Leprotic Iritis and Blindness

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The number of patients blind from the ocular lesions of leprosy is unknown, as would be expected with a disease whose victims still exist in some countries, mainly in seclusion. That it is of considerable magnitude, cannot be doubted by anyone who comes in contact with the disease in the field, in leprosaria or even today in the eye departments of hospitals in England. The frequency with which the eyes are involved has evidently varied at different times, for although Lopez in 1891 estimated an incidence of 50% for uveal infection in leprosy, and Chance in 1916 thought that it might amount to 75% in the cases which he examined; Elliott in 1920 considered that the lack of agreement among current opinions in his day was so great as to suggest real variations in differing circumstances and parts of the globe. The fact that blindness complicates the later stages of the disease in many patients cannot be doubted by any who undertake their care.

In recent years the hope that the effect of improved systemic treatment upon the ocular lesions would control them has given rise: first, to optimistic forecasts, then to conflicting reports and, latterly, to some degree of pessimism.

Cochrane, in 1955, considered that with such therapy it should now be possible to prevent blindness, and Amendola in the same year spoke of the sulphones as having "revolutionized the outlook for ocular lesions." But Bucalossi, as early as 1950, had observed the development of leprotic iritis in patients under intensive sulphone treatment. Kirwan had seen relapses of the same condition in patients supposed to be cured, and although Ebenezer and Holmes considered that with sulphone treatment ocular complication were less likely, Balakrishnan found that "the longer the treatment the greater the percentage of ocular complications." Choyce reported an increase in the number of cases of iritis in patients so treated and under observation at the Hospital for Tropical Diseases. This he attributed to the effect upon the uveal tissue releasing into the circulation increasing quantities of bacterial protein through the greater efficacy of the drugs presently employed.

My own experience at the Hospital and Homes of St. Giles agrees with that of Choyce: namely, active disease—usually iritis—is found on routine inspection of ocularly symptomless patients whose condition is regarded as fully under control, and in some of whom further treatment is considered unnecessary. These are lepromatous cases, of course, and although lagophthalmos is seen, it is the iritis which arises as the unsuspected, potentially blinding lesion. Potentially blinding because in the absence of ocular treatment its symptomless course is transformed into manifest visual loss—even then without pain or ocular injection—when complicating irreversible lens opacification has begun. Leprotic iritis, hence, merits special consideration.

Leprotic iritis. How the iris becomes involved in leprosy is still a matter for debate, whether by blood stream spread, transconjunctivally, or from contiguous lesions. That it is the common and visually dangerous lesion, has been known for a century, overlooked for a time, and once more has received emphasis in recent years. The earliest account of it, as a complication of a bacterial disease, is given, as would be expected, by the Norwegians, Bull and Hansen who, regretting the omissions of the early descriptions in preophthalmoscopic days (1842-1848), describe ocular lesions in some detail. They say, "if we examine a somewhat large number of leprous patients we shall find in 30% of cases traces of iritis, in the form of fringes around the pupillary margin, or of

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deposits on the capsule of the lens. . . .” Terminal staphylomata are mentioned and it is clearly recognized that: “... as the iritis occurs without violent symptoms, it is not uncommon to find exudations around the borders of the pupil and adhesions to the capsule of the lens in patients who have not complained of pain or derrangement of sight.

Until the slit-lamp microscope became a clinical tool upon which the ophthalmologist relied, in the 1920’s, opinions on the incidence of iritis in leprosy differed greatly. Naked eye inspection offered uncertain evidence and inspection with the loupe was little used by the leprologist. Now, with opinions based upon the biomicroscopic evidence, ophthalmologists with experience of leprosy see the role of uveitis more clearly as: “the commonest cause of blindness in leprosy, Kirwan (14); as responsible for most of the blindness” in the disease, Choyce (6); or as “the cause par excellence of blindness,” Weerekoon (18).

Of the several leprotic manifestations of iritis which are seen, it is the most insidious, chronic form of the condition which is the most dangerous to sight. The acutely painful, congestive form of iritis which may complicate the lepra, or erythema nodosum leprosum, reactions attracts attention and treatment at once. Iris nodules and even "pearls" (Fig. 4, Plate 1) are visible to the naked eye and the visual hazard is apparent at a relatively early stage, but when the muted reactions of the leprotic eye suppress pain and provide no tell-tale ocular injection, the initial active stage of the condition is to be diagnosed only by biomicroscopic examination of a “white” eye in a patient who may be visually symptomless. Such routine examination of symptomless patients is a simple matter in the ophthalmic department, but may be much more difficult in the leprosarium or control center.

