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Terson hemorrhage in patients suffering aneurysmal subarachnoid hemorrhage: predisposing factors and prognostic significance

Kostas N. Fountas, M.D., Ph.D.,[1,4] Eftychia Z. Kapsalaki, M.D., Ph.D.,[2] Gregory P. Lee, Ph.D.,[3] Theofilos G. Machinis, M.D.,[4] Arthur A. Grigorian, M.D., Ph.D.,[4] Joe S. Robinson Jr., M.D.,[4] Ioannis Vergados, M.D., Ph.D.,[5] and Panagiotis G. Theodosiadis, M.D.[5]

Departments of [1]Neurosurgery and [2]Diagnostic Radiology, University Hospital of Larisa, University of Thessaly, School of Medicine, Larisa; [5]Department of Ophthalmology, Attiko General Hospital of Athens, University of Athens, Greece; [3]Department of Neurology, Medical College of Georgia, Augusta; and [4]Department of Neurosurgery, Medical Center of Central Georgia, Mercer University, School of Medicine, Macon, Georgia

Object. The association of vitreous and/or subhyaloid hemorrhage with aneurysmal subarachnoid hemorrhage (SAH) has been frequently identified since the original description by Terson in 1900. In this prospective clinical study the authors examined the actual incidence of Terson hemorrhage in patients suffering aneurysmal SAH, they attempted to identify those parameters that could predispose its development, and they evaluated its prognostic significance in the overall patients' outcome.

Methods. A total of 174 patients suffering aneurysmal SAH were included in this study. The admitting Glasgow Coma Scale scores (GCS), World Federation of Neurological Societies (WFNS) scale scores, Hunt and Hess grades, and Fisher grades were recorded. A careful ophthalmological evaluation was performed in all participants. The exact anatomical locations and the largest diameter of the dome of the ruptured aneurysms were also recorded. Surgical clipping or endovascular coiling was used in 165 patients. Clinical outcome was evaluated at discharge from the hospital by using the Glasgow Outcome Scale and the modified Rankin Scale. Periodic ophthalmological evaluations were performed for 2 years.

Results. In this series, the observed incidence of Terson hemorrhage was 12.1%. Statistical analysis of our data demonstrated that patients with low GCS scores and high WFNS scores, Hunt and Hess grades, and Fisher grades had an increased incidence of Terson hemorrhage. The mortality rate for patients with Terson hemorrhage was 28.6%, whereas that for patients without Terson hemorrhage was 2.0%. Moreover, patients with Terson hemorrhage who survived had significantly worse outcomes than those in patients without Terson hemorrhage.

Conclusions. Terson hemorrhage constitutes a common SAH-associated complication. Its incidence is increased in patients with low GCS and high WFNS scores, and high Hunt and Hess and Fisher grades. Its presence is associated with increased mortality and morbidity rates. *(DOI: 10.3171/JNS/2008/109/9/0439)*

Key Words • aneurysm location • aneurysm size • clinical outcome • mortality rate • subarachnoid hemorrhage • Terson hemorrhage

The development of ophthalmological signs in patients suffering aneurysmal SAH has been adequately described in the literature. Since the original description of subhyaloid hemorrhage secondary to intracerebral hemorrhage by Albert Terson in 1900,[23] numerous studies have been published regarding the incidence of not only subhyaloid but more generally of vitreous and retinal hemorrhages associated with aneurysmal SAH. The incidence, pathogenetic mechanism, and prognostic significance of Terson hemorrhage have been explored in these studies. However, the identification of any factors predisposing the occurrence of Terson hemorrhage has not been adequately explored.

In this clinical study we attempted to estimate the exact incidence of Terson hemorrhage among patients suffering aneurysmal SAH, identify those parameters that could be positively or negatively associated with Terson hemorrhage, and examine its association with newer clinical outcome markers. Additionally, we reviewed the pertinent literature.

Abbreviations used in this paper: ACoA = anterior communicating artery; DS = digital subtraction; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; mRS = modified Rankin Scale; SAH = subarachnoid hemorrhage; WFNS = World Federation of Neurological Societies.

