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Acute closed-angle glaucoma: an investigation into the effect of delay in treatment

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SUMMARY A retrospective study of 212 eyes with acute closed-angle glaucoma is reported. A peak incidence in the sixth decade was noted and an increased incidence in females confirmed statistically. A surprising and often marked delay occurred in the presentation of many patients for treatment, but visual outcome was not influenced by such delay. Despite good control of intraocular pressure, many of the eyes suffered visual loss from optic nerve damage, and the visual outcome was not related to the height of intraocular pressure at presentation. Damage to the visual system occurred very early in the disease, probably with the initial acute rise of intraocular pressure, and eyes appear to vary in their susceptibility to such an insult. It does not appear that earlier presentation of the patient with acute glaucoma would significantly improve the visual outcome in terms of visual acuity. The short critical time before damage occurs to the eye suggests a role for preventive ophthalmology in the detection and surgery of eyes at risk with shallow anterior chambers and narrow angles before they develop acute closed-angle glaucoma.

Acute glaucoma has long been known as a potentially blinding disease which was at first incurable, but treatment became possible just over a century ago with the introduction of iridectomy and miotics. The disease is regarded as an ophthalmological emergency, and the lowering of the intraocular pressure to physiological levels is considered a matter of urgency to minimise visual loss. Modern management involves initial medical control of the intraocular pressure with pilocarpine, acetazolamide, and osmotic diuretics followed by iridectomy or fistulising surgery when the pressure has been controlled. Several studies have confirmed a significant risk of acute glaucoma following in the fellow eye when treated conservatively (Bain, 1957; Lowe, 1962; Ghoshal and Blaxter, 1969), and so consideration is given to the need for prophylactic peripheral iridectomy in that eye also. In view of the severe pain and marked fall in vision which occurs in acute closed-angle glaucoma it is surprising for how long many patients tolerate the disease before presenting for treatment.

This study was undertaken to assess the effect of delay in patient presentation, to seek a critical time within which the disease should be controlled for best visual result, and to assess the results of the modern management of acute closed-angle glaucoma.

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Material and methods

This study was made retrospectively by review of the case notes of 191 patients who presented with acute closed-angle glaucoma in the years 1971–74 and who have been the subject of a previous study of aetiological factors (Hillman and Turner, 1977).

Results

Of the 191 patients included in the study 21 (11·0%) presented with bilateral acute glaucoma, giving a total of 212 eyes studied; 60 (31·4%) of the patients were male and 131 (68·6%) female, and their ages ranged from the fourth to the ninth decade (Fig. 1). In comparison with population statistics for 1971 a chi-squared analysis shows a statistically significant increased proportion of females ($P<0·001$). The number of days the patients tolerated the disease before presentation appears in Fig. 2, which shows that 30·9% presented on the first day and progressively smaller numbers on the succeeding days, with a second peak of presentation after a week.

The visual acuities of the eyes on presentation are compared with their final visual acuities in Fig. 3.

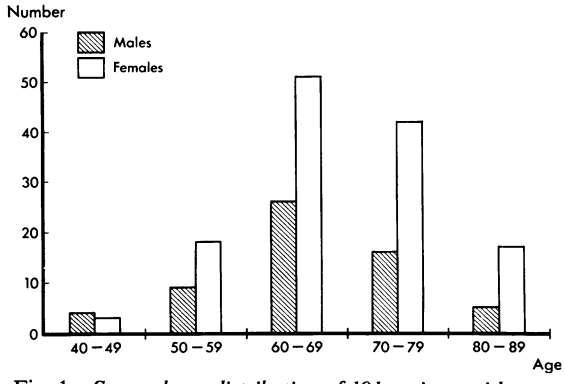

Fig. 1 *Sex and age distribution of 191 patients with acute closed-angle glaucoma*

