Compulsive polydipsia following menigioma resection: an epileptic phenomenon?

Case report

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The authors report the case of an individual who developed compulsive polydipsia following resection of a left sphenoidal ridge meningioma. The episodic, stereotyped nature of his symptoms, response to treatment, and electroencephalographic and magnetic resonance imaging findings are all highly consistent with temporal lobe-onset epilepsy. The pathophysiology of this underrecognized phenomenon is discussed.

KEY WORDS • polydipsia • hypothalamus • temporal lobe • epilepsy

Polydipsia may be a manifestation of diabetes insipidus or diabetes mellitus, but when no organic cause for polydipsia is found the patient is often diagnosed as having psychogenic polydipsia. We report a patient who presented with a compulsion to drink as the primary manifestation of temporal lobe epilepsy. We conclude that temporal lobe epilepsy may be an unusual cause of "psychogenic" polydipsia.

Case Report

This 42-year-old man complained of occasional episodes of loss of consciousness and impulse to drink liquids beginning 3 years after he had undergone craniotomy for a left sphenoidal ridge meningioma with involvement of the suprasellar area. Five months later, an evaluation for hypertension identified hyperaldosteronism. An abdominal computerized tomography scan disclosed a 1.7-cm left adrenal tumor; an adrenalectomy was performed and the pathological findings were consistent with an adenoma. His pituitary adrenal axis had been studied several times and the biochemical profile was always within the normal range.

On questioning, the patient reported drinking 12 glasses of water, several cans of diet cola, and multiple cups of coffee daily. This behavior started 3 years after the resection of his meningioma, and he described an "urge to drink" even if he was not thirsty. These recurrent paroxysmal episodes lasted 20 to 25 seconds, and resolved regardless of whether he drank. Physical and neurological examination was normal, except for mild depression. Because of his prior history, an endocrine cause was suspected at first. A 24-hour urine collection demonstrated a total volume of 5.8 liters, urine osmolality of 150 mOsm/kg, and specific gravity 1.005. The serum sodium level was 138 mmol/kg, and serum osmolality 275 mOsm/kg. A 24-hour fluid deprivation test revealed no hyperosmolality or hypernatremia, and urine volume decreased appropriately to between 20 and 30 cc/hr. Following administration of 2 μg desmopressin acetate, the patient's urine osmolality level did not increase.

Electroencephalographic (EEG) studies revealed left temporal epileptiform discharges, and magnetic resonance (MR) imaging showed encephalomalacia in the left temporal and left frontal lobes (Fig. 1). Ambulatory EEG monitoring was performed for 24 hours, showing occasional left temporal spikes during the episodes; however, because of the limited number of channels, its contribution is unclear. The patient was initially taking phenobarbital, which was tapered because of malaise, depression, and lack of efficacy. Carbamazepine therapy was initiated with resolution of the urge to drink and the associated polyuria.
Compulsive polydipsia

FIG. 1. Magnetic resonance T2-weighted image demonstrating encephalomalacia in the left anterior and mesial temporal lobe.

Discussion

Temporal lobe epilepsy is one of the most common localization-related epilepsies; 70% to 80% of such cases arise from the hippocampus and amygdala.2,3 Typical auras include an epigastric rising sensation and olfactory or gustatory sensations, but may include autonomic phenomenon such as pupillary dilatation, facial flushing, and tachycardia.4 Temporal lobe seizures can spread to different structures, resulting in different auras and clinical signs. Deep structures such as the hypothalamus may be involved in an ictus.1,5 The hypothalamus is a complex structure that governs endocrine, autonomic, and homeostatic mechanisms such as eating and drinking. Spread of electrical activity to the hypothalamus is thought to be the mechanism causing the elevation of prolactin which frequently occurs following a complex partial or generalized tonic-clonic seizure, especially originating from the mesial temporal lobe.6,9 Prolactin elevation following electroconvulsive therapy has also been postulated to result from stimulation of the ventromedial hypothalamus.10 In cats, stimulation of the hypothalamus elicits a drinking response, and there is considerable evidence in animals and humans that the lateral hypothalamus mediates drinking behavior.9

In the early 1930's, Lennox and Cobb4 first reviewed the auras of patients with complex partial seizures and demonstrated that an urge or compulsion to drink was an aura in a small number of patients. Rémillard, et al.,7 later documented with depth electrodes that the urge to demand, pour, and drink water may be a manifestation of temporal lobe epilepsy. More recently, Cascino and Sutula1 reported thirst and compulsive water drinking in a patient similar to ours with mesial temporal onset epilepsy.

It is accepted that the best way to accurately characterize an ictal event in this case would have required implantation with intracranial electrodes; however, because of his response to carbamazepine, the procedure was not performed. We hope physicians will include temporal lobe epilepsy in the differential diagnosis of psychogenic polydipsia.

References

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