Case Reports

Palinopsia Due to Nonketotic Hyperglycemia

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PALINOPSIA is a distortion of processing in the visual system in which images persist or recur after the visual stimulus has been removed.[1,2] In a related condition, allesthesia, visual images are transposed from one half-field of vision to the other.[3] Both conditions are well described but rare.[1-13] Controversy has arisen about whether the pathophysiologic mechanism of palinopsia is a partial seizure[3,4] or due to a "release phenomenon."[5,6] The transitory nature of palinopsia associated with persistent neurologic lesions is also remarkable, suggesting the interaction of more than one causative factor. We report here the case of a patient with visual hallucinations, palinopsia, allesthesia, and loss of spatial orientation. These symptoms were caused by focal seizures due to nonketotic hyperglycemia. Hyperglycemia may be a frequent causative factor in palinopsia. This has important practical and theoretic consequences.

Report of a Case

The patient, a 57-year-old right-handed rancher, had the sudden development of a right homonymous hemianopia. During the subsequent three days, he had peculiar spells from one to three times per hour and persistence of the field cut. The spells lasted three to six minutes each and usually consisted of visual hallucinations followed by a left parietal headache that was intensified by coughing. On several occasions, including one witnessed in the emergency ward, the spells culminated in a motor seizure, manifested by jerking, eye deviation leftward, interruption of consciousness, and postictal confusion. A partial seizure arising from the left occipital lobe was confirmed by an electroencephalographic (EEG) recording during one of the patient's typical visual hallucinations. He described a feeling of spatial disorientation and poor memory, particularly severe while the episodes were in progress. He could not give simple directions to his home nor find his way out of the physician's office. Because he could give an accurate medical history and carry on a normal conversation between seizures and because he could clearly describe the visual phenomena while they were occurring, he seemed to have focal brain dysfunction rather than a more generalized impairment or an acute confusional state.

The visual phenomena were complex and occurred together. All of the spells included flickering spots resembling a 4th of July fireworks display of "beautiful red, gold, and black sparkling lights" that would increase in size and number in the right visual field. Frequently during the time the "fireworks" were present, he had hallucinations of objects that had actually been present in his left visual field also appearing and persisting in the blind right visual field. For example, on one occasion while his physician was standing quietly on the left side of his bed during a spell, the physician's image suddenly appeared on the right side of the bed as well, in the patient's sightless hemifield. The image persisted on the blind side even when the physician moved behind the patient. Other objects the patient could see on his left shifted to the blind right side and persisted in a similar fashion during other attacks. The palinopsia (persistence) and allesthesia (transposition) always occurred together, and they did not occur in the absence of flickering lights, although the flickering lights did at times occur without the other phenomena.

Overshadowed by his dramatic visual symptoms was a history of a 10-kg (22-lb) weight loss and excessive thirst in the preceding month. He had a past history of hypertension. By examination, his neurologic abnormalities were limited to the perceptual phenomena and dense right homonymous hemianopia to confrontation testing already described. Visual acuity, extraocular movements, pupillary reactions, and other cranial nerve, motor, reflex, and sensory testing were normal.

A computed tomographic head scan was normal. His blood glucose level was 609 mg per dl (33.8 mmol per liter), serum sodium was 130 mEq per liter (130 mmol per liter), and serum potassium was 4.7 mEq per liter (4.7 mmol per liter). A glycosylated hemoglobin was 15.3% (0.153) (normal range, 5% to 9% [0.05 to 0.09]). Other biochemical testing was unremarkable. His visual symptoms and seizures ceased with control of the blood glucose level, first with insulin, then with oral hypoglycemic agents, and finally with diet alone. No anticonvulsant drugs were required. His visual field returned to normal in two days after control of the blood glucose. In four years of follow-up, he has had no recurrence of seizures or palinopsia. His diabetes remains controlled. On neurologic examination there are no abnormalities and formal visual field testing is normal.

