

LETTER TO EDITOR (VIEWER'S CHOICE)

A RARE CASE OF PRIMARY POLYDIPSIA IN A CHILD

Madhav Kamath, Dinesh Nayak,* Chandrika D. Nayak***

Key Words: Primary polydipsia, Central diabetes insipidus, Nephrogenic diabetes insipidus, Osmolality, Arginine vasopressin

A 2 year 11 months old boy was admitted with complaints of polydipsia and polyuria. It had started about 11 months back with an intervening asymptomatic period of about 6 months. Recurrence of symptoms was observed in the last two months and more since last 1 week before admission to the hospital. Past history was not suggestive of any trauma, CNS infections, renal disorder or drug intake except for post measles local abscess. A significant gross motor developmental delay was present in early infancy; however is functioning age appropriately presently. Family history for similar symptomatology and unexplained deaths was unremarkable. Clinical examination revealed weight of 11.9 kgs (on 10th centile), height of 87cms (just below 5th centile) and head circumference of 51cms (just above the mean). Rest of clinical examination was unremarkable. He was allowed ad lib fluids for a 24hr period and his 24hr urine output was documented for 2 consecutive days during the hospital stay. His fluid intake was 5.5L/m²/day, and urine output was 14ml/kg/hour suggestive of polydipsia and polyuria. Urinalysis revealed low specific gravity (1.005) and urine osmolality of 144 mosm/kg indicating water diuresis. Initial blood investigations revealed normal total and differential count, urea, creatinine, sodium, potassium, calcium, phosphorus and alkaline phosphatase. His blood gas was not showing acidosis and random blood sugar was normal (125mg/dl). Serum osmolality done during ad lib fluid intake was 262 mosm/kg. Water deprivation test was administered under close supervision, to delineate other causes revealed blood osmolality returning to normalcy, with serially increasing urine osmolality up to 837mosm/kg and no further significant increase (less than 9%) of osmolality noticed following administration of vasopressin. Hence a clinical diagnosis of psychogenic polydipsia was made. MRI scan of the head was done and was found to be normal. Thyroid function test revealed thyronormalcy state. Mother was counseled and advised to restrict fluids, following which she reported less demanding of fluids by the child over a period of time.

Disorders of water balance presenting with polydipsia and polyuria could be caused due to central diabetes insipidus (CDI), nephrogenic diabetes insipidus or primary polydipsia (1, 2). Central diabetes insipidus is due to the defect in the production of arginine vasopressin (AVP) while nephrogenic diabetes insipidus occurs due to the defect in the renal response to vasopressin (1). Primary polydipsia (PPD) is an uncommon clinical disorder characterized by excessive water-drinking in the absence of a physiologic stimulus to drink (3). Primary polydipsia (PPD) can be due to inappropriate thirst drive (Psychogenic polydipsia) or can be due to hypothalamic damage that alters thirst (neurogenic polydipsia). However in neurogenic

polydipsia AVP secretion is normal. Primary polydipsia (PPD) is an uncommon clinical disorder characterized by excessive water-drinking in the absence of a physiologic stimulus to drink (3). The excessive water drinking is well tolerated unless hyponatremia intervenes (3). However PPD is reported to be a common occurrence in adult inpatients suffering from psychiatric disorders wherein the underlying pathophysiology is unclear and multiple factors are indicated including hypothalamic defect and adverse medication effects (4). PPD is rare in infants and children (5). The diagnosis of PPD is one of exclusion and requires special investigation and management. The most important test is the water deprivation test which should be undertaken carefully. Polydipsia and polyuria can result in chronic changes or complications, such as bowel and bladder dilation, hydronephrosis and renal failure (6,7,8). Still unclear is why only a subgroup of polydipsic patients become hyponatremic (9). Hyponatremia does not usually develop from over ingestion of water, since the renal capacity for free water excretion (about 28 liters a day) is usually more than adequate to handle large fluid volume loads (10, 11). Hyponatremia in PPD can progress to water intoxication and is characterized by symptoms of confusion, lethargy, and psychosis, and seizures or death (4). This case reveals, it is prudent to contemplate a diagnosis of primary polydipsia in children though overtly it may seem to be diabetes insipidus/mellitus as evidenced by huge amount fluid intake and polyuria.

REFERENCES

1. Di Dio G, Zanetta F, Zanetta S, Petri A, Bellone S, Bona G. Polyuria and polydipsia in a 5 year-old child: diagnostic problems. *Minerva Pediatr.* 2007; 59: 813-816.
2. Nichols R. Polyuria and polydipsia. Diagnostic approach and problems associated with patient evaluation. *Vet Clin North Am Small Anim Pract.* 2001; 31: 833-844.
3. Cronin RE. Psychogenic polydipsia with hyponatremia: report of eleven cases. *Am J Kidney Dis.* 1987; 9: 410-416.
4. Dundas B, Harris M, Narasimhan M. Psychogenic polydipsia review: etiology, differential, and treatment. *Curr Psychiatry Rep.* 2007; 9: 236-241.
5. Grunberg J, Gazzara G, Lopez P, Esquivel N. Psychogenic polydipsia in a child. *Bol Med Hosp Infant Mex.* 1981; 38: 807-815.
6. Blum A, Friedland GW. Urinary tract abnormalities due to chronic psychogenic polydipsia. *Am J Psychiatry.* 1983; 140: 915-916.
7. Harrison RB, Ramchandani P, Allen JT. Psychogenic polydipsia: unusual cause for hydronephrosis. *AJR Am J Roentgenol.* 1979; 133: 327-328.
8. Singh H, Linas SL. Compulsive water drinking in the setting of anticholinergic drug use: an unrecognized cause of chronic renal failure. *Am J Kidney Dis.* 1995; 26: 586-589
9. Illowsky BP, Kirch DG. Polydipsia and hyponatremia in psychiatric patients. *Am J Psychiatry.* 1988; 145: 675-683

10. Dubovsky SL, Grabon S, Berl T, Schrier RW. Syndrome of inappropriate secretion of antidiuretic hormone with exacerbated psychosis. *Ann Intern Med.* 1973; 79: 551-554
11. Gillum DM, Linas SL. Water intoxication in a psychotic patient with normal renal water excretion. *Am J Med.* 1984; 77: 773-774

From: *Department of Pediatrics, **Department of Biochemistry, Melaka Manipal Medical College (Manipal Campus), Manipal University, Manipal, India.

Address for Correspondence: Dr. Madhav Kamath, Assistant Professor in Pediatrics, Dr. T.M.A. Pai Hospital, Udipi. 576101, India. Email: drmadhavkamath@gmail.com

E-published: 1st April 2011. **Art#25**
