Noninfectious Thrombosis of the Superior Sagittal Sinus in a Patient With Iron Deficiency Anemia

To the Editor:

Noninfectious thrombosis of cerebral veins is commonly found in patients with hereditary coagulation or immunologic disorders and during pregnancy or intake of contraceptive drugs. Severe cases are often lethal.

A 46-year-old male patient, who had been previously well, was admitted to our intensive care unit with symptoms of nausea, dizziness, pulsatile tinnitus, bradycardia, and iron deficiency anemia. On the fifth day after admission, the patient experienced a paresis of his left arm and, on the seventh day, a paresis of the right abducens nerve. Angiography of the cerebral veins, computed tomography, and magnetic resonance imaging (MRI) showed an approximately 5-cm-long thrombosis of the superior sagittal sinus, beginning at the confluens sinuum. Our further examinations did not reveal any other known causes of cerebral vein thrombosis.

This patient's iron deficiency anemia was caused by chronic blood loss due to a prolapse of the lower rectum. After anticoagulant treatment with high-dose intravenous heparin for 3 weeks, MRI showed recanalization of the sagittal sinus. Paresis of the arm and the abducens nerve, pulsatile tinnitus, bradycardia, and anemia all disappeared and were retrospectively interpreted as signs of high intracerebral pressure. Following surgical resection of the rectal prolapse, the patient received further anticoagulant therapy with phenprocoumon and was discharged from the hospital in good health.

Iron deficiency anemia coinciding with cerebral vein thrombosis has been reported in four patients: in a 22-month-old boy with iron deficiency anemia and thrombocytosis; in a young woman with hypochromic anemia and thrombocytosis following surgery; and in two women with anemia due to chronic bleeding from myoma uteri. In two cases, thrombocytosis associated with anemia was assumed to cause cerebral vein thrombosis. However, in the 2 women with myoma uteri and in our patient, thrombocyte counts were normal. Other pathogenetic considerations might include rheological phenomena resulting from a relative anemia in the venous sinus system where negative pressure values prevail, or a hypercoagulable state due to chronic blood loss. However, in neither case were parameters indicating coagulation disorders found.

With this report, we have sought to focus attention on iron-deficiency anemia, a frequent disorder, coinciding with thrombosis of the cerebral veins, a rare and sometimes lethal disease.

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References


Vascular Headaches and the Anticardiolipin Antibodies

To the Editor:

Your recent publications from The Antiphospholipid Antibodies in Stroke Study Group, and Coull and Goodnight reflect the important role of antibodies against phospholipids in many different pathological conditions. In recent years, interest has been growing in neurological syndromes, including migraine, that are associated with antiphospholipid antibodies. Since Brandt and Lessell first reported the presence of antiphospholipid antibodies in two of 11 patients with migraine and systemic lupus erythematosus, 27 more cases of migraine with antiphospholipid antibodies have been described.

However, some investigators have felt that headache might simply be a common feature in patients with antiphospholipid antibodies, and that these patients would present primarily to neurologists. In order to test whether antiphospholipid antibodies play a role in primary headaches, we recently measured anticardiolipin antibody levels in migraine patients during and between acute attacks and in cluster headache patients during cluster periods and in acute attacks. All suffered from only one or the other of these illnesses, without evidence of any associated disease, particularly of any autoimmune disease or other known manifestations of antiphospholipid antibodies such as thromboses, episodes of thrombocytopenia, or strokes.

In 94 consecutive migraine patients aged 18-50 years (11 studied during acute attacks) and 20 consecutive male cluster headache patients aged 32-48 years (all in the cluster period, three during acute attacks; R. Hering, E.G.M. Couturier, R.A. Asherson, and T.J. Steiner, unpublished observations), no elevations were found of anticardiolipin antibody levels assayed by the method of Gharavi et al. The 95% confidence intervals for these zero observations in 94 migraine patients and 20 cluster headache patients were 0.3-9.9% and 0.1-17.8%, respectively. Manoussakis et al. found high anticardiolipin antibody levels in 23% of healthy blood donors (95% confidence interval, 0.9-5.1%). These findings suggest no specific association between anticardiolipin antibody and migraine or cluster headache, and certainly no importance of anticardiolipin antibody in their etiopathogenesis.