Comment

Palinopsia is a rare phenomenon, and its mechanism is disputed. There may be a number of mechanisms for palinopsia in general, and more than one factor may be involved in any single patient. Much of the trouble in unraveling the causes of palinopsia lies in its rare and transient occurrence. Discussions in the literature are based on incomplete investigation, which is unavoidable in most instances.

Bender and colleagues[2] suggested four possible mechanisms for palinopsia: visual aftersensations, sensory seizures, hallucinations, and psychogenic elaborations or fantasies. Subsequent observers have raised other possible mechanisms, including "release hallucinations"[5,6] and migraine.[11]

We think that our patient's hallucinations, palinopsia, allesthesia, visual field defect, and spatial disorientation were caused by seizures triggered by nonketotic hyperglycemia. A number of reversible neurologic disorders are associated with nonketotic hyperglycemia, including confusion, hallucinations, hemianopia, sensory deficits, hemiparesis, aphasia, and

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focal motor seizures (epilepsia partialis continua).[14-16] Our patient's neurologic abnormalities seem to be part of that array. Most of the neurologic abnormalities associated with nonketotic hyperglycemia revert to normal with correction of the hyperosmolar state, as in our patient.

Although hyperglycemia has been observed with palinopsia before,[5,7] its actual significance seems to have been little appreciated. In the 39 cases of palinopsia reviewed in this discussion,[1-13] the reports specifically mentioned the presence or absence of diabetes mellitus or hyperglycemia in only five instances.[5-7] Of those, two of the patients had normal glucose levels, one had diabetes mellitus but normoglycemia during palinopsia, and two had pronounced elevations of blood glucose levels. These latter hyperglycemic patients had reported blood glucose values as high as 699 mg per dl (38.8 mmol per liter) and 534 mg per dl (29.6 mmol per liter), respectively.[5,7] These elevated glucose levels are within the range known to cause epilepsia partialis continua.[14] Adding our case, 50% of patients with palinopsia in whom the glucose level was reported had substantial elevations. This suggests that palinopsia not uncommonly may be triggered by hyperglycemia.

In our patient, the mechanism of palinopsia appeared to be ictal. Palinopsia associated with seizures has frequently been noted in the articles reviewed here. Of 39 patients reported with palinopsia, 25 either had EEG evidence of or were observed to have seizures associated with palinopsia or responded to anticonvulsant therapy. Of these 25, 13 certainly had ictal palinopsia and another 7 had abnormal but nonparoxysmal EEGs. Considering that a surface EEG may be false-negative even during an ictus, an epileptic mechanism appears to be operative in most patients with palinopsia.

In addition to clinical observations, experiments indicate that serum hyperosmolarity and brain dehydration can induce seizures, given a potentially epileptogenic focus.[17] Our patient had no identifiable structural abnormality of his brain, in contrast to the experimental situation and most patients with epilepsia partialis continua due to nonketotic hyperglycemia.[14-17] The two previously reported cases of hyperglycemia with palinopsia did show structural pathology.[5,7] In our patient, the hemianopia and spatial disorientation persisted longer than the "fireworks," palinopsia, and allesthesia. We assume the more persistent manifestations represented a "Todd's paralysis." All of the visual phenomena cleared within two days after treatment of hyperglycemia, supporting this explanation.

Nearly all reported cases of palinopsia (34/39 or 87%) are associated with a homonymous field defect. The defect is often caused by tumor, arteriovenous malformation, hemorrhage, stroke, or trauma. When an anatomic lesion is found by computed tomography or a pathologic examination, it is usually situated at the junction of the occipital, parietal, and temporal lobes, more often in the right hemisphere than the left. When palinopsia is associated with migraine[11] or hyperglycemia (this report), however, no identifiable anatomic lesion need be present.

