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Renal medullary solute depletion resulting from psychogenic polydipsia in a rhesus monkey

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A 9-year-old 9-kg male rhesus monkey was observed to have polydipsia and an unthirsty appearance. The monkey was being fed a commercial primate ration, with fresh water provided ad libitum by automatic waterer, and was housed in individual caging. Physical examination revealed extensive bilateral periorbital edema and patchy alopecia over the lower trunk and limbs. Blood cell counts were unremarkable, but serum biochemical analysis revealed hyponatremia (116 mEq/L; normal, 145 to 158 mEq/L) and hypochloremia (81 mEq/L; normal, 106 to 115 mEq/L) and the plasma osmolality was calculated to be 247 mosm/kg of H2O (normal, 295 to 330 mosm/kg of H2O)1,2 Urinalysis revealed a specific gravity of 1.000.

The only environmental alteration recorded for this monkey was a change in feeding regimen. The monkey had been inadvertently fed almost twice his daily requirement of biscuits for nearly 4 months, resulting in obesity. Four weeks before the onset of polydipsia, the diet was restricted to 100% of the calculated daily caloric requirement, to diminish further unnecessary weight gain. This apparently was the triggering factor for the polydipsia.

The monkey’s water consumption was monitored while he was given free access to bottled water. The monkey’s mean daily water consumption was 3.75 L, or 47 mℓ/kg of body weight, which is approximately 5 times the normal water consumption for a rhesus monkey fed a commercial dry diet.3

To establish the cause of the hyposthenuria, a water deprivation test was performed. The urine specific gravity after 10 hours was 1.000. This lack of renal concentrating ability indicated either a lack of antidiuretic hormone (ADH) production or diminished renal responsiveness to ADH. Although renal failure was considered, normal BUN and creatinine values, loss of anemia, and normal hydration tended to rule it out. An ADH stimulation test was performed, with 5 U of aqueous vasopressin administered intramuscularly. Urine specific gravity was monitored hourly for 5 hours and again did not exceed 1.000. The lack of ability to concentrate urine after exogenous administration of ADH would normally indicate nephrogenic diabetes insipidus, however, one would expect the serum osmolality to be greater than normal4.

On the basis of the low serum osmolality, renal medullary solute depletion was suspected. To verify the diagnosis, water intake was restricted to 1.5 L/day, and the monkey was closely monitored. Signs of distress or dehydration were not observed. The urine specific gravity remained at 1.000. After 7 days of restricted water intake, the ADH stimulation test was repeated and resulted in a urine specific gravity of 1.010. Also, the serum electrolyte values had returned to within normal limits. The water deprivation test was repeated 3 days later and resulted in a urine specific gravity of 1.020. Water intake was restricted to 1 L daily for 2 months, during which time serum electrolyte concentrations remained normal during weekly monitoring. Initially, urine specific gravity remained constant at 1.000. On day 5, the specific gravity was 1.005, and continued to slowly increase until values ranged from 1.015 to 1.025 on a daily basis. At the end of the 2 months, a water deprivation test resulted in an increase in urine specific gravity from 1.020 to 1.030 within 2 hours.

The normal response to water deprivation verified a normal concentrating ability and the diagnosis of psychogenic polydipsia with renal medullary solute depletion. One year after diagnosis and treatment, there was no evidence of recurrent polydipsia.

Various abnormal behaviors, including thumb-sucking, cage-chewing, abnormal vocalization, aggression, and polydipsia have been attributed to cage confinement of nonhuman primates.5-4 Renal medullary solute depletion develops when prolonged diuresis results in a loss of the normal solutes, including urea, from the medullary interstitium.6

When the osmotic gradient between the medulla and the cortex decreases, the renal concentrating ability decreases concurrently, resulting in the inability to respond to either water deprivation or ADH stimulation. The rhesus monkey kidney does not have a well developed inner medulla with long loops of Henle.7 It has been suggested that because of this anatomic variation, urine formation in rhesus...
rmonkeys is not so dependent on the medullary osmotic gradient as in other species. However, in this case, medullary solute depletion did develop in response to prolonged diuresis and resulted in a lack of renal concentrating ability.

Reversal of renal medullary solute depletion may be attempted by gradual water restriction with concurrent monitoring of hydration, urine specific gravity, and BUN concentration. In this case, after 1 week of water restriction to 1500 ml/day, there was a minimal response to ADH administration because of restoration of hyperoncotic to the renal medullary interstitium. Correction of renal medullary solute depletion must be accomplished before the cause of polyuria may be established.  