

OPHTHALMIC IMPRESSIONS OF A LEPROSARIUM

By IDA MANN,

C.B.E., M.A. OXON., D.SC. LOND., M.B.B.S., F.R.C.S. ENG.

*Ophthalmic Consultant to the Government of Western Australia.
Neuro-Ophthalmologist, Royal Perth Hospital.*

To the average ophthalmologist who has only worked in large cities, ocular leprosy is a rarity and a curiosity. To the leprologist, eye disease in general is often somewhat baffling, and though he may be familiar with ocular leprosy, his attitude is the logical one of attacking primarily the general disease. It is therefore of interest to consider whether ophthalmology has anything to offer in the matter of detailed local treatment in a leprosarium, and whether any observations of interest to oculists can be made there. The following notes were compiled after a stay at the Derby Leprosarium in Western Australia, where 273 patients were examined and some treated for ocular complications.

The ocular complications of leprosy are well known but may be briefly summarised as:

1. Neural complications. The common one is facial paralysis from leprosy involvement of the 7th nerve. This prevents proper closure of the eye during sleep and is a source of danger to the cornea. The effect on the eye is an indirect one. The 5th nerve may be involved more rarely but the effect is similar because the anaesthesia of the cornea abolishes the normal blink reflex and dust tends to collect in the eye and produce serious keratitis in the same way as does exposure.
2. Lepromatous leprosy involving the eye itself. This is usually seen as an iritis, a keratitis or both. Sometimes the lacrimal passages are involved in nasal leprosy.

In addition to ocular leprosy, other diseases of the anterior segment of the eye were found in the Derby Leprosarium. Trachoma is endemic in the northern part of Western Australia occurring in approximately 42% of all the inhabitants. It is the disease of greatest sociological and economic importance in that part of the State. Therefore in an ophthalmic survey of the Derby Leprosarium one would expect to find a large number of cases of trachoma. When leprosy occurs in a trachomatous area and when no detailed ophthalmic survey has been done, there is a tendency to confuse the

two diseases, so that blind persons in a leper hospital are often looked on as blind from leprosy when in fact they are blind from the secondary complications of trachoma.

The following table shows the incidence of diseases of the anterior segment of the eye found in the 273 patients examined at Derby:—

Trachoma	172
Ocular complications of leprosy	47
Corneal scars not due to leprosy or trachoma	22
Pterygium	15
Cataract	13
Conjunctival concretions not associated with trachoma	3
Unequal pupils of unknown etiology						
				(?syphilitic)		3
Glaucoma	2
Papilloma of the lid	2
Meibomian cyst	1
Conjunctival Melanoma	1
Fly bite of lids	1
Strabismus	1
Conjunctivitis (mild)	1

Of the cases of ocular leprosy, 22 had leprotic keratitis, 21 iritis and 4 facial palsy.

The cases of trachoma far outnumbered the cases of ocular leprosy, and a fact of great interest and importance was brought to light by the survey. No cases of active trachoma were found. All the cases except four showed no signs of active infectious trachoma with follicles. These four had each a few remaining follicles, practically healed. Of the rest, 136 were completely healed with fine lid scars and preservation of good sight. Eight were healed with some impairment of sight from corneal scarring. Twenty-four were blind from trachoma (gross scarring of cornea, ingrowing lashes and secondary corneal ulceration) and had been blind when admitted to the leprosarium.

These results, in view of the results of the trachoma survey elsewhere, were surprising, and it was immediately obvious that the chemotherapy (Sulphetrone and D.A.D.P.S.) used in the treatment of leprosy had entirely killed the trachoma virus and had also cured any secondary septic infection. No local treatment whatever had been given to any of the eyes. This finding was subsequently confirmed by a clinical experiment with D.A.D.P.S. on a group of

children with active trachoma but no leprosy. Administration of sulphones by mouth cures trachoma but not as rapidly as does sulphadiazine or sulphadimidine by mouth combined with local eye treatment with terramycin or aureomycin ointment. Oral sulphone treatment is not therefore recommended as a routine treatment for trachoma but, conversely, no treatment for trachoma need be given in a leprosarium where sulphones are in use, since the trachoma will heal automatically.