Methods

In our current prospective clinical study, all patients admitted or transferred to the Medical Center of Central Georgia in whom spontaneous SAH had been diagnosed were considered for ophthalmological evaluation. The diagnosis of SAH had been established either by obtaining a head CT scan or by performing a lumbar puncture. All patients with SAH underwent 4-vessel cerebral DS angiography within 12 hours of their admission to our institution. A standard femoral catheterization technique was used with the aide of a 4 or 5 Fr wire-guided catheter. The patients in whom no aneurysm was noted on their DS angiogram were excluded from our study. Those patients who denied participating in our study were also excluded. This study was approved by our institutional review board. A detailed written consent form was obtained from the participant or the patient's legal representative in cases in which the participant was unable to sign. The data collection and analysis were performed according to the current Health Insurance Portability and Accountability Act regulations.

A total of 174 patients (103 men and 71 women) harboring 181 cerebral aneurysms were included in our cohort. Their mean age was 56.7 years (range 21–90 years). The admitting GCS scores, Fisher grades, Hunt and Hess grades, and WFNS scores were recorded at the time of the patients' admission. The anatomical location and the largest diameter of the dome (calculated on the DS angiogram) of the studied aneurysms were recorded. All patients were admitted to the neurointensive care unit, and a meticulous ophthalmological examination including visual acuity, visual field, pupillary reflexes, external ocular movement, and funduscopic appearance after induced mydriasis was performed by an experienced neuroophthalmologist within 24 hours of their admission. The ophthalmologist performing the examination was not aware of the patient's Fisher grade or the head CT scan details. Surgical clipping or endovascular coiling was performed within 48 hours of admission in the vast majority of our patients (165 of 174 [94.8%]). Two patients (1.1%) were transferred for further treatment to another facility. The remaining 7 patients (4.0%) either refused further treatment, or it was decided that they would not undergo surgical or endovascular intervention.

Outcome was assessed using the GOS and the mRS at the time of the patients' discharge from the hospital. The patients' follow-up included head CT scanning, DS angiography, MR imaging/MR angiography (when indicated), and detailed neurological examination at 1, 4, and 12 weeks after discharge and then at 6, 12, 18, and 24 months postoperatively.

Follow-up ophthalmological examinations were performed at postictal Weeks 1, 4, 8, and 12 and then at postictal Months 6, 12, 18, and 24. The ophthalmological follow-up evaluation included visual acuity and visual field examinations as well as a detailed funduscopic examination.

Statistical analysis of our data was performed using the chi-square method. The level of statistical significance was set at 0.0005.

Results

The diagnosis of Terson hemorrhage (subhyaloid and/or vitreous hemorrhage) was established in 21 patients (12.1%). Thirteen patients were men and the remaining 8 were women (Table 1). Analysis of our data revealed no statistically significant difference between men and women in regard to the incidence of Terson hemorrhage ($\chi^2 = 0.0011$, df = 1, p = 0.9739). Bilateral hemorrhages were present in 9 patients (42.9%), whereas 12 patients (57.1%) had unilateral hemorrhage (7 cases on the left and 5 cases on the right side).

The admitting GCS scores, Fisher grades, Hunt and Hess grades, and WFNS scores of our patients are summarized in Table 1. The vast majority of patients with Terson hemorrhage had GCS scores < 8 at admission. The difference in the incidence of Terson hemorrhage among patients with admitting GCS scores < 8 and those with GCS scores ≥ 8 reached levels of statistical significance ($\chi^2 = 52.9081$, df = 1, p = 0.0000). Terson hemorrhage was also found to be more common among patients with Fisher grades > 3 in a statistically significant fashion ($\chi^2 = 50.7877$, df = 1, p = 0.0000). The incidence of Terson hemorrhage was higher among those patients whose Hunt and Hess grades were > III at admission. This difference was statistically significant ($\chi^2 = 54.1764$, df = 1, p = 0.0000). Likewise, Terson hemorrhage occurred more commonly among patients with admitting WFNS scores > III in a statistically significant fashion ($\chi^2 = 29.6548$, df = 1, p = 0.0000).

The data regarding the occurrence of Terson hemorrhage in association with the location of the aneurysms in the anterior or posterior cerebral circulation and their exact anatomical locations are summarized in Table 2. Terson hemorrhage was more common among patients harboring anterior cerebral circulation aneurysms. However, this difference did not reach levels of statistical significance ($\chi^2 = 2.2873$, df = 1, p = 0.1304). Anterior communicating artery aneurysms were more commonly associated with Terson hemorrhage in this study. This association, however, was not statistically significant ($\chi^2 = 11.2216$, df = 15, p = 0.7367). We could not establish any statistically significant association between the size of the ruptured aneurysm (given that the size was estimated by measuring the largest diameter of the aneurysm dome) and the occurrence of Terson hemorrhage ($\chi^2 = 0.4807$, df = 2, p = 0.7864) (Table 3).

