Cortical Blindness after Correction of Symptomatic Hyponatremia: Dynamic Cerebral Dysfunction Visualized Using Serial SPECT Scanning

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A 70-yr-old woman developed cortical blindness after correction of hyponatremia. Regional hyperperfusion was noted on SPECT scans obtained in the acute phase. One month later when symptoms had largely resolved, a repeat examination was normal. This regional hyperperfusion, which was not associated with any apparent structural damage, may have represented either luxury perfusion or a transient increased metabolic requirement of the dysfunctional cortical area. SPECT scanning may be a useful method to study cerebral dysfunction resulting from an osmotic disturbance.

Key Words: single-photon emission computed tomography; cortical blindness; hyponatremia

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Cortical blindness following correction of hyponatremia has been noted, but its incidence and pathophysiology are unknown (*1*). We present a patient for whom a SPECT scan was the only diagnostic test to pinpoint the area of clinical dysfunction.

CASE REPORT

Three weeks prior to neurologic consultation, this 73-yr-old woman was involved in a motor vehicle accident and required emergency nephrectomy and splenectomy. There was no loss of consciousness reported at that time by the patient and no evidence of head trauma was noted on physical examination. She was at home for 1 wk when her family noticed she was drinking water excessively and was confused. She was brought to the emergency ward where she had a generalized seizure. Her serum sodium level was 107 mEq/liter. Serum and urine osmolarity were 225 and 465 mOsm, respectively. Hypertonic followed by isotonic saline was administered. Although she remained somnolent, her serum sodium level increased to 123 mEq/liter. After 72 hr, her sodium level was 132 mEq/liter, she opened her eyes when her name was spoken and appeared to recognize her family members by sight. On the fifth hospital day, however, her family and hospital staff realized that she could not see.

On neurological evaluation, she was attentive and cooperative, although her conversation was rambling and repetitious. She could not give the month, date, name of the hospital or her length of stay. Her speech was fluent with good comprehension, but she made paraphasic errors. She was aware she had a visual problem but underestimated its severity in thinking her corrective lenses needed adjustment. In addition, she was unable to identify coins or a safety pin placed in either hand, and, when given a pen, described it as "something like a line." Fundoscopic examination, pupillary reflexes, extraocular movements and the remainder of the neurological examination were normal.

Routine chemistries, including electrolytes and CBC, were within the normal ranges. T1-weighted MRI scans before and after gadolinium enhancement and T2-weighted MRI scans were obtained on the sixth and fifteenth hospital days. Both MRI studies were normal. Visually evoked potentials using flash stimulation were normal. An EEG revealed diffuse slowing, absence of an alpha rhythm and occasional triphasic waves. Spinal fluid analysis was unremarkable.

A SPECT scan was obtained using 25 mCi ^{99m}Tc-HMPAO administered intraveneously with the patient resting quietly with eyes open in a dimly lit room. Sixty minutes later, tomographic images were obtained on a three-detector SPECT system over 22 min of continuous rotation using ultra-high resolution fanbeam collimators. Images were processed via transverse reconstruction followed by three-dimensional filtering using a low-pass (5.8/3.2) filter. This was followed by uniform (Chang) attenuation correction using manually-adjusted per slice skull edge detection. Images are normally interpreted by normalizing the cerebellum to 100% of relative brain activity and using it as an internal reference. Relative perfusion (rCBF) was initially determined by displaying images in a discrete decile (i.e., 10% incremental) color scale and comparing relative perfusion of occiput to cerebellum. These rCBF interpretations were then verified by comparing the average counts per pixel of circular regions of interest (ROIs) of fixed size placed over the occiput and cerebellum. The ratios of occiput-to-cerebellar perfusion were then compared to published normal files (*2*). These images demonstrated an abnormal (~30%)

