

Traumatic Hyphema in Children

Risk Factors for Complications

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Objective: To identify risk factors associated with higher rates of ocular complications in children with traumatic hyphema.

Methods: Consecutive inpatient records from July 1990 through December 1997 were retrospectively reviewed for all children (aged ≤ 18 years) who were admitted to the Wilmer Ophthalmological Institute, Baltimore, Md, within 48 hours of a closed-globe injury leading to hyphema. Data obtained included age, sex, race, sickle cell status, initial and final visual acuities, hyphema size and intraocular pressure at presentation, the occurrence of a secondary hemorrhage, subsequent intraocular pressure elevations, and therapeutic interventions.

Results: Forty children fulfilled the inclusion criteria: 20 African American, 1 Asian American, and 19 white. Five of the 20 African American children had sickle cell

trait, and 1 had sickle cell anemia. The rate of secondary hemorrhage was statistically higher in the African American population ($P = .05$), but no statistical difference existed between the rate of secondary hemorrhage in patients with and without sickle cell hemoglobinopathy. Sickle cell hemoglobinopathy was associated with a higher intraocular pressure at presentation ($P = .03$) and during inpatient follow-up ($P = .02$).

Conclusions: In the setting of traumatic hyphema, African American children appear to be at greater risk for developing a secondary hemorrhage. In our patients, sickle cell hemoglobinopathy increased the risk of intraocular pressure elevation, but did not seem to increase the risk of rebleeding beyond that associated with race. Larger studies are needed to validate these observations.

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IN EYES WITH traumatic hyphema following closed-globe injury, a secondary hemorrhage is associated with a worse visual outcome.^{1,2} Recurrent bleeding increases the risk of vision-threatening complications, including corneal blood staining, secondary glaucoma, and optic atrophy. It is, therefore, generally accepted that a major goal in the treatment of traumatic hyphema is to prevent secondary hemorrhage and its associated sequelae. However, the large variability in the reported rates of recurrent hemorrhage (3%-38%) and associated ocular complications³⁻¹⁰ has led to controversy over the optimal management of traumatic hyphema.⁷⁻¹⁸

Numerous studies^{5-7,19-22} have attempted to identify risk factors for recurrent bleeding and associated ocular complications in eyes with traumatic hyphema, which would guide medical and surgical interventions. The influence of race on these issues has not been fully explored.^{4,8,23} Patients with sickle cell hemo-

globinopathy require special attention, because they are at higher risk for developing ocular complications.²⁴⁻²⁶ As a result of the mechanical obstruction of the trabecular meshwork by sickled erythrocytes, eyes of these patients are more likely to develop intraocular pressure (IOP) elevations. Furthermore, these eyes are more likely to develop central artery occlusion and optic nerve damage, with only marginal increases in the IOP.²⁴ It is unclear, however, whether the presence of sickle cell hemoglobinopathy actually increases the risk of secondary hemorrhage in these patients. The goals of this study were to determine which factors, including but not limited to race and sickle cell hemoglobinopathies, were associated with an increased risk of secondary hemorrhage and related ocular complications.

RESULTS

Sixty-two consecutive inpatient records coded for hyphema were reviewed for the period between July 1990 and December

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MATERIALS AND METHODS

Consecutive inpatient records from July 1990 through December 1997 were retrospectively reviewed for children (aged ≤ 18 years) admitted to the Wilmer Ophthalmological Institute at The Johns Hopkins Hospital, Baltimore, Md, within 48 hours after a closed-globe injury leading to hyphema. Patients were excluded if they had a microhyphema (ie, visible only with a slitlamp as dispersed, non-settled erythrocytes), associated open-globe injury, preexisting eye pathological features, or prior intraocular surgery. Aspirin, unlike other nonsteroidal anti-inflammatory drugs, irreversibly inhibits the platelet enzyme responsible for aggregation.²⁷ Hemostasis is impaired for at least 5 to 7 days following the administration of a single dose of aspirin. Therefore, patients with a history of aspirin use within 2 weeks of presentation were excluded, while those with documented use of other nonsteroidal anti-inflammatory drugs were not. Patients referred by outside ophthalmologists for management were excluded to prevent any referral bias to the study population. The following information was collected: age, sex, race, sickle cell status, mechanism of injury, initial and final visual acuities, hyphema size and IOP at presentation, subsequent IOP elevation during hospitalization, the occurrence of a secondary hemorrhage, and medical or surgical interventions. Because this was a retrospective review of clinical outcomes, and not a prospective clinical trial, informed consent was not obtained for inclusion in this study.

