

# Characteristics and Functional Outcome of Traumatic Hyphema without Routine Administration of $\epsilon$ -Aminocaproic Acid

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## Abstract

**Background:** The prevalence of traumatic hyphema as well as the distribution of its severity varies among different patient populations. Treatment recommendations in the literature differ significantly among various published reports. This lack of a uniformly accepted treatment probably reflects the different characteristics of this pathology among the populations investigated and calls for a population-adjusted treatment recommendation.

**Objectives:** To report the characteristics and functional outcome of patients with traumatic hyphema and to discuss possible recommendations regarding the use of  $\epsilon$ -aminocaproic acid.

**Methods:** A prospective, non-randomized study was conducted in 154 consecutive patients with traumatic hyphema, including data collection of ophthalmic status at various time points, the presence or absence of secondary hemorrhage, and final visual acuity.

**Results:** Of the 154 eyes studied over 3 years, nearly 90% had hyphema of grade 1 or less, 5 (3.25%) experienced rebleeding, and 2 (1.3%) – neither of which rebled – needed surgical intervention. None of the four patients who experienced final visual acuity of 6/40 or less suffered rebleeding.

**Conclusion:** The use of  $\epsilon$ -aminocaproic acid in the studied population was unjustified and routine use of  $\epsilon$ -aminocaproic acid in our patient population is probably not indicated. A treatment policy regarding  $\epsilon$ -aminocaproic acid use should be adjusted to the population being treated.

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The prevalence of traumatic hyphema has been estimated at 17–20 per 100,000 per year [1,2]. The ophthalmic insult in cases of traumatic hyphema depends on the severity of the hyphema itself as well as on additional damage caused to other eye organs during the trauma. Hyphema severity can be defined by five stages: a) microscopic, b) stage 1: less than one-third the volume of the anterior chamber, c) stage 2: one-third to one-half the volume of the anterior chamber, d) stage 3: more than half and less than total hyphema, and e) stage 4: total hyphema.

The protocol of treating traumatic hyphema patients is not uniformly accepted. Different recommendations are presented in the literature, and no single agreed-upon policy exists. Restricted activity for 1–2 weeks is advised, but rest has not proved to be of benefit [2,3]. Hospitalization is accepted in some medical centers but has not been proven to influence prognosis [2]. Other means include raising the head of the bed by 30°, a metal cover for the eye, the use of acetaminophen, restriction of aspirin, and administration of atropine and prednisolone in eyedrops.

$\epsilon$ -Aminocaproic acid has been recommended as an efficient treatment for preventing secondary hemorrhage in patients with traumatic hyphema [4–8]. This recommendation was based on

studies in which the prevalence of secondary hemorrhage diminished following the use of  $\epsilon$ -aminocaproic acid. In the studied populations the prevalence of secondary hemorrhage was 25–33% without treatment, compared with 2–3% in the treated groups [4–8].

The present study aimed at determining the prevalence of secondary hemorrhage following traumatic hyphema, the prognosis after such an event, and an estimation of whether the use of  $\epsilon$ -aminocaproic acid was justified in our patient population.

## Patients and Methods

Our study group comprised all patients diagnosed as suffering from traumatic hyphema who were hospitalized in our department over a 3 year period. At that time our department's policy included hospitalization for each traumatic hyphema. We collected data regarding the ophthalmic status upon admission, at discharge, and at follow-up after 1 year; the presence or absence of secondary hemorrhage; and final visual acuity (as a marker of ophthalmic prognosis); as well as demographic data and data regarding other complications.

## Results

Altogether, 154 eyes of 154 patients were studied. Of these patients 40.2% were 11–20 years old and 79.2% were under age 30.

Table 1 summarizes the distribution of patients by severity of hyphema. Nearly 90% of the patients had hyphema of grade 1 or less. Secondary hemorrhage was observed in five patients (3.25%). Table 2 lists these patients with regard to the severity of their initial

**Table 1.** Distribution of patients by severity of hyphema

Grade of hyphema	No. of patients	Percentage
Microscopic	73	47.4
1	63	40.9
2	10	6.5
3	4	2.6
4	4	2.6
Total	154	100.0

**Table 2.** Grade of initial and secondary hemorrhage and final visual acuity in patients with secondary hemorrhage

Patient no.	Severity grade of initial hemorrhage	Severity grade of secondary hemorrhage	Final visual acuity
1	1	1	6/9
2	3	4	6/10
3	1	2	6/7.5
4	1	2	6/7.5
5	1	1	6/7.5

and secondary hyphema and their final visual acuity at the 1 year follow-up visit.

In the study population, only two patients (1.3%) needed surgical intervention, both of whom had grade 4 hyphema at admission and did not rebleed. None of the patients with hyphema of grade 3 or less needed surgery, including those with secondary hemorrhage. Only four patients had final visual acuity of 6/40 or less, all of whom had retinohoroidal damage accounting for this loss of visual acuity. One of the latter had only microscopic hyphema (with choroidal tear and anterior vitreous face rupture), two had grade 1 hyphema (one with choroidal tear and foveal hemorrhage and the other with severe macular edema and later scar formation), and one had grade 4 hyphema (with subfoveal choroidal tear).

