Vitreous hemorrhages and sudden increased intracranial pressure

R. GRAHAM VANDERLINDEN, M.D., F.R.C.S. (C), AND LIONEL D. CHISHOLM, M.D., F.R.C.S. (C)

Departments of Surgery (Neurosurgery) and Ophthalmology, Toronto Western Hospital, University of Toronto, Toronto, Ontario, Canada

Six cases of bilateral hemorrhage into the vitreous body related to intracranial hypertension are presented. Four were associated with ruptured cerebral aneurysms, and the others followed head injury. The onset of vitreous hemorrhage was delayed in all cases, and in five patients subhyaloid hemorrhages were present from 2 to 27 days prior to their extension into the vitreous. Visual acuity was greatly reduced. The ophthalmoscopic and slit lamp appearance of the vitreous are described. The hemorrhages usually cleared spontaneously within 24 months, and vision returned to normal. Surgical treatment to remove residual vitreous blood in selected cases is outlined.

Key Words • vitreous hemorrhage • subarachnoid hemorrhage • head injury • visual deficit • spontaneous resolution • increased intracranial pressure

Subhyaloid hemorrhages are frequently seen following ruptured intracranial aneurysms,17,33,34 and occasionally after craniocerebral trauma.18,20 The demonstration of subhyaloid hemorrhages is important prognostically as the mortality rate of subarachnoid hemorrhage is 60% when they are present compared to 27% when they are absent;17,26 moreover, the mortality rate is higher in bilateral than in unilateral hemorrhages. The evidence suggests that the presence of subhyaloid hemorrhages is directly related to the severity of the intracranial bleeding and presumably to the concomitant elevation of intracranial pressure. In patients who survive, the subhyaloid hemorrhages invariably resolve and are considered little more than an ophthalmological curiosity. The extension of pre-retinal hemorrhages through the hyaloid membrane into the vitreous body, however, is a rare complication of subarachnoid hemorrhage in the experience of Walsh and Hoyt.25 The early related ophthalmological reports were concerned with the pathogenesis of intraocular bleeding resulting from brain hemorrhages, and most of the papers were based on postmortem studies. Very little has been written on the clinical course of patients who have survived a subarachnoid hemorrhage or craniocerebral injury with vitreous hemorrhages as a complication. There are also contradictory views regarding the resolution of hemorrhages and treatment of the visual deficit.

We are reporting six cases of vitreous
hemorrhage to draw attention to this condition as a complication of suddenly raised intracranial pressure.

Case Reports

The first four cases of vitreous hemorrhage were associated with subarachnoid hemorrhage. The last two were associated with craniocerebral injury.

Vitreous Hemorrhage Associated with Subarachnoid Hemorrhage

Case 1. A 53-year-old woman became comatose with a left hemiplegia 10 days following an unrecognized minor subarachnoid hemorrhage (SAH). The fundi appeared normal on admission to hospital. A large right temporal clot was removed, and as the posterior communicating aneurysm ruptured during the dissection of its neck, temporary carotid occlusion was maintained for 10 minutes until the aneurysm was clipped successfully. The patient recovered, but 11 days later examination revealed that she had a residual left homonymous hemianopsia and in addition her visual acuity had suddenly become reduced to light perception in the left eye and finger counting on the right. Red reflexes were obscured by bilateral vitreous hemorrhages but the pupils reacted to light directly and consensually, and ocular tension was normal (Fig. 1). Six months later visual acuity was 20/80 on the right and 20/125 on the left corrected for hyperopia. The red reflex was seen only on the right but detail of the fundus was obscured. Apart from a mild anxiety-depression she was then well neurologically. Seventeen months after the SAH, visual acuity was corrected to 20/25 in both eyes. At 3½ years following the SAH, examination of both eyes revealed that the formed vitreous had collapsed forward, anterior to the midpoint of the vitreous cavity. The white crumb-like remains of extravasated blood lay on the posterior surface of the formed vitreous inferiorly at the 6 o'clock position, and clear fluid filled the posterior portion of the vitreous cavity.