Because active iritis in general responds so well to local treatment in the early stage, it appeared to me very desirable to have up-to-date information of the present incidence of its "silent" form, of the frequency with which it is complicated by cataract formation or ocular degeneration; and, hence, of its relation to blindness among leprosy patients. To this end two complementary investigations were undertaken: a smaller survey at Chikankata, Zambia, and a larger one at Sungei Buloh, Malaysia.

MATERIALS AND METHODS

At Sungei Buloh, 507 patients (297 males and 210 females) of ages ranging from under 14 to over 80 were examined (12); ten percent of them being drawn from permanently bedridden patients among whom were most of the blind. Not unexpectedly, the total prevalence of ocular disorder of all kinds was high (32.5%), and potentially blinding nonleprotic lesions, chiefly pterygium (3.3%) and primary cataract (9.3%), figured prominently among them.

Leprotic eye lesions, lagophthalmos and exposure keratitis, intrinsic keratitis, corne-

### Table 1. Age incidence of iritis and its complications (Sungei Buloh).

<table>
<thead>
<tr>
<th>Age groups</th>
<th>0-14</th>
<th>15-19</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases (507)</td>
<td>13</td>
<td>42</td>
<td>57</td>
<td>63</td>
<td>102</td>
<td>99</td>
<td>80</td>
<td>51</td>
</tr>
<tr>
<td>Signs of iritis (39)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>10</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td>Percentage of cases in age group</td>
<td>4.9</td>
<td>9.8</td>
<td>12.1</td>
<td>10</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Secondary cataract (21)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>5</td>
<td>6</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Percentage of iritis cases</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>50</td>
<td>50</td>
<td>75</td>
<td>66.6</td>
</tr>
<tr>
<td>Phthisis bulbi or staphylomata (7)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Blind from effects of iritis (11)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Total blind* (leprotic &amp; non-leprotic lesions) (36)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>15</td>
</tr>
</tbody>
</table>

* The total number blind from all causes are included for comparative purposes.
al lepromata and iridocyclitis, with or without secondary cataract, formed roughly one half of the ocular lesions. Evidence of iritis, posterior synechiae or pigment deposits upon the lens or of its complications, secondary cataract with synechiae, phthisis bulbii or ciliary staphylomata were noted in about 50% of lepromatous lesions (males 54%, females 44%). The age incidence of these signs and complications (Table 1) which, unless blindness had led to identifiable incapacity, had escaped notice, was of considerable interest in view of the known tendency of iritis to relapse in the absence of local treatment and to lead to complications if its effects are unrelieved. Signs of the condition were not seen under the age of 30, but after that age the incidence of iritis rose to about ten percent. However, the proportion of complicated cataract and "blindness" increased more sharply in the later age groups.

Some of these patients had been under constant supervision and treatment going on 30 years, while others were for only a year or so. In view of demonstrating how these varying periods of treatment might be related to the signs of iritis detected, Figure 1 was constructed.

The fact that signs of activity were detected in only one case at the time of examination (treated for 25 years), and that the interval between the attack(s) and the date of examination were quite unknown, would diminish the value of this evidence even if treatment had been known to be continuous during the periods recorded. In many cases, it had not been so if only on the account of the hazards of war.

The most that can be said, therefore, is that in these circumstances prolonged control did not prevent the onset of inflammatory changes in the uveal tracts of patients showing positive signs of intraocular disease.

The expected preponderance of uveal lesions in patients with lepromatous leprosy (90%) is apparent from Table 2.

In the smaller group at Chikankata (191 cases), the fact that although all of the men were examined and only a certain number of women (unselected) could be seen, renders an analysis of the findings of less value. These are, however, shown in Table 3.

The interpretation of these figures is the more difficult since all of the cases of iritis.
TABLE 2. The incidence of iritis in tuberculoid, borderline and lepromatous cases.

<table>
<thead>
<tr>
<th></th>
<th>Total cases</th>
<th>Tuberculoid</th>
<th>Borderline</th>
<th>Lepromatous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>297</td>
<td>50</td>
<td>27</td>
<td>220</td>
</tr>
<tr>
<td>Female</td>
<td>210</td>
<td>48</td>
<td>21</td>
<td>141</td>
</tr>
<tr>
<td>Signs of iritis</td>
<td>(39 cases)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1</td>
<td>1</td>
<td></td>
<td>19</td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td></td>
<td></td>
<td>16</td>
</tr>
</tbody>
</table>

TABLE 3. Age incidence of iritis and its complications (Chikankata).

