Short report

Postictal blindness in adults

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SUMMARY Cortical blindness following grand mal seizures occurred in five adult patients. The causes of seizures included idiopathic epilepsy, vascular accident, brain cyst, acute encephalitis and chronic encephalitis. Blindness was permanent in one patient, but the others recovered within several days. Since most of the patients were either unaware of or denied their blindness, it is possible that this event often goes unrecognised. Cerebral hypoxia is considered the most likely mechanism.

Cortical blindness is a rare complication of epileptic seizures. In 1903 Ashby and Stephenson summarised the clinical features of this entity in 11 children and infants (five in their own series). In addition to blindness some patients were aphasic and hemiplegic; vision returned in all except one. Since this early report there have been only a few descriptions of postictal blindness and six cases of postictal hemianopia. Blindness has been noted in infants and children, except for one adult case, while hemianopia has been described in adults and children. We report the sudden development of blindness in five adults immediately after grand mal seizures of various etiologies. The postictal blindness was transient in four of the five cases.

Case reports

Case 1. A 33-year-old woman had grand mal seizures since the age of 18. She was treated with anticonvulsant drugs and had a seizure approximately once a year. She was admitted to another hospital in status epilepticus. Upon regaining consciousness she was restless and confused. She was transferred to the Department of Neurology when she was found to be disoriented in time and place. Pupillary reactions and fundoscopic examination were normal but she was totally blind. She did not respond to any visual stimuli and no optokinetic nystagmus could be elicited. She denied her blindness. Plantar reflexes were extensor but otherwise the neurological examination was normal. The EEG showed diffuse high voltage slow waves and irregular bifrontal sharp wave activity. Results of lumbar puncture were normal. A CT head scan showed dilated ventricles and enlarged sulci and subarachnoid cisterns. Bilateral carotid and vertebral angiography was normal. Routine blood and urine analyses were also normal. The patient remained listless and generally hypoactive throughout 3 months of follow-up. Repeated examinations confirmed her persistent total blindness. Subsequently she died for unknown reasons elsewhere, but a necropsy was not performed.

Case 2. A 70-year-old woman had a transient left hemiparesis seven years before admission. Three months prior to admission she presented with convulsions involving her right arm and leg and right hemiparesis. EEG showed irregular slow waves in the left temporal area. CT head scan and lumbar puncture findings were normal. An embolus was observed in the right occipital fundus involving the lower branch of the central retinal artery. She was admitted with an acute confusional state. During the examination a grand mal seizure was witnessed. After regaining consciousness she became surprisingly alert and well oriented. However, while denying any visual problem, examination revealed her to be totally blind. She did not respond to any visual stimulus such as optokinetic flag. Pupillary reactions were normal. An embolus was seen in the lower branch of the right retinal artery as noted previously. The neurological examination was otherwise normal. The next morning she could count fingers at a distance of 2 m with a gradual improvement of her vision in three days to 20/100 in the right eye and 20/50 in the left. There was no record of her previous visual acuity. Perimetry revealed an upper temporal field defect in the right eye. On follow-up there was no change in her visual acuity, visual fields and fundoscopic findings.

Case 3. A 25-year-old woman developed a grand mal seizure disorder at the age of 20. Paranoid behaviour and brief episodes of total blindness were reported by her fam-

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ily following several of the epileptic seizures. She was admitted for evaluation of a mild right hemiparesis and treatment of frequent seizures. She was alert and well oriented. General physical examination was normal. Some slow activity was evident in the EEG in the left temporal area. A CT head scan showed a small low density lesion in the left temporal lobe not enhancing after contrast injection. Left carotid angiography was normal. During hospitalisation she had frequent advergade and grand mal seizures. Visual testing was normal until the tenth day when she was found to be completely blind following a prolonged grand mal seizure. Although alert and well oriented she appeared undisturbed by her visual loss. She did not respond to any visual stimuli and no optokinetic nystagmus could be elicited. Pupils were equal and reactive and fundoscopic examination was normal. Vision cleared gradually after four days with return to normal vision and visual fields. On follow-up CT head scan the lesion was unchanged and appeared to be a low-grade astrocytoma.

**Case 4.** In July 1976 a 23-year-old previously healthy woman suffered a grand mal seizure. On regaining consciousness she complained that she could not see. She was alert, well oriented and cooperative. General physical and neurological examinations were normal, except for visual testing which confirmed her blindness. Pupils reacted well to light and the ocular fundi were normal. EEG showed diffuse high voltage slow waves. Cerebral angiography was normal (CT scan was not then available). A day after admission her temperature rose to 39°C and she became somnolent and developed a mild expressive aphasia. Lumbar puncture was normal except for slightly elevated protein level of 0.56 g/l. Over the next two days the patient became gradually more alert. Her aphasia cleared and her vision returned to normal. Subsequent hospital course was uneventful. Her fever abated over the ensuing several days and she made a complete recovery. The presumed diagnosis was a viral encephalitis although this was never confirmed.

**Case 5.** A 20-year-old woman had presented a year previously for investigation of mental changes over the previous year. These included several episodes of confusion. On admission she was disorientated and had a markedly impaired memory function. The EEG showed periodic discharges of generalised high amplitude slow waves every 10 to 20 seconds. The CT head scan was normal. Measles antibody titre was 1/256 in the blood and 1/32 in the CSF. A diagnosis of subacute sclerosing panencephalitis was made and she was treated with isoprenosine. The current admission was precipitated by a grand mal seizure which occurred at her home. At the hospital she was awake and alert but completely blind although unaware of this. No response to optokinetic flag or any other visual stimuli was observed. Pupils reacted normally to light and the ocular fundi were normal. When examined five hours after the seizure, normal vision returned. Subsequent hospital course was slowly progressive with typical clinical and laboratory findings of subacute sclerosing panencephalitis.

