CASE REPORT

Sudden vision loss after repeated retching: a case of Valsalva retinopathy

Valerie Chiang,[1] Kendrick Co Shih,[2,3] MRCSEd, Jimmy Shiu-Ming Lai,[2,3] FHKAM, FRCSEd, FRCOphth, Ian Yat-Hin Wong,[2,3] FHKAM, FRCSEd, FRCOphth

[2]*Li Ka Shing Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong SAR, China.*
[2]*Department of Ophthalmology, LKS Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong SAR, China.*
[3]*Research Centre of Heart, Brain, Hormone & Healthy Aging, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong SAR, China.*

Valerie Chiang and Kendrick Co Shih contributed equally as the first authors to the paper.

Correspondence and reprint requests:
Dr. Ian Yat-Hin Wong, Department of Ophthalmology, LKS Faculty of Medicine, The University of Hong Kong, 301B, Cyberport 4, 100 Cyberport Road, Pokfulam, Hong Kong SAR, China.
Email: wongyhi@hku.hk

Abstract

We report on a patient who developed unilateral retinal and vitreous hemorrhages after repeated retching and vomiting. A 22-year-old man of South Asian descent was seen at the Accident and Emergency Department, Queen Mary Hospital, for sudden loss of vision in his right eye. The episode was acute, painless and preceded by repeated retching and vomiting after a spicy curry meal. Examination revealed fresh intra- and pre-retinal hemorrhage involving the inferior macula. The condition resolved spontaneously with conservative measures and did not require specific treatment or intervention. Repeated vomiting as a result of a poorly tolerated spicy meal resulted in unilateral Valsalva retinopathy. This case report details the course of recovery and what patients and physicians should expect and rule out. It is important to reassure patients during this course that they will regain full visual function.

Key words: *Eye hemorrhage; Retinal diseases*

Introduction

Valsalva retinopathy is a form of premacular hemorrhage that typically occurs after strenuous physical activity. Initial presentation is usually a unilateral, painless, acute vision loss that is preceded by a sudden increase in intra-abdominal pressure, hence the term 'Valsalva' retinopathy. The name may be misleading in that it is not a disease of the retina, but simply a pressure-induced hemorrhage caused by ruptured sub-retinal capillaries. The hemorrhage tends to obscure vision severely and suddenly, and is alarming for most patients. The advantage of this is that patients tend to seek help early, and this prevents complications of prolonged blood-trapping in the premacular space.

Case report

In September 2015, a 22-year-old man of South Asian decent, with good past health, presented with sudden onset loss of vision in his right eye. The episode was immediately preceded by repeated retching and vomiting, as he had been unable to tolerate a particularly spicy curry meal that evening. There was no associated ocular pain or headache and no preceding head or ocular trauma. Vision in his left eye vision was unaffected. The patient denied any history of easy bruising, or use of anticoagulants or antiplatelet medication. There was also no family history of bleeding tendencies or disorders although both of his parents had type II diabetes mellitus.

On physical examination, the patient had unaided Snellen Chart visual acuity of hand movements at 1 m in his right eye and 0.7 m in his left eye. Intraocular pressure was measured at 14 mm Hg in both eyes via Goldmann

applanation tonometry. His pupils were equal and reactive, with no relative afferent pupillary defect. He had full-range extraocular muscle movements and there was no proptosis. Anterior segment examination of both eyes using a slit lamp revealed no abnormalities. A dilated fundus examination of both eyes revealed a large pre-retinal hemorrhage of at least 5 disc-diameters over the inferior macula in the right eye. The disc was pink with a cup-disc ratio of 0.5. Multiple intra-gel clots along with minor dot and blot hemorrhages were present. Retinal vessels were not tortuous and there was no evidence of retinal neovascularization (**Figure**). The left eye fundus examination was unremarkable, with no evidence of vasculopathy or retinal neovascularization.

Differential diagnoses

In considering the differential diagnoses, common causes of intraocular hemorrhage include proliferative diabetic retinopathy, retinal artery macroaneurysm, and retinal vein occlusion.[1] Other causes include bleeding tendency, posterior vitreous detachment, retinal break, vasculitis, Terson's syndrome, trauma etc. It is important to note that these conditions are typically found in middle-aged to elderly patients. With the clinical history and lack of clinical features to indicate underlying vasculopathy or intracranial hemorrhage, it was most likely that the repeated vomiting had caused a retinal arteriole to rupture, leading to premacular hemorrhage. The patient was noted to have approximately -1.0D of uncorrected myopia in his right eye and -0.5D in his left eye that explained the suboptimal vision for his age even in the unaffected eye.

Management

To look for possible underlying vasculopathy, blood was taken for complete blood count, clotting profile (prothrombin time and activated partial thromboplastin time), erythrocyte sedimentation rate, C-reactive protein, fasting glucose and lipid profile. The blood tests were normal with no evidence of bleeding tendencies or diabetes mellitus (**Table 1**).

