


Research Article

Experiencing Acute Blindness: An Elderly Man's Rosette Formation in a Blind Eye Treated Successfully by Immediate Globe Massage

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Abstract

Background: Observation in acute central retinal artery occlusion

Methods: Ocular massage of a blind eye

Results: A patient's unique observation in an episode of acute unilateral blindness is described. Blindness happened when the patient suddenly rose from a sitting position to standing. The patient immediately lay flat on his back and began to digitally massage the closed eye. By closing his normal seeing right eye, he noticed with the left blind one a nearly complete black visual field that was interrupted by many light oval spaces resembling a rosette which rapidly flickered continuously and rhythmically. Within seconds, and after intense ocular massage, his clearly visible peripheral visual field returned and during those seconds, he noticed that the black central visual field (with an unchanged rosette formation) was smaller than before. After another brief but intense ocular massage, the black visual field center disappeared completely. The left eye had recovered its normal vision, which persisted.

Conclusions: A central retinal artery occlusion in the left eye is quite probably the cause of his temporary blindness. The appearance of a rosette formation in a blind eye has never been described in the literature to the best of our knowledge.

Bullet points: Amaurosis, orthostatic failure, defective blood coagulation, central retinal artery occlusion, rosette, ocular massage

Keywords: Central retinal artery occlusion; Amaurosis; Phosphenes; Ocular massage; Arterial hypertension; Atrial fibrillation

Introduction

Central retinal artery occlusion (CRAO) is a devastating ocular emergency with a poor visual prognosis. Arterial retinal obstructions can be embolic, atherosclerotic, ischemic, or inflammatory [2]. CRAO is believed to be caused due to an embolic or thrombotic occlusion of the central retinal artery, followed by retinal ischemia [18]. Patients present with an acute painless loss of vision of a varying severity depending on the absence or presence of a cilioretinal artery and the duration of blood flow deprivation. Tobalem & Schutz [24] reported that retinal infarction is most likely to occur after only 12-15 minutes of complete CRAO. The retinal ganglion cells depend entirely on oxygen supplied by the central retinal artery's end-artery circulation. According to a study on the vascular risk factors of CRAO, the main risk factor is arterial hypertension affecting 197 patients (79.8% out of 247 patients) with CRAO. Retinal emboli were detected in 28 out of 253 patients with CRAO (11.1%) [22]. Retinal arterial occlusions are seldom caused by

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an embolism originating from cardiac tumors [21]. Hayreh & Zimmerman [12] highlighted another important but less often acknowledged vascular CRAO risk factor, namely that a fall in perfusion pressure below the critical level in the retinal vascular bed can provoke CRAO. Several factors can trigger such a drop in perfusion pressure: that is identical the mean blood pressure in the artery minus the intraocular pressure. A marked fall in mean arterial blood pressure can be caused by factors such as nocturnal arterial hypotension, severe shock, during hemodialysis, a CRA spasm, marked stenosis or occlusion of the internal carotid or ophthalmic artery, or ocular ischemia. Aggressive antihypertensive therapy with very potent antihypertensive drugs is another cause of arterial hypotension resulting in an abnormal drop in blood pressure [10]. The general opinion is that a patient suffering from acute blindness perceives only gray or black in the amaurotic eye, which is not necessarily true. In this article, I describe a patient with acute blindness who saw a rapidly fluctuating black and white pattern in his visual field. The elderly patient suffered a unilateral acute blindness event; he had two risk factors: an orthostatic failure in a stress situation and a defective blood coagulation. I describe this patient's unique observation in his episode of acute blindness.

First report of an unusual episode with the patient's description

One morning, six weeks ago, an 83-year old ophthalmologist experienced acute blindness in his left eye. He had been sorting books, and had risen from his chair several times to arrange heavy books. He then suddenly felt faint after rising quickly from a sitting position and noticed blindness in his left eye. Closing his right eye, he saw with the left one a nearly complete black visual field that was interrupted by many light oval spaces resembling a rosette (Figure 1) which flickered continuously, rapidly and rhythmically, and which was faster than his pulse. The patient immediately lay flat on his back and, within a few seconds, pressed his lid-closed left eye three times back into the orbit with his fingers. He then opened his left eye again, but still perceived the black fluctuating center, but the peripheral vicinity of the black visual center - the peripheral visual field - was now clearly visible in a circle around the black center of the visual field. There was no shadow or obscuration in the peripheral visual field. The black center had become smaller than before. Within seconds, the patient then again squeezed the closed left eye twice and then re-opened his left eye. To his enormous reassurance, the black center had disappeared completely, now; he was able to see normally with his left eye. The left eye's visual acuity was equal to that of the right eye. In the following weeks his vision remained normal and he had no more visual problems. In the following month, the patient had no visual problems. An eye examination revealed - besides slight cataracts - a normal visual acuity and visual field.