All patients were admitted to the Wilmer Ophthalmological Institute inpatient unit for bed rest. The affected eye was covered with a metal shield, and cycloplegic eye drops were administered. All patients were treated with oral aminocaproic acid (Amicar) (37 of 40 patients) or oral prednisone (3 of 40 patients) at the discretion of the admitting physician. All affected eyes were treated with topical corticosteroids. Eyes with an IOP greater than 21 mm Hg were also treated with a combination of either of the following: levobunolol hydrochloride, apraclonidine

hydrochloride, dorzolamide hydrochloride, methazolamide, or acetazolamide. All African American children were screened for sickle cell hemoglobinopathy, using a sickle hemoglobin solubility test at The Johns Hopkins Hospital.²⁸ Hemoglobin electrophoresis was then used to differentiate between sickle cell trait and other sickle cell hemoglobinopathies.

The hyphema size was graded according to the percentage of the anterior chamber filled with blood.¹ A grade 1 hyphema occupied less than one third of the anterior chamber; grade 2, greater than one third but less than one half of the anterior chamber; and grade 3, one half or more of the anterior chamber. A grade 4 hyphema was a complete blood clot ("8-ball hyphema") in the anterior chamber. A *secondary hemorrhage* was defined as an increase in the measured quantity of layered blood in the anterior chamber on slitlamp examination during follow-up. In the setting of a grade 4 hyphema, a secondary hemorrhage was defined as the appearance of fresh blood over old clots in the anterior chamber. An *IOP elevation* was defined as an IOP greater than 21 mm Hg that developed during inpatient follow-up and resulted in the addition of pressure-lowering agents to the preexisting medical regimen.

Associations between the incidence of a secondary hemorrhage and potential risk factors were examined using χ^2 , Fisher exact, and *t* tests, where appropriate. $P \leq .05$ was considered to be statistically significant. Separate multivariate logistic regressions were used to explore the risk factors for secondary hemorrhage and for IOP elevation. Odds ratios and 95% confidence intervals were calculated. The results of 3 separate sets of analyses are reported. In the first set, all patients with sickle cell hemoglobinopathy (sickle cell trait or disease) were compared with those with a normal hemoglobin level. Because there was only 1 patient in the study with sickle cell disease, a second set of analyses was then performed in which this patient was excluded. The third set of analyses was restricted solely to African American patients, and those with sickle cell hemoglobinopathy were compared with those with a normal hemoglobin level.

1997. Seven patients with delayed presentation to the hospital, 6 with open-globe injuries, 5 with preexisting pathological features in the affected eye, 2 referred by outside ophthalmologists for management, and 1 with a microhyphema were excluded. Only 1 patient had documented use of aspirin within 2 weeks of admission and was excluded from the study. A total of 40 patients fulfilled the inclusion criteria: 20 (50%) were African American; 1 (2%), Asian American; and 19 (47%), white. Of the 20 African American children, 5 (25%) had sickle cell trait and 1 (5%) had sickle cell disease (**Figure 1**). There were 36 male and 4 female patients (mean age, 10.6 years; range, 2.5-18.0 years). Thirty-one patients were admitted within 24 hours of sustaining injury to the eye, and 9 (6 African Americans and 3 whites) were admitted more than 24 hours after the initial injury.

Thirty-four (85%) of the eyes had a grade 1 hyphema; 1 (2.5%), grade 2; 1 (2.5%), grade 3; and 4 (10%), grade 4. The overall secondary hemorrhage rate was 10.3% (4/39). One patient with sickle cell trait developed a secondary hemorrhage 3 days following an anterior cham-

ber washout for a grade 4 hyphema. Because the surgical intervention may have contributed to the rebleed, this patient was excluded from the analyses of the rate and risk factors for secondary hemorrhage. Sex and age were not found to be statistically associated with the incidence of rebleeding ($P = .36$ and $P = .91$, respectively). All of the rebleeds occurred in patients with a grade 1 hyphema and within 5 days of the initial injury. Of the 3 patients who were treated with oral corticosteroids, 1 developed a secondary hemorrhage. This patient, who did not have sickle cell hemoglobinopathy, was switched to treatment with aminocaproic acid after developing the rebleed on hospital day 1, but subsequently developed another rebleed on hospital day 5. Of the 37 patients treated with aminocaproic acid, 4 developed a secondary hemorrhage.