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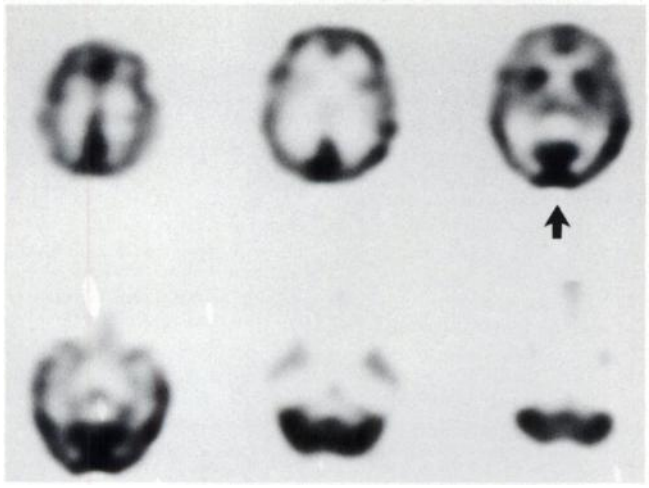

FIGURE 1. Initial SPECT scan. Transaxial slices demonstrate the highest relative perfusion in the occiput near the midline (arrow). The occipital-to-cerebellar ratio averages 1.2, which is approximately 30% increased from normal.

increase in perfusion of the occipital and posterior parietal lobes bilaterally (Fig. 1).

Over the remaining 10 hospital days, the patient slowly improved. On discharge, she was oriented, spoke without paraphasias and could reliably localize a moving visual stimulus. The patient was able to describe the shape, size and color of stationary visual objects, but was unable to identify them. One month later, she reported improved vision and fewer complex visual hallucinations. On examination, her visual acuity was 20/200 in both eyes and she showed no impairment of either tactile or visual perception. A repeat SPECT scan performed under similar conditions showed normal distribution of perfusion, with return of the previous occipital hyperperfusion to within normal ranges (~70% of cerebellum) (Fig. 2).

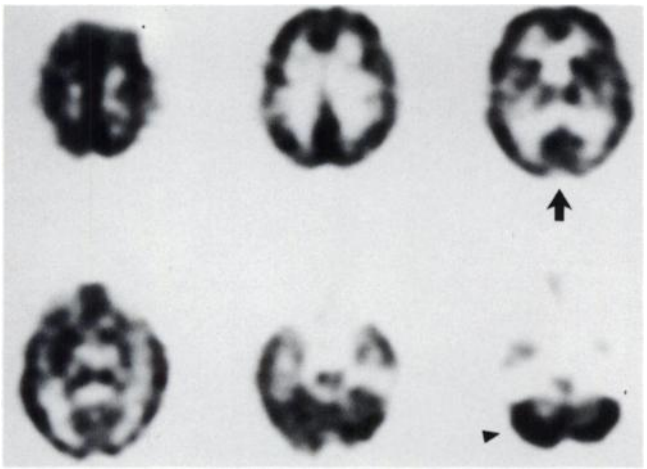

FIGURE 2. Repeat SPECT scan obtained 1 mo later. Transaxial slices similar to those of the prior study now show normal distribution of perfusion, with highest uptake in the cerebellum (arrowhead). Occipital uptake is now within the normal range at 70% of cerebellar uptake (arrow).

DISCUSSION

This patient developed cortical blindness and astereognosis following the correction of symptomatic hyponatremia. As she recovered, she showed visual agnosia and complained of visual hallucinations similar to other reported cases of transient cortical blindness (*2,3*).

The rate of correction and the absolute increase of this patient's serum sodium levels were not in the ranges usually associated with cerebral demyelination or infarction (*4–6*). Neither MR imaging nor visually evoked potentials revealed evidence of infarction or demyelination along the visual pathways. By contrast, a SPECT scan done in the acute phase showed a regional perfusion abnormality in the areas correlated with the neurological deficits.