<table>
<thead>
<tr>
<th>Age groups</th>
<th>0-14</th>
<th>15-19</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases</td>
<td>44</td>
<td>18</td>
<td>26</td>
<td>37</td>
<td>33</td>
<td>22</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Signs of iritis</td>
<td>1</td>
<td>2</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Secondary cataract</td>
<td></td>
<td></td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Blind from effects of iritis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total blind</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

and its complications were in men and, although a higher incidence of disease among them would be in accord with the experience of many, the fact that only a part of the female population of the leprosarium was examined must cast doubt on the assumption that such a sex-determined difference is revealed.

The total incidence of ocular disorders here (34.4%) was a little greater than at Sungei Buloh, perhaps because trachoma as well as senile cataract and pterygium contributed to nonleprotic ocular abnormalities. Leprotic lesions formed a smaller proportion of the total (24.2%), but to them iritis and its complications contributed 55.5%; a figure comparable with that of the Sungei Buloh series.

That the lower incidence of leprotic eye lesions at Chikankata is more apparent than real is strongly suggested by the fact that this group included a larger number of cases in the age group which at Sungei Buloh showed fewer leprotic eye lesions: 46.1% of patients under 30 at Chikankata and 22.1% at Sungei Buloh; and a smaller number of patients over 50 where the highest incidence of iritis, 40%, was found at Sungei Buloh, while Chikankata had 17.3% cases.

It would seem, therefore, that the incidence of 'silent' iritis in these two areas is probably similar, and that the threat of later blindness which it holds is present to a comparable degree in both places.

DISCUSSION

It is clear that in these two groups of patients a large proportion of the leprotic ocular involvement is iritis. That this should have, in some cases, resolved spontaneously is fortunate; that in other, complicated cases it recurred is likely, in view of this general tendency in the condition when it is seen in association with other diseases. The complications resulting from iris adhesions and toxic aqueous (Fig. 3, Plate 1), secondary cataract (Fig. 1, Plate 1), ciliary staphylomata (Fig. 2, Plate 1), and secondary glaucoma and phthisis bulbi are such as would be expected to follow untreated iritis in general, and the contribution which they make to blindness in leprosy is evidently a considerable one.

Diagnosis. Reference has already been made to the 'silence' of the condition in its early treatable stages. As a cause of delay in diagnosis, this feature of insidious leprotic iritis has been repeatedly emphasized by Somerset (17), Kirwan (14), McKie Reid (16), Weerekoon (18), Choyce (4), and Hobbs (11). It is exemplified in these groups, in which a history of relevant ocular disorder was recorded in only two cases.

Examination of the apparently normal eyes of patients with no symptoms of eye trouble provides the only certain means of diagnosis in the early, active stage of the disease, and when the appropriate technic is employed the degree of certainty can be
Fig. 1. A mature, complicated cataract resulting from longstanding iritis in a patient with inactive lepromatous disease. There is no history of ocular pain, no ciliary injection and the posterior synechiae which are present are invisible to the naked eye. Vision is reduced to the ability to perceive light only. (Coincidental lagophthalmos and exposure keratitis also.)

Fig. 2. The sightless, painless, degenerating fellow-eye of the patient depicted in Fig. 1. Total synechiae have been followed by secondary glaucoma and then by thinning and degeneration of the ocular coats. Herniation of the ciliary body ("ciliary staphyloma") has finally resulted in the swelling seen below the cornea. (The eye is directed upward.)

Fig. 3. Acute iritis in a patient in reaction. Severe pain, with intense ciliary injection and diminished vision, attract the attention of patient and doctor at once. The pupil is partly dilated with atropine and a posterior synechia—invisible until then—is seen. The slit-lamp microscope displayed a dense aqueous flare.

Fig. 4. Iris "pearls" in a patient with controlled lepromatous leprosy who was visually symptomless. Unless they are accompanied by other evidence of ocular disease these remarkable deposits may remain for long periods without giving rise to complications which threaten sight.
FIG. 5. The anterior chamber of a patient with controlled lepromatous leprosy as seen by slit-lamp microscopy. The sole ocular symptom was vague discomfort; vision was unimpaired and ciliary injection minimal. Nevertheless, iritis of considerable severity is present, since the slit-beam displays dense turbidity of the aqueous from the numerous leucocytes exuded into it from the surface of the inflamed iris. ("Aqueous flare.")

FIG. 6. The slit-lamp microscope picture of the anterior chamber of a normal eye (the "optical section"). The clear aqueous reflects no light from the slit-beam into the observer's eye and hence the interval between cornea and lens appears dark—"optically empty."

FIG. 7. The optical section in iritis. In this case not only is there aqueous flare, but from the masses of leucocytes in the aqueous many have been deposited as clumps upon the posterior surface of the cornea and are seen as whitish dots—so-called "keratic precipitations" or "K.P."

