The suggestion that clofibrate (Atromid-S; ethyl 2p (chlorophenoxy) -2-methylpropionate) is effective in the treatment of acute angle-closure glaucoma was recently made by Orbán, Hanisch, and Vereb (1966). These authors had previously observed a rise in the concentration of free fatty acids and in blood viscosity just before and during an attack of congestive glaucoma. Although clofibrate effectively controlled such attacks in ten cases, no details are given of the results in one case of chronic simple glaucoma (CSG) and in three cases of secondary glaucoma.

Cullen (1967) reported the effect of clofibrate in ten patients with CSG or secondary glaucoma. The intraocular pressure (IOP) fell significantly in seven eyes of five patients; these, however, had not responded to other treatments and operation was envisaged. No details of other investigations on these patients were given.

In a recent report, Orbán (1968) stated that, of five cases of CSG, the IOP was “normalized” in one, decreased in two, and unaltered in two; clofibrate therefore was not markedly effective in cases in which the angle was open. Orbán suggested three possible modes of action in those cases where the drug was effective; antilipaemic effect (but no change in IOP observed in normotensive animals eyes); cholinesterase inhibition (but no change in pupil diameter); carbonic anhydrase inhibition (but no diuresis observed). Since the last-named effect is not likely to be striking this would not seem to be a valid criticism.

It seemed useful, therefore, to observe the effect of clofibrate on untreated proven cases of CSG with gonioscopic and tonographic control; this investigation was begun in September, 1967.

Patients and methods

Ten consecutive patients who presented at the Oxford Eye Hospital with previously undiagnosed and untreated CSG were used in the trial. All eyes under consideration had open angles (confirmed by gonioscopy), cupped discs, and raised IOP.

Applanation tonometry followed by tonography was performed on all eyes. Patients were then given clofibrate capsules (Atromid-S) in a total dose of 2 g. daily. The observations were repeated one month later.

Results

One patient reported gastrointestinal side-effects; no other systemic effects were noted, apart from one patient who was firmly convinced that the treatment had caused him to stop smoking.

Age, sex, initial and final applanation IOP, and coefficient of outflow (“C”) are given in the Table, which shows that, except for Cases 8 and 10, the IOP was not reduced to an acceptable level. In Case 8 the IOP fell to within the normal range and the patient is still
Clofibrate in chronic simple glaucoma

maintained on the drug. In Case 10, although the IOP appeared to be controlled on the drug for 6 months, it later rose and other treatment was begun. In the other eight cases, treatment with clofibrate was discontinued after one month. Despite the marked reduction of IOP in Case 1, the resultant level was clinically unacceptable; in any event the side-effects precluded further treatment with clofibrate.

It is noteworthy that (except for one eye of Case 8) the coefficient of outflow did not improve appreciably in any eye under investigation. No change in pupil diameter was observed. Any slight action of clofibrate may therefore be due to decrease in secretion of aqueous (possibly carbonic anhydrase inhibition). It is possible that this is the mechanism of acute congestive glaucoma (Orbán and others, 1966), since the pupil diameter does not change.

Table Results in each eye in ten cases

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Initial IOP</th>
<th>Final IOP</th>
<th>Eye</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>38</td>
<td>M</td>
<td>46 0.12</td>
<td>22 0.06</td>
<td>L</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>F</td>
<td>48 0.07</td>
<td>38 0.12</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>62</td>
<td>F</td>
<td>42 0.02</td>
<td>22 0.27</td>
<td>R</td>
<td>Gastrointestinal discomfort</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>M</td>
<td>36 0.12</td>
<td>20 0.25</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>M</td>
<td>66 0.03</td>
<td>44 0.02</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>33</td>
<td>M</td>
<td>46 0.10</td>
<td>30 0.15</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>M</td>
<td>46 0.08</td>
<td>44 0.12</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>M</td>
<td>25 0.02</td>
<td>18 0.08</td>
<td>R</td>
<td>Still on therapy</td>
</tr>
<tr>
<td>9</td>
<td>60</td>
<td>F</td>
<td>28 0.07</td>
<td>26 0.08</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>59</td>
<td>F</td>
<td>30 0.14</td>
<td>21 0.14</td>
<td>R</td>
<td></td>
</tr>
</tbody>
</table>

Mean initial pressure = 37.5 (S.D. ± 11 mm. Hg)
Mean final pressure = 31.8 (S.D. ± 12 mm. Hg)

Summary

In a trial of clofibrate treatment in ten patients with proven chronic simple glaucoma, only three showed a definite fall in intraocular pressure. Only one of these has normal intraocular pressure after 6 months' treatment, and except for this one case the co-efficient of outflow did not improve on treatment. Overall there is no significant difference between intraocular pressure before and after treatment (0.2 < P < 0.3).
References

ORBÁN, T. (1968) *Lancet*, 1, 47

Addendum

Since this paper was accepted this work has been confirmed by Gloster, Hartley, and Perkins (1968).
Clofibrate in chronic simple glaucoma.

T G Ramsell and J A Roth

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