

Case Report

ACUTE SUBMACULAR HAEMORRHAGE: A CASE OF EARLY SPONTANEOUS DISPLACEMENT

Izwan Kamal Tan^{1,2}, Mushawiahti Mustapha,¹ Ropilah Abdul Rahman³

¹Department of Ophthalmology, Faculty of Medicine, University Kebangsaan Malaysia Medical Centre, Jalan Yaacob Latif, Bandar Tun Razak, 56000 Kuala Lumpur, Malaysia.

²Department of Ophthalmology, Hospital Selayang, Lebuhraya Selayang-Kepong, 68100 Batu Cave, Selangor, Malaysia.

³Kulliyah of Medicine & Health Sciences, Universiti Islam Antarabangsa Sultan Abdul Halim Mu'adzam Shah, 09300 Kuala Ketil, Kedah, Malaysia.

ARTICLE INFO

Corresponding author:
Dr. Izwan Kamal Tan

Email address:
izwankamaltan@yahoo.co.uk

Received:
November 2021
Accepted for publication:
December 2021

Keywords:

Submacular haemorrhage;
polypoidal choroidal vasculopathy;
spontaneous blood displacement;
choroidal neovascular membrane

ABSTRACT

A case report of submacular haemorrhage that spontaneously displaced within 24 hours, thereby improving visual acuity. A case of subretinal haemorrhage over the macula of the right eye was presented to the eye clinic. The visual acuity of the affected eye was "counting fingers" at presentation. The patient was asked to return the next day for a pneumatic displacement of subretinal blood and was instructed on how to properly propped her head at home. Visual acuity of the affected eye improved dramatically to 6/24 the next day. Fundus examination showed a smaller submacular haemorrhage which was away from the foveal area. A pigment epithelial detachment was identified superior to the macula. Fundus fluorescein angiography showed a pulsatile hyperfluorescent spot near the macula with branching vascular network. A diagnosis of submacular bleed secondary to polypoidal choroidal vasculopathy (PCV) was made, and the patient was treated with intravitreal aflibercept. Submacular hemorrhages secondary to PCV can affect the vision severely and while the natural progression is variable, the vision can be improved simply by proper and adequate positioning of the patient's head to relocate the accumulation of the blood away from the fovea.

INTRODUCTION

Subretinal hemorrhage occurs when there is bleeding originating from the choroidal and/or the retinal circulation. The blood collects in a potential space between the neurosensory retina and the retinal pigment epithelium (RPE). The hemorrhages of choroidal origin may arise directly from the choriocapillaries via discontinuities in the Bruch's membrane, or from choroidal neovascular membranes (CNVs) which proliferate through breaks in the Bruch's membrane.

CASE REPORT

A 62-year-old lady presented with a sudden onset of loss of vision in the right eye. It was painless and affected the central field of vision. It was not preceded by floaters or flashes of light. Best-corrected visual acuity (BCVA) was 6/12 in the left eye and counting fingers in the right eye. Anterior segment examination revealed a nuclear sclerotic cataract in both eyes. There was no relative afferent pupillary defect detected. Fundus examination revealed a large dense subretinal haemorrhage in the posterior pole involving the macula and extending to the superior and inferior vascular arcades (Figure 1). The optic disc was

visible and pink in color with apparently healthy neuroretinal rim. An Optical Coherence Tomography confirmed the haemorrhage to be subretinal (Figure 2). She was planned for pneumatic displacement of the subretinal blood the next day. Instructions on how she ought to prop up her head while at home were clearly demonstrated to her. On her return to the clinic the next day, the visual acuity of her affected eye showed a dramatic improvement to 6/24 from the previous "counting fingers". On further inquiry, she attested that she had followed the instructions to keep her head propped up at all times, as directed.

Fundus examination showed a smaller area of subretinal haemorrhage which was away from the foveal (Figure 3). A pigment epithelial detachment was identified superior to the macula. Fundus fluorescein angiography showed a pulsatile hyperfluorescent spot near the macula with branching vascular network (Figure 4). A diagnosis of Acute submacular haemorrhage secondary to polypoidal choroidal vasculopathy (PCV) was made. Following confirmation with Indocyanine green angiography, patient was subsequently treated with intravitreal aflibercept. Her right BCVA remained good, 6/12 at six months follow up.