**Discussion**

Cortical blindness is associated with normal pupillary reactions and absence of abnormal findings on fundoscopic examination. It may be denied by the patient (Anton syndrome). Cortical blindness has rarely been reported as a consequence of seizure. Walsh and Hoyt compared the blindness to postictal (Todd’s) paralysis, the cause of which was thought to be exhaustion of neurons by hyperactivity; this analogy, however, raises several questions. Postictal paralysis appears in patients suffering from focal seizures. A transient paralysis occurs after many of these seizures for minutes or hours, with full recovery and without remaining pathological neurological signs. The severity and the duration of the paralysis are not correlated with the severity of the convulsions or with the occurrence or absence of loss of consciousness. In fact, with abortive seizures the palsy may even be more pronounced.

As opposed to postictal paralysis, postictal blindness follows a generalised grand mal seizure and often a prolonged episode of status epilepticus. Blindness is bilateral and continues for days, weeks or months and only exceptionally for hours, sometimes being accompanied by other neurological deficits such as weakness, deafness, aphasia or dementia. If postictal blindness were analogous to postictal paralysis, a typical case would be one with a unilateral occipital lobe focus having “positive phenomena” during activation of the occipital lobe and the adjacent areas, such as light flashes, colour visions and visual hallucinations. These events would perhaps be followed by the “negative phenomena” hemianopia and blindness, or by secondary generalisation to a grand mal seizure. Sometimes the negative phenomena would occur without the positive ones.

Cases similar to this idealised description have in fact been documented. Penfield reported a case with post-traumatic scar in the right occipital pole. Seizures generally started with light flashes which were followed by left hemianopia, blindness, stiffness and occasionally by a grand mal seizure. Episodes of blindness not accompanied by the positive phenomena have been described by Straus in an 11-year-old boy. EEG showed bilateral synchronous occipital discharges; however, no recording was obtained during the ictal phase. Huot described five cases with epileptic activation of the occipital lobes. The ictal events included flashing lights, visual hallucinations, fixation of gaze, oculoclonic movements and visual field defects or blindness. In one case, blindness preceded loss of consciousness and in another it either preceded a grand mal seizure or occasionally followed it. Engel described a transient cortical blindness during prolonged partial complex status epilepticus. In these cases, the use of the term “ictal blindness” appears to be adequate since blindness occurred during an
epileptic activity of the occipital lobes and represented an inhibitory seizure. Blindness may appear during any stage of the seizure as there is no distinct border between ictal or postictal inhibition.\(^5\)

The hypothesis that postictal paralysis is caused by an active inhibitory mechanism was first suggested by Gowers,\(^6\) who objected to the "exhaustion theory" that had been proposed by Todd.\(^7\) Efron\(^8\) criticised the vague term "exhaustion" and claimed that no evidence supporting this idea stood critical analysis. He offered further evidence for Gowers' arguments and suggested that a hyperpolarisation mechanism developing during the epileptic activity was responsible for ictal or postictal inhibition. This mechanism could well explain ictal blindness as well as a short transient postictal blindness or hemianopia, such as those described by Kosnik \textit{et al.}\(^5\) In their cases, vision loss for several hours was associated with focal seizures and epileptic discharges involving the occipital lobe. However, this mechanism can not account for the relatively long duration of blindness following grand mal seizures or status epilepticus.

Fiume\(^4\) and later Olurin\(^7\) proposed intra-ictal anoxia as the basis for postictal blindness. During seizures, severe hypoxia, acidosis, rise in PCO\(_2\) and lactic acidemia have been found.\(^8\) These changes are induced mostly by respiratory spasm, obstruction of airways and muscular activity during the seizure. Prevention of hypoxia and muscle contractions by artificial respiration and curarization have markedly reduced the mortality of convulsing cats and rats and avoided acidosis, accumulation of lactic acid and cardiac arrhythmias.\(^9\) Adequate oxygenation enabled the brain temporarily to meet the increasing energetic demands during seizures.\(^10\)

Brain anoxia could well account for the bilaterality of the blindness and its relatively long duration since the occipital lobes are especially sensitive to anoxia, being located in the cerebral blood supply border zone.\(^21\) In the last two decades with the wide use of cardio-pulmonary resuscitation, many severe hypoxaemic episodes and hypotensive events have occurred. Not uncommonly they have been complicated with cortical blindness.\(^6\)\(^22\) Thus, anoxia has been recently considered to be one of the most frequent immediate causes of bilateral cortical blindness.\(^23\)

In two of our patients the underlying cerebral disease was focal and unilateral and therefore it is not possible to relate directly the location of the lesions to the bilateral blindness. Four patients were blind for hours or days after a single grand mal seizure. Patient 1 was permanently blind after status epilepticus lasting several hours, and it may be assumed that the irreversibility of the blindness was due to severe prolonged anoxia. Permanent postictal blindness was described by Ashby and Stephenson in one of their cases.\(^1\)

Anton's syndrome was present in at least three patients and only one complained spontaneously of visual loss. It is therefore possible that postictal blindness occurs more frequently than has been reported, going unrecognised with the accompanying behavioural changes being attributed to postictal confusion.

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References

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17 Todd RB. Clinical lectures on paralysis, disease of the brain and other affections of the nervous system. London, 1856.