History of patient's vascular diseases

According to the patient's medical history, he had been taking antihypertensive drugs and anti-thrombotic drugs (Phenprocoumon, "MarcumarR"), for atrial fibrillation for more than 20 years. He had never suffered a transient ischemic attack (TIA) or amaurosis fugax. He usually has his "International Normalized Ratio" (INR) measured to assess the prothrombine time, approximately every two weeks. The patient had normal intraocular pressure. A Duplex ultrasound exam of his carotid artery revealed normal blood flow. The patient had no diabetes mellitus. He did not smoke and avoided alcohol. His blood sample revealed no hyperviscosity or hypercholesterolemia. At the time of the blindness event he had neither infection or migraine.

Two explanations for visual loss

The loss of sight in the patient's episode of acute blindness may be attributable to: orthostatic failure by rising repeatedly and abruptly standing up from his chair. His blood pressure was 110/70 mm Hg, as measured 15 minutes after the episode. His blood pressure had previously been higher than in this situation, but still within normal limits. There was a defective blood coagulation with an elevated INR-value of 3.7 (normal INR between 2 and 3). The INR-value was measured ten minutes after the episode.

The combination of orthostatic failure and relative low blood pressure and an increased INR may have caused his acute but temporary blindness.

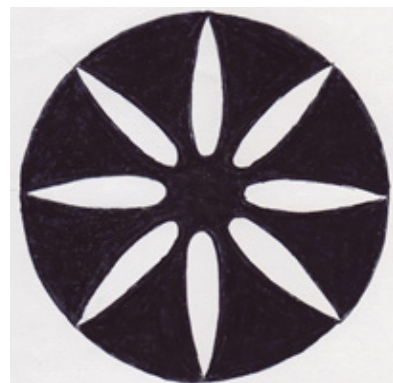


Figure 1: The center of the black visual field may represent the macula area. The black areas between the center and the peripheral field may correspond to the retinal radiating structures, which are reminiscent of Henle's fiber layer's structures. This area was not entirely black as he observed regularly inserted light oval spaces radiating over the entire field. It resembled a rosette that was rapidly flickering continuously and rhythmically. These oval spaces may represent light scleral areas. The light oval spaces may be caused by contracting of retinal tissue - presumably because of the lack of oxygen. In such an emergency situation, lasting a few seconds, it was not possible to count the number of the light oval spaces. There may have been more than shown in the figure.

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To the best of our knowledge this is the first description of such a visual perception during an episode of unilateral blindness.

Discussion

Visual changes occurring during indentation of the temporal eyeball region at different intervals

With simultaneous indentations of both eyeballs in the temporal region Grüsser & Landers [9] observed flickering patterns, some with radiation direction of structures, mostly with a slightly bowed direction of structures, beginning from a gray center. These “phosphenes” occurred after about 10 sec of indentation. The radiating pattern was not interrupted by bright oval spaces. The observation by Grüsser & Landers [9] may be explained by a too weak indentation of the eyeball without complete blind eye

Cases of brief or prolonged occlusion of the central retinal artery

Episodes of brief amaurosis without visual sequelae were reported by Knapp & coworker [15]. They described gaze-induced amaurosis in a 13-year-old girl. Blindness occurred on abduction of the left eye. Her visual acuity dropped to no light perception, and the left pupil became unreactive to light stimulation when the patient abducted the eyes. Her vision recovered within 10 seconds once her eye returned to its primary position. Late vision recovery was reported in a 70-year-old man who suffered from a combined obstruction of both the central retinal artery and the lateral posterior ciliary artery in his left eye. Duker & Brown [4] treated the patient intensively, but were unsuccessful. However, one week later, his vision had suddenly recovered to almost normal. Reperfusion occurred after four days. His visual acuity improved from mere light perception to 20/30.