Case 2. This 44-year-old woman arrived in the emergency department approximately 15 minutes after an SAH. She complained of headache, stiff neck, and vomiting; bilateral subhyaloid hemorrhages surrounding swollen disc margins were already present. The same day she had a grand mal seizure; after return of consciousness a left abducens palsy was the only residual sign. Eight hours later she rebled, became restless, mute, incontinent, and developed extensor plantar responses. Angiography revealed a basilar bifurcation aneurysm. Two days later she was sufficiently alert to comment on transient episodes of blindness and periods of red vision akin to flashing stop lights. The peripheral visual fields were full but bilateral central scotomata were present. The aneurysm was clipped on the tenth day, but postoperative angiography revealed that the Mayfield clip used had slipped and part of the sac was still filling; this was obliterated 15 days later by a long Kerr-Lougheed clip. Six days after the first craniotomy visual acuity had decreased to finger counting in the right eye and hand movements in the left. Details of the right fundus were partially obscured by vitreous hemorrhage, and the red reflex was absent on the left (Fig. 1). Eight months later she could read newspaper headlines; visual acuity was 20/100 on the right and 20/400 on the left. The right vitreous hemorrhage now obscured only the disc and macula; the nasal retina could be seen in the left eye. Fourteen months after the hemorrhage, vision was 20/25 on the right and 20/30 on the left, and white remains of extravasated blood were observed on the vitreous inferiorly. Four years following the SAH, the patient's vision had improved to 20/20 bilaterally. The formed vitreous had collapsed forward in its cavity, leaving clear fluid posteriorly. White debris lay on the

Fig. 1. Cases 1 and 2. Graph showing delayed onset of vitreous hemorrhages following SAH.
posterior surface of the remaining formed vitreous at the 6 o’clock position.

Case 3. A 50-year-old woman with a 10-year history of hypertension suddenly suffered headache and temporary coma. Two days later she noted visual impairment and was transferred to a neurosurgical unit. She was disoriented and drowsy with bilateral abducens palsies and extensor plantar responses. Large subhyaloid hemorrhages reduced visual acuity to light perception in the right eye and counting fingers on the left, but optic disc margins were sharp and gross testing of visual fields was normal. A retrograde brachial arteriogram revealed a right superior cerebellar artery aneurysm that was clipped successfully on the sixth day. Eight days after surgery the craniotomy site was tense, lumbar CSF pressure was 290 mm H2O, and daily spinal taps were instituted. The subhyaloid hemorrhages were described as dense, red, and central with a sharply outlined margin. There were multiple irregular intraretinal hemorrhages; however, the retina appeared normal at the extreme periphery of the hemorrhages. There was complete right external ophthalmoplegia. Two days later the subhyaloid hemorrhages penetrated into the vitreous, and the red reflexes were abolished. The left retina could not be seen; under direct ophthalmoscopic examination, the retina could be seen at the periphery of the large black central vitreous from which black wispy filaments radiated. The slit lamp revealed dark red areas most dense at the posterior pole of the vitreous. Seven months later the visual acuity was limited to light perception in the right eye and to 20/200 vision in the left; the red reflex was still absent. There was a right-sided tremor, and she required a cane to walk. Eleven months following the SAH she was readmitted for treatment of recurrent depression. Visual acuity was still markedly impaired on the right and on the left was 20/100. At the last follow-up, 26 months following SAH, the patient required electroconvulsive therapy after a suicidal attempt. Visual acuity was 20/400 on the right and on the left corrected to 20/20. Dark material floating in the vitreous obscured fundus detail on the right.