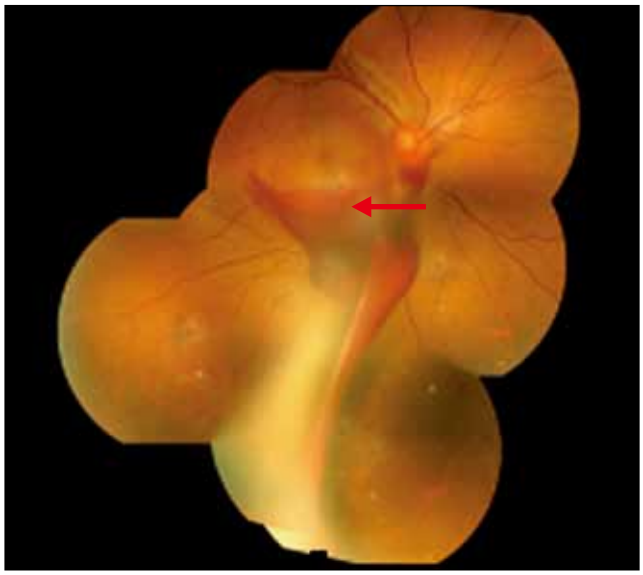

Figure. Fundus photograph of the right eye showing a large well circumscribed area of pre-retinal hemorrhage (arrow) involving the macula.

In view of the above findings, the patient was advised to allow time for the blood to resolve spontaneously. He was instructed to avoid physical exertion, and to prop his head up with 3 pillows while in bed. No further ocular treatment was recommended. Follow-up sessions were scheduled for the following morning, in 3 days and in 10 days. His vision began improving as early as day 2 with no complications (**Table 2**). Initially, a large pre-retinal hemorrhage covering the inferior macula was seen and there were no breaks or holes found in the peripheral retina. Minor dot and blot hemorrhages were present, as expected with sudden increases in intraretinal vascular pressure. Many intra-gel clots also contributed to obscuring the patient's vision. The blood spread out into the vitreous by the 3rd day and was seen to be resolving well by day 10, with a corresponding visual acuity of 0.5 in his right eye. After this, the patient defaulted from further follow-up visits.

Discussion

Vision is the most dominant sense for humans. Sudden changes in vision, especially loss, can be intimidating for patients. Nonetheless not all causes of vision loss need specific ocular treatment or intervention. This case provides a timeline for which Valsalva retinopathy spontaneously resolves. It is important for physicians to offer security and reinforce the patient's trust in conservative management.

Valsalva retinopathy occurs when a sudden increase in intrathoracic or intra-abdominal pressure causes an abrupt increase in peripheral intraocular venous pressure, leading to rupture of sub-retinal capillaries. It is often seen in younger patients with no history of retinal disease.[2] Detailed history taking is essential for diagnosis as a wide range of activities that involve forced expiration can lead to Valsalva retinopathy. These may include heavy lifting, labor, constipation, sneezing, sexual activity and vomiting.[3,4] It is also important to exclude risk factors for ocular hemorrhage such as diabetes and bleeding disorders.

Vision loss is often unilateral, acute and painless.[2] It is a result of the macula being obscured by hemorrhage that may be sub-retinal, intra-retinal, pre-retinal, sub-hyaloidal, or in the intra-vitreal cavity.[5] In our case the presentation was predominantly a pre-retinal hemorrhage that covered the inferior macula. The prognosis of Valsalva retinopathy is excellent. Patients usually return to baseline visual function after resolution although the recovery process can take several months.[6,7]

In our case, although the patient presented with a severe loss of vision, he recovered as early as the 3rd day after presentation. Vision is regained as the pre-macular blood is absorbed. It is important during this time to reassure the patient that the symptoms are transient and will resolve

Table 1. Baseline blood results.

Blood parameter	Data	Reference range
Metabolic profile		
Sodium (mmol/L)	138	136-148
Potassium (mmol/L)	4.3	3.6-5.0
Chloride (mmol/L)	100	100-109
Urea (mmol/L)	5.9	2.5-6.3
Creatinine (μmol/L)	66	67-109
R-glucose (mmol/L)	5.0	
Total protein (g/L)	84	68-84
Albumin (g/L)	49	39-50
Globulin (g/L)	35	24-37
Total bilirubin (μmol/L)	14	4-23
Alkaline phosphatase (U/L)	65	42-110
Alanine transaminase (U/L)	29	8-58
Aspartate transaminase (U/L)	27	15-38
Coagulation		
Prothrombin time (sec)	13.3	11.3-13.5
Activated partial thromboplastin time (sec)	29.8	25.9-33.7
Complete blood count		
White blood cell count (x 10^9/L)	7.99	3.89-9.93
Red blood cell count (x 10^{12}/L)	4.59	4.46-5.71
Hemoglobin (g/L)	133	133-171
Hematocrit (%)	40.1	39.5-48.8
Mean corpuscular volume (fL)	87.4	82.0-95.5
Mean corpuscular hemoglobin (pg)	29.1	27.0-32.4
Mean corpuscular hemoglobin concentration (g/dL)	33.2	32.6-35.7
Red cell distribution width (%)	13.0	11.2-13.9
Platelets (x 10^9/L)	231	154-371
Differential count		
Neutrophil (x 10^9/L)	5.20	2.01-7.42
Lymphocyte (x 10^9/L)	1.93	1.06-3.61
Monocyte (x 10^9/L)	0.48	0.18-0.65
Eosinophil (x 10^9/L)	0.18	0.02-0.45
Basophil (x 10^9/L)	0.03	0.01-0.07

Table 2. Physical examinations findings.

