Long-term outcome of primary acute angle-closure glaucoma

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SUMMARY Factors influencing the long-term cure of 116 consecutive cases of acute angle-closure glaucoma, with at least one year's follow-up were examined. Delay in presentation and the time needed to terminate the attack were found to have a detrimental effect on the final outcome, while the height of the intraocular pressure at the time of the attack was found to be unimportant in predicting the long-term prognosis of the disease.

It is not clear whether primary acute angle-closure glaucoma (AACG) has a favourable outcome when iridectomy is performed during the acute stage of the disease. Factors like duration of the attack have not been thoroughly investigated. A late increase in the intraocular pressure (IOP) after an apparently successful terminated attack of AACG by iridectomy has been reported.¹⁻⁵ The rate of this delayed rise in IOP is reported to be between 19%³ and 24%.² The reason for it has not been elucidated. Some authors have blamed the surgical procedure of peripheral iridectomy,^{1,3} while others could not report on any positive correlation between various factors and the late outcome.^{4,6}

The almost unique arrangement by which our department and institute render ophthalmological services exclusively to a large population offers an excellent opportunity for long-term studies. Taking advantage of this we analysed the follow-up records over an 11-year period of all patients treated for AACG who had been followed up for at least one year after the attack, and we attempted to analyse the influence of factors prior to surgery on the late outcome of AACG.

Materials and Methods

The files of 116 consecutive patients treated in our department for AACG between 1 January 1972 and 31 December 1982 were retrieved and the following data recorded: (1) the time which elapsed between

the onset of the AACG attack and the diagnosis was made and treatment started; (2) the IOP reading at the time of the diagnosis; (3) attack terminated (or not) by conventional medical therapy; (4) how long after beginning treatment the attack was terminated; (5) final outcome one year or later after the attack, namely, 'cured' with no additional medical treatment; 'not cured' needing continuous medical therapy, and/or having glaucomatous damage, and/or needing further glaucoma surgery.

All data were fed into the computer and the relationship between the various parameters analysed. Statistical significance was tested by the χ^2 method.

In this study we examined the final outcome of each case and related it to three preoperative data, namely, delay in presentation, height of the IOP during the attack, and the time needed to terminate the attack by conventional medical therapy.

Results

Delay in presentation. For 94 patients who were followed up for at least one year information was available on the time elapsing between the onset of symptoms and start of treatment. Table 1 presents

Table 1 Relationship between presentation time and final outcome

	Cured	Not cured
<24 h	45	7
≥24 h	20	22

$\chi^2=16.49$; $p<0.001$.

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Table 2 Effect of the height of IOP at presentation on the final outcome of AACG

IOP at presentation	Total	Final outcome			
		Cured		Not cured	
		n	%	n	%
≤40 mmHg	16	10	62.5	6	37.5
>40 mmHg	81	58	71.6	23	29.4

$\chi^2=0.53$; $p>0.5$.

the correlation between delay in presentation and the final outcome. Patients with less than 24 hours' delay in presentation had a more favourable outcome than those with 24 hours' delay or more. This difference between the two groups was found to be statistically significant ($p<0.001$).

Height of the IOP during the AACG attack. The height of the IOP when the patient first presented to the emergency room or to the referring ophthalmologist had practically no effect on the final outcome (Table 2).

Time needed to terminate the attack. This information was available for just over half of the cases. Although the numbers were small, a clear correlation was apparent. The longer it took to terminate the AACG attack, the worse the long-term prognosis. Six out of seven patients who could not be relieved of their acute attack with the usual medical treatment prior to iridectomy developed glaucomatous damage and/or needed filtration surgery.

Discussion

In our department a prophylactic iridectomy was performed on all fellow eyes (surgical in the first years and laser in later years), so we could evaluate the effect of a possible surgical insult on these eyes. None of these fellow eyes developed such a late rise of IOP, and therefore, when this occurs in the AACG eye, it cannot be attributed to the procedure of iridectomy but must be due to the disease process of AACG, and it is our belief that this is connected with events prior to surgery.

The importance of the delay in presentation as seen in Table 1 indicates that patients arriving, diagnosed, and treated within 24 hours less often have a late increase in IOP than those who present later. The fact that the height of the IOP during the

acute attack has no effect on the final outcome has been reported previously⁴ and was confirmed in this study.

Our results also indicate the importance of the duration of the attack, as expressed both by the delay in presentation and by the time elapsing between initiation of treatment and until the termination of the attack. The longer the time between presentation and termination of the attack, the higher is the risk for a future elevation of the IOP. Analysis of the data showed that most of the cases with delayed presentation were also relieved of their attack after a longer period of time. We therefore assume that the time factor plays an important role in the formation of peripheral anterior synechiae, iris atrophy with pigment deposits in the angle, etc, factors which later interfere with the normal angle filtration.

The effect of delay in presentation was previously investigated by Hillman,⁶ who did not find it to be related to the final IOP. Our study re-examined the effects of delay in presentation and the time needed to terminate the attack, and we found that this duration of the AACG attack certainly has a detrimental effect on the long term outcome of the affected eye.

Playfair and Watson⁴ reported that all the changes in their patients occurred within four months after the attack. Though we examined our results at least one year later, in retrospect we can state that a careful follow-up of 6–12 months is probably sufficient in those 70–75% of cases which will be trouble-free and may be discharged from follow-up, as they can be considered cured.

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