Surgical clipping was performed in 95 patients (97 aneurysms), and endovascular coiling was performed in 70 patients (75 aneurysms). The observed mortality rate among patients with Terson hemorrhage was 28.6% (6 of 21 patients), whereas among patients without Terson hemorrhage the rate was only 2.0% (3 of 153 patients). This difference was statistically significant ($\chi^2 = 16.1729$, df = 1, p = 0.0001). Patients with Terson hemorrhage had worse outcomes (GOS score ≤ 3) than those without Terson hemorrhage (Table 3). This difference reached the levels of statistical significance in our cohort ($\chi^2 = 14.0149$, df = 1, p = 0.0002). However, when the mRS score was used for evaluating clinical outcome in the same series (Table 3), no statistically significant difference was found between the 2 groups of patients ($\chi^2 = 0.5784$, df = 1, p = 0.4469).

TABLE 1

*Characteristics in 174 patients with aneurysmal SAH**

Characteristic	No. of Patients	
	w/o Terson hemorrhage	w/ Terson hemorrhage
sex		
male	90	13
female	63	8
admission GCS score		
≥8	133	3
<8	20	18
admission Fisher grade		
≤3	135	4
>3	18	17
admission H & H grade		
≤III	127	1
>III	26	20
admission WFNS score		
≤III	100	0
>III	53	21

* H & H = Hunt and Hess.

TABLE 2

*Locations of 181 aneurysms in 174 patients**

Anatomical Location of Aneurysm	No. of Aneurysms	
	Not Associated w/ Terson Hemorrhage	Associated w/ Terson Hemorrhage
anterior cerebral circulation	116	19
posterior cerebral circulation	44	2
exact location		
ICA	16	4
OphA	4	1
PCoA	37	3
AChA	4	0
A_1	2	1
ACoA	36	9
A_2	3	0
M_1	9	1
M_2	5	0
P_1	1	0
P_2	1	0
P_3	1	0
BA	29	2
SCA	3	0
AICA	3	0
PICA	6	0

*AChA = anterior choroidal aneurysm; AICA = anterior inferior cerebellar artery; BA = basilar artery; ICA = internal carotid artery; OphA = ophthalmic artery; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery.

In regard to the ophthalmological treatment of our patients with Terson hemorrhage, it was decided that all patients would be conservatively treated. Our policy in the treatment of patients with Terson hemorrhage secondary to aneurysmal SAH is to conservatively treat their ophthalmological condition unless no improvement occurs after 6 months of observation. In these cases, a pars plana vitrectomy can be considered if the patient is medically stable for undergoing a surgical procedure. In our current study, 15 patients with Terson hemorrhage were alive and able to undergo an ophthalmological evaluation at 4 weeks after their admission. In 12 (80%) of these patients there was improvement of their ophthalmological condition, whereas in the remaining 3 (20%) an accurate visual evaluation was not possible due to their neurological condition, although there was no change in the size of their subhyaloid hemorrhage. No difference was found in the ophthalmological outcome of patients with bilateral versus unilateral Terson hemorrhage. Six-month follow-up data were available in 15 patients. Improvement in ophthalmological status was evident in 13 patients (86.7%), and complete resolution of subhyaloid and/or vitreous hemorrhage occurred in 3 patients (20%). In the remaining 2 (13.3%) of these 15 patients accurate examination was not feasible, although the size of the subhyaloid hemorrhage had been significantly decreased. Twelve-month data were available in 11 patients. Further improvement was evident in all examined patients, and complete resolution of retinal hemorrhage was demonstrated in 5 patients (45.5%). The 24-month follow-up data were available for 9 patients. Full restoration of visual acuity along with complete resolution of the subhyaloid hemorrhage was evident in all examined patients.