As for other pathophysiologic mechanisms, there are similarities between palinopic images and normal visual afterimages in some instances, but there are also differences.[2,8,12] Even if palinopsia is due to "exaggeration" of the normal afterimage process, that implies some abnormal mechanism—but what is it? The analogy does not really broaden our understanding of palinopsia or its management. Likewise, the idea of "release hallucinations," similar to the hallucinations of sensory deprivation,[5,6,8,12,18,19] serves mostly to help define a nonictal category of palinopsia but does not give a clue about the neurophysiology involved. No cases of palinopsia have been reported in psychosis, so delusions or psychiatric fantasies are unlikely explanations. As the neurophysiologic basis of migraine is better defined, the results will undoubtedly be relevant to some cases of palinopsia. It is clear at the present level of our knowledge, however, that palinopsia is a dysfunction of the association areas at the junction of temporal, occipital, and parietal lobes, is frequently an ictal phenomenon, and can be triggered by nonketotic hyperglycemia.

REFERENCES

1. Critchley M: Types of visual perseveration: 'Palinopsia' and 'illusory visual spread.' Brain 1951; 74:267-299
2. Bender MB, Feldman M, Sobin AJ: Palinopsia. Brain 1968; 91:321-338
3. Jacobs L: Visual allesthesia. Neurology (NY) 1980; 30:1059-1063
4. Swash M: Visual perseveration in temporal lobe epilepsy. J Neurol Neurosurg Psychiatry 1979; 42:569-571
5. Brust JCM, Behrens MM: 'Release hallucinations' as the major symptom of posterior cerebral artery occlusion: A report of 2 cases. Ann Neurol 1977; 2:432-436
6. Cummings JL, Syndulko K, Goldberg Z, et al: Palinopsia reconsidered. Neurology (NY) 1982; 32:444-447
7. Michel EM, Troost BT: Palinopsia: Cerebral localization with computed tomography. Neurology (NY) 1980; 30:887-889
8. Cleland PG, Saunders M, Rosser R: An unusual case of visual perseveration. J Neurol Neurosurg Psychiatry 1981; 44:262-263
9. Meadows JC, Munro SSF: Palinopsia. J Neurol Neurosurg Psychiatry 1977; 40:5-8
10. Lance JW: Simple formed hallucinations confined to the area of a specific visual field defect. Brain 1976; 99:719-734
11. Klee A, Willanger R: Disturbances of visual perception in migraine. Acta Neurol Scand 1966; 42:400-414
12. Kinsbourne M, Warrington EK: A study of visual perseveration. J Neurol Neurosurg Psychiatry 1963; 26:468-475
13. Landis T, Cummings JL, Benson DF, et al: Loss of topographic familiarity. Arch Neurol 1986; 43:132-136
14. Singh BM, Strobos RJ: Epilepsia partialis continua associated with nonketotic hyperglycemia: Clinical and biochemical profile of 21 patients. Ann Neurol 1980; 8:155-160
15. Guisada R, Arieff AI: Neurologic manifestations of diabetic comas. Metabolism 1975; 24:665-679
16. Maccario M: Neurologic dysfunction associated with nonketotic hyperglycemia. Arch Neurol 1968; 19:525-534
17. Vastola EF, Maccario M, Homan R: Activation of epileptogenic foci by hyperosmolality. Neurology (Minneapolis) 1967; 17:520-526
18. Cogan DG: Visual hallucinations as release phenomena. Albrecht von Graefes Arch Klin Exp Ophthalmol 1973; 188:139-150
19. Heron W, Scott TH: Visual disturbances after prolonged perceptual isolation. Can J Psychol 1956; 10:13-18

An Unusual Cause of D-Lactic Acidosis

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D-LACTIC ACIDOSIS has been well documented in patients with the short-bowel syndrome.[1-4] Abnormal colonic bacterial flora have been found to be responsible for the D-lactic acid production, and symptoms can be treated with enteric antibiotics. We report an unusual case of D-lactic acidosis caused by a mechanism not previously described.

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