

# The Blinding Lesions of Leprosy

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## Introduction

Estimates of the frequency with which the eyes are affected by leprosy have varied considerably in reports from widely separated places during the century or so in which the nature of the disease has become more clearly apparent. Climate, race and skin pigmentation are prominent among the factors which have been thought possibly to be responsible for these differences, and there can be no doubt of course that ocular involvement is everywhere seen more frequently among patients with lepromatous or near lepromatous leprosy (Choyce, 1969; Hobbs, 1971). Another factor which appears to us to be of probable importance in giving rise to these differences is the method of ocular examination employed.

Some workers fail to indicate how their observations were made; others have used a magnifier, but in diffuse light when definition is less good. Only a minority have been able to employ biomicroscopy with the slit-lamp microscope and, not surprisingly, the incidence of ocular lesions detected by them has been higher than in others examined by less critical methods (Beretti and Cahuzac, 1970). With this instrument the detection of early ocular lesions with ease and certainty has become routine practice in the ophthalmic clinic. Realization of the increased gravity of eye complications, especially in India and the East, makes it highly desirable that the most accurate means of diagnosis should be available where it is most needed, that is, in the leprosarium.

Two types of ocular lesion, in general very different in their clinical presentation, can be distinguished as the causes of the vast majority of blindness attributable to leprosy. The first type comprises chiefly conditions involving the superficial tissues of the eye and eyelids. Lagophthalmos from involvement of the facial nerve in the tuberculoid form of leprosy is the commonest. Impaired corneal sensation, due to trigeminal nerve damage, frequently aggravates the effects of inadequate corneal protection from this cause; but whether or not this is the case, exposure keratitis with vascularization and opacification lead to loss of vision. Intrinsic corneal lesions—corneal lepromata or interstitial keratitis occurring either alone or in association with other ocular disease—are also to be included in this group, the common characteristic of which is the fact that the abnormalities are apparent to the naked eye. Iritis, when it occurs with severe

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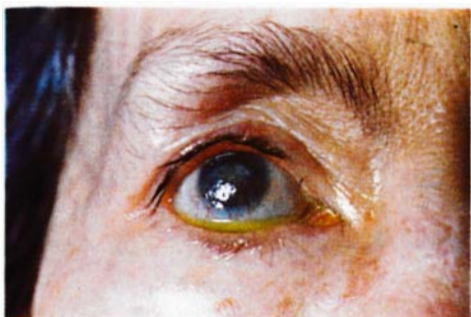


Fig. 1. A mature, complicated cataract resulting from long-standing iritis in a patient with inactive lepromatous disease. There is no history of ocular pain, no ciliary injection and the posterior synechiae which are present are invisible to the naked eye. Vision is reduced to the ability to perceive 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.

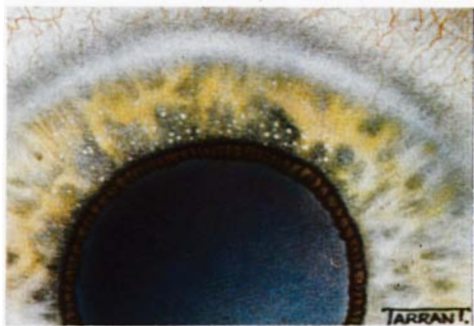


Fig. 4. Iris "pearls" in a patient with controlled lepromatous leprosy who was visually symptomless. Unless they are accompanied by other evidence of ocular disease these remarkable deposits may remain for long periods without giving rise to complications which threaten sight.

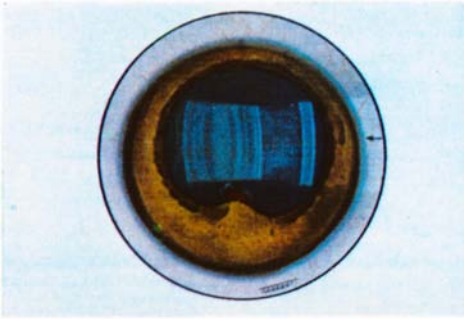


Fig. 5. The anterior chamber of a patient with controlled lepromatous leprosy as seen by slit-lamp microscopy. The sole ocular symptom was vague discomfort; vision was unimpaired and ciliary injection minimal. Nevertheless, iritis of considerable severity is present, since the slit-beam displays dense 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".