Discussion

The incidence of Terson hemorrhage varies significantly among previously published series.[7,8,11,12,17,18,21,24,25,29] It has been reported to range between 2.6 and 27% among patients suffering aneurysmal SAH.[7,11,12,18,21,24,25,29] In our current series 12.1% of our patients developed Terson hemorrhage. Our rate is comparable to that reported by Kuhn et al.,[11] who found that 8% of their patients with SAH of aneurysmal origin developed Terson hemorrhage. Frizzell et al.[7] reported an 8% incidence of Terson hemorrhage in their prospective series. Similarly, Roux et al.[18] reported Terson hemorrhage in 10.5% of their patients with SAH, whereas the respective percentage was 14.6% in the study by Wietholter et al.[29] in which the patients suffered spontaneous SAH of aneurysmal and nonaneurysmal origin. Tsementzis and Williams[25] reported a significantly lower incidence (7.1%) of Terson hemorrhage in a very limited series of patients with aneurysmal SAH. On the other hand, Manschot[12] reported a 20% incidence, Timberlake and Kubik[24] reported a 23.5% occurrence, and Garfinkle et al.[8] described a 27% incidence of Terson syndrome among patients with SAH. This significant variation may be explained by the fact that the term "Terson hemorrhage" was originally used for describing a solely vitreous hemorrhage. In several studies, however, this term has been used for describing retinal hemorrhages in addition to vitreous hemorrhages.[1,8,13,24] Furthermore, the observed variation in incidence of Terson syndrome may be associated with the fact that the vast majority of the previously published studies were retrospective ones.[13] Despite the existent variation in its reported incidence, Terson hemorrhage is an underreported SAH-associated complication.[8,21] The fact that the vast majority of the pertinent literature references can be found in ophthalmological and not in neurosurgical journals is noteworthy.[29] Such underreporting of Terson hemorrhage in cases of

spontaneous SAH may be explained by the fact that its diagnosis can be easily missed.

Careful ophthalmological examination including meticulous funduscopic evaluation remains the method of choice for detecting Terson hemorrhage. The ideal timing for performing an ophthalmological examination remains to be defined. In our series all ophthalmological evaluations were obtained within 24 hours of the patient's admission. Manschot[12] reported that all Terson hemorrhages were present within 1 hour from the ictal event. However, Vanderlinden and Chisholm[26] reported that delayed Terson hemorrhage might occur up to 47 days after the ictal event. The role of visual evoked potential examination in detecting Terson hemorrhage is very limited due to the extremely low sensitivity of this method.[29] Swallow et al.[22] evaluated the potential of obtaining CT scans of the orbits for detecting Terson hemorrhages. They found that in the majority (66.7%) of patients with Terson hemorrhage, characteristic retinal nodularity and retinal crescentic hyperdensities were evident on their CT scans.[22] Interestingly, the interobservational concordance rate among different radiologists reading the obtained CT scans in their study was quite high.[22] It has to be emphasized, however, that the reported CT findings are subtle and can be easily overlooked, especially in an acutely ill patient, or can be misdiagnosed as streak osseous or motion artifacts.[22] The diagnostic role of multidetector, high-resolution CT scanning in detecting a Terson hemorrhage remains to be prospectively evaluated in a large-scale clinical study.

Several pathogenetic theories have been proposed for explaining the development of Terson hemorrhage since its original description.[2–4,8,10,12,14–17,20,21,26,27] The most widely accepted pathogenetic mechanism suggests that a suddenly increased ICP is propagated from the intracranial compartment to the orbit through a rapid effusion of cerebrospinal fluid via the optic nerve sheath.[2,3,8,10,12,14,15,17,19,26,27] Experimental confirmation of this mechanism has been provided by Manschot.[12] In a human cadaveric study, he injected a mixture of gelatin and India ink in the cerebral subarachnoid space. After the injection, the optic canal was dissected and the injected stain was found in the entire length of the subarachnoid space of the optic canal.[12] The dilated retrobulbar portion of the optic nerve mechanically compresses and obstructs the central retinal vein and the existent retinochoroidal anastomoses.[2,3,8,14,17] The venous obstruction causes venous stasis, which results in distention and rupture of the fine retinal capillaries.[3,8,14,17] This pathogenetic mechanism explains the association of Terson hemorrhage with pathological conditions other than SAH of increased ICP. The occurrence of Terson hemorrhage has been described in cases of moyamoya disease,[1] severe closed-head injuries,[8,14,26] intracerebral hemorrhage,[14,20,27] carotid artery occlusion,[14] cranial subdural hematoma,[20] lumbosacral myelomeningocele,[28] and as a complication associated with intraarterial angiography.[10] The extravasated blood may hydrodissect the inner limiting membrane from the underlying neurosensory retina.[15] This is suggested by the fluorescein angiographic findings of Ogawa et al.[15] in a case of Terson hemorrhage in which they noticed stain leakage at the demarcation between the inner limiting membrane of the retina and the inner limiting membrane of Elschnig.

