

Valsalva Retinopathy Associated with Choking and Heimlich Maneuver

Abstract

Valsalva is "a particular form of retinopathy, pre-retinal and hemorrhagic in nature, secondary to a sudden increase in intrathoracic pressure". Increasing intrathoracic pressure against a closed glottis diminishes venous return to the heart, decreasing stroke volume and subsequently increasing the venous system pressure. Here we report a case of 52-year-old male who presented with a sudden central vision loss because of macular hemorrhage. The case occurred following the suffocation, which is not often seen; hence we conducted investigative tests to determine the cause of retinal bleeding.

Case Report

A 52-year-old Chinese man complained a "black spot" in the vision of his left eye. There was no previous ophthalmic history. He was under investigation for hypertension.

The man inhaled food three days before seeing the doctor, causing choking, which prompted his wife to beat his chest like doing "Heimlich maneuver". His unaided visual acuity was 1.0 and 1.0 in both eyes. Anterior segment examination was unremarkable. Pupil reactions were normal. Fundus examination of the right eye was normal whereas examination of the left eye revealed a subretinal hemorrhage involving the posterior pole of the left eye (Figure 1). Fundus fluorescein angiography (FFA) showed hypofluorescence in the macular region, due to blockage by hemorrhage. The total size of the lesion was about six disc areas; no large area of fluorescein leakage was found (Figure 2).

One week later his left visual acuity suddenly dropped to 0.05. We found macular and vitreous hemorrhage during fundus examination. Two months later, indocyanine green angiography (ICGA) showed that vitreous hemorrhage, partial low fluorescence, no fluorescence leakage. After six months, his best-corrected visual acuity recovered to 1.0, examination indicated that in the left eye the vitreous was clear, and there were pigmentary disorders in macular area. Fifteen months later, the corrected visual acuity of the left eye remained at 1.0 with some pigmentary changes in the macular area.

Discussion

Vitreous hemorrhage is one of the most common causes of sudden, painless loss of vision. We have reviewed the related case reports from 2002 to 2012 in PubMed, and summarized reported literature about vitreous hemorrhage and macula hemorrhage in the Table 1.

Valsalva retinopathy is a unilateral or bilateral condition that occurs when increased intra-thoracic or intra-abdominal pressure transmitted to the eye causes a sharp rise in the intraocular venous pressure and rupture of superficial retinal capillaries [1,2]. Patients usually give a history of sudden visual loss following a bout of heaving lifting, coughing or vomiting [3]. These hemorrhagic lesions are typically self-limiting and resolve without any complications. There

is no evidence of increased risk of recurrence. A diagnosis of Valsalva retinopathy is only made after exclusion of other causes of retinal hemorrhages [1,2].

Valsalva retinopathy classically presents with pre-retinal/subhyaloid hemorrhages and in this case, it's subretinal. Subretinal hemorrhage refers to blood located between the neurosensory retina and the retinal pigment epithelium (RPE). Blood in this space can arise from the choroidal and/or the retinal circulation.

Subretinal hemorrhage may arise simply from choriocapillary hemorrhage through discontinuities in Bruch's membrane. Most of them may arise directly from choroidal neovascular membranes or from a break in the Bruch's membrane/RPE complex. In the case, we have not found any evidence of choroidal neovascularization (CNV) and choroidal rupture. As we know, the outer capillary network runs from the inner plexiform layer to the outer plexiform layer through the inner nuclear layer [4]. The inner capillaries lie in the ganglion

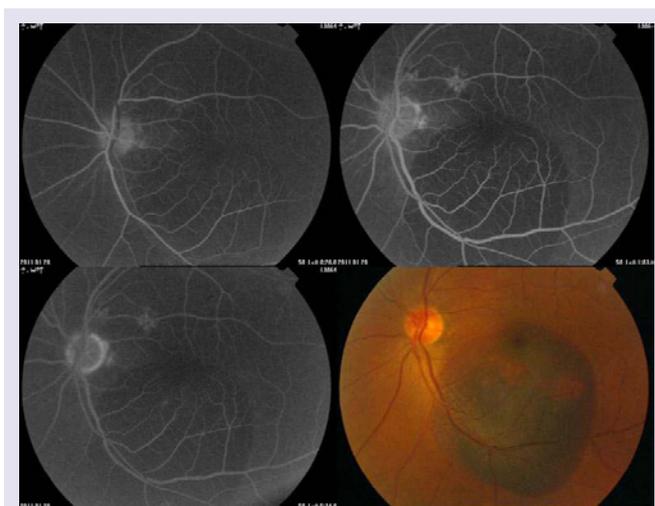


Figure 1: The left eye showed a partial subretinal hemorrhage in the posterior pole and macula one day after asphyxia and no fluorescence leakage. UPPER LEFT: The early phase of FFA; UPPER RIGHT: subretinal hemorrhage blocked fluorescence; LOWER LEFT: the later phase of FFA: no leakage. LOWER RIGHT: color image.