The rate of secondary hemorrhage was statistically higher in the African American population (4 [21%] of 19) compared with the non-African American population (0 [0%] of 20) ($P = .05$) (**Figure 2A**). Of the 4 African American patients who rebled, 1 had sickle cell dis-

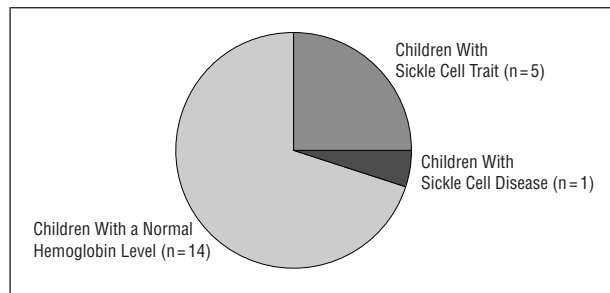


Figure 1. Of the 20 African American children, 1 (5%) had sickle cell disease, 5 (25%) had sickle cell trait, and the remainder (14 [70%]) had a normal hemoglobin level.

ease and 3 did not have sickle cell hemoglobinopathy (**Table 1**). None of the 4 patients with sickle cell trait developed a rebleed. The rate of secondary hemorrhage among patients with sickle cell hemoglobinopathy (1 [20%] of 5) compared with all those without sickle cell hemoglobinopathy (3 [9%] of 34) was not statistically significant ($P=.44$) (Figure 2B). Furthermore, when the analysis was restricted to only African American patients, there was no statistical difference between the rate of secondary hemorrhage among African American patients with sickle cell hemoglobinopathy (1 [20%] of 5) compared with those without it (3 [21%] of 14) ($P>.99$) (Figure 2C). Of the 14 African American children who were seen within 24 hours of sustaining injury to the eye, 2 patients, 1 of whom had sickle cell disease, developed a secondary hemorrhage. In comparison, 2 of the 6 African American patients who sought medical attention more than 24 hours after their injury developed a secondary hemorrhage. When excluding the 1 patient with sickle cell disease from analysis, a delay of more than 24 hours to treatment did not result in a statistically significant increase in the rate of rebleeding (2 [33%] of 6 vs 1 [8%] of 13; $P=.11$). Within the white population, no secondary hemorrhages (0/3) developed in the group of patients who were seen more than 24 hours after the initial injury.

The difference in mean (\pm SEM) IOP at presentation between patients with sickle cell trait (43 ± 9 mm Hg) and patients without sickle cell trait (20 ± 1 mm Hg) approached statistical significance ($P=.06$) (**Figure 3A**). When the patient with sickle cell disease was included in the analysis, the mean (\pm SEM) IOP of patients with sickle cell hemoglobinopathy on admission remained 43 ± 7 mm Hg (Figure 3B). With this additional patient, the IOP at presentation of patients with sickle cell hemoglobinopathy was statistically higher than that of those without it ($P=.03$). In the analysis restricted solely to African American patients, those with sickle cell hemoglobinopathy had a statistically higher IOP at presentation than African American patients with a normal hemoglobin level (43 ± 7 vs 20 ± 2 mm Hg; $P=.03$) (Figure 3C).

An initially higher IOP at presentation was not, however, found to be associated with a higher rate of secondary hemorrhage in the analysis comparing patients with and without sickle cell hemoglobinopathy ($P=.13$). Similarly, when the analysis was restricted to only African American patients, there was

still no statistically increased risk for secondary hemorrhage in patients who were initially seen with a higher IOP ($P=.40$).