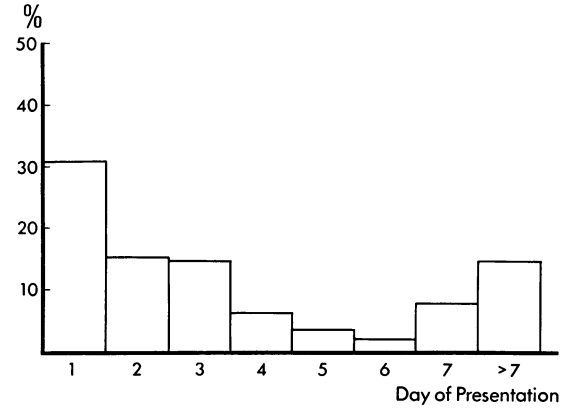

Fig. 2 *Delay in presentation by patients with acute closed-angle glaucoma*

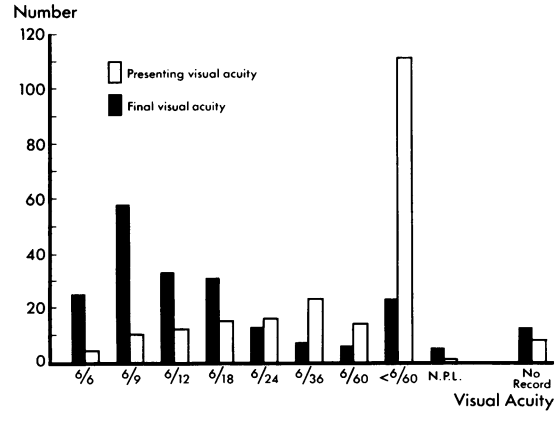

Fig. 3 *Presenting visual acuity compared with final visual acuity in eyes treated for acute closed-angle glaucoma*

which shows that 158 (74·5%) of these eyes regained '6/18 or better' vision, 127 (59·9%) regained '6/12 or better', and only 23 eyes (10·8%) achieved 6/6 vision. The response of these eyes to the initial medical treatment is shown in Table 1. In 156 (73·6%) eyes control of intraocular pressure was achieved by acetazolamide together with pilocarpine delivered as drops or by a saturated hydrophilic contact lens (Hillman *et al.*, 1975). In a further 53 (25·0%) eyes control was achieved by the added use of oral glycerol and in only 3 (1·4%) eyes medical control failed. Table 2 shows that the response to medical control was not directly related to the level of intraocular pressure on presentation.

Surgery was performed as shown in Table 3 on all eyes except 10, when the patients refused surgery

Table 1 *Response of 212 eyes with acute closed-angle glaucoma to initial medical treatment*

Medical treatment	*1971*	*1972*	*1973*	*1974*	*1971–74*
Control by pilocarpine and acetazolamide	30	30	51	45	156 (73·6%)
Control by pilocarpine, acetazolamide, and glycerol	18	4	15	16	53 (25·0%)
Failed medical control	0	3	0	0	3 (1·4%)

Table 2 *Response of eyes with acute closed-angle glaucoma to initial medical therapy considered with regard to the height of intraocular pressure on presentation*

Presenting IOP	*Pilocarpine and acetazolamide* (%)	*Pilocarpine, acetazolamide, and glycerol* (%)	*Failed control* (%)
20 to 40 mmHg	81·3	18·7	—
41 to 60 mmHg	71·9	26·5	1·6
61 to 70+ mmHg	73·9	23·9	2·2

Table 3 *Surgical procedures performed after medical control of 212 eyes with acute closed-angle glaucoma*

Surgical treatment	*1971*	*1972*	*1973*	*1974*	*1971–74*
Peripheral iridectomy	23	17	35	33	108 (50·9%)
Trabeculectomy	12	14	25	18	69 (32·6%)
Broad iridectomy	11	0	1	2	14 (6·6%)
Lens extraction	0	3	2	4	9 (4·2%)
Scheie's procedure	0	1	1	0	2 (0·9%)
No surgery	2	2	2	4	10 (4·7%)

Table 4 *Final visual acuities achieved for each day of presentation of 212 eyes with acute closed-angle glaucoma*

Day	*6/6*	*6/9*	*6/12*	*6/18*	*6/24*	*6/36*	*6/60*	*<6/60*	*Blind*	*No record*
1	7	23	7	6	6	3	2	6	0	3
2	2	9	5	6	0	2	1	4	0	3
3	3	8	11	5	2	1	1	2	0	0
4	2	1	5	2	1	0	1	0	1	0
5	0	3	0	3	1	0	0	1	0	0
6	0	2	1	1	1	0	0	0	0	0
7	2	3	0	2	1	0	0	2	3	2
>7	6	9	2	2	1	0	1	7	1	4
No record	1	0	2	4	0	1	0	1	0	1

or were considered too ill. As the patients were under the care of a number of different surgeons varying criteria were applied to select the type of surgery, and approximately one-half of the eyes had peripheral iridectomy and approximately one-third had trabeculectomy.