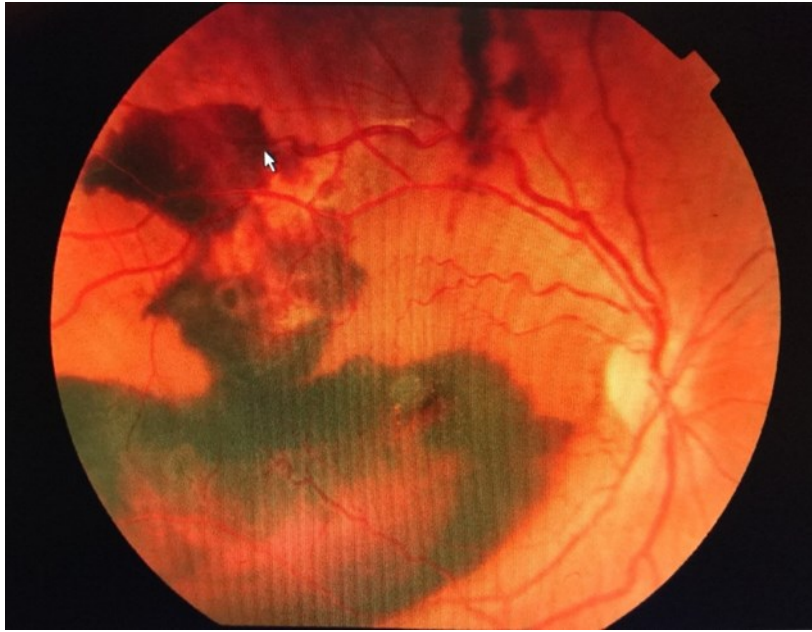


Figure 1: Right fundus photograph showing submacular haemorrhage at the initial presentation.

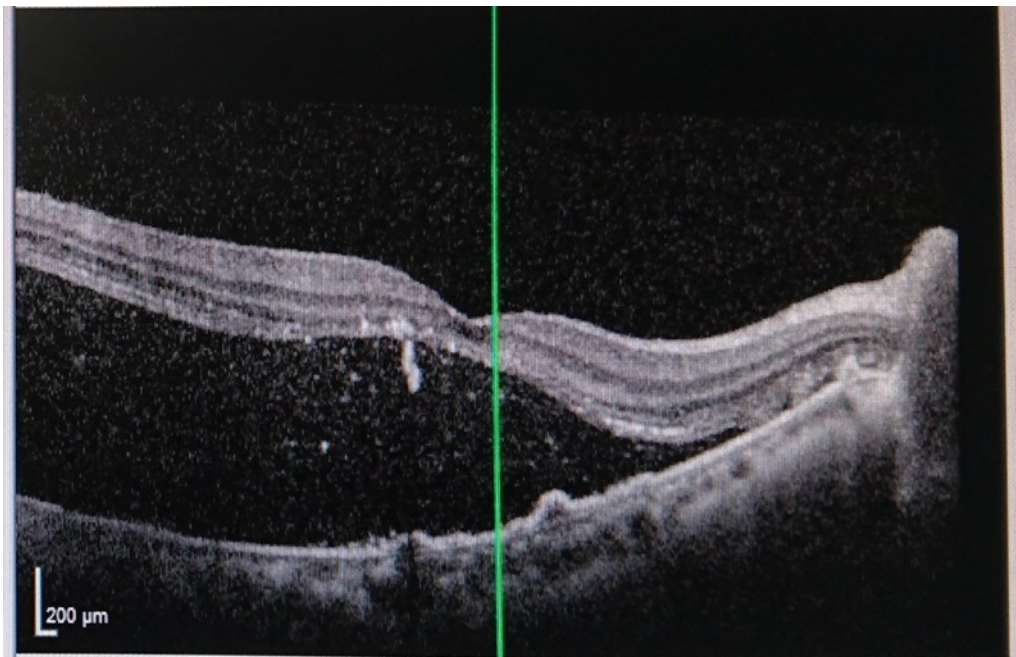


Figure 2: Optical Coherence Tomography showing subretinal haemorrhage involving the foveal region and RPE detachment.

DISCUSSION

Subhyaloidal or macular haemorrhages are known to occur secondary to retinal or choroidal vascular diseases. The pathology include arteriosclerosis, hypertension, retinal vessel occlusion, diabetic retinopathy, chorioretinitis, retinal microaneurysm, age-related macular degeneration and several other blood disorders [1]. The condition may also be precipitated by trauma or occur spontaneously.