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cell layers under and parallel to the RPCs [4]. Thus, any retinal vascular abnormality or retinal trauma that causes retinal endothelial compromise (inner blood-retinal barrier) will predispose to formation of subretinal hemorrhage. The retinal vessels bleed, with resultant dissection into the subretinal space, forming subretinal hemorrhage and, at times, intraretinal hemorrhage, vitreous hemorrhage and choroidal hemorrhage. Asphyxia or chest compressions may increase the circulation pressure. After asphyxia, the intrathoracic pressure increases against a closed glottis. This diminishes venous return to the heart, decreasing stroke volume and subsequently increasing the venous system pressure [5,6]. A sudden rise in intraocular venous pressure causes retinal capillaries to spontaneously rupture.

In this case, there was no typical manifestation of reddish-orange, spheroidal, polyp-like structures or polypoidal vascular lesions at the

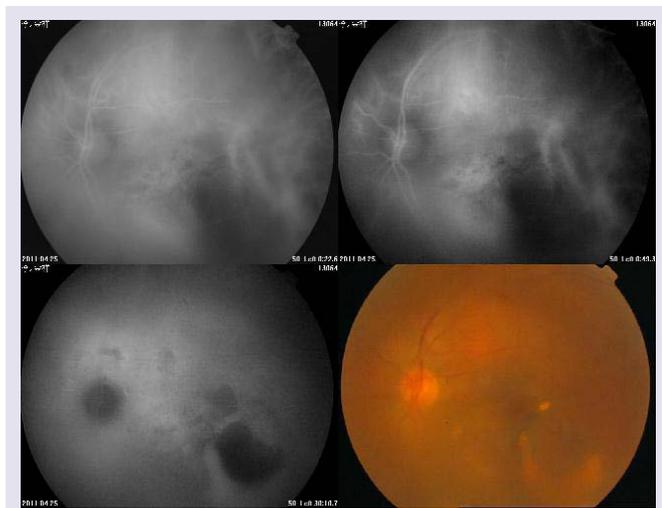


Figure 2: Indocyanine green angiogram showed macular hemorrhage (and mild vitreous hemorrhage) three months after asphyxia. UPPER LEFT: The early phase of ICG; UPPER RIGHT: No polyp-like structures or polypoidal vascular lesions at the termination of a network of large choroidal vessels in the patient's fundus photograph; LOWER LEFT: the later phase of ICG showed only hemorrhage block. LOWER RIGHT: color image.

Table 1: The reported literatures about vitreous hemorrhage and macula hemorrhage from 2002 to 2012 in PubMed.

	Reason (Number)
Original research	Age-related macular degeneration (16), Polypoidal choroidal vasculopathy (9), Proliferative diabetic retinopathy (8), Retinal vasculitis and branch retinal vein occlusion (8), Terson syndrome (7), Eales disease (5), Thrombocytopenic purpura (3), Valsalva retinopathy (3)
Case report	Uveal melanoma, Systemic anticoagulants, Choroidal melanoma, Retinal detachment, Postoperative endophthalmitis, Multiple sclerosis-associated uveitis, (2, respectively) Persistent hyaloid artery, Proliferative sickle cell retinopathy, whole-body vibration training, Retinal vasculitis associated with mixed connective tissue disease, Retinal astrocytic hamartoma, Vitamin K deficiency bleeding, Lung cancer from choroidal metastasis, Antiphospholipid syndrome, Unilateral frosted branch angiitis, Leber congenital amaurosis, Wegener granulomatosis, Aplastic anemia, retinal artery macroaneurysms, Autosomal dominant syndrome of retinal arterial tortuosity, Acquired factor VIII inhibitor, Acute posterior vitreous detachment, Subarachnoid hemorrhage, Retinoblastoma Retinoblastoma, (1, respectively)

If two articles reported results from the same group of patients, they are regarded as one article.