Case 4. About 6 weeks following a sudden occipital headache, this 54-year-old woman suffered transient loss of consciousness. Twelve days later a similar episode led to hospital admission and the discovery of bloody spinal fluid. There was mental confusion, neck stiffness, mild left hemiparesis, and bilateral subhyaloid hemorrhages. Angiography revealed a right posterior inferior cerebellar aneurysm. Two days after admission a fourth SAH resulted in deterioration of consciousness and impairment of function of the right lower cranial nerve. Posterior fossa exploration was undertaken and the aneurysm clipped; there was elevated ventricular pressure and fresh blood clot around the brain stem. Rapid postoperative recovery was interrupted by two transient episodes of sudden headache, drowsiness, and increased right facial weakness on the fourth and eighth days. Lumbar CSF pressure was 240 mm H2O between these attacks. On the twelfth postoperative day, examination revealed a deterioration of visual acuity to hand movements in the right eye and counting fingers in the left. The right red reflex was absent and the left fundus detail was obscured by vitreous hemorrhages. Two months following the craniotomy visual acuity was 20/60 in the right eye and limited to counting fingers in the left. The vitreous was opaque bilaterally. At 18 months the acuity was 20/40 on the right and 20/60 on the left. Fundus details were clear on the right; and a slight red reflex was visible in the left eye. At 38 months vision was 20/20 bilaterally. White remains of blood lay inferiorly in the vitreous cavity but the left disc was slightly obscured. In the right eye the vitreous had collapsed forward and downward as in the previous cases. In the left eye, however, a strong adhesion to the disc held the vitreous up, preventing complete collapse. This accounted for the haziness of the disc on fundus examination but allowed normal vision.

Vitreous Hemorrhage Associated with Craniocephalic Injury

Case 5. A 35-year-old woman was unconscious immediately after an occipital head injury, but recovered in the ambulance and on admission showed no lateralizing neurological signs. There were blurred optic disc margins, engorged retinal veins, and a linear temporoparietal skull fracture. Seven hours later she became drowsy and developed mild...
Fl. 2. Cases 5 and 6. Diagrams showing development of vitreous hemorrhage after acute subdural hematoma following head injury. In Case 5 the vitreous hemorrhage was delayed for 2 days on the right and 9 days on the left following SAH. In Case 6 the vitreous hemorrhages followed a third craniotomy 47 days after the original trauma.

right arm weakness, left hyperreflexia, and a left Babinski sign; bilateral subhyaloid hemorrhages had developed. Following bilateral carotid angiography a left extradural and a right subdural hematoma were evacuated. Forty-eight hours later, the patient developed a right vitreous hemorrhage and on the ninth postoperative day a more massive left vitreous hemorrhage. Lumbar CSF pressure was found to be 230 mm H$_2$O. Repeat angiography revealed a recurrent right subdural hematoma, and this was evacuated (Fig. 2). Three months following trauma, vision was 20/200 on the right and 20/400 on the left. The fundus was seen in the periphery on the right but there was no red reflex on the left. At 17 months after trauma, vision was 20/70 on the right and 20/30 on the left when corrected for myopic astigmatism. By 23 months vision on the right was 20/30. Vitreous examination (Fig. 3) of the right eye revealed a white material lying in front of the macula and at the 6 o'clock position. In the left eye vitreous opacities were scattered at the periphery, particularly inferiorly. In both eyes the formed vitreous was collapsed far forward leaving clear fluid posteriorly. There was a small equatorial horse-shoe-shaped break in the left retina at the 3 o'clock position.

Case 6. This 38-year-old man became unconscious after he fell, striking the back of his head against a curb. By the time of admission he was talking and moving all limbs. Neurological examination was otherwise unremarkable except for slight blurring of the optic disc margins. A linear left occipital fracture extended into the petrous bone. Five hours later his level of consciousness suddenly deteriorated, the right pupil dilated, and he became decerebrate. A massive right subdural hematoma was evacuated, and his con-