Retinal vascular changes in alternating head posture

Furlan & coworker [7] reported on a patient with a carotid artery's occlusion. The patient complained of unilateral visual loss on changing from a sitting or upright position. His episodes probably had a hemodynamic rather

than embolic cause. Patients may experience an orthostatic drop in the retinal artery pressure without a corresponding decrease in brachial blood pressure. Intraocular pressure (IOP) can be altered by changing our body position. Findings have confirmed the effect of body position on IOP. In ocular normotensives, intraocular pressure increased by 2 – 4 mm Hg when the body positions changed from standing or sitting erect to supine [17]. Burger & coworker [1] reported on a 78-year-old woman who experienced five episodes of visual loss in the left eye over several days. Each episode began with sudden decrease in central vision after she bent over. During an ophthalmoscopic examination, these authors observed blanching of the central retinal artery that lasted three minutes before clearing suddenly. Jehn & coworker [14] characterized amaurosis fugax as a sudden, monocular, painless, temporary visual loss due to hypoperfused retinal circulation. A 65-year-old man experienced recurrent exercise-induced transient monocular blindness. His visual symptoms consisted of a rapidly progressing visual field constriction in his right eye. The events lasted from 30 seconds to three hours. They appeared regularly during strenuous sport activities, such as jogging or biking.

Ultrastructural retinal findings in CRAO or in retinal ischemia

Functional loss usually occurs much earlier and more severely than the anatomic loss of axons [11]. Shakib & Ashton [23] reported on ultrastructural changes in focal retinal ischaemia after micro-embolization of retinal vessels in animal studies. They showed the degeneration's progress up to five hours after embolization, 24 hours to four days, and four to 25 days after embolization. Electron microscopy at an early stage revealed focal swelling of nerve fibers in the lesion's center. Depleted cytoplasmic organelles involving swollen mitochondria and enlarged endoplasmic reticulum in the ganglion cells were observed. The inner plexiform layer revealed marked swelling and the outer nuclear layer contained swollen cells with almost empty cytoplasm. About 24 hours after embolization, white areas occurred resembling cotton-wool spots in ischaemic areas. About four days after embolization cotton-wool spots began to fade. The ischaemic area ultimately appeared as a glial scar with a few persistent nerve fibers and vessels. The retina's outer layers – including Henle's fiber layer – are believed to derive their nutrition from the choriocapillaris. Advanced degenerative anomalies and interrupted neurites in Henle's fiber layer were observed in a patient ten days after a total occlusion of the CRA [25]. Cogan [3] reported that the outer reticular layer is modified in the macula to form radiating fibres, or Henle's fiber layer. This radiating pattern may constitute a star-shaped figure with exudates, and it sometimes hemorrhages in the central area. Gold [8] reported ultrastructural findings in eyes after CRAO. Ruptured cytoplasmic membranes have been observed and a loss of cytoplasm, marked swelling of all cellular elements,

accumulation of cytoplasmic organelles and their degradation products.

Ocular massage therapy

Ocular massage as emergency treatment has been described in numerous reports on patients with CRAO. Firm massage entailing globe compression for up to 15 seconds at a time followed by sudden release was recommended by Ffytche [6]. This proved to be a very effective method to improve retinal blood flow. In their experimental study, Ffytche & coworker [5] showed that globe compression/decompression is more effective in improving retinal volume flow than aqueous puncture. Gold [8] emphasized that digital ocular compression or massage is quite effective in producing significant retinal arterial dilatation and in raising increased blood flow velocity and volume. Nielsen [19] pressed and depressed abruptly and rhythmically the globe of patients suffering from acute obstruction of retinal arteries. Digital ocular massage was experimentally carried out in rabbits by Jay & coworker [13], and they showed that the blood flow increased significantly after massage of the globe. Schmidt [20] reported on a 50-year-old man with unilateral CRAO who was treated by massage. After a delay of several minutes, the fundus vessels became increasingly filled with blood. During this procedure, the patient noticed the gradual recovery of vision in his left eye.

Animal experiments involving CRAO

The central retinal arteries of monkeys were ligated within the orbit by Kroll [16]. He then detected histologically massive swelling of the inner retinal layers, followed by necrosis. Fine-structurally, CRAO produced mitochondrial swelling in neurons of the inner retinal layers within 15 minutes. This was followed by swelling of the endoplasmic reticulum and cell cytoplasm. In arterial hypertensive monkeys, Hayreh & Jonas [11] observed that a CRAO lasting less than 100 minutes produced no apparent morphometric evidence of optic nerve damage; however, in CRAO lasting 105 minutes but under 240 minutes yielded a variable degree of damage; 24 minutes of CRAO, or more, triggered total or almost total optic nerve atrophy and nerve fiber damage. Hayreh & Jonas [11] argued that there is no evidence that the retina in humans and rhesus monkeys are different structurally or physiologically.

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