condition characterized by polydipsia and polyuria due to a defect of arginine vasopressin. Known conditions for this disorder include inflammatory, autoimmune, traumatic brain injury, and brain tumors. Brain tumors such as craniopharyngioma<sup>1</sup> and germinoma<sup>2</sup> are associated with Central DI. Up to 50 percent of cases of central DI are thought to be idiopathic in nature.<sup>3,4</sup> Herein we describe an interesting case of a 17-year-old man who was diagnosed with central DI, initially thought to be idiopathic but found to have pituitary teratoma.

A 17-year-old male was admitted to our hospital with a 4-month history of polyuria and polydipsia with severe thirst. Initial laboratory work revealed serum BUN 8.4 mg/dL, creatinine 0.65 mg/dL, sodium 147 mEq/L, potassium 3.9 mEq/L, chloride 108 mEq/L, calcium 9.1 mg/dL, phosphate 4.6 mg/dL, fasting plasma glucose 109 mg/dL, and hemoglobinA1C 6.0%. A thyroid function test was normal. Plasma osmolality was 291 mOsm/kgH<sub>2</sub>O and urinary osmolality was 79 mOsm/kgH<sub>2</sub>O. His urine output was 4 to 5 liters per day without glycosuria.

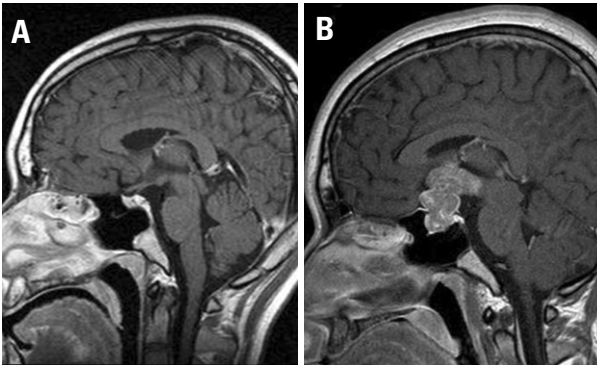
Urine osmolality increased from 115 to 297 mosm/Kg H<sub>2</sub>O after 6 hours of water deprivation. Two hours later, urine osmolality was still under 300 mosm/KgH<sub>2</sub>O (296 mosm/KgH<sub>2</sub>O) and the serum level of the antidiuretic hormone (ADH) level was < 0.4 pg/mL. After using nasal spray with 1-desamino-8-D-arginine vasopressin (DDAVP) 10 µg, his urine osmolality increased from 296 to 600 mosm/KgH<sub>2</sub>O.

His clinical course and laboratory finding led us to suspect Central DI. No abnormality was detected in a brain MRI (Fig. 1A). A complete work-up result to find the cause of Central DI was negative and his central DI was thought to be due to idiopathic cause.

He was treated with 10 µg of desmopressin nasal spray every night. His urine volume decreased to around 2 L/day. He was discharged with DDAVP and was followed monthly in an outpatient clinic.

After 45 months, he presented with a headache and diplopia persisting for 1 month. A brain MRI was completed, which showed a 3.3 cm sized lobulated solid mass involving the sellar and suprasellar area (Fig. 1B). He underwent a transphenoidal excision biopsy and the biopsy result was confirmed by pituitary teratoma. He received chemotherapy and radiation therapy.

Magnetic resonance imaging (MRI) is the best imaging technique for detecting subtle abnormalities of the hypothalamic-pituitary region.<sup>5</sup> Idiopathic central DI is a diagnosis of exclusion, and is diagnosed when central DI occurs in the absence of any alteration that is known to be responsible for DI.<sup>2,5</sup>



**Fig. 1.** (A) Initial brain MRI showed normal signal intensity in the posterior pituitary gland. (B) Follow-up MRI showed 3.3 cm sized lobulated solid mass on the sella and suprasellar area involving hypothalamus and optic chiasm.

Central DI can precede overt brain tumors. Therefore, even though idiopathic central DI is diagnosed initially with a normal brain MRI, we may need a periodic clinical follow-up and thus serial brain MRIs to detect occult brain lesions.<sup>2,6</sup>

## REFERENCES

1. Maghnie M, Cosi G, Genovese E, Manca-Bitti ML, Cohen A, Zecca S, et al. Central diabetes insipidus in children and young adults. *N Engl J Med* 2000;343:998-1007.
2. Mootha SL, Barkovich AJ, Grumbach MM, Edwards MS, Gitelman SE, Kaplan SL, et al. Idiopathic hypothalamic diabetes insipidus, pituitary stalk thickening, and the occult intracranial germinoma in children and adolescents. *J Clin Endocrinol Metab* 1997;82:1362-7.
3. Blotner H. Primary or idiopathic diabetes insipidus: a system disease. *Metabolism* 1958;7:191-200.
4. Wang LC, Cohen ME, Duffner PK. Etiologies of central diabetes insipidus in children. *Pediatr Neurol* 1994;11:273-7.
5. Shin JH, Lee HK, Choi CG, Suh DC, Kim CJ, Hong SK, et al. MR imaging of central diabetes insipidus: a pictorial essay. *Korean J Radiol* 2001;2:222-30.
6. Leger J, Velasquez A, Garel C, Hassan M, Czernichow P. Thickened pituitary stalk on magnetic resonance imaging in children with central diabetes insipidus. *J Clin Endocrinol Metab* 1999;84:1954-60.