The rate of IOP elevation in the eyes of subjects with sickle cell trait (4 [80%] of 5) was significantly higher than that in eyes of patients without sickle cell trait (5 [15%] of 34) ($P=.006$) (**Figure 4A**). The 1 patient with sickle cell disease had an elevated IOP on admission but did not develop further episodes of IOP elevation during hospitalization. When this patient was included in the analysis, the rate of IOP elevation for patients with sickle cell hemoglobinopathy decreased to 67% (4/6) (Figure 4B). The association between sickle cell hemoglobinopathy and IOP elevation remained significant ($P=.02$). When the analysis was restricted to African American patients, the difference in the rate of IOP elevation between patients with and without sickle cell hemoglobinopathy approached statistical significance (4 [67%] of 6 vs 3 [21%] of 14; $P=.05$) (Figure 4C).

Using a multivariate logistic regression analysis, the association between sickle cell hemoglobinopathy and secondary hemorrhage was not found to be statistically significant ($P>.05$). Patients with sickle cell hemoglobinopathy, however, were approximately 8 times more likely to have developed an IOP elevation (odds ratio, 8.33; 95% confidence interval, 1.03-66.67), even after controlling for the development of secondary hemorrhage. However, when the analysis was restricted to African American patients, this association was no longer statistically significant ($P>.05$) (odds ratio, 6.21; 95% confidence interval, 0.62-62.50).

Three patients, all with a sickle cell hemoglobinopathy, required surgical intervention. Two patients, 1 with sickle cell disease and the other with sickle cell trait, underwent an anterior chamber washout following the development of a secondary hemorrhage. The third subject with sickle cell trait underwent an anterior chamber washout for a grade 4 hyphema and an elevated IOP. This patient subsequently developed a secondary hemorrhage 3 days following the procedure. The association between sickle cell hemoglobinopathy and surgical intervention approached statistical significance ($P=.05$). Race, initial hyphema size, initial visual acuity, initial IOP, and subsequent elevation of IOP were not associated with an increased risk of surgical intervention ($P=.23$, $P=.39$, $P=.20$, $P=.31$, and $P=.12$, respectively).

On admission, African American patients had a visual acuity of 20/40 or better in 9 (45%) of 20 patients compared with 8 (40%) of 20 non-African American patients. Of the African American patients with sickle cell hemoglobinopathy, 2 (33%) of 6 had a visual acuity of 20/40 or better compared with 7 (50%) of 14 African American patients without sickle cell hemoglobinopathy. On discharge from the hospital, the visual acuity was 20/40 or better in 13 (65%) of 20 patients in the African American and the non-African American groups. Of African American patients with sickle cell hemoglobinopathy, 3 (50%) of 6 had a visual acuity of 20/40 or better compared with 10 (71%) of 14 African American patients without sickle cell hemoglobinopathy. Because many patients were lost to follow-up (15 at the 1-week