FIG. 8. The eyes of a patient aged 14 who had been under treatment for lepromatous leprosy for some 8 years and in whom, 6 years previously, the condition depicted in Fig. 5 had been detected. Following continuous general and ocular treatment he retains excellent vision and shows no sign of ocular activity.
very high. Naked eye inspection has value, as Weerekoon (18) has recently pointed out. If, however, it reveals a pupil fixed by adhesions to the lens, diagnosis probably comes too late to prevent serious complications. Iris atrophy affecting the anterior iris layer patchily, or the sphincter iridis to produce unequal, poorly reacting pupils can also be seen in this way. It is sometimes a sequel to the attack of iritis but occurs in other cases without inflammatory associations.

Inspection with a loupe magnifying six or eight times, if suitably illuminated, displays the iris changes well and in experienced hands may show the "aqueous flare" (Fig. 5, Plate 2) which is the cardinal (and may be the only) sign of the early active condition. Clumps of leucocytes exuded from the iris into the aqueous may be seen circling in its convection currents, as well as the deposits which they produce (keratic precipitates) on the posterior corneal surface (Fig. 7, Plate 2).

With the slit-lamp microscope, however, these signs are seen with ease and certainty. This instrument is the ophthalmologist's most important diagnostic aid and the changes revealed in the optical section of the transparent tissues which it displays provide data which are quite invaluable. In its modern form, the technic of examination with it is simplified and the leprologist can easily be taught to use it in routine examination of his patients. Its cost should not be too great to prevent it from being installed in at least the principal leprosaria and centers of leprosy control.

**Treatment.** "The treatment of iritis... is not so hopeless as for disease of the cornea. The chief point is, of course, in this case to prevent synechiae by the timely instillation of atropine." This was the advice of Bull and Hansen (4) and, in respect of insidious iritis with which we are concerned, it remains sound. Atropinization by means of drops or ointment (1%) applied often enough to maintain full mydriasis and cycloplegia, is required. Twice daily applications are usually necessary if drops are used, and if these can be supplemented by weak steroids (guttæ or oculent hydrocortisone [1%] B.P.C.), this is helpful. Stronger steroids should not be employed because their therapeutic effect in uveitis is little greater than those mentioned, and in long term use they are known to provoke glaucoma. Ointment is more easily applied in children and daily application is usually sufficient.

Continued atropinization is needed for as long as cells can be detected on biomicroscopy of the aqueous humor. This may be for a period of years and, of course, the treatment imposes a disability—photophobia from the mydriasis and blurred near vision from the cycloplegia—which the patient must be enabled to tolerate if the aims of therapy are to be achieved. Dark glasses are usually welcomed for the photophobia, and simple spherical lenses for the visually-exacting tasks of near vision (reading, sewing, etc.) make these possible.

The acute congestive attacks which may occur; e.g., in reactions, may well, of course, call for sterner measures of treatment with subconjunctival injections of mydriatics and steroids as well as local heat.

**SUMMARY**

1. The iritis which is responsible for a major part of the blindness due to leprosy is at present largely unrecognized in the early, active stage when, by the application of simple treatment, it could be controlled and its blinding complications be prevented.

2. Early diagnosis involves: first, recognition of the insidious nature of the condition and also the fact that the active stage is usually silent.

3. Signs of inflammation in the active stage are to be elicited only with the aid of magnification and critical illumination, and these are best provided with the slit-lamp microscope.

4. The installation of this instrument in centers of leprosy control and leprosaria is a matter of considerable importance. In those countries where decentralization of leprosy patients is contemplated, it becomes a matter of urgency if the unwitting hospital discharge of potentially blind patients is to be avoided.
RESUMEN

1. La irisitis, que es la responsable de la mayor parte de las cegueras debidas a la lepra, no se reconoce actualmente en la etapa precoz y activa, cuando mediante la aplicación de un tratamiento simple podría ser controlada y podrían prevenirse las complicaciones que conducen a la ceguera.

2. El diagnóstico precoz envuelve: primero, el reconocimiento de la naturaleza insidiosa de la condición y también del hecho que la etapa activa generalmente es silenciosa.

3. Los signos de inflamación en la etapa activa son puestos en evidencia solamente con la ayuda de magnificación y de iluminación crítica y éstas se obtienen mejor mediante el microscopio de lámpara de hendidura.

4. La instalación de este instrumento en los centros de control de lepra y en los leprosarios de considerable importancia. En aquellos países en los cuales se contempla la decentralización de los pacientes de lepra, se transforma en algo de suma urgencia si se quiere evitar el alta hospitalaria de pacientes potencialmente ciegos.

REFERENCES


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