Consideration was next given to the spectrum of visual outcome for each day of presentation (Table 4) and also to the proportion of eyes achieving '6/12 or better' vision for each day of presentation (Fig. 4). There is a significant regularity of results which appears independent of the day of presentation for treatment. In view of this, consideration was given to the presenting visual acuities of those eyes which presented promptly on day 1 compared with those which presented late on day 7 or later and which finished with visual acuity '6/12 or better' (Fig. 5). It is interesting that there is no significant difference in the presenting visual acuities associated with this delay in presentation for treatment. The same applies to the comparison of presenting visual acuities for eyes which presented early or late and finished with visual acuity 'worse than 6/12' (Fig. 6).

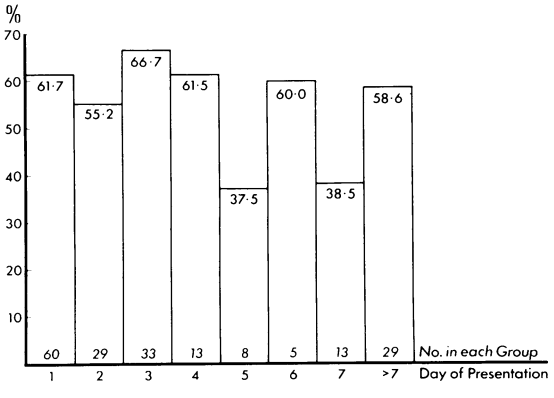

Fig. 4 *Proportion of eyes with acute closed-angle glaucoma which achieved final visual acuity '6/12 or better' for each day of presentation*

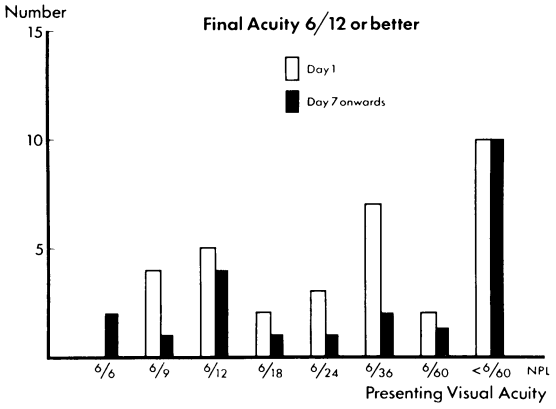

Fig. 5 *Comparison of the presenting visual acuities of eyes presenting on day 1 with those presenting on day 7 onwards with acute closed-angle glaucoma which achieved final visual acuity '6/12 or better'*

The final visual acuity was also considered with regard to the level of intraocular pressure on presentation, and Fig. 7 shows that there appears to be no strong relationship.

In this series 34 eyes (16·0%) had a poor outcome with final visual acuity '6/60 or worse' despite apparently good medical and surgical control of the intraocular pressure in most cases. Table 5 presents the apparent reasons for poor vision in these eyes. The major cause of glaucomatous visual loss appeared to be atrophic change in the optic nerve, and this was not associated with very high intraocular pressure on presentation. Of these 19 eyes 10·5% presented with pressures between 20 and 40 mmHg, 63·2% presented with pressures between

41 and 60 mmHg, and 21·0% presented with pressures between 61 and 70+ mmHg. As expected in an elderly population of patients a number of eyes had coexisting disease—macular degeneration, cataract, arterial occlusion, and in 1 case amblyopia. Three eyes in this series failed to recover from hyphaema associated with trabeculectomy surgery.

Discussion

The statistically significant predominance of females over males and the peak incidence in the sixth decade is as expected from clinical experience with acute glaucoma. The sexual difference is attributable to the shallower anterior chamber in the female as shown in optical and ultrasound studies (Törnquist, 1953; Jansson, 1963). The 11·0% of bilateral cases in this series is similar to the 8·5% reported by Bain (1957) and the 11·5% reported by Lowe (1962) in series of similar size.

