Anoxic Brain Injury Secondary to Metabolic Induced Encephalopathy

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Abstract

Syndrome of inappropriate anti-diuretic hormone (SIADH) is a metabolic syndrome causing hyponatremia, encephalopathy and anoxic brain injury which may lead to visual dysfunction. Rehabilitative vision therapy can provide improvements in patients’ quality of life.

I. Case History
   a) Demographics
      i) 24 year old active duty Caucasian male
   b) Chief Complaint
      i) Initial cortical blindness that resolved with subsequent right sided peripheral vision loss & difficulty reading
   c) Ocular History
      i) Intermittent accommodative right esotropia since childhood
         ii) Congenital overacting inferior obliques
   d) Medical History
      i) Initial emergency department visit
         (1) SIADH with severe hyponatremia
         (2) Acute encephalopathy
         (3) Anoxic brain injury from suspected seizure
         (4) Acute respiratory failure
         (5) Leukocytosis (white cell count 26,000)
         (6) Rhabdomyolysis
         (7) Dysphagia
      ii) Inpatient rehabilitation
         (1) Anoxic encephalopathy
         (2) Mild amnesia
         (3) Cortical blindness with homonymous right visual field defect
   e) Medications
      i) None currently

II. Pertinent Findings
   a) Clinical
      i) Initial outpatient ophthalmology visit
         (1) BCVA: OD 20/30\textsuperscript{+1}, OS 20/30\textsuperscript{+1}
         (2) Saccadic dysfunction
         (3) Oculomotor (fixation) dysfunction
         (4) Right homonymous hemianopsia
      ii) Initial traumatic brain injury eye visit
         (1) BCVA: OD 10/16, OS 10/10\textsuperscript{+1}
         (2) Saccadic dysfunction – overshooting worse in right gaze
(3) Visual information processing dysfunction
(4) Incongruous Homonymous Right Hemianopia

iii) Traumatic brain injury eye visit s/p five 1-hour vision therapy sessions
(1) BCVA: OD 10/12.5−2, OS 10/10−1
(2) Saccadic dysfunction – overshooting worse in right gaze, improved
(3) Accommodative insufficiency
(4) Visual information processing dysfunction, resolved
(5) Incongruous Homonymous Right Hemianopsia OD>OS

b) Physical
i) Initial emergency department visit
   (1) Mental disorientation progressing to loss of consciousness
   (2) Rigid with flexor posturing
   (3) Febrile
ii) Inpatient rehabilitation
   (1) Aphasia
   (2) Ataxia
   (3) Mild right hemiparesis, primarily right hand

c) Radiology Studies
i) Initial emergency department visit
   (1) CT angio of the brain – no abnormalities
   (2) CT scan of the head – normal
   (3) MRI of the brain x 2 – normal
   (4) Chest x-ray – no acute cardiopulmonary process
   (5) Panorex x-ray – grossly normal
   (6) Echocardiogram – grossly normal ejection fraction w/o abnormality
   (7) Lower extremities venous Doppler – negative for DVTs
ii) Initial Veteran’s Affairs medical in-processing
   (1) MRI of brain w/ and w/o contrast & MRA
      (a) Gyriform cortical hyperintensity on FLAIR and T2-weighted images
          involving the left temporal, parietal and occipital lobes
      (b) Slight asymmetric hyperintensity of the medial left temporal lobe and
          hippocampus

III. Differential Diagnosis
   a) Primary/leading
      i) Anoxic Brain Injury
   b) Others
      i) Traumatic Brain Injury, Cerebral Vascular Accident, Cerebral Tumor,
         Infection, Iatrogenic

IV. Diagnosis/Discussion
   a) SIADH is an abnormal metabolic condition resulting in hyponatremia. Its onset is
      characterized by the following:
      i) effective serum osmolality < 275 mOsm/Kg
      ii) urinary osmolality >100 mOsm/Kg
      iii) urinary sodium >40 mOsm/Kg
      iv) normal adrenal cortical function
      v) normal body water concentration (envolaemia)
b) SIADH is the most common electrolyte disorder in hospital patients, accounting for 1/3 of all hyponatremia cases. SIADH induced hyponatremia can occur and resolve suddenly, or exist in a chronic state. Symptoms range from mild headaches to more advanced neurological disorders such as confusion, limb weakness, seizures and respiratory insufficiency. Acute severe hyponatremia typically causes encephalopathy due to the sudden change in intracellular vs extracellular osmolality. If not treated rapidly, the metabolic encephalopathy can result in permanent brain damage and death.

c) Anoxic brain injuries (ABIs) result from the deprivation of oxygen to the brain. Symptoms range from loss of consciousness and coma to persistent neurological damage consistent with the location and severity of brain tissue damage. Common symptoms include short-term memory loss, higher level processing deficits, anoma and visual disturbances. Rehabilitative therapy to target the sequale of ABIs is the key to maximum recovery. When the damage involves the visual processing centers, treatment should include vision therapy to increase the patient’s adaptation to the new visual state and enhance remaining ocular skills so as to improve visual function.

V. Treatment, Management
   a) Vision Therapy
      a. Focusing on oculomotor skills and visual exploration training
   b) Observation
      a. Symptoms may partially resolve over time due to the brain’s neuroplasticity and patient adaptation

VI. Conclusion
   a) Metabolic induced brain injuries resulting in visual sequale should be assessed similar to traumatic brain injuries, and the specific sequale managed with the appropriate rehabilitative therapy, such as vision therapy. Brain injury often affects multiple systems: thus, ensuring the patient is receiving comprehensive rehabilitative assessment and treatment is important in ensuring maximum adaptability and recovery.