Of the ocular leprosy cases, four were facial palsy in adult males. One was recovering under general treatment with sulphones, two were mild with sufficient covering of the cornea. One was bilateral and severe, the patient being unable to cover his corneae in sleep. I performed a bilateral lateral tarsorrhaphy under local anaesthesia with great relief to his symptoms and improvement in his appearance. I saw him again many months later and the condition was still satisfactory. This operation is simple and should be done in every case where the cornea is exposed at its lower border with the eyes as closed as possible. Novocaine or other local anaesthetic is injected along both lids from the outer canthus. The lid margin is pared with a sharp knife and stitched together for from a third to half of its length from the outer end. No trouble will be encountered from the lashes, which grow through the line of union. The Meibomian glands apparently cease to function after the union. If the condition improves, the tarsorrhaphy can easily be reopened by one snip with sharp scissors. Untold discomfort can be relieved and all risk of loss of the eye from exposure keratitis will be removed by this simple plastic operation, which is not sufficiently practised.

Twenty-two cases had leprotic keratitis. The typical minute bright white dots in the grey infiltrated margin of the cornea make the diagnosis certain. These have been described by King from observations in the leper colony in England. They can be seen with good focal illumination. A corneal loupe is helpful. Three of the 22 cases were active, with circumcorneal redness and discomfort. The margin of the cornea was swollen, especially below. There was a diffuse greyish peripheral corneal haze with minute discrete very white dots scattered through it. These dots persist after the acute stage has subsided. They were seen in the other 19 quiescent cases. These often showed a definite raised grey area along the lower margin of the cornea. This may become vascularised and looks like a pannus with a sharp upper edge.

The three active cases were treated with cortone eye drops but I was only able to observe them for a week. If I had stayed longer I would have tried subconjunctival injection of cortisone. This drug,

which blocks the manifestations of disease but has no therapeutic action, is a very great benefit in the treatment of many acute inflammatory diseases of the eye. It is suitable for use in leprosy since in the sulphones we have a weapon against the infecting organism and what is required in the eye is something to decrease the oedema and exudative processes which are much more harmful to sight than is the disease itself.

The twenty-one cases of leprosy iritis are of interest. One was acute, with cloudy aqueous, contracted sluggish pupil, and great pain. Atropine 1% and cortone eye drops were given half hourly. Within four hours the pupil dilated fully and the pain subsided. Treatment was continued two hourly next day and the patient made an excellent recovery with no synechiae. This case also demonstrates the great use of cortone in ocular leprosy. It must, of course, be combined with atropine and if used at once all cases of iritis have an excellent chance of complete recovery. Even if cortone cannot be obtained, atropine must be used liberally since the bad results of leprosy iritis are practically all due to blocking of an undilated pupil with exudate.

The other 20 cases of iritis were quiescent, with synechiae of varying extent, and the above mentioned typical white dots on the iris. Three of the healed cases required iridectomy (see footnote), as their pupils were almost completely occluded. In one eye of these, the occlusion was the most complete I had ever seen, the iris stroma running right across, and no pupil being visible at all. Yet there was no iris bombé and no rise of tension. No attempt had been made to treat these cases with atropine in the acute stage. If it had been used, the results would have been very much better. It appears that the use of atropine and cortone is highly to be recommended in all cases of leprosy iritis in the acute or reactive stage. It will prevent the formation of synechiae and lead to restoration of vision. There seems little danger in the use of atropine, as glaucoma is admittedly rare among lepers.

On the whole, therefore, the ocular picture in this leprosarium is a cheerful one. No one is blind from leprosy alone and ocular leprosy is not common in any case. The new drug treatments appear to prevent any serious ocular complications.

Of the other eye diseases found at Derby, little need be said.

Pterygia are common throughout Australia. Their exact cause is unknown. Very few among the leprosy patients required operation, though one very large one covering the pupil was seen. They

should, of course, be removed when they approach the pupil margin.

Corneal scars among Australian aboriginals are common. They are usually due to injury.

Cataract (nuclear sclerosis of the lens) is found among old people but is rare in Australia compared with the Asiatic countries.

Conjunctival concretions are of no great importance. They were merely noted for interest.

The cases of unequal pupils were probably syphilitic but just possibly neural leprosy could have been responsible. They were not investigated.

The cases of chronic glaucoma and papilloma of the lid presented nothing unusual.