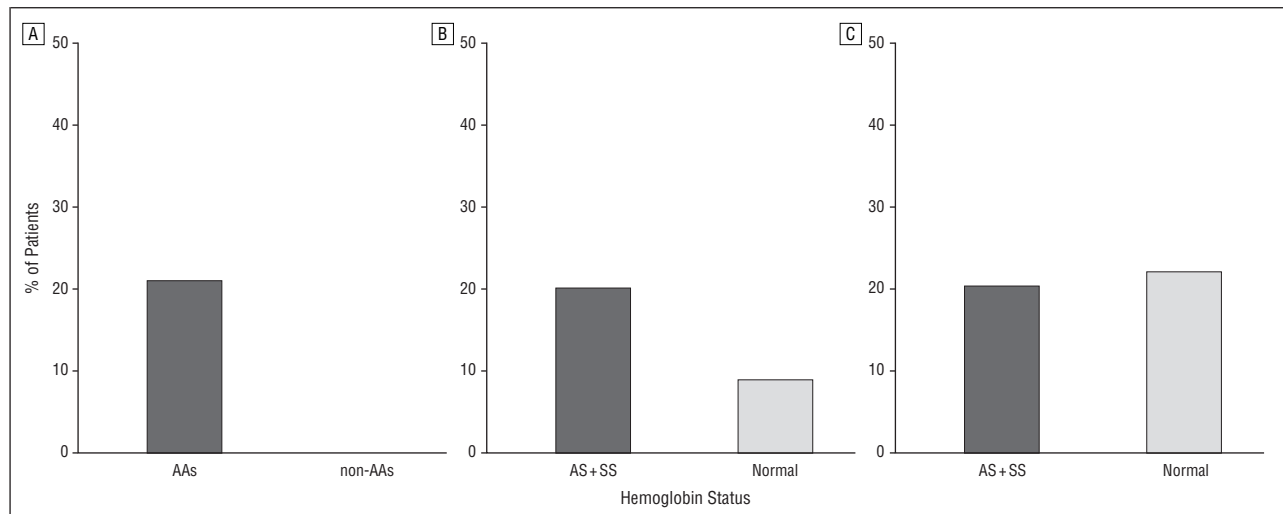


Figure 2. A, The rate of secondary hemorrhage was statistically higher in the African American (AA) population compared with the non-AA population (4 [21%] of 19 vs 0 [0%] of 20; $P = .05$). B, The rate of secondary hemorrhage among patients with sickle cell hemoglobinopathy (AS+SS) compared with those without AS+SS was not statistically significantly different (1 [20%] of 5 vs 3 [9%] of 34; $P = .44$). C, When the analysis was restricted to only AA patients, the rate of secondary hemorrhage among AA patients with AS+SS and without AS+SS was not statistically significantly different (1 [20%] of 5 vs 3 [21%] of 14; $P > .99$).

Table 1. Patients With Secondary Hemorrhage*

Variable	Patient				
	1	2†	3	4	5
Age, y	12	5	18	6	8
Sickle cell status	SS	AS	AA	AA	AA
Sex	F	M	M	M	M
Eye	OD	OD	OS	OS	OD
Mechanism of injury	Egg	Fist	Fist	Corn cob	Shovel
Initial VA	LP	LP	20/50	20/30	20/25
Initial IOP, mm Hg	40	60	17	10	12
Initial hyphema grade	1	4	1	1	1
Subsequent IOP elevation‡	No	Yes	Yes	Yes	No
Time after injury of rebleed, d	2	5	2	1 and 5	1
Grade of rebleed	3	1	3	1	1
Intervention (days after injury)	AC washout and ECCE (7)	AC washout (2)	AC washout (9)	None	None
VA on discharge	2/200	CF at 120 cm	20/60	20/30	20/25
Follow-up, mo	>36	>36	None	None	<1
Best-corrected VA at follow-up	20/150	20/70	NA	NA	20/25

*All patients were African American. SS indicates sickle cell disease; AS, sickle cell trait; AA, normal hemoglobin level; VA, visual acuity; LP, light perception; IOP, intraocular pressure; AC, anterior chamber; ECCE, extracapsular cataract extraction; CF, counting fingers; and NA, not available.

†This patient was not included in the analyses of rate and risk factors for secondary hemorrhage because the rebleed occurred after an anterior chamber washout.

‡Defined as an IOP greater than 21 mm Hg that developed during inpatient follow-up and resulted in the addition of pressure-lowering agents to the preexisting medical regimen.

follow-up and 27 at the 4-week follow-up), no meaningful analysis of long-term visual acuity could be performed. However, no cases of corneal blood staining, optic atrophy, or vascular occlusions developed during the follow-up that was recorded.

COMMENT

The presence of a sickle cell hemoglobinopathy in a patient with traumatic hyphema is associated with an increased risk of ocular complications.²⁴⁻²⁶ Within the stagnant environment of a hyphema, the erythrocytes in these eyes are predisposed to sickling and to clogging the tra-

becular outflow tract. The resultant IOP elevation leads to hypoperfusion and hypoxia of the anterior and posterior segments of the eye, thereby perpetuating a cycle in which further sickling and sludging of erythrocytes occur. The end result is that these patients are more likely to develop central retinal artery occlusion or optic nerve damage at only modest elevations in IOP compared with patients with normal erythrocytes.