TABLE 3

Outcomes after surgery

Parameter	Value	
	w/o Terson Hemorrhage	w/ Terson Hemorrhage
aneurysm dome diameter (cm)*		
<1	43	7
1–2.5	111	13
>2.5	6	1
GOS score at discharge†		
>3	136	12
≤3	15	9
mRS score at discharge‡		
≤3	136	12
>3	15	3

*Values represent the number of aneurysms.

†Values represent the number of patients who were treated at our institution.

‡Values represent the number patients who were treated at our institution and survived.

Bilateral Terson hemorrhages occurred in 42.9% of our cases. Pfausler et al.[17] reported that bilateral Terson hemorrhages represented the majority (60%) of their cases. Castren[4] reported that bilateral Terson hemorrhages represented 62.5% of his cases. Similarly, Garfinkle et al.[8] found in their series that 66.7% of their patients suffered bilateral vitreous and/or subhyaloid hemorrhages. The presence of bilateral versus unilateral hemorrhages has not been associated with worse visual outcome in our current or any of the previously published clinical series.[4,8,17] The only exception was the series reported by Shaw et al.,[21] who found that bilateral Terson hemorrhages were associated with 58.1% mortality rates, whereas the mortality rates in patients with unilateral hemorrhage was 48%. The described difference in mortality rates, however, was not statistically significant.[21]

No sex difference in the patients suffering Terson hemorrhage was found in our current series. In his series Castren[4] reported equal distribution of Terson hemorrhages among male and female patients. Similarly, Fahmy[6] found no statistically significant difference between males and females regarding the incidence of fundal hemorrhages, although he found a statistically significant difference in the occurrence of vitreous hemorrhage with male preponderance. Contrariwise, Pfausler et al.[17] and Garfinkle et al.[8] reported that females more commonly developed Terson hemorrhage. It has to be emphasized, however, that both of these prospective series included significantly smaller numbers of patients than those in our current series.

The previously described association of poor clinical condition at admission with Terson hemorrhage was confirmed by our findings.[8,13,17] In our series, Terson hemorrhage was significantly more common among patients with GCS score < 8, Hunt and Hess grade > III, WFNS score > III, and Fisher grade > 3. Similarly, Pfausler et al.[17] reported that increased incidence of Terson hemorrhage was found among patients with Hunt and Hess grade ≥ III. This association was statistically significant, as it also

was in our current series. Garfinkle et al.[8] also reported increased incidence of Terson hemorrhage among patients with higher Hunt and Hess grade SAH (> III), although that difference did not reach levels of statistical significance in their study. The significantly increased incidence of Terson hemorrhage among patients with Fisher grade > III hemorrhages documented in our series had also been reported by Fahmy.[6] A massive SAH, as expressed by a higher Fisher grade, may suddenly increase the ICP and cause an acute obstruction of the central retinal vein and a subsequent subhyaloid hemorrhage.

Aneurysms of the anterior cerebral circulation, and particularly aneurysms of the ACoA, were more commonly involved in the development of Terson hemorrhage in our study. However, this difference did not reach the level of statistical significance. Fahmy[6] reported in his study that ACoA aneurysms were more commonly associated with Terson hemorrhage than any other aneurysm. He also reported that this difference was statistically significant.[6] Garfinkle et al.[8] reported an increased incidence of Terson hemorrhage among patients with ACoA aneurysms without mentioning the statistical significance of their finding. As far as we know no association between the site of the ruptured aneurysm and the laterality of the Terson hemorrhage has been reported.[6,17] Interestingly, no statistically significant association between the size of the aneurysm (given that its size was estimated by measuring the largest diameter of the aneurysm dome) and the occurrence of Terson hemorrhage could be established in our series. The occurrence of Terson hemorrhage was the same in patients with small or giant aneurysms.

In our study, the mortality rate among patients with Terson hemorrhage was significantly higher ($p = 0.0001$) than that in patients without Terson hemorrhage. Likewise, Gutierrez Diaz et al.[9] reported that the presence of Terson hemorrhage increased the mortality rate from 20 to 50%. In his study, Manschot[12] reported that the mortality rate among patients with Terson syndrome was 50%, whereas that for patients without Terson hemorrhage was 25%. Vanderlinden and Chisholm[26] reported an increase in mortality rate from 27 to 60% in cases in which Terson hemorrhage was present. Pfausler et al.[17] found that the mortality rate among their patients with Terson hemorrhage was 90%, whereas it was only 23% among patients with no Terson hemorrhage. Similarly, Shaw et al.[21] reported that the mortality rate among patients with Terson hemorrhage was 53.6%, whereas the respective rate among patients without was 19.7%. Garfinkle et al.[8] found an increased mortality rate among patients with Terson hemorrhage compared with those without, however, this difference was not statistically significant. On the other hand, Roux et al.[18] found that the presence of Terson hemorrhage did not increase the mortality rate in their series.