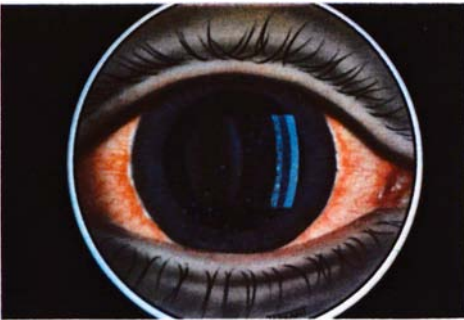


Fig. 7. The optical section in iritis. In this case not only is there aqueous flare, but from the masses of leucocytes in the aqueous many have been deposited as clumps upon the posterior surface of the cornea and are seen as whitish dots—so-called "keratic precipitations" or "K.P."



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.

congestion, pain, and loss of vision (as in the lepra reaction, for example (see Fig. 3)) may be included in this group; and iris pearls and lepromata may also be large enough to attract the attention of the naked eye. Treatment in the majority of these patients can be very effective when they present early to the leprologist, and it is perhaps for this reason that they appear to be making a smaller contribution to the total of those blinded by leprosy.

Detailed analyses of large series of the leprotic blind are few; but recent opinions suggest that the greater proportion of cases of blindness arise from causes in the second group. These consist of purely endogenous ocular lesions—usually anterior uveitis (iritis or iridocyclitis) of an insidious type which, unless it becomes complicated by secondary cataract (Fig. 1) or ciliary staphyloma (Fig. 2), is likely to remain invisible to the naked eye. Certainly, in its early stage when it is readily amenable to simple treatment, naked eye signs are absent (Fig. 5); the muted sensations of the leprotic eye cause the patient little in the way of discomfort in an eye which may appear almost white, and the turbidity of the aqueous humour, the sole observable abnormality at this stage, offers little or no hindrance to vision.

Bull and Hansen (1873) first drew attention to this insidious form of leprotic iritis which they found occurring “without violent symptoms” and often with “exudations around the borders of the pupils and adhesions to the capsule of the lens in patients who have not complained of pain or derangement of sight”. Such signs were observed by them in some 30% of their patients. Thereafter the condition seems to have passed largely unnoticed until the development of the slit-lamp microscope provided the ophthalmologist with a diagnostic tool on which he could rely, *inter alia*, for the detection of the earliest signs of uveal inflammation: i.e., the exudation of leucocytes into the normally clear aqueous of the anterior chamber (Figs 6 and 7). Now, with opinions based upon this evidence, ophthalmologists with experience of leprosy see the rôle of this type of uveitis in the production of blindness more clearly. Kirwan (1955) refers to it as “the commonest cause of blindness in leprosy”; Choyce (1964) as “responsible for most of the blindness”, and Weerekoon (1969) as “the cause *par excellence* of blindness” in leprosy patients. In contrast to the first group of causes its association is predominantly with the lepromatous or near lepromatous forms of the disease.

### Personal Observations

In the ophthalmic clinics of the Hospital for Tropical Diseases, London, and the Hospital and Homes of St. Giles at East Hanningfield in Essex, we detect the early signs of insidious uveitis, sometimes associated with active systemic disease. Not infrequently, however, signs of the latter are absent, skin biopsies are negative, and the disease, on these grounds, would be looked upon as controlled, if not cured. In both instances, however, local treatment with atropine and hydrocortisone drops, as well as any general treatment dictated by the systemic disorder, is needed urgently and when so applied is consistently effective in preventing the blinding complications of secondary cataract and ocular degeneration (Hobbs, 1963). However, unless a leprosarium is equipped with a slit-lamp microscope and the need to employ this in the routine examination of all patients with active disease is realized, these early signs will be overlooked, so

that complications and visual loss will gradually develop. Treatment, if at this stage still possible, may then involve complex surgical procedures.

In these circumstances—evidence, ancient and modern, of the importance of insidious iritis in leprosy and of its clinically “silent” presentation, with the earliest diagnostic signs visible only on bio-microscopy—it appeared at least possible that, in the field, a proportion of patients with signs of iritis might exist unsuspected, and that patients with the complications of the condition might be found to contribute notably to blindness among sufferers from leprosy. The fact that the condition, in its early active stage, had been seen to be readily amenable to simple treatment rendered the problem the more interesting, with its indication of potential therapeutic rewards.

### The Sungei Buloh Survey

One of us, therefore (H.E.H.), has recently undertaken a brief survey in the large leprosarium at Sungei Buloh in Malaysia with the primary object of detecting signs of iritis—active, healed or complicated—and of estimating the frequency with which the complications resulted in blindness. The results of this study have been reported in detail elsewhere (Hobbs, 1971).

The table below summarizes the important ocular observations in the series of 507 patients examined.

