CASE REPORT

Diabetes Insipidus in A 8-Year Old Balinese Boy

A A N Prayoga, I N Westra, Sudaryat Suraatmadja

(Department of Child Health
Medical School, University of Udayana, Denpasar)

ABSTRACT We report a case of diabetes insipidus in a 8 years old Balinese boy. The diagnosis was based on clinical features, laboratory findings, and a good result to chlorpropamide therapy. Common differential diagnoses, i.e., were hypercalcemia, potassium deficiency, psychogenic polydipsia, and diabetes mellitus, could be excluded. The treatment of first choice for diabetes mellitus is desmopressin acetate; however, since the drug was not available, we gave chlorpropamide instead. The patient responded well to 125 mg of clorpropamide twice daily. **[Paediatr Indones 1994; 34:57-61]**

Introduction

Diabetes insipidus is a rare disease characterized by polyuria and polydipsia, resulted from lack of antidiuretic hormone, arginine vasopressin.[1,2,3,4] This disease is more commonly found in males than in females. Although may occur at any age, diabetes insipidus usually manifests clinically in young adults. The diagnosis is usually based on clinical and laboratory findings.[2,3,5] Report of this disease in Indonesia is very scanty; only two adult cases are reported by Yo Kian Tjay and Darmojo, four cases by Rizal Sunaryo et al., and three by Piliang.[6]

We report this first case of diabetes insipidus in a Balinese boy treated in our department.

Accepted for publication: October 8, 1993. For correspondence: A A N Prayoga, MD, Department of Child Health, Medical School, Udayana University - Sanglah Hospital, Denpasar, Indonesia.

Report of a Case

A 8-year old Balinese boy was admitted to the Child Health Department of Denpasar General Hospital on 7th September, 1992 with the main complaints of thirsty and polyuria. These complaints appeared suddenly one month prior to admission. He urinated about 15 times per day with large amount of urine. His

urine was pale. He drank water about 7 liters a day. His body weight had decreased about 4 kg in one month, and he lost his appetite. He has never had other disease.

Physical examination on admission disclosed an alert boy with a pulse rate of 80/min, regular. The respiratory rate was 20/min. The blood pressure was 100/60 mmHg, the body temperature was 36,6°C. He was well nourished with the body weight of 22 kg. The heart and lungs were normal, the liver and spleen were not palpable. He was slightly dehydrated.

Laboratory data on admission showed Hb concentration of 12,1 g/dl, WBC 10,700/μL, PCV 36.7%, and casual blood sugar level 125 mg/dl. Urinalysis showed spesific gravity of 1.000, and pH 6,0; leukocyte, albumin, glucose, keton, urobilin, bilirubin, or blood was not detected.

The patient was diagnosed to have diabetes insipidus. He was advised to drink water or orange juice as much as possible. Urine production, water intake, and body weight were evaluated daily. On September 8, 1992, the blood sugar level was 101 mg/dl, and the urine pH was 5 with a specific gravity of 1.000. On fecal examination ascaris and trichocephalus eggs were found. The urine production as 6 l/day, with the water intake of 6 l/day. His body weight was 22 kg. Chest and head rontgenograms showed no abnormality. The sella turcica was essentially normal. Pyrantel pamoate was given for eradicating intestinal worms.

On September 10, 1992, the blood sugar level was 108 g/dl. Liver function tests disclosed a total bilirubin of 0,44 mg/dl, normal serum transaminases, alkaline phosphatase, and total cholesterol. Albumin and globulin concentrations were 5.21 g/dl and 2.75 g/dl, respectively. Blood urea nitrogen, creatinine, and blood electrolytes were within normal limits. His body weight was 22 kg, the urine production was 6.25 l, with the water intake of 6.7. The electrocardiogram was normal.

The patients was treated with a single daily dose of 100 mg chlorpropamide for 6 days, with special attention to the possibility of hypoglycemia. On September 16, 1992, his body weight was 22 kg, the urine production was 4 l and his water intake was 6.75 l. The urine specific gravity was 1,008, and the blood sugar level was 108 mg/dl. The dose of chlorporpamide was increased to 150 mg daily single dose.

On September 22, 1992 he weighed 23 kg. He drank 4.7 l and urinated 3 ml. His blood sugar was 109 mg/dl. The dose of chlorpropamide was changed to 125 mg, twice daily. On September 26, 1992 the urine production was 1 l/day and the water intake was 2.25 l/d. Three days later the urine production was 1 l/day and the water intake was 1.5 l/day. His body weight remained 23 kg, the blood sugar level was 91,6 mg/dl. The patient was discharged in a good condition. He was maintained on chlorpropamide 125 mg twice a day, and was further followed-up at the OPD.

Discussion

The diagnosis of diabetes insipidus is based on clinical and laboratory findings. Polyuria, polydipsia, hyperthermia, rapid loss of weight, vomiting, constipation, growth failure, dehydration, excessive thirst, dry skin, and anorexia, dehydration, and collapse are frequently observed. Daily urine volume may exceed 4-10 liters or even more; the urine is pale or colorless with the specific gravity varies from 1.001 to 1.005, and corresponding osmolality of 50 to 200 mOsm/kg water. Skull rontgenogram may reveal evidence of an intracranial tumor, such as calcification, enlargement of sella turcica, erotion of the clinoid processes or increased with of the sutura lines.[3]

In our case the clinical manifestations were polyuria, polydipsia, excessive thirst, weight loss, anorexia, and slight dehydration. He urinated about 15 times per day with large amount of urine, and he drank water about 7 l/day. The urine spesific gravity was 1.001. Rontgenogram of the skull showed normal limits.