As a result of the location of the haemorrhage at the macula, patients usually experience a sudden occurrence of reduced vision. Visual acuity may

deteriorate within seconds to minutes [2]. However, in patients who have a concurrent subfoveal disciform scarring, a condition which is often related to eyes with age-related macular degeneration, the haemorrhage itself may go unnoticed as the patient's visual acuity may be limited to begin with [3]. Similarly, a subretinal haemorrhage which occurs away from the fovea could present with minimal symptoms and preserve the patient's central vision. Other symptoms of visual deterioration in association with macular hemorrhages include a visual field defect with or without metamorphopsia [4].



Figure 3: Fundus photograph one day after the presentation: The subretinal haemorrhage is inferiorly displaced.



Figure 4: Fundus Fluorescein Angiography of the right eye documenting a polyp as a hyperfluorescent spot in the macula area supero-temporal to the fovea.

In fundusoscopic examination, submacular hemorrhages appear as an elevation of the overlying neurosensory retina which may be associated with retinal folds. The borders of the haemorrhage are usually sharply demarcated, resulting in a dome-like shape in the area of haemorrhage [5]. The colour of the blood within the haemorrhage may vary, ranging from bright or dark red in early stages, or yellow or tan in later stages once the blood content becomes devoid of its haemoglobin content [6].

Without active intervention, the haemorrhage may undergo spontaneous reabsorption, a process that could take a duration of 1 to 2 months duration [7]. This scenario carries the risk of permanent visual loss from irreversible retinal damage caused by the formation of pre-retinal tractional membranes and proliferative vitreoretinopathy as well as the toxic effect of longstanding haemorrhage to the photoreceptors [5]. Submacular hemorrhages thus carry a poorer prognosis of visual recovery compared to subhyaloidal hemorrhages for these particular reasons [8].

Hayasaka et al retrospectively reviewed 24 eyes with pathologic myopia and subretinal haemorrhage and found that the subretinal haemorrhage spontaneously reabsorbed within 1 year in 15 eyes without CNV, with the visual acuity improving or remaining unchanged [9]. The visual acuity was unchanged or worsened in 9 other eyes which had CNV without AMD. A non-consecutive retrospective study by Berrocal et al showed spontaneous improvement in visual acuity correlating to spontaneous reabsorption of subretinal haemorrhage over a period of 3 to 56 months [10]. The eyes with AMD-related subretinal hemorrhages were more likely to improve compared to those that were not AMD related. Another non-consecutive retrospective study by Bennet et al reviewed 29 cases of subretinal haemorrhage of at least one disc-diameter in size resulting in visual loss [11]. Cases were reviewed over an average period of 3 years. Mean visual acuity was seen to improve more in cases that were not AMD related, while AMD related eyes showed no improvement. In addition, eyes with a thick subretinal haemorrhage carried a worse visual prognosis and the final visual acuity correlated with the size of the initial subretinal haemorrhage.

Based on the preceding studies, there is a variable natural history of subretinal haemorrhage [12]. Treatment of the condition must therefore aim to remove the haemorrhage before any permanent retinal damage manifests. Observation of the condition for spontaneous clearing of the haemorrhage is a clinically accepted practice up to a duration of 3 months, although early surgery can also be considered within this time period.

Among the treatment modalities for submacular hemorrhages include laser drainage, also known as laser membranotomy and laser puncturing. This technique was introduced by Heydenreich and Fechner in 1973 [13]. This treatment has been recommended for use within the first 3 to 4 days after the onset of haemorrhage and works by

creating a focal opening into the vitreous cavity through which the entrapped blood may drain [14]. A common complication of this procedure is the formation of epimacular membranes which are theorized to occur due to the presence of growth factors responsible for stimulating proliferation of entrapped cells between the inner limiting membrane (ILM) and the retinal surface. Formation of macular hole and retinal detachments are rarely reported. Recombinant tissue plasminogen activator and gas has also been used in management of submacular hemorrhages where drainage of the blood was achieved via separation of the vitreous [15].

Immediate and complete removal of the haemorrhage can be accomplished via vitrectomy, with the added benefit of prompt recovery of vision. De Maeyer et al treated 5 cases of submacular haemorrhage with excellent visual recovery in all subjects [16]. Intraoperatively, the location of the hemorrhages were all identified as sub-ILM, which was achieved by ILM biostaining [17]. Timely vitrectomy may also prevent irreversible retinal damage which could potentially arise from a longstanding haemorrhage. The procedure carries a number of risks and side effects which should be informed to the patient prior to surgery. This includes cataract formation, intraoperative breaks and postoperative proliferative vitreoretinopathy which may result in a retinal detachment, necessitating a second procedure [18,19].

