Transient amaurosis under decreased atmospheric pressure with sphenoidal sinus dysplasia

Case report

KENICHIRO SUGITA, M.D., TOSHIYUKI HIROTA, M.D., IKUZO IGUCHI, M.D., NAOKI KAGEYAMA, M.D., AND AKIKAZU ITO, M.D.

Departments of Neurosurgery and Otorhinolaryngology, Nagoya University School of Medicine, Nagoya, Japan

The authors report a unique case of transient amaurosis occurring every time the patient flew in a jet plane and frequently when he drove up a mountain. X-ray examination showed dysplasia of the sphenoidal sinus and optic canals. The pathogenesis and the treatment of the amaurosis is discussed.

KEY WORDS • transient amaurosis • sphenoidal sinus • optic canal • atmospheric pressure • hyperbaric chamber

Transient amaurosis is usually observed in association with vascular insufficiency due to atherosclerosis or a vasospasm of the extra- and intracranial arteries. We describe a unique case of transient amaurosis occurring each time the patient was in a jet plane and often when he was in an automobile driving up a mountain. Transient amaurosis in such situations has not been reported in the literature except in one case of postirradiation pneumatocele in a massive chromophobe adenoma reported by David, et al.

Case Report

This previously healthy 26-year-old man had first noted transient bilateral blindness upon arising in the morning at the age of 19 years. He had the same episodes a few times in the following 2 years. During the 6 years before presentation, he experienced transient bilateral blindness on five occasions while driving up a mountain. During this period he also experienced the same phenomenon eight times in a jet liner. On jet flights he lost his sight several minutes after taking off and recovered it completely within 30 to 60 minutes. Every attack had the same pattern of sudden onset, complete bilateral blindness, and recovery of the peripheral visual field within 1 hour. Prior to January, 1974, his vision remained normal except for these attacks. In February, 1974, he experienced the same episode on a ski-tour bus while traveling up a mountain. This time vision returned only partially, and he had continued visual disturbance thereafter.

Examination. On February 15, 1974, 2 weeks after the last attack, the patient visited our hospital. His visual acuity with full cor-
K. Sugita, et al.

Fig. 1. Tomograms of lateral view of pneumoencephalography. Left: Lateral view shows the large sphenoidal sinus without cisternal communication. Right: Anteroposterior view showing the large sphenoidal sinus without septum.

rection was finger counting at 10 cm in the right eye and 20/30 in the left. Visual field examination showed the defects of a temporal half and inferior temporal quadrantanopsia with a centrocecal scotoma in the left eye. The right optic disc was slightly pale and the left was normal. Except for the visual disturbance, physical examination revealed nothing abnormal. On March 6, 1974, the vision was 20/200 in the right eye and 20/30 in the left. Plain x-ray films and tomograms of the skull showed the sphenoidal sinus to be extremely large in size (Fig. 1). Bone defects of the sphenoidal sinus around the optic canals were suspected. The structures of the medial and inferior part of the optic canals were not clear bilaterally in the x-ray anteroposterior view or the tomograms, the right optic canal being more radiolucent than the left. The sella turcica was relatively small and slightly deformed. The tuberculum and chiasmatic sulcus were normal in size and shape. Neither arteriogram nor air study showed any pathological findings.

On March 10, 1974, the patient was placed in a hyperbaric chamber to test whether a hypoxic factor was a cause of the visual disturbance. While atmospheric pressure was increased and maintained at 2.0 atm for 1 hour, no neurological change was observed. When the pressure was lowered from 2.0 to 1.9 atm, the patient complained of a sudden blindness bilaterally and this symptom was identical to that of his preceding episodes. The light reflex was abolished on both sides. Extraocular eye movements were normal bilaterally. No other neurological defects were observed. Thirty minutes later the peripheral visual field returned to its previous level.

Operation. On March 17, 1974, sinusotomy of the sphenoidal sinus using a transmaxillary approach was performed. The sphenoid sinus had a capacity of 40 ml when filled with air. The septum between the sphenoidal sinuses was absent. The mucous membrane was histologically normal. The roof of the sinus near the optic canals was not touched. It could not be confirmed whether or not the aperturae sinus sphenoidalis communicated. Bone defects, 2 cm in diameter, were made on both sides so that the sphenoidal and maxillary sinuses communicated freely.

Postoperative Course. Two weeks after operation, the patient was again placed into the hyperbaric chamber. No visual disturbance occurred during the whole course of increase, maintenance, and decrease of atmospheric pressure. The maximum pressure reached was 2.5 atm. Vision and visual field improved gradually after the operation (Fig. 2). The visual acuity was 20/70 in the right eye and 20/20 in the left eye 2 months postoperatively. No episode of transient blindness has occurred since surgery.

Discussion

Except for the several episodes observed during the first 8 years, all episodes in the 6 years before surgery occurred only in jet air-
Atmospheric pressure and transient amaurosis

Craft or in automobiles while driving up a mountain. The air pressure in the cabin of a jet aircraft usually drops to 0.9 or 0.8 atm during its ascent. In the case of an automobile, air pressure at an altitude of 1000 meters is approximately 0.9 atm. The patient was more accurately observed during his attack in the hyperbaric chamber when the pressure was decreased from 2.0 to 1.9 atm. It was apparent that the pressure drop of 0.1 atm from adapted pressure, regardless of oxygen tension, caused the attacks. A 0.1 atmospheric pressure, equivalent to 76 mm Hg, is close to the normal pressure of the retinal artery.

In addition to pressure decrease, the symptom was precipitated by three anatomical factors: 1) the sphenoidal sinus dysplasia had a capacity three to four times greater than normal capacity; 2) there were bone defects around the optic canals; and 3) the apertura sinus sphenoidalis was obstructed (although the last factor was only suspected before surgery). It was speculated that the air inside the abnormally large sphenoidal sinus expanded under a decreased atmospheric pressure and pushed the optic nerve up through the bone defects, thus causing a disturbance of the regional blood flow. Renn and Rhoton reported that bone defects around the optic canal were observed in 2% of their autopsy cases studied. After surgery, the air in the sinus was presumably absorbed slowly through the mucous membrane while the pressure was equilibrated with the surrounding tissues. The reason for the slow absorption of air in the sinus before operation was suspected to be poor vascularity in the wall of the sinus in comparison with that of the lung, intestine, and other organs.

Speculation concerning the pathogenesis was confirmed by the fact that attacks of blindness did not occur in the hyperbaric chamber after the bilateral sinusotomy, which

![Diagram](image-url)
gave free communication between the sphenoidal sinus and the maxillary sinus.

Acknowledgments

The authors wish to express their appreciation to Drs. W. F. Hoyt and H. Sasaki for their advice and encouragement, and also to Drs. K. Sakakibara and H. Takahashi for their assistance in operating the hyperbaric chamber.

References


Address reprint requests to: Kenichiro Sugita, M.D., Department of Neurosurgery, Nagoya University School of Medicine, Tsurumai 65, Showa ku, Nagoya, Japan 466.