## Discussion

$\epsilon$ -Aminocaproic acid has been found in several controlled studies to be efficient in the prevention of secondary hemorrhage following traumatic hyphema [4–8]. Prevalent side effects of treatment with  $\epsilon$ -aminocaproic acid include nausea, vomiting, and hypotension. Less prevalent side effects are tinnitus, numbness, skin rash, myalgia, and hematuria. Up to 50% of patients treated with  $\epsilon$ -aminocaproic acid were found to suffer from one or more of these side effects [1,2,5,9,10]. In some cases the side effects are severe enough to necessitate cessation of treatment [11]. Discontinuation of treatment before completion of a 5 day course is associated with increased prevalence of secondary hemorrhage [6].

The estimated risk for secondary hemorrhage in the populations serving the controlled studies, upon which the recommendation for  $\epsilon$ -aminocaproic acid use was based, varied between 3.5 and 38% [12,13]. The highest prevalence was found in the non-white population in the United States [2,14,15]. A high severity score (stage 4) was found in 25% of the patients in the populations on which the recommendation for  $\epsilon$ -aminocaproic acid was based [5,16].

The small numbers of patients with secondary hemorrhage reported in the literature have made the establishment of predictive factors for such an event impossible [8]. For this reason, a policy of  $\epsilon$ -aminocaproic acid use should be established for the entire patient population uniformly.

Witteman [17] described 400 white patients from a rural area with similar ethnic origin and found a rebleeding rate of 3.8%. In his report nearly 90% of the patients with rebleeding had their original hyphema graded as 3–4. Based on these data, Witteman suggested that  $\epsilon$ -aminocaproic acid should be limited in routine use to grades 3 and 4 only.

In four of five patients with rebleeding in our series, the original trauma was graded 1 in severity; in only one was the original bleeding graded as 3. Based on this distribution of the “rebleeders,” we believe that the correct recommendation in our population is not to treat with  $\epsilon$ -aminocaproic acid, regardless of the severity rank of the original hyphema. Our data strongly support the conclusion of the above-mentioned report [17] – namely, that the recommendation for using  $\epsilon$ -aminocaproic acid

to treat traumatic hyphema should be adjusted to the specific population [17].

In view of the low incidence of secondary hemorrhage and the final visual outcome of the few patients with this complication on the one hand, and the high prevalence of side effects of the treatment on the other, we believe that treatment with  $\epsilon$ -aminocaproic acid was not indicated in our population. The fact that discontinuation of the treatment might have been indicated because of side effects exposing the patient to higher risk for secondary hemorrhage, along with the very high cost of the treatment, further strengthens this conclusion.

We recommend that the decision regarding the use of  $\epsilon$ -aminocaproic acid for traumatic hyphema should be made by every ophthalmic authority based on the specific characteristics of this event in the relevant population.

## References

1. Agapitos PJ, Noel L-P, Clarke WN. Traumatic hyphema in children. *Ophthalmology* 1987;94:1238–41.
2. Kennedy RH, Brubaker RF. Traumatic hyphema in a defined population. *Am J Ophthalmol* 1988;106:123–30.
3. Wright KW, Sunalp M, Urrea P. Bed rest versus activity ad lib in the treatment of small hyphemas. *Ann Ophthalmol* 1988;20:143–5.
4. Hill K. Cryoextraction of total hyphema. *Arch Ophthalmol* 1968;80:368–70.
5. Crouch ER Jr, Frenkel M. Aminocaproic acid in the treatment of traumatic hyphema. *Am J Ophthalmol* 1976;81:355–60.
6. McGetrick JJ, Jampol LM, Goldberg MF, Frenkel M, Fiscella RG. Aminocaproic acid decreases secondary hemorrhage after traumatic hyphema. *Arch Ophthalmol* 1983;101:1031–3.
7. Palmer DJ, Goldberg MF, Frenkel M, Fiscella R, Anderson RJ. A comparison of two dose regimens of epsilon aminocaproic acid in the prevention and management of secondary traumatic hyphemas. *Ophthalmology* 1986;93:102–8.
8. Wilson TW, Jeffers JB, Nelson LB. Aminocaproic acid prophylaxis in traumatic hyphema. *Ophthalmic Surg* 1990;21:807–9.
9. Varnek L, Dalsgaard C, Hansen A, Klie F. The effect of tranexamic acid on secondary haemorrhage after traumatic hyphaema. *Acta Ophthalmol (Copenh)* 1980;58:787–93.
10. Goldberg MF. Antifibrinolytic agents in the management of traumatic hyphema [Editorial]. *Arch Ophthalmol* 1983;101:1029–30.
11. Kutner B, Fourman S, Brein K, et al. Aminocaproic acid reduces the risk of secondary hemorrhage in patients with traumatic hyphema. *Arch Ophthalmol* 1987;105:206–8.
12. Kearns P. Traumatic hyphaema: a retrospective study of 314 cases. *Br J Ophthalmol* 1991;75:137–41.
13. Volpe NJ, Larrison WI, Hersh PS, Kim T, Shingleton BJ. Secondary hemorrhage in traumatic hyphema. *Am J Ophthalmol* 1991;112:507–13.
14. Cassel GH, Jeffers JB, Jaeger EA. Wills Eye Hospital traumatic hyphema study. *Ophthalmic Surg* 1985;16:441–3.
15. Thomas MA, Parish RK II, Feuer WJ. Rebleeding after traumatic hyphema. *Arch Ophthalmol* 1986;104:206–10.
16. Marcus M, Biedner B, Lifshitz T, Yassur Y. Aspirin and secondary bleeding after traumatic hyphema. *Ann Ophthalmol* 1988;20:157–8.
17. Witteman GJ. Traumatic hyphema [Letter]. *Arch Ophthalmol* 1984;102:356–8.

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