Fig. 3. Case 5. Diagrams showing results of vitreous examination of both eyes 17 months after vitreous hemorrhage. Left: Right eye shows formed vitreous collapsed forward leaving clear fluid posteriorly; white material lying in front of macula accounts for the 20/70 visual acuity. Right: Left eye, similar to right but with clear macular area and 20/30 visual acuity.
Vitreous hemorrhages and increased intracranial pressure

dition improved only to deteriorate again 24 hours later. A large frontal bone flap was removed to decompress the swollen brain and a tracheostomy was done. Following a stormy course characterized by pulmonary insufficiency, atelectasis, and *Pseudomonas* pneumonia, he gradually recovered with a residual left hemiplegia, left hemianopsia, and dysarthria. Twenty days after the head injury a few subhyaloid hemorrhages were seen surrounding the discs. Six weeks after injury the bone flap was replaced; this was followed by immediate temporary drowsiness and increased dysarthria. Two days later the visual acuity was reduced to light perception on the left and finger counting on the right. There was no left red reflex and the right disc and macula were obscured by blood in the vitreous (Fig. 2). At 18 months after the vitreous hemorrhages were first noted, vision on the right was 20/400 and on the left limited to perception of hand movements. The fundus detail was obscured on the right, and the red reflex was present only at the extreme periphery on the left. He exhibited mild dementia, dysarthria, and an ataxic gait.

**Discussion**

**Incidence**

The syndrome of bleeding into the vitreous body following an SAH has been named after Terson who described it in 1912. Freud in 1884 reported vitreous hemorrhage associated with subdural hematoma in a case of scurvy. The incidence of vitreous hemorrhage associated with SAH is unknown, but is so rare that Paunoff found only 16 cases in the world literature up to 1962. Castrén reported eight more cases in 1963 but did not mention the total number of SAH from which the series was gleaned. In 1952 Timberlake and Kubik reported a 4% incidence of vitreous hemorrhage in their series of 280 patients with SAH; 20% of their cases also had "retinal" hemorrhages of unspecified type. The incidence of subhyaloid hemorrhage appears to vary from 11% to 33% in the few series reporting this complication of SAH.

**Pathogenesis**

The pathogenesis of subhyaloid hemorrhage due to spontaneous intracranial bleeding has been a subject of controversy. One group of authors believed that blood from the subarachnoid space was forced into the optic nerve sheath and penetrated the lamina cribrosa to appear within the globe. A much larger body of opinion supports the hypothesis that subhyaloid hemorrhage is the result of rupture of retinal veins secondary to retinal venous hypertension. Lehman, et al., have recently shown that experimentally induced intracranial hypertension results in elevation of intraocular pressure which they attribute to increased ophthalmic venous pressure. The exact mechanism whereby suddenly raised intracranial pressure creates retinal venous hypertension is unknown and a discussion of the principle theories is beyond the scope of this paper.

**Appearance**

The retinal veins responsible for the subhyaloid leakage of blood lie beneath the so-called internal limiting membrane of the retina. A tiny leakage of blood contained by the internal limiting membrane may track along nerve fiber bundles to form the familiar flame-shaped retinal hemorrhage, or lie between the nerve fiber layer and the internal limiting membrane and be seen as a bright red punctate mass on the retinal surface. More profuse bleeding which splits or ruptures the internal limiting membrane may track along nerve fiber bundles to form the familiar flame-shaped retinal hemorrhage, or lie between the nerve fiber layer and the internal limiting membrane and be seen as a bright red punctate mass on the retinal surface. The exact mechanism whereby suddenly raised intracranial pressure creates retinal venous hypertension is unknown and a discussion of the principle theories is beyond the scope of this paper.

The vitreous is a clear gel-like substance composed principally of a network of collagen fibrils in which long chains of hyaluronic acid molecules are enmeshed. The collagen fibrils are more dense near the retinal surface and form the cortical vitreous layer or posterior hyaloid lamina. The most peripheral fibrils of this layer coalesce with the internal limiting membrane of the retina.
and it is this interface that may be split by a subhyaloid hemorrhage. A hemorrhage at the subhyaloid stage may rupture through the posterior hyaloid barrier and disperse into the neighboring vitreous substance causing visual impairment and obscuring fundus detail. The funduscopic appearance varies from a mere haziness of the vitreous to complete blackness depending on the amount of blood that bursts into the vitreous substance.