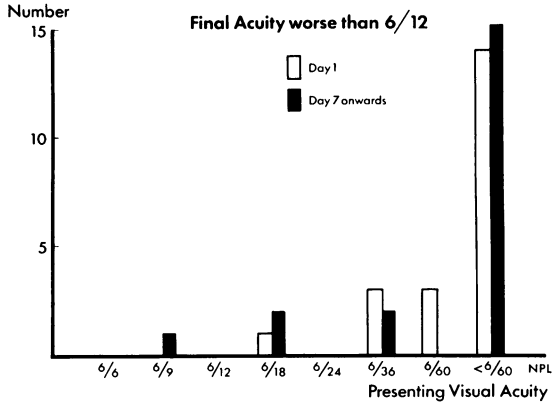

Fig. 6 *Comparison of the presenting visual acuities of eyes presenting on day 1 with those presenting on day 7 onwards with acute closed-angle glaucoma which achieved final visual acuity 'worse than 6/12'*

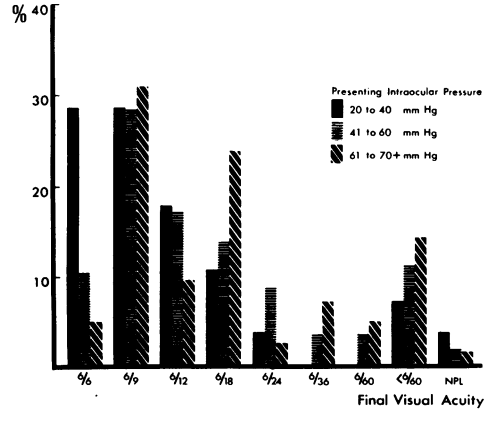

Fig. 7 *Final visual acuity for eyes with acute closed-angle glaucoma considered with regard to the height of intraocular pressure on presentation*

Table 5 *Reasons for poor visual outcome in 34 eyes which achieved final visual acuity '6/60 or worse' after acute closed-angle glaucoma*

Blind (5 eyes)	Optic atrophy		2
	Trabeculectomy haemorrhage		2
	Other pathology		1
	retinal detachment	1	
6/60 or less (29 eyes)	Optic atrophy		17
	Trabeculectomy haemorrhage		1
	Other pathology		11
	macular degeneration	4	
	cataract	3	
	arterial occlusion	1	
	amblyopia	1	
	mental confusion	2	

At the time of presentation most of the patients had severe reduction in visual acuity to about 6/60 or less and only 12·3% with 6/12 or better. Most of the patients responded well in terms of control of intraocular pressure to both the initial medical and later surgical treatment, the response to initial treatment being independent of the height of intraocular pressure on presentation. After this apparently successful treatment, however, only 74·5% eyes achieved 6/18 or better vision, only 59·9% eyes achieved 6/12 or better, and only 10·8% achieved 6/6 vision. These figures may be compared with the 76·7% which achieved 6/18 or better in the series of Mackie and Rubinstein (1954) and the 84% which achieved 6/12 or better in the small series reported by McNaught *et al.* (1974).

Table 4 shows that there is a group of eyes which presented late after 1 week but did particularly well, achieving visual acuities of 6/6 or 6/9. These have behaved differently from the main population and are possibly cases of subacute closed-angle glaucoma rather than true acute closed-angle glaucoma. The overall unsatisfactory visual results despite apparently good response of intraocular pressure to treatment demonstrate the permanent damage which occurs to the visual system in most cases and which is marked in some.

The patients show a surprising and often considerable delay in presentation, with less than one-third presenting on the first day of the attack. The average delay of 3·5 days is less than the delay of 10·9 days reported by Lloyd (1948) and may reflect an improvement in patient education. It is interesting to consider the eyes which achieved a good visual outcome with regard to this delay in presentation. Fig. 4 shows that approximately 60% of eyes achieved 6/12 or better irrespective of the day of presentation. The eyes which did well presented with a similar spectrum of visual acuities whether they presented on the first day or the seventh day of the attack. There was no association between the height of the intraocular pressure on presentation and visual outcome. Consideration of the 16% of eyes which had a bad visual outcome suggested that the main cause of visual loss in acute glaucoma is neuronal damage.

The independence of visual outcome from the speed of presentation of the patient suggests that neuronal damage occurs very early in the disease, possibly within hours of the initial high rise in intraocular pressure. The independence from the height of intraocular pressure at the time of presentation suggests that there is individual variation in the ability of different eyes to withstand the insult of sudden pressure rise just as eyes differ in their ability to withstand the sustained pressure rise of chronic glaucoma.

The risk to the 'fellow' eye in acute glaucoma is well appreciated, and the place for prophylactic peripheral iridectomy is firmly established. The results of this study show that earlier presentation of the patient would not significantly improve the visual outcome in the eye with acute glaucoma. The short critical time before damage occurs to the eye suggests a role for preventive ophthalmology in the detection and surgery of 'first' eyes which have shallow anterior chambers and narrow angles placing them at risk before they develop acute closed-angle glaucoma.

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