TABLE 1  
*Ocular lesions and blindness at Sungei Buloh*

No. of patients examined: 507	
	male 297 female 210
Incidence of ocular lesions of all types <sup>a</sup>	32.5%
Percentage of leprotic eye lesions in the total <sup>b</sup>	50%
Percentage of leprotic eye lesions due to iritis (males 54%, females 44%)	50%
Total no. of blind patients	36 (7.1% of sample)
Blind from leprotic lesions	18 (50% of total)
Blind from leprotic iritis	11 (61% of leprotic lesions)

<sup>a</sup> Pterygium, senile cataract, primary glaucoma and leprotic lesions.

<sup>b</sup> Lagophthalmos, exposure and intrinsic keratitis, corneal leproma, iridocyclitis and its complications.

In the majority of patients the disease was under control and the signs of iritis were those of the old healed or complicated condition, evidence of activity being noted in only a few cases. Such signs were absent in patients below the age of 30, but thereafter the incidence increased from 4.9% in the 30-39 age-group to 11.7% in the 70+ age-group. No complications were seen in patients under the age of 40, but later age-groups showed an incidence of some 7% to 10%. The incidence of blindness from all causes rose steadily from 1.5% in the 30-39 age-group to 22% in the 70+ age-group, but that due to leprotic iritis showed a maximum incidence (6% in the 60-69 age-group) at a slightly earlier age. The known association of lepromatous leprosy with iritis was confirmed, 35 out of a total of 39 cases of iritis (90%) occurring in lepromatous patients. No relationship could be found

between the duration of treatment and the presence of signs of iritis. Choyce's (1970) figures also demonstrate the virtually exclusive association of leprosy iridocyclitis (and keratitis) with lepromatous and borderline leprosy.

From these findings it is clear that a large proportion of ocular involvement in this group of patients is as iritis, as has been increasingly emphasized (Somerset, 1962; Kirwan, 1955; McKie Reid, 1966; Weerekoorn, 1969; Choyce, 1964; Beretti and Cahuzac, 1970). That in the Sungei Buloh series the iritis should have resolved spontaneously or been controlled by systemic treatment without local ocular measures is probably no more than fortunate, given the general tendency for the condition to relapse and become complicated in its later stages. The complications observed—secondary cataract, secondary glaucoma, ciliary staphylomata and phthisis bulbi—are such as would be expected to follow untreated iritis, whatever its cause. The contribution which these complications make to blindness in leprosy is evidently a considerable one.

### Conclusions

The treatment of early leprotic iritis presents difficulties in only a minority of cases—principally those in which episodes of acute exacerbation attract attention during a "reaction", or in which massive destruction of the iris by a localized leproma occurs. In the important group of cases in which insidious iritis leads gradually to loss of vision and eventual blindness, it is early diagnosis which is needed to interrupt this silent and sinister sequence; and for this, the naked eye is insufficient. The modern equivalent of Bull and Hansen's "focal light" and "magnifying glass", i.e. a loupe and lens, or the Hobbs illuminated slit-loupe (Hobbs, 1963) may elicit early signs in practised hands; but for the ophthalmologically inexpert an up-to-date slit-lamp microscope is the most certain way of demonstrating unequivocally the signs of early uveitis.

The need for more expert ophthalmological advice and skill in dealing with problems of potentially blinding leprotic ocular conditions has been stated on many occasions, recently and notably at the Ninth International Leprosy Congress in London in 1968. This need is perhaps emphasized by the high incidence of ocular lesions of various types observed in the Sungei Buloh series, a large proportion of which would now be remediable only by surgery. Our aim here, however, is to draw attention to the at least equally important prophylactic rôle of ophthalmic medical treatment in treating leprotic iritis and preventing its blinding complications. Early and accurate detection of exudation into the anterior chamber from the inflamed iris is the essential preliminary diagnosis here, and in the modern slit-lamp microscope the means to do this are available. It has been suggested that full ophthalmological training is necessary for the use of this invaluable diagnostic tool but, whilst in no way wishing to decry the clinical value of the sophisticated techniques which may be developed in the use of the instrument, we should like to emphasize our belief that for the purpose in mind—primarily the early diagnosis of iritis—only a short course of instruction by a trained observer is necessary.

The installation of these instruments at strategically sited centres where suitable personnel could be trained to make regular surveys of infected individuals and so detect insidious leprotic iritis in its early remediable stage, is, in our opinion, a necessary and important measure in the prevention of blindness in leprosy.

*Acknowledgement*

Figures 1, 2, and 7 are reproduced from *Principles of Ophthalmology* by H. E. Hobbs. London, 1965, Heinemann Medical Books Ltd., to whom we are indebted for permission to use them.

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