Visual Hallucinations in Recovery From Cortical Blindness

Imaging Correlates

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Objective: To investigate the cerebral metabolic and functional patterns during recovery from cortical blindness.

Design: Follow-up study with serial clinical, metabolic, and functional imaging and visual evoked potentials.

Case Presentation: A 24-year-old woman suffered from cortical blindness after cardiac arrest and recovered over a 6-month period. During recovery, she experienced complex visual hallucinations that could be initiated by visual imagery.

Results: Initially, the regional cerebral metabolic rate of glucose was severely reduced in the visual and parieto-occipital cortex bilaterally but recovered almost completely. Visual hallucinations led to significant increases of the regional cerebral blood flow in the initially severely hypometabolic parieto-occipital and temporolateral cortex.

Conclusions: Recovery of vision was related to normalization of the postlesionally dysfunctional cortex. Visual hallucinations appeared as the clinical correlate of the electrophysiological hyperexcitability of the recovering partially damaged visual cortex.

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CORTICAL BLINDNESS refers to loss of vision caused by damage of the geniculo-calcarine pathways. Cerebrovascular disease is the most common cause, followed by cardiac surgery and cerebral angiography. Cardiac arrest usually leads to global brain anoxia or to more focal neurological abnormalities, such as cortical blindness, resulting in a variable spectrum of diffuse or multifocal cerebral atrophy due to neuronal loss and cortical and subcortical gliosis or even severe cortical damage. Accordingly, positron emission tomography (PET) has shown that global ischemic anoxia results in diffuse hypometabolism with preferential localization of the metabolic alterations in the parieto-occipital cortex. Nevertheless, most patients recover from cortical blindness, but the mechanisms of this recovery are poorly understood.

We describe a patient who experienced vivid visual hallucinations during recovery from prolonged cortical blindness. Using multitracer PET imaging, we demonstrated that the hallucinations were associated with activations of the parieto-occipital cortex, which was severely metabolically compromised initially but which regained normal metabolic function as vision recovered.

REPORT OF A CASE

A 24-year-old right-handed woman with allergic asthma but an otherwise unremarkable medical history had a generalized epileptic seizure after undergoing diagnostic bronchoscopy. While the seizure was promptly interrupted by the administration of diazepam, a few minutes later the patient needed cardiopulmonary resuscitation (CPR) because of cardiac arrest. She was then given respirator therapy for 1 day before sedation was discontinued. After she awakened, it became obvious that she had complete cortical blindness.

On neurological examination, she had a mild tetraparesis and myoclonic jerks but no pyramidal signs. The mydriatic pupils showed a prompt reaction to light, and the corneal and vestibulo-ocular reflexes were intact. No optokinetic nystagmus could be elicited. Furthermore, the patient was disoriented and complained of retrograde amnesia and slight motor aphasia. She never denied cortical blindness (Anton syn-
drome), nor did she have a neglect. An electroencephalogram revealed high-voltage delta activity mainly over bifrontal leads, as well as paroxysmal generalized sharp slow-wave complexes compatible with a complex partial status. Initial treatment with clonazepam followed by carbamazepine (800 mg/d) stopped the seizures promptly.

Two weeks after CPR, some vision recurred such that the patient was able to discriminate light from dark shadows. However, at this stage, no visual evoked potentials could be recorded (Figure 1, A). Simultaneously, the patient developed vivid visual hallucinations consisting of brightly colored, mostly moving objects of dysmorphic shape, which in part appeared in clusters showing interactions of scenic character. The hallucinations were taken for real by the patient and caused fear. Most interestingly, it seemed that these hallucinations could in part be initiated by the patient herself. When asked, she reported that the imagination of visual contents together with perception of light and dark shadows resulted in bizarre and dysmorphic objects of bright color. After such initiation and onset, she was overwhelmed by the vividness of the hallucinatory phenomena, which usually lasted for 10 to 15 minutes. Neuropsychological testing revealed an average verbal IQ and a marked memory deficit, with the affection of verbal memory being the most severe. Furthermore, mild dyscalculia was detected, whereas executive functions, such as cognitive flexibility and concept making, were not affected. The patient was not apraxic. Eight weeks later, the visual hallucinations disappeared. Her vision had recovered to a degree that she was able to differentiate objects nominally, but