Relatively few studies^{8,12,19,21,29} have examined the risk of secondary hemorrhage in patients with traumatic hyphema and sickle cell hemoglobinopathy. Reported rates of secondary hemorrhage in patients with sickle cell hemoglobinopathy range from 0% to 80% (**Table 2**). It re-

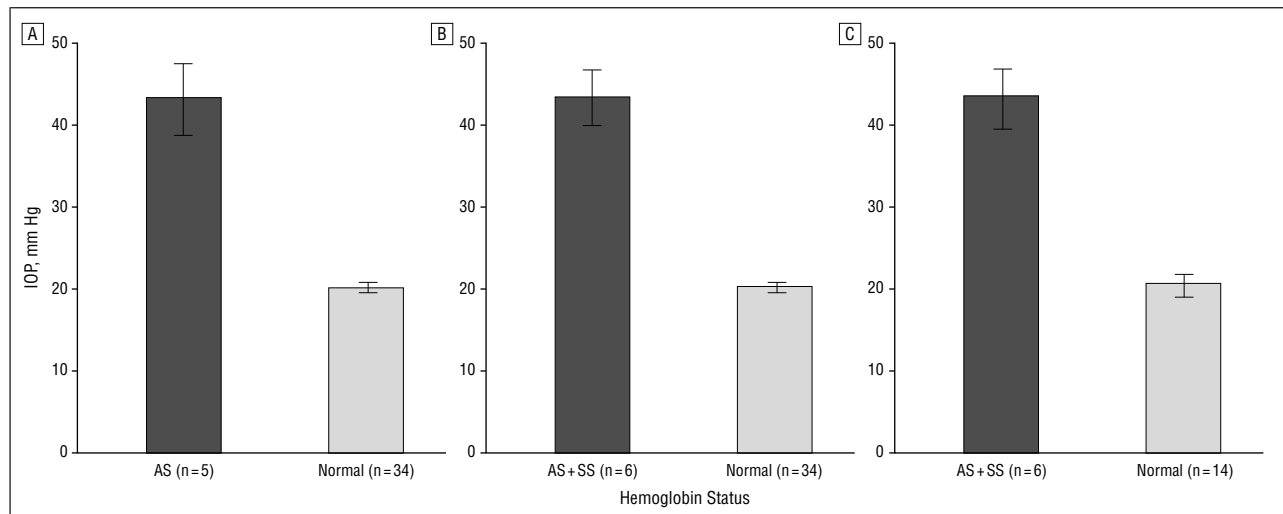


Figure 3. A, The difference in mean (\pm SEM) intraocular pressure (IOP) at presentation between patients with sickle cell trait (AS) and without AS approached statistical significance (43 ± 9 vs 20 ± 1 mm Hg; $P = .06$) (the patient with sickle cell disease was excluded from this analysis). B, When the patient with sickle cell disease was included in the analysis, the difference in mean IOP at presentation between patients with sickle cell hemoglobinopathy (AS+SS) and without AS+SS was statistically significant (43 ± 7 vs 20 ± 1 mm Hg; $P = .03$). C, In the analysis restricted solely to African American patients, those with AS+SS had a statistically higher IOP at presentation than those with a normal hemoglobin level (43 ± 7 vs 20 ± 2 mm Hg; $P = .03$).