In our study, significantly worse clinical outcome (as measured using GOS scores) occurred among patients with Terson hemorrhage ($p = 0.0002$) compared with outcomes observed among patients without Terson hemorrhage. Our finding is in agreement with those of previously published studies.[11,18] Roux et al.[18] found that increased morbidity was associated with Terson hemorrhage. Kuhn et al.[11] observed that the incidence of coma among patients with Terson hemorrhage was 89%, whereas it was 46% among patients without Terson hemorrhage. Interestingly, analysis of our data regarding the clinical outcome, as measured by mRS scores, demonstrated no statistically significant difference in outcome between patients with or without Terson hemorrhage ($p = 0.4469$). The observed discrepancy between the GOS and the mRS scores and their relation with Terson hemorrhage may be related to the different sensitivities of these 2 outcome scales. A larger clinical study may be necessary to address this issue. It is apparent that the increasing usage of mRS score in future clinical studies may confound any comparisons with previously published studies in which the GOS scale had been used.

Conservative ophthalmological treatment is the method of choice in managing Terson hemorrhage.[3,8,11,20,21,26] The visual outcome was excellent in the majority of cases of Terson hemorrhage in our series. Our findings are in agreement with those of previously reported series.[3,8,11,20,21,26] In rare instances, however, Terson hemorrhage can be associated with the development of proliferative retinopathy, retinal breaks, retinal detachment, and cataract.[29] Surgical intervention is reserved for those cases in which there is no visual acuity improvement within 6 months from the ictal event.[3,8,26] On the other hand, Roux et al.[18] recommended earlier surgical intervention. They suggested that in some occasions ophthalmological surgical intervention should be performed even before the neurosurgical intervention.[18] However, pars plana vitrectomy was used in only 15.3% of their patients (4 of 26 cases).[18] The surgical treatment of choice, when indicated, is pars plana vitrectomy. Clarkson et al.[5] reported excellent results in their series of 5 patients undergoing vitrectomy for persistent vitreous hemorrhage. It has to be noted, however, that vitrectomy has been occasionally associated with troublesome complications such as retinal damage, retinal detachment, cataract, endophthalmitis, and retinal hemorrhage recurrence.[11] Complete resolution of Terson hemorrhage occurred in all our patients (9 of 9 patients with available follow-up data). Interestingly, complete resolution of vitreous hemorrhage was observed in 20% of our cases within 6 months from the ictal event. Likewise, Weaver and Davis[28] reported complete resolution in their case within 11 weeks from the ictal event, whereas Pfausler et al.[17] observed complete resolution of vitreous hemorrhage in 1 of their cases in 4 weeks. Similarly, Vanderlinden and Chisholm[26] reported that complete resolution of Terson hemorrhage occurred in their series, although this occurred 15–29 months from the ictal event (mean resolution time 20.4 months). Contrariwise, Timberlake and Kubik[24] reported that they never observed a complete resolution of vitreous hemorrhage in any of their patients.

Conclusions

Terson hemorrhage constitutes a common complication of aneurysmal SAH, occurring in 12.1% of our prospectively studied patients. Early identification of Terson hemorrhage by performing a meticulous ophthalmological examination is of paramount importance not only

for establishing its diagnosis and promptly managing it, but also for its prognostic significance in the overall outcome of these patients. An increased incidence of Terson hemorrhage was associated with low admitting GCS and high WFNS scores, Fisher and Hunt and Hess grades in our current study. The anatomical location or the size of the ruptured aneurysm did not influence the incidence of Terson hemorrhage in a statistically significant fashion. Patients with Terson hemorrhage had a higher mortality rate, whereas those who survived had significantly worse outcomes than patients with no Terson hemorrhage. Conservative treatment of Terson hemorrhage resulted in its complete resolution in the vast majority of our cases.

Disclaimer

The authors do not report any conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Address correspondence to: Kostas N. Fountas, M.D., Ph.D., Department of Neurosurgery, University Hospital of Larisa, University of Thessaly, School of Medicine, Lambrou Katsoni str., Terspithea-Larisa, 41500, Greece. email: knfountasmd@excite.com.