In other studies in which antiphospholipid antibody levels have been elevated in migraine, all affected patients have had other neurological or systemic complications in addition. The most reasonable conclusion is that the elevated antiphospholipid antibody levels were related to these other conditions, migraine being coincidentally associated, with a normal prevalence of approximately 10%.

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References

Bilateral Loss of Vision in Bright Light

To The Editor,

We were interested in the communication of Wiebers et al.1 reporting four patients with episodic bilateral vision impairment related exclusively to light exposure, who revealed angiographically documented bilateral high-grade stenosis or occlusion of the internal carotid arteries. Because there are few such observations, we would like to report two other cases, the two particular clinical features of which complement the communication of Wiebers et al.

**Case 1.**

In May 1988, a 48-year-old man who had arterial hypertension and who smoked heavily experienced two kinds of visual symptoms. While extending his head to shave, he experienced blurring of the upper altitudinal field of vision in both eyes, followed by spread of the blurring to the entire visual field, which lasted for 10 minutes and was followed by 5 minutes of blindness. Vision returned first in "black and white" and then was normal. A similar course of events occurred in three separate episodes. The second pattern consisted of sudden bilateral blindness of 5–7 minutes duration. He recovered from this blindness, but the pattern returned, with three additional spells followed sequentially by 10 minutes of "black and white" vision and then normal vision.

On examination, blood pressure was 180/100 mm Hg and visual acuity was normal. Ophthalmoscopy revealed bilateral venous stasis retinopathy, and ocular pneumoplethysmography recorded very low values of 25 mm Hg bilaterally. Carotid ultrasonography showed a near-80% stenosis of both internal carotid arteries, confirmed on angiography, that was associated with a recent reversal of flow in both ophthalmic arteries. While taking aspirin and when the flow reversal of the bilateral ophthalmic arteries was major, the visual symptoms disappeared.

**Case 2.**

A 74-year-old hypertensive, diabetic man suffered three episodes of exposure to bright light with blurring of bilateral upper altitudinal vision for 10 minutes, then total blindness for 15 minutes. Vision returned first in "black and white" and then was normal. Clinical examination revealed bilateral systolic carotid bruits while visual acuity was normal bilaterally. Ophthalmoscopy revealed mild venous engorgement bilaterally while the intracranial tension was bilaterally low, 20 mm Hg on the right and 25 on the left. Carotid ultrasonography revealed a high-grade stenosis at the origin of both internal carotid arteries, with mild flow reversal of the ophthalmic arteries, confirmed by cerebral angiography. After starting on ticlopidine, this patient's symptoms also disappeared although the flow reversal of the bilateral ophthalmic arteries was major.

Our two cases are similar to the four reported by Wiebers et al.1 The phenomenon of vision loss on exposure to bright light is usually associated with choriodoretinitis or retinal pigmentary degeneration, but may be related to atherosclerotic carotid artery disease, either with unilateral vision loss2 or bilateral vision loss.3 This phenomenon appears to be related to bilateral simultaneous retinal ischemia, delaying regeneration of visual pigments in the pigment epithelial layer, and must be distinguished from bilateral occipital lobe ischemia of vertebrobasilar system insufficiency.4

Our two observations contribute to the understanding of this phenomenon by adding two clinical features not described in the four observations of Wiebers et al.1 In our two cases, blurring of vision began in the upper part of the visual field and then reached all fields. This evolution usually characterizes retinal ischemia, while the defect of the lateral part of the visual field characterizes disease of central pathways.6 In addition, carotid ultrasonography showed that, during these visual symptoms, the flow reversal of the ophthalmic arteries was mild and uneven, and became strong and steady when the symptoms disappeared.

We conclude that bilateral loss of vision in bright light related to severe carotid atherosclerotic disease indicates low perfusion in the retinal vessels until the flow reversal of both ophthalmic arteries corrects it. This hypothesis would explain why the visual defect begins in the upper part of visual field.

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References


The following is in reply.

We appreciate the interest and comments of Giroud and colleagues. None of our four patients had noted "black and white" vision prior to the return of color vision, and none of them manifested altitudinal defects. Nevertheless, these symptoms are undoubtedly part of the clinical spectrum of visual loss with exposure to bright light resulting from bilateral carotid occlusive disease.

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