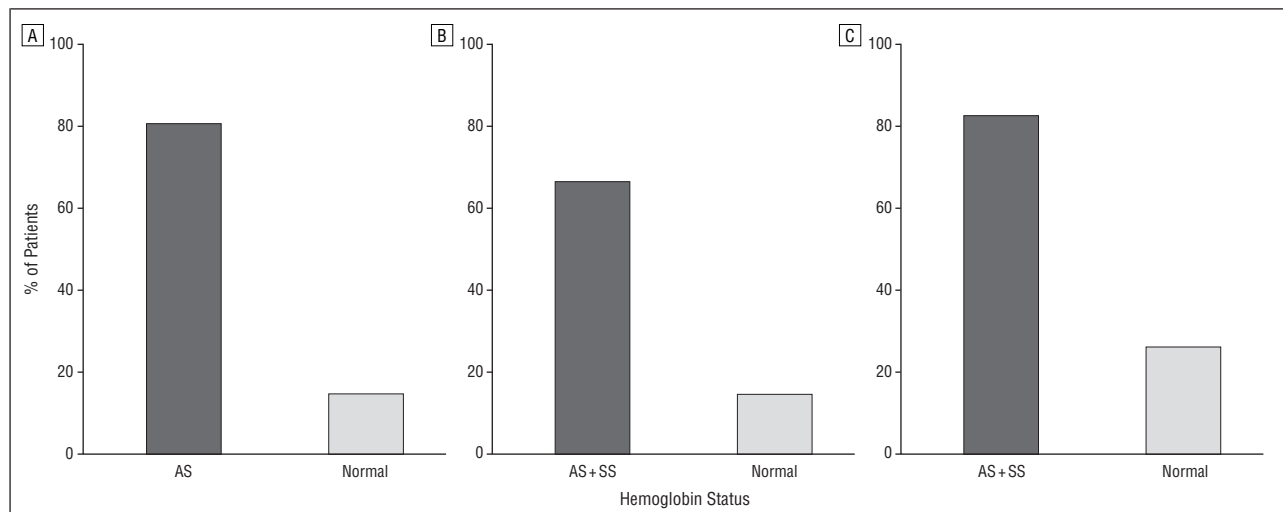


Figure 4. A, The rate of intraocular pressure (IOP) elevation in subjects with sickle cell trait (AS) was significantly higher than that in subjects without AS (4 [80%] of 5 vs 5 [15%] of 34; $P = .006$). B, The rate of IOP elevation for patients with sickle cell hemoglobinopathy (AS+SS) was significantly higher than that for patients with a normal hemoglobin level (4 [67%] of 6 vs 5 [15%] of 34; $P = .02$). C, When the analysis was restricted to African American patients, the difference in the rate of IOP elevation between patients with and without AS+SS approached statistical significance (4 [67%] of 6 vs 3 [21%] of 14; $P = .05$).

remains unclear whether the presence of a sickle cell hemoglobinopathy increases the likelihood of secondary hemorrhage.

Our study retrospectively reviewed the inpatient records of all children admitted to the Wilmer Ophthalmological Institute from July 1990 through December 1997. This 7½-year period was chosen because the department's databases were computerized beginning in 1990, and because pediatric patients with traumatic hyphema were routinely admitted for treatment during this period. Our study population consisted of an equal distribution of African American and non-African American patients. The overall rate of secondary hemorrhage in our study was 10.3%. The rate of rebleeding in African Americans in our study was significantly higher than that of non-African Americans (21% vs 0%); ($P = .047$).

This finding is consistent with other reports^{4,7,8,21,23,29} that have found a higher rate of rebleeding in the African American population (**Table 3**). It is unclear what accounts for this racial difference in the rate of secondary hemorrhage. One hypothesis is that confounding socioeconomic issues, such as increased severity of disease or delay in seeking medical attention, may increase the likelihood of recurrent bleeding in the African American population.^{4,8,23} Other reports^{4,23} have postulated that there may simply be a predisposition of unknown type, possibly enhanced uveal inflammation and vascular congestion, for African American individuals to develop a rebleed.

Whether the increased frequency of sickle cell hemoglobinopathy in African American individuals contributes to their propensity for developing a secondary

hemorrhage has been unclear. Our data suggest that sickle cell hemoglobinopathy may not increase the risk of secondary hemorrhage. In this study, the rate of rebleeding among patients with sickle cell hemoglobinopathy was 20% (1/5) and the rate of rebleeding in those without sickle cell hemoglobinopathy was 9% (3/34). This was not a significant difference ($P=.44$). Furthermore, when the analysis was restricted to African American patients, those with sickle cell hemoglobinopathy (1 [20%] of 5) did not have a statistically higher rate of rebleeding compared with those with a normal hemoglobin level (3 [21%] of 14) ($P>.99$). The lack of a significant association may have been the result of the sample size not being statistically powerful enough to detect small differences among groups of patients (power=9% when the patient with sickle cell disease was excluded, and power=25% when the patient with sickle cell disease was included). Additional studies with larger sample sizes may clarify the possible relation between sickle cell hemoglobinopathy and the development of secondary hemorrhage. However, because the difference in the rate of secondary hemorrhage between African American patients with and without sickle cell hemoglobinopathy is so small, it is unlikely that a clinically significant difference in the rates would exist even with larger sample sizes.

