To the Editor:

A 3-year-old, 12.5-kg girl, who had a late diagnosis of transposition of the great vessels with atrial and ventricular septal defects, underwent pulmonary artery banding along with a Blalock-Taussig shunt for left ventricular retraining in case of a possible arterial switch operation. She had an unstable course in the initial days in the intensive care unit, requiring inotropic and ventilator support. She was started on a vasopressin infusion at 0.0003 units/kg/min intravenously to maintain blood pressure, along with epinephrine at 0.1 μg/kg/min and milrinone at 0.75 μg/kg/min. As her clinical condition stabilized it was decided to taper off vasopressin on day 4 over 6 to 8 hours. She was maintaining adequate urine output of 1 to 2 mL/kg/hour with intravenous furosemide administered at 0.05 to 0.1 mg/kg/hour. The patient’s serum sodium had fallen from 142 mEq/L to a low of 120 mEq/L when started on vasopressin infusion for which slow correction was done with 3% hypertonic saline. She had a sodium level of 130 mEq/L when weaning of vasopressin was started. After discontinuation of vasopressin the child became polyuric with urine output of 1,300 mL over the next 7 hours despite stopping the furosemide infusion (Fig 1). The child was hemodynamically stable during this period with good peripheral perfusion. Serum sodium increased to 142 mEq/L over 6 hours. Vasopressin infusion was resumed at an antidiuretic dose of 0.0003 units/kg/hour and the urine output immediately came down to 1 to 3 mL/kg/hour. Subsequently, the patient was weaned off vasopressin over 24 hours with normal urine output.

Vasopressin, a neurohypophyseal antidiuretic hormone, is a major regulator of water balance in the body. Early initiation of low-dose vasopressin has been reported to decrease the inotropic and fluid requirement in neonates undergoing complex cardiac surgery. Hyponatremia has been reported to occur during vasopressin therapy in children after cardiac surgery. It is related to dose and duration of administration and usually is due to dilutional effect from increased water resorption. Transient diabetes insipidus has been described after discontinuation of vasopressin therapy in a septic patient. Our patient had adequate urine output with low-dose furosemide infusion at 0.05 to 0.1 mg/kg/hour while on vasopressin. Upon cessation of vasopressin the urine output for the next 7 hours increased significantly, amounting to 100 mL/kg in 7 hours despite discontinuing furosemide infusion. The urine specific gravity (1.005) and osmolality, along with plasma osmolality, were consistent with the diagnosis of diabetes insipidus. Blood glucose, potassium, and calcium levels were acceptable. The
finding that polyuria occurred when vasopressin was discontinued and ended when a small dose of vasopressin was restarted suggested that the cause of diuresis was transient diabetes insipidus related to the antidiuretic hormone. It could have been due to a suppression of endogenous production of vasopressin caused by exogenously administered vasopressin (central diabetes insipidus) or development of resistance to vasopressin at the receptor (nephrogenic diabetes insipidus). Though it is logical that these patients may excrete the excess water retained after discontinuation of vasopressin and resolution of cardiac failure, polyuria amounting to diabetes insipidus is very uncommon and has not been reported. Our experience suggests that reinstitution of vasopressin in very small doses may help in the management of such patients if polyuria does not resolve spontaneously. Hyponatremia has been shown to occur in patients receiving vasopressin, and a rebound hypernatremia can result from abrupt discontinuation of vasopressin. Our patient also showed a jump in sodium level by 12 mEq/L over 6 hours after discontinuation of vasopressin. Sudden changes in serum sodium level can cause demyelination and neurologic morbidities.

In summary, we report a rare incidence of transient diabetes insipidus after discontinuation of vasopressin infusion in a child following cardiac surgery. Reinstatement of an antidiuretic dose of vasopressin was effective in controlling the rebound polyuria. Caution should be exerted while correcting hyponatremia in patients receiving vasopressin because sudden rebound hypernatremia can occur after discontinuation of vasopressin.

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