In summary, submacular haemorrhage can result in a sudden severe drop in vision and several methods can be applied for its management. Spontaneous reabsorption or relocation of the haemorrhage may occur in the early period of the disease, but the natural progression without treatment is variable. Vitrectomy has shown good results in prompt clearing of the haemorrhage with good potential recovery of vision, while laser drainage has also achieved good functional results. In patients who present with severe visual loss at the outset, a simple procedure like the positioning of the head is recommended. This is to attempt relocation of the site of accumulated blood to an area that spares visual loss.

CONCLUSION

Submacular hemorrhages secondary to PCV can affect the vision severely and while the natural progression is variable, the severity may be improved upon simply by proper and adequate positioning of the patient's head. This procedure is non-invasive, inexpensive, and without any ill effects.

PATIENT CONSENT

Consent to publish the case report has been obtained from the patient. The anonymity of the patient is preserved in this report

REFERENCES

1. Russell S R, Hageman G S. Hemorrhagic detachment of the internal limiting membrane after penetrating ocular injury. *Retina* 1992;12:346–350.
2. Bloome MA, Ruiz RS: Massive spontaneous subretinal hemorrhage. *Am J Ophthalmol*,86:630–637,1978
3. Gass JDM: Pathogenesis of disciform detachment of the neuroepithelium: III. Senile disciform macular degeneration. *Am J Ophthalmol*, 63:617–644,1967
4. Abdel-Khaled MN. Richardson 1: Retinal macroaneurysm: natural history and guidelines for treatment. *Br J Ophthalmol* 70(1): 2-11, 1986
5. Kroll P, Busse H. Therapy of preretinal macular hemorrhages. *Klin Monatsbl Augenheilkd* 188:610–612,1986
6. Avery RL, Fekrat S, Hawkins BS, Bressler NM. Natural history of subfoveal hemorrhage in age-related macular degeneration. *Retina* 16(3):183–9,1996
7. Messmer E P, Wessing A, Ruprecht K, Naumann GO. Solitary intraretinal macular hemorrhage. *Graefes Arch Clin Exp Ophthalmol*. 222(1):9–12,1984.
8. Iijima H, Satoh S, Tsukahara S. Nd: YAG laser photodisruption for preretinal hemorrhage due to retinal macroaneurysm. *Retina* 18(5):430 – 4, 1998
9. Hayasaka S, Uchida M, Setogawa T: Subretinal hemorrhages with or without choroidal neovascularization in the maculas of patients with pathologic myopia. *Graefes Arch Clin Exp Ophthalmol* 228(4):277-80, 1990
10. Berrocal MH, Lewis ML, Flynn Hw: Variations in the clinical course of submacular hemorrhage. *Am J Ophthalmol* 122:486-493,1996
11. Bennett SR, Blodi CF, Folk JC: Factors prognostic of visual outcome in patients with subretinal hemorrhage involving the fovea. *Am J Ophthalmol* 109:33-37,1990
12. Kuhn F, Morris R, Witherspoon C D. Mester V. Terson syndrome. Results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. *Ophthalmology* 105(3):472–77. 1998.
13. Fechner P U. Premacular hemorrhage: a new indication for argon-laser-therapy. *Klin Monatsbl Augenheilkd*,177(4): 502–5, 1980
14. Kroll P, Le Mer Y. Treatment of preretinal retinal hemorrhage: value of early argon laser photocoagulation. *J Fr Ophthalmol* 12(1): 61–6, 1989
15. Hesse L, Schmidt J, Kroll P. Management of acute submacular hemorrhage using recombinant tissue plasminogen activator and gas. *Graefes Arch Clin Exp Ophthalmol* 237(4):273–7,1999
16. De Maeyer K, Van Ginderdeuren R, Postelmans L, Stalmans P, Van Calster. Sub-inner limiting membrane hemorrhage: causes and treatment with vitrectomy. *Br J Ophthalmol* 91(7): 869-872,2007.
17. Burk SE, Da Mata AP, Snyder ME, Rosa RH, Foster RE. et al Indocyanine green-assisted peeling of the retinal internal limiting membrane. *Ophthalmology* 107(11):2010–4. 2000.
18. Thompson J T. The role of the patient age and intraocular gas use in cataract progression after vitrectomy for macular holes and epiretinal membranes. *Am J Ophthalmol* 137(20): 250 – 7, 2004.
19. Cherfan G M, Michels R G, de Bustros S, Enger C, Glaser BM. Nuclear sclerotic cataract after vitrectomy for idiopathic epiretinal membranes causing macular pucker. *Am J Ophthalmol* 111(4): 434- 8, 1991.