Physical examination	Day 1	Day 3	Day 10
Snellen Chart visual acuity (unaided)	RE: hand movements only; LE: 0.7	RE: 0.3; LE: 0.7	RE: 0.5; LE: 0.7
Intraocular pressure (mm Hg)	RE: 14; LE: 14	RE: 12; LE: 14	RE: 11; LE: 10
Pupillary reflex	Pupils equal and reactive to light, no relative afferent pupillary defect		
Anterior segment exam	Clear corneas with formed and quiet anterior chambers; clear crystalline lenses		
Dilated fundus examination of the RE	Large pre-retinal hemorrhage covering posterior pole, at least 5 disc diameters in size tracking towards inferior retina; right supra-temporal arcade blot hemorrhage; no retinal breaks or holes	Large pre-retinal hemorrhage covering posterior pole; no retinal breaks/holes seen	Pre-retinal hemorrhage resolving and shifting inferiorly; no more intra-retinal hemorrhage; no retinal breaks/holes seen

Abbreviations: LE = left eye; RE = right eye.

with conservative management, as loss of vision can be debilitating and alarming for most. Patients should be informed that no permanent damage or further complications should be expected. To help with the recovery process, they should be advised to prop the head up and avoid strenuous physical activity.

Conservative management with close observation is the primary initial treatment for Valsalva retinopathy and is followed with good recovery.[8] In patients with very large and dense hemorrhages, blood trapped in the subhyaloid or sub-inner limiting membrane space for prolonged periods of time can cause visual impairment. This can be due to toxic damage to the retina by hemoglobin and iron, pigmentary macular changes or formation of epiretinal membranes. These complications, however, are typically uncommon unless the hemorrhage persists for over 6 weeks.[9]

Should the pre-macular hemorrhage persist after a reasonable period of observation, alternative treatments can be considered. Laser therapy is advised when conservative management is unsuccessful.[8] Nd:YAG laser treatment has been reported to help diffuse the hemorrhage into the vitreous gel, and can speed up its clearance.[1] However, the procedure comes with risks such as unsealed internal limiting membrane and epiretinal membrane formation.[10,11] In more severe cases of Valsalva retinopathy, vitreoretinal surgery has also been cited to be useful when spontaneous resorption is incomplete and visual acuity is significantly affected.[9]

Conclusion

Valsalva retinopathy is a self-limiting condition associated with sudden severe loss of vision in the affected eye, leading patients to seek immediate medical attention at accident and emergency departments. This case report provides a natural history of Valsalva retinopathy caused by repeated vomiting after a spicy meal; patient reassurance and conservative measures are effective first-line strategies for Valsalva retinopathy.

Declaration

All authors have disclosed no conflicts of interest.

References

1. *Rennie CA, Newman DK, Snead MP, Flanagan DW. Nd:YAG laser treatment for premacular subhyaloid haemorrhage. Eye (Lond). 2001;15:519-24.*
2. *Waikar S, Srivastava VK. Valsalva retinopathy in a young healthy individual. Med J Armed Forces India. 2013;69:193-5.*
3. *Ahmadabadi MN, Karkhaneh R, Mirshahi A, et al. Premacular hemorrhage in Valsalva retinopathy: a study of 21 cases. Iran J Ophthalmol. 2009;21:11-6.*
4. *Garcia Fernandez M, Navarro JC, Castano CG. Long-term evolution of Valsalva retinopathy: a case series. J Med Case Rep. 2012;6:346.*
5. *Duane TD. Valsalva hemorrhagic retinopathy. Trans Am Ophthalmol Soc. 1972;70:298-313.*
6. *Kilincalp S, Coban S, Yuksel I. An alarming condition after upper gastrointestinal endoscopy: valsalva retinopathy. Gastroenterol Nurs. 2013;36:465-6.*
7. *Sahu DK, Namperumalsamy P, Kim R, Ravindran RD. Argon laser treatment for premacular hemorrhage. Retina. 1998;18:79-82.*
8. *Liu Z, Pan X, Bi H. Treatment of Valsalva retinopathy. Optom Vis Sci. 2014;91:e278-81.*
9. *De Maeyer K, Van Finderdeuren R, Postelmans L, Stalmans P, Van Calster J. Sub-inner limiting membrane haemorrhage: causes and treatment with vitrectomy. Br J Ophthalmol. 2007;91:869-72.*
10. *Zou M, Gao S, Zhang J, Zhang M. Persistent unsealed internal limiting membrane after Nd:YAG laser treatment for valsalva retinopathy. BMC Ophthalmol. 2013;13:15.*
11. *Kwok AK, Lai TY, Chan NR. Epiretinal membrane formation with internal limiting membrane wrinkling after Nd:YAG laser membranotomy in valsalva retinopathy. Am J Ophthalmol. 2003;136:763-6.*