Onset

In four of our cases subhyaloid hemorrhages were present on the day of admission. In Case 6 they were first noted 20 days later, and in Case 1 they were never seen, and the appearance of vitreous hemorrhages 11 days following the SAH was the first observed evidence of intraocular bleeding. In all six cases the development of vitreous hemorrhage was delayed following the initial episode of intracranial hypertension. This delay varied from 2 days to 47 days although in most cases it was approximately 2 weeks. Another patient not included in this series, who died of recurrent SAH, developed papilledema and retinal hemorrhages 20 days after the initial SAH; however, subhyaloid hemorrhages with extension into the vitreous appeared 4 days later following rebleeding. This delayed phenomenon was also observed by Walsh and Hoyt in two patients in whom the vitreous hemorrhages occurred 5 and 7 days after an initial SAH but were related to rebleeding. Another of their patients developed massive vitreous hemorrhages in both eyes following carotid angiography.

The evidence suggests that if a further episode of sudden intracranial hypertension supervenes in a patient with subhyaloid hemorrhages, such as the rebleeding of an aneurysm or the development of postoperative cerebral edema, the resulting elevation of intraocular venous pressure can facilitate the extension of subhyaloid blood into the substance of the vitreous. This point is well illustrated by our Case 4 in which two postoperative episodes of impairment of consciousness probably caused by pressure waves preceded the appearance of the vitreous hemorrhages.

The exact moment when blood invaded the vitreous was not precisely documented in any of our cases. All of the patients except Case 2 were extremely ill so that mydriatics were contraindicated and in addition the postoperative periorbital swelling made ophthalmoscopic examination difficult.
Vitreous hemorrhages and increased intracranial pressure

Course of Vitreous Hemorrhages

Experimental Studies. The course of vitreous hemorrhages has been studied experimentally in rabbits by isotopic labelling of various components of blood.10,25,30 These studies reveal that spontaneous lysis of collagen takes place at the site of the hemorrhage, allowing the red blood cells and debris to spread throughout the vitreous and later migrate toward the posterior pole following the forces of fluid circulation within the body of the vitreous.28 When they reach the retinal vessels, the soluble derivatives are absorbed and the insoluble particles are phagocytosed and carried out along the perivascular spaces. As the hemoglobin molecule breaks down, iron also escapes, but up to 25% is trapped in ocular tissues. Iron is generally accepted as responsible for liquefaction of the vitreous body and for the retinal damage seen in rabbits following vitreous hemorrhage.25,33 In addition to iron, the end product of the porphyrin chain is a bilirubin-like substance that produces the observed residual yellowish bands and veils previously thought to be fibrin formations.33

Clinical Studies. The long-term effects of vitreous blood in patients suffering SAH have been documented occasionally but the number of survivors is few. In their 12 cases of vitreous hemorrhage, Timberlake and Kubik34 noted that there was appreciable though never complete resolution of the hemorrhages in patients still living, but they did not mention the length of time they followed their patients. Walsh has noted clearing of massive intraocular hemorrhages presumably in the vitreous, with recovery of normal vision after more than a year. Riddoch and Goulden77 followed two cases for 10 months and one for 19 months with recovery of useful vision to 20/30, 20/40, and 20/60 respectively, although a few vitreous "floaters" were apparent. In the case that Paton23 followed for 18 months the vision returned to 20/20 and 20/30. In the remarkable case of Weaver and Davis38 in which the vitreous hemorrhages resulted from sudden intracranial hypertension by compression of a lumbosacral myelomeningocele, vision returned to 20/25 and 20/70 in 11 weeks.

We are not aware of any previously documented cases of vitreous hemorrhage following head injury. Mock,20 however, refers to this condition and states that the hemorrhages usually disappear in a few weeks or months although some permanent vitreous opacities may remain. Walsh and Hoyt36 have also observed "severe retinal hemorrhages, vitreous hemorrhages, and subsequent retinal detachment in patients with major injuries to both head and thorax," and felt that the causes of the intraocular complications were difficult if not impossible to unravel.