Figure 1. Visual evoked potentials 2 (A) and 8 (B) weeks after cardiopulmonary resuscitation.
the visual perception and spatial orientation deficit that affected feature perception (e.g., detail of form) as well as perception of color and contrast in association with delusional experience of colored stripes. At that time, visual evoked potentials of almost normal appearance and P100 latency could again be elicited (Figure 1, B). The cranial magnetic resonance imaging scan showed no structural lesion in the parieto-occipital cortex and no atrophy. The T2-weighted images revealed bilateral hyperintensities only in the basal ganglia. Six months later, the patient had normal memory functions and was able to read. The visual perception deficit and the visual field defects were less severe, although formal testing of visual acuity again demonstrated normal values (20/20) in both eyes, reflecting normal central vision. One year after CPR, the visual perception deficit had also disappeared.

**RESULTS**

The first PET scan obtained 1 month after CPR revealed a significantly reduced rCMRGlut of 2 to 3 SDs compared with values of controls bilaterally in almost all brain regions. In the temporomesial area and the striatum, the rCMRGlut was only slightly reduced. However, the most severely depressed rCMRGlut was found bilaterally in the visual cortex, with 13.2 (controls, 40.7) µmol/min per 100 g on the left and 12.6 (controls, 41.1) µmol/min per 100 g on the right, and in the parieto-occipital cortex, with 16.0 (controls, 37.4) µmol/min per 100 g and 16.1 (controls, 37.5) µmol/min per 100 g, respectively (Figure 2, B). Five months after CPR, the rCMRGlut had improved in all areas, with the exception of the bilateral parieto-occipital cortex, which still was markedly hypometabolic (22.9 [37.4] µmol/min per 100 g and 24.6 [37.5] µmol/min per 100 g, respectively). Eighteen months after CPR, the rCMRGlut was within the normal range in all brain regions (Figure 2, C).

**COMMENT**

We have described a patient with hypoxemia-induced cortical blindness who recovered over a period of 12 months. It should be stressed that cortical blindness occurred as a consequence of hypoxemia after cardiac arrest and CPR but not after the preceding generalized seizure, although both conditions have been reported to cause cortical blindness. Similar to other patients who have been rescued from cardiac arrest, our patient also showed a severe hypometabolism in the parieto-occipital visual-association cortex. The prognosis of cortical blindness is usually favorable, if it is not caused by ischemia and no biocapital abnormalities are present on computed tomographic scans. In our patient, the magnetic resonance imaging scan was normal and the rCMRGlut returned to normal in parallel to clinical recovery.

Visual hallucinations occur frequently during recovery from cortical blindness. We observed in our patient that they were associated with activations in secondary visual areas, which initially showed a severely depressed rCMRGlut, but recovered to normal values. Since in the ischemic cortex the balance between excitatory and inhibitory neurotransmission is disturbed, leading to hyperexcitability, the visual hallucinations are likely to be the correlate of postlesional hyperexcitability. In the acute disease stage, this hyperexcitability was so severe...
that the patient suffered a complex partial epileptic status, which responded favorably to anticonvulsant therapy.

Remarkably, our patient could trigger her hallucinations by barely viewing visual objects and by visual imagery. This gave us the opportunity to perform an activation study while she was hallucinating. It should be noted that mental visual imagery is an internal representation that gives rise to the perceptlike experience,\textsuperscript{14} while visual hallucinations occur in the absence of an adequate external stimulus, with constant experience of concrete reality and lack of insight into the situation. Our patient was not able to voluntarily separate these 2 conditions. Nevertheless, the visual hallucinations yielded activation areas in the severely hypometabolic but structurally preserved visual, parieto-occipital, and temporolateral cortex. There is accumulating evidence from neuroimaging studies in healthy volunteers that both the primary visual cortex\textsuperscript{14,15} and the visual association (parieto-occipital and temporo-occipital) areas subserve visual imagery as well as the perception of illusory contours and attention to visual motion.\textsuperscript{16-19} Recently, ffytche and coworkers\textsuperscript{20} reported rCBF increases during visual hallucinations in patients with affection of the afferent visual pathways. Since the hallucinations in our patient ceased as vision returned, they appeared as the clinical correlate of a transient cortical hyperexcitability during recovery of the partially damaged and dysfunctional visual cortex.

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