A recent SPECT study in patients with cortical blindness from infarction, global hypoxia, trauma and Alzheimer's disease showed hypoperfusion of the occiptoparietal regions (*7*). In our patient, SPECT showed a selective hyperperfusion of the occipital cortex. Furthermore, a repeat SPECT study performed after clinical recovery revealed a return to normal occipital perfusion. There are two alternative explanations for this finding: (a) either the regional hyperperfusion reflected an increase in metabolism of the dysfunctional cortical areas or (b) a mismatch existed between flow and metabolism (i.e., the hyperperfusion represented luxury perfusion) (*8*).

Luxury perfusion is felt to reflect regional acidosis and is usually associated with ischemia or structural damage. In the initial SPECT scan, the areas of hyperperfusion extended beyond vascular territories and the prolonged time course of the patient's clinical deficit was not consistent with a transient ischemic event. If the hyperperfusion represented luxury perfusion, then this case illustrates that luxury perfusion may occur in the absence of ischemia and structural damage.

If hyperperfusion reflected increased metabolism, then the question remains as to why clinical dysfunction was observed. In the presence of hyponatremia, brain tissue loses both inorganic and organic solutes. With correction of serum osmolarity, the cells reaccumulate inorganic as opposed to most organic solutes more rapidly (*9*). The reaccumulation of organic solutes is likely an energy consuming process which could create a transient increased metabolic requirement. The dysfunction may then have resulted from an impairment of the occipital and parietal areas to respond to an osmotic disturbance and restore equilibrium and normal function, thus implying a selective vulnerability of the occipital region to hyponatremia and its correction.

PET scanning using ^{18}FDG would have been particularly useful in this case since this would have added direct information about metabolism to the perfusion information obtained using ^{99m}Tc-HMPAO. Low occipital uptake on PET-FDG at the time of the initial HMPAO study would have indicated depressed occipital metabolism with luxury perfusion. High occipital FDG uptake, on the other hand,

would have suggested that the occipital hyperperfusion reflected a hypermetabolic process as might exist in an accelerate metabolic correction. This case suggests a role for this imaging study in the evaluation of patients with neurologic dysfunction resulting from osmotic disturbances.

REFERENCES

1. Arieff AI, Griggs RC. *Metabolic brain dysfunction in systemic disorders*. Boston: Little, Brown & Co.; 1992:55–87.
2. Rubin RT, Villanueva-Meyer J, Ananth J, Trajmar PG, Mena I. Regional ^{133}Xe cerebral blood flow and cerebral ^{99m}Tc-HMPAO uptake in unmedicated patients with obsessive-compulsive disorder and matched normal control subjects. *Arch Gen Psychiatr* 1992;49:695–702.
3. Silverman SM, Bergman PS, Bender MB. The dynamics of transient cortical blindness: report of nine episodes following vertebral angiography. *Arch Neurol* 1961;4:333–348.
4. Aldrich MS, Alessi AG, Beck RW, Gilman S. Cortical blindness: etiology, diagnosis and prognosis. *Ann Neurol* 1987;21:149–158.
5. Ayus JC, Arieff AL. Pathogenesis and prevention of hyponatremic encephalopathy. *Endocrinol Metab Clin North Am* 1993;22:425–446.
6. Tien R, Arieff AL, Kucharczyk W, Wasik AJ, Kucharczyk J. Hyponatremic encephalopathy: is central pontine myelinolysis a component? *Am J Med* 1992;92:513–522.
7. Brunner JE, Redmond JM, Hagger AM, Kruger DR, Elias SB. Central pontine myelinolysis and pontine lesions after rapid correction of hyponatremia: a prospective magnetic resonance imaging study. *Ann Neurol* 1990;27:61–66.
8. Silverman IE, Galetta SL, Gray LG, Moster M, Atlas SW, Maurer AH, Alavi A. SPECT in patients with cortical visual loss. *J Nucl Med* 1993;34:1447–1451.
9. Lassen NA. The luxury perfusion syndrome and its possible relation to acute metabolic acidosis localized within the brain. *Lancet* 1966;2:1113–1115.
10. Gullans SR, Verbalis JG. Control of brain volume during hyperosmolar and hypo-osmolar conditions. *Ann Rev Med* 1993;44:289–301.