

Transient Bilateral Cortical Blindness in an Adult after an Acute Episode of Asthma Exacerbation

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Cortical blindness is a clinical syndrome characterized by loss of vision, normal pupillary response and normal fundoscopic examination. It is a complication mainly described in infants and children and occurs after various hypoxemic events, such as asphyxia, infections, surgery, hypovolemia, etc. Common etiologies in young adults include preeclampsia/eclampsia, migraine, epilepsy and post-traumatic conditions [Table 1].

The cortical blindness phenomenon is usually accompanied by other serious neurologic deficits. Only a few cases have been described as a post-asthma attack. We report a case of acute transient bilateral cortical blindness as a solitary neurologic defect due to hypoxemia in a woman after a severe asthmatic episode and respiratory failure. We discuss the differential diagnosis of asthma and visual loss, and examine the cortical blindness phenomenon and its possible pathophysiology, as well as imaging and prognosis.

Patient Description

A 19 year old female with a history of bronchial asthma since childhood arrived at our clinic in progressive respiratory distress due to asthma exacerbation. Shortly after her arrival she became unconscious. Initial resuscitative treatment was given, including ventilation via an endotracheal tube, oxygen supplement, intravenous glucocorticosteroids and adrenaline. She was transferred to the hospital.

At the hospital (35 minutes after beginning our treatment) she was treated with intravenous glucocorticosteroids and magnesium. Initial arterial blood gases and pH showed: pO₂ 182 mmHg, pCO₂ 64.5 mmHg, and pH 7.17. Several hours after admission the patient regained consciousness, was extubated and her arterial blood gases returned to normal (pO₂ 377

Table 1. Etiologic classification of cortical blindness

<p>Obstetric Eclampsia Preeclampsia HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count) Pregnancy toxemia Postpartum posterior leukoencephalopathy syndrome</p>	<p>Antiphospholipid antibodies syndrome Migraine</p>
<p>Pediatric Perinatal hypoxia (hypoxic encephalopathy) Infectious Meningitis Encephalitis Hydrocephalus Status epilepticus</p>	<p>Iatrogenic Post-aortic arch surgery Post-cardiac catheterization or coronary artery bypass graft Post-arteriography, aortography Post-cardioventricular pacing</p>
<p>Vasculitic Henoch-Schönlein purpura Churg-Strauss syndrome Cerebral angiitis</p>	<p>Toxic Heroin Cyclosporin A Snake bite Vindesine Gastrografin</p>
<p>Traumatic Head trauma Cervical trauma Parietal contusion and transient superior sagittal sinus occlusion</p>	<p>Elevated intracranial pressure Intracranial hemorrhage Brain tumor</p>
<p>Ischemic Stroke Cardioembolic event (bilateral posterior cerebral artery distribution)</p>	<p>Miscellaneous Creutzfeldt-Jakob disease Liver cirrhosis and encephalopathy Acute glomerulonephritis Cerebral malaria Acute intermittent porphyria</p>

mmHg, pCO₂ 38.4 mmHg, and pH 7.39).

She complained of loss of vision, and examination revealed severe bilateral visual impairment (inability to follow light or other objects). No other neurologic deficits were evident. Pupillary reflexes and fundoscopic examinations were normal, as was a computerized tomography scan. A diagnosis of bilateral cortical blindness was made.

During the next few days her vision improved gradually. The patient was discharged 5 days later with normal vision and good respiratory function.

Comment

Status asthmaticus increases the risk of neurologic complications induced by hypoxia because of the high susceptibility of central nervous system cells to oxygen deficit. Visual injury is also described in this context.

A few entities in the differential diagnosis of visual loss and acute asthma not induced by hypoxia were all ruled out. These include:

- Reversible or permanent monocular blindness, as a rare systemic vasculitic

complication of Churg-Strauss syndrome

- Valsalva hemorrhagic retinopathy, explained by a sharp rise in intrathoracic/intraabdominal pressure against a closed glottis transmitted to the eye causing a rupture of superficial retinal capillaries
- Anterior ischemic optic neuropathy, partially related to hypoxia, that occurs when low oxygen pressure induces vasospasm of the optic nerve arteries.

The pathophysiology of CB is hypoxia of the visual pathways at the territories supplied by the distal posterior cerebral arteries. The possibility that vascular factors (a limited capability of the posterior vascular system to autoregulate blood flow) rather than cortical tissue vulnerability are responsible for the hypoxic affect on the visual cortex was investigated in a few studies on cerebral hemodynamics. Decreased compensatory dilation response of the basilar arterial system to occlusion of the carotid system was demonstrated in the dog electromagnetic flow-meter study during hypoxia and hypercapnea (but not during normocapnea [1]). Another study described brain CT scan findings in the persistent vegetative state. The most common vascular brain infarctions occurred at border zones and in the posterior cerebral artery territory.

The latest information on cerebral hemodynamics was obtained from an ultrasonographic study on fetal brain circulation, which demonstrated a 'sparing effect' in the anterior cerebral artery under chronic hypoxia. This suggests that the frontal lobes are spared longer than the lateral and occipital regions [2].

Several methods of imaging are used to

assess the extent of damage and prognosis, but there is no widely accepted laboratory or imaging finding with a significant positive predictive value for the visual outcome.

Whereas visual evoked potentials and electroencephalography are used as prognostic tools in cases of cortical blindness in infants and children, the most common neuroimaging modalities used in young adults are computed CT and magnetic resonance imaging. CT scans performed during the first 24 hours showed low density areas in the bilateral occipital lobes, suggesting cerebral edema formation [3]. Abnormality in the striate and parastriate cortices was associated with poor outcome. Xenon/CT cerebral blood flow measurement in a patient with cortical blindness due to eclampsia showed decreased local blood flow in the area of the posterior cerebral arteries [3].

Although it is sometimes difficult to use MRI to diagnose cortical blindness at the acute stage, several studies demonstrated abnormal T2-hyperintensity signals in the bilateral occipital lobes, mainly in the white matter [3,4]. Recovery of vision was accompanied by complete resolution of the radiologic abnormalities, both in CT and MRI follow-up imaging studies. Significant hypometabolism in the visual and parieto-occipital cortices was found in positron emission tomography studies during the acute phase [5]. The cerebral metabolic rate of glucose recovered almost completely with the recovery of vision.

Recovery time from cortical blindness varies from several days to 3 years and is not influenced by the extent of the damage.

Younger age is associated with poor prognosis. Even though the infant brain has greater regenerative potential than the adult brain, the visual cortex lacks plasticity, resulting in greater damage.

Cortical blindness, as in our patient, is scarce as a solitary neurologic complication, especially in adults. The pathophysiology is not clear, although abnormal vascular autoregulation of posterior cerebral artery blood flow under certain circumstances is currently the preferred theory. Neither laboratory nor imaging scans are accepted as reliable prognostic tools.

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