In the present series (Fig. 5) the vision became normal following vitreous hemorrhage in four cases in 15, 17, 23 and 29 months respectively. The vision in Case 3 was normal in the left eye in 26 months but acuity in the right remained at counting fingers. In Case 6 the follow-up has been relatively short but at 18 months recovery appears to have been arrested.

The eight eyes that we studied with the slit lamp and Goldmann three-mirror contact lens showed strikingly similar vitreous pictures at the time of the most recent examinations. The remains of formed vitreous had collapsed forward leaving clear fluid in the posterior vitreous cavity. White products of blood breakdown lay inferiorly in the plane of the posterior surface of the formed vitreous. In Case 5, the white material still lay on the right optic axis thus accounting for the reduced visual acuity at that time.

In our cases the initial vitreous hemorrhage probably occupied only the posterior portion of the vitreous. Blood breakdown resulted in liquefaction of the vitreous gel, condensation of the remaining collagen fibrils, and further separation at the vitreo-retinal interface. The white material resulting from degraded blood usually lay on the posterior face of the formed vitreous and at the 6 o'clock position. This indicates that vitreous liquefaction was most marked in the immediate vicinity of the blood extravasated into the posterior vitreous and that gravitational effects were present. With this slow process of vitreous liquefaction and collapse, the blood previously held in mid-vitreous by an intact gel-like structure settled below the optic axis and vision improved.

Treatment

Our experience indicates that vitreous hemorrhage following acute intracranial hypertension will clear spontaneously in 18 to 24 months in the majority of cases. Accord-
ingly, we advocate nonintervention for at least 18 months or until no progress in the rate of vitreous clearance has been observed for a period of 6 months.

Surgical treatment to remove residual masses of vitreous blood is available. Maberley and Chisholm hastened the gross clearance rate of artificial vitreous hemorrhage in rabbits by irrigation of the vitreous cavity through the pars plana with a balanced salt solution and restored the vitreous volume with the irrigating fluid. This

![Graphs showing amount of recovery of vision following bilateral vitreous hemorrhage. Case 1: Recovery of vision at 17 months. Case 2: Recovery of vision at 15 months. Case 5: Recovery of vision at 23 months. Case 6: No improvement in visual acuity 18 months following vitreous hemorrhage; vitreous surgery recommended.](image-url)

Fig. 5. Graphs showing amount of recovery of vision following bilateral vitreous hemorrhage. Case 1: Recovery of vision at 17 months. Case 2: Recovery of vision at 15 months. Case 5: Recovery of vision at 23 months. Case 6: No improvement in visual acuity 18 months following vitreous hemorrhage; vitreous surgery recommended.
Vitreous hemorrhages and increased intracranial pressure

technique has been used over the years by many ophthalmic surgeons employing a variety of replacement materials. Should this simple approach fail to remove sufficient vitreous debris, the procedure of posterior vitrectomy may be employed. Machemer and co-workers have developed a sophisticated device which is inserted into the vitreous cavity through a pars plana incision. At the tip of the cylindrical instrument, suction draws solid elements into a round sharp edged opening where an inner rotating cutting edge breaks the material in scissors fashion. As a replacement, normal saline or balanced salt solution is infused into the vitreous cavity by means of the same system. By careful manipulation under indirect ophthalmoscopic control, the ophthalmic surgeon can remove solid material from the vitreous and replace the volume with infusion fluid without damaging the lens or retina.

Acknowledgments

The authors thank Dr. H. Schutz and Dr. S. Schatz for permission to publish their cases and Dr. J. L. Silversides and Dr. J. F. R. Fleming for their interest and encouragement.

References

25. Regnault FR: Vitreous hemorrhage: an experimental study. 1. A macroscopic and
33. Timberlake WH, Kubik CS: Follow-up report with clinical and anatomical notes on 280 patients with subarachnoid hemorrhage. Trans Amer Neurol Assoc 77:26-30, 1952

Address reprint requests to: R. G. Vanderlinden, M.D., F.R.C.S., Department of Neurosurgery, Toronto Western Hospital, Toronto, Ontario, Canada.