Table 2. Rate of Secondary Hemorrhage in African American Patients With Sickle Cell Hemoglobinopathy*

Source, y	Patients Without Sickle Cell Hemoglobinopathy	Patients With Sickle Cell Hemoglobinopathy
Cassel et al, ¹⁹ 1985	Not available	0 (. . .)
Spoor et al, ⁸ 1990†	24 (43/178)	0 (0/4)
Crouch and Frenkel, ¹² 1976‡	6 (2/31)	37 (3/8)
Nasrullah and Kerr, ²¹ 1997	0 (0/58)	64 (9/14)
Crouch et al, ²⁹ 1997‡	23 (6/26)	80 (4/5)
Present study†§	21 (3/14)	20 (1/5)

*Data are given as the percentage (number/total) of patients in whom a secondary hemorrhage occurred. Ellipses indicate data not provided.
 †Includes patients treated systemically with oral aminocaproic acid.
 ‡Placebo group only.
 §Difference between rates is not statistically significant ($P = .44$).

Our study did confirm the finding in previous reports²⁴⁻²⁶ that patients with a sickle cell hemoglobinopathy were at a higher risk for developing an elevation in IOP. The mean IOP at initial examination of patients with sickle cell hemoglobinopathy was significantly higher than that of patients without hemoglobinopathy (43 ± 7 vs 20 ± 1 mm Hg; $P=.03$). Furthermore, the subsequent rate of IOP elevation during hospitalization was significantly higher in patients with a sickle cell hemoglobinopathy (66% vs 15%; $P=.02$). Of 5 patients with sickle cell trait, 4 developed an IOP elevation during hospitalization. The 1 patient with sickle cell disease initially had an IOP of 40 mm Hg and was immediately prescribed an aggressive pressure-lowering regimen, which probably prevented any subsequent IOP elevation. The increased risk for the development of an IOP elevation in patients with sickle cell hemoglobinopathy is likely due to the clogging of the trabecular meshwork by sickled erythrocytes.^{24,25} Unlike other studies,^{6,22} however, a higher IOP at presentation was not associated with an increased risk of secondary hemorrhage in our study.

The identification of patient characteristics predictive for secondary hemorrhage may assist in guiding management decisions in the treatment of patients with traumatic hyphema. In this study, we found that African American patients are at a higher risk of developing a rebleed. These patients may deserve closer clinical scrutiny than non-African American patients. Based on evidence that non-African American patients are less likely to rebleed and, therefore, less likely to benefit from aminocaproic acid treatment, other studies^{15,17,30} have recommended that aminocaproic acid treatment be reserved only for patients at greater risk for secondary hemorrhage. In our study, the decision to initiate oral aminocaproic acid treatment was not influenced by the patient's race. However, based on the results of this and previous studies,^{15,17,30} we recommend that aminocaproic acid treatment be based on individual patient characteristics, including race.

We have also found that patients with sickle cell hemoglobinopathy are more likely to have a higher IOP and to develop subsequent elevations in IOP. It is unclear whether this translates into an increased risk of secondary hemorrhage, but our data do not support this rela-

Table 3. Increased Rate of Secondary Hemorrhage in African American Patients

Source, y	Study Population That Was African American, %	Secondary Hemorrhage*	
		African American Patients	Non-African American Patients
Wittman et al, ⁷ 1985	0.8	67 (2/3)	3 (11/368)
Skalka, ⁴ 1978	25	38 (19/50)	8 (15/200)
Palmer et al, ²³ 1986†	52	20 (6/30)	0 (0/28)
Crouch et al, ²⁹ 1997‡	57	32 (10/31)	9 (2/23)
Nasrullah and Kerr, ²¹ 1997	72	12 (9/72)	0 (0/58)
Spoor et al, ⁸ 1990§	74	24 (43/178)	5 (3/63)
Present study§	50	21 (4/19)	0 (0/20)

*Data are given as the percentage (number/total) of patients.
 †All patients were treated with oral aminocaproic acid.
 ‡Placebo group only.
 §Includes patients treated systemically with oral aminocaproic acid.
 ||Difference between rates is statistically significant ($P = .05$).

tion. Until this question is resolved, we recommend that patients with sickle cell hemoglobinopathy who have traumatic hyphema continue to receive aggressive management to lower their IOP, because they are at greater risk for developing other associated ocular complications such as IOP elevations, central retinal artery occlusions, and optic neuropathies, as demonstrated by this and previous studies.²⁴⁻²⁶

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