Diabetes insipidus is due to lack of antidiuretic hormone, anginine vasopressin. It is a peptide hormone synthesized in the cell bodies of neurons in the supraoptic and paraventricular nuclei hypothalamus, and secreted into the circulation by nerve cells.[7] Vasopressin binds to receptors on the blood side of responsive renal tubular cells, activating adenylate cyclase and thus causing the formation of additional cyclic AMP in the cells. The cyclic AMP produces a striking increase in the per- meability of the membrane on the luminal side of the cell to water, urea, and some other solutes, so that water enters the hypertonic interstitium of the renal pyramids. The urine becomes concentrated and its volume decreases. The effect is retention of water in excess of solute; consequently, the effective osmotic pressure of the body fluids is decreased. In the absence of vasopressin, the urine is hypotonic to plasma, urine volume is increased, and there is a net water loss. Consequently, the osmolality of the body fluids rises.

Vasopressin secretion is controlled by feedback mechanism that operates continuously to defend the osmolality of the plasma. When the effective osmotic pressure of the plasma is increased above normal (290 mOsm/ kg), the rate of discharge of these neurons increases and vasopressin secretion is increased, when the effective osmotic pressure is decreased, discharge is inhibited.[7]

A highly sensitive radioimmunoassay is capable of measuring as litle as 0,1 pg/ml of arginine vasopressin.[3] We were unable to perform this assay. Some authors beileve that the diagnosis of diabetes insipidus is established when a patient with characteristic clinical findings shows a good response to vasopressin or other drug like chlorpropamide.[2,5] Sudaryo et al.[5] reported 4 diabetes insipidus cases, one of them was diagnosed by using this method. Good response to chlorpropamide in our patient favoured the diagnosis of diabetes insipidus.

Though polyuria and polydipsia are important clinical findings in diabetes insipidus, other diseases such as hypercalcemia, potassium deficiency, compulsive water drinking (psychogenic polydipsia), diabetes mellitus, or chronic renal disease may show these findings,[2-4] and should be considered as a differential diagnosis.

Normal serum electrolyte concentrations has virtually excluded hypercalceor hypokalemia. Psychogenic polydipsia

usually occurs in young women with psychiatric problem, which was not the case in our patient. The urine specific gravity of psychogenic polydipsia varies according to water intake.[5] In our case the urine specific gravity was 1.001. Diabetes mellitus could be excluded by the absence of hyperglycemia or glycosuria. Similarly, chronic renal disease was excluded by normal renal fuction test.

Diabetes insipidus is due to vasopressin insufficiency, therefore replacement is the treatment of first choice. The drug of first choice is desmopressin acetate (1-desamino-8-D-arginine vasopressin, DDAVP) given intranasally. The dosage must be adjusted, but the duration of action is generally at least 12 hours.[3] Replacement with lypressin (lysine-8-vasopressin, Syntopressin Spray, lysine-8-vasopressin drops), vasopressin tannate (Pitressin Tannate), or posterior pituitary powder for nasal insufflation is of less value because of its short duration of action, impurities, less uniform activity, or need for injection.

Chlorpropamide has been found to have an antidiuretic effect through its augmentation of endogenous ADH effect. Chlorpropamide, clofibrate, and carbamazepine stimulate the release of ADH. They may be tried with the dose of 100-250 mg once or twice daily. The maximal effect may take 1-2 weeks.

Unfortunately desmopressin and other derivates were not available in Denpasar, therefore the patient was treated by chlorpropamide. Chlorpropamide is an oral hypoglycemic drug; however, hypoglycemia is an uncommon side effect. The dose varies from patient to patient. In this case we started with a single dose of 100 mg of chlorpropamide. Nine days after the initiation of treatment there only little response observed, where the urine production was 4 l with water intake of 6.5 l. For that reason we decided to give a single daily dose of 150 chlorpropamide; however until the 15th day the result was still unsatisfactory. By increasing the dose of chlorpropamide to 125 mg twice daily, the urine production decreased hastily.

Normal urine production in a child is 500 to 1000 ml/day, and water intake for an 8 year old boy is normally 1.8 to 2 l/day. In our case these normal values were observed on the 19th day, and were maintained until he was discharged.

The prognosis of diabetes insipidus depends on the etiology, which includes tumor of the suprasellar and chiasmatic regions, particularly craniopharyngiomas and optic gliomas, infections such as encephalitis, sarcoidosis, tuberculosis, actinomycosis, head injury, or operative procedures in the region of pituitary or hypothalamus. In neonatal asphyxia, intraventricular hemorrhage, sepsis, meningitis are common.[3] We were unable to determine the etiology of diabetes insipidus in our patient; therefore, the prognosis is still to be observed. Periodic reevaluation is mandatory in this situation.

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