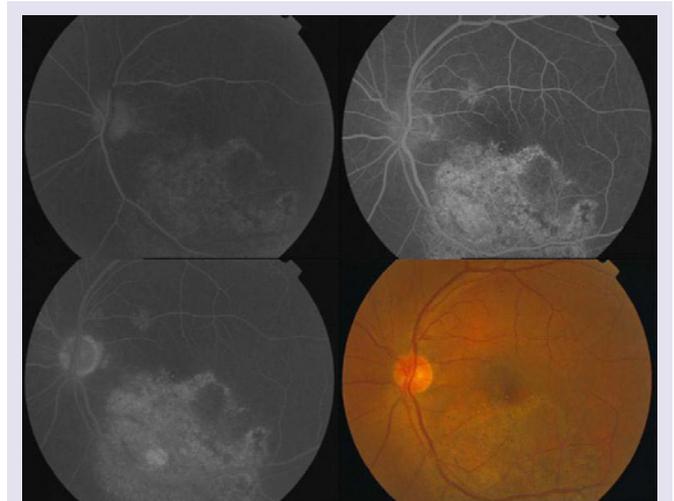


Figure 3: The left eye in FFA fifteen months later showed some pigmentary disorder below the macula. UPPER LEFT: The early phase of FFA; UPPER RIGHT: The venous phase; LOWER LEFT: the later phase of FFA: no leakage. LOWER RIGHT: color image.



Figure 4: Indocyanine green angiogram showed that the inferior macula was surrounded by chaotic retinal pigment epithelia fifteen months after asphyxia. UPPER LEFT: The early phase of ICG 23 seconds; UPPER RIGHT: 6 minutes later; LOWER LEFT: 10 minutes later. LOWER RIGHT: 30 minutes later.

termination of a network of large choroidal vessels in the patient's fundus photograph. We did not discover any evidences of PCV in ICGA. Also, no characteristics of AMD or pathological changes of neovascularization and effusion appeared. We reckoned that since the patient was overweight and had a history of hypertension, during the process of rescue, the superior vena cava pressure suddenly lifted, induced the reflux of retinal vein blood, and caused acute deep subretinal hemorrhage.

The data suggested Valsalva retinopathy with sub-hyaloid haemorrhage, probably secondary to coughing on a background of hypertension. This report highlights the need to perform a basic systemic examination in similar cases, as incidental findings requiring systemic treatment may be detected. There is no evidence

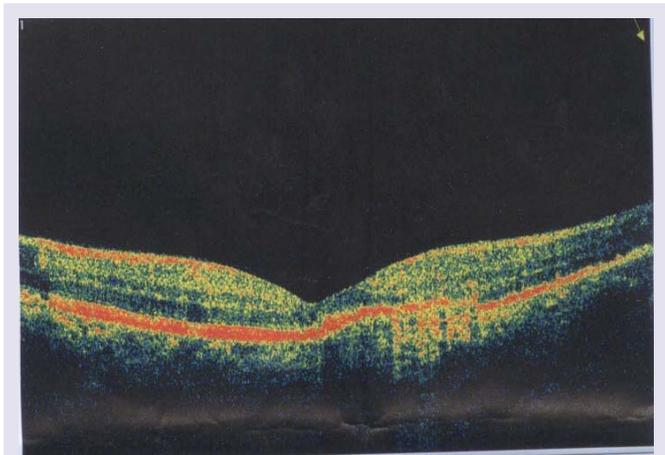


Figure 5: OCT indicated the disordered retinal pigment epithelial layer fifteen months after asphyxia.

of increased risk of recurrence [7]. In the relevant reports about Valsalva retinopathy, it happened in rare condition [2,8]: A 36-year-old Caucasian woman had Valsalva retinopathy after vomiting during pregnancy [8]; A 52-year-old Caucasian man had premacular bleeding due to vomiting after general anaesthesia [2]; A 24-year-old Caucasian man had a macular hemorrhage after thoracic trauma [2]; some men developed a premacular bleed after vigorous dancing [2,9]; some persons developed a premacular hemorrhage after weightlifting [2,3]. A 17-year-old man presented in his recent oratorical contest [10]. There are many case reports of Valsalva. The reasons are different such as choking asthma attacks, motorcycle riding, colonoscopy and others [11-18]. A Diagnosis of Valsalva retinopathy is only made after exclusion of other causes of retinal hemorrhages [1,2].

In summary, several reasons will lead to vitreous hemorrhage or macular hemorrhage and it is important to find out the reason. ICGA is a useful tool for the identification and differentiation of Valsalva, PCV and AMD. And we should ask for the patients' history carefully. It is very useful for the diagnosis.

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