Influence of untreated chronic plastic iridocyclitis on intraocular pressure in leprous patients

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SUMMARY The intraocular pressures of a total of 1015 eyes of leprous patients who never had ophthalmological care or local eye treatment were measured. The patients were categorised according to the type of leprosy they had and the eyes were categorised as without or with chronic plastic iridocyclitis. In patients with the tuberculoid and lepromatous types of leprosy the intraocular pressure was significantly lower in eyes with chronic plastic iridocyclitis than in unaffected eyes. It has been shown that chronic plastic iridocyclitis which remains untreated for many years results in significantly lower intraocular pressure.

In leprous patients chronic plastic iridocyclitis 'leads to the gradual failure of the ocular physiology, resulting in complicated cataract and phthisis bulbi'.¹ But not all eyes become blind. Most of them still have visual function after many years of suffering from chronic plastic iridocyclitis. Since there is no report dealing with the influence of untreated chronic plastic iridocyclitis on the intraocular pressure, it is the purpose of this paper to describe the natural course of the intraocular pressure during that disease process.

Material and methods

The intraocular pressures of the 460 patients in the Khokana Leprosarium² were included as well as those of 71 outpatients in the Nepal Eye Hospital, Kathmandu. The ages and the duration of the disease in these patients are shown in Table 1.

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During the long course of the disease there was no regular medical check-up and no control whatsoever of the dispensing of the oral sulphonamide therapy. An ophthalmological examination had never been done, and local treatment had not been given before. All patients had received sulphonamide therapy for more than 10 years except for 29 patients who had suffered from leprosy for less than 10 years, and these 29 patients received therapy for longer than 5 years. Eyes with an intraocular pressure of less than 5 mmHg and those with intraocular pressure of more than 22 mmHg were not considered in this study.

Intraocular pressure was measured with a Schiötz tonometer. Because a slit-lamp was not available, all examinations had to be performed with a magnifying lens and a pocket torch. Owing to this fact, many of the discrete signs of chronic plastic iridocyclitis could not be observed. Even though chronic plastic iridocyclitis leads to the formation of posterior synechiae only after a considerable time, we have considered these synechiae to be the only criteria for the diag-

Table 1 Distribution of patients with different types of leprosy according to their age and duration of disease

Type	Age (years)			Total	Duration of disease (years)			Total
	<20	20-40	>40		<10	10-20	>20	
Tuberculoid	2	87	212	301	14	91	196	301
Lepromatous	5	54	161	220	15	67	138	220
Total	7	141	373	521	29	158	334	521

Table 2 Distribution of the intraocular pressure in the different types of leprosy in eyes without and with signs of chronic plastic iridocyclitis

Type		Intraocular pressure mmHg									Average mmHg	Total number of eyes
		4-5	6-7	8-9	10-11	12-13	14-15	16-17	18-19	20-21		
Tuberculoid	Without CPI*	0	4	4	54	84	147	189	65	15	15.5	562
	With CPI	1	1	1	3	10	7	5	1	1	13.5	30
	Total	1	5	5	57	94	154	194	66	16	15.4	592
Lepromatous	Without CPI	0	1	6	54	53	96	76	27	5	14.8	318
	With CPI	2	7	12	18	17	13	31	4	1	13.4	105
	Total	2	8	18	72	70	109	107	31	6	14.4	423
Total	Without CPI	0	5	10	108	137	243	265	92	20	15.2	880
	With CPI	3	8	13	21	27	20	36	5	2	13.4	135
	Total	3	13	23	129	164	263	301	97	22	15.0	1015

*Chronic plastic iridocyclitis.

nosis of 'eyes with iridocyclitis' in the present study.

Because chronic plastic iridocyclitis is known to be a relatively early complication of leprosy,³ it can be assumed that these patients, all of whom had suffered from the disease for many years, had probably also had manifest chronic plastic iridocyclitis for many years.

Results

There was no significant difference between the patients in the 2 types of leprosy in age or duration of disease.

The distribution of intraocular pressures in the different types of leprosy is shown in Table 2. Of 592 eyes examined with the tuberculoid type 30 (5.1%) showed signs of iridocyclitis. The mean intraocular pressure was 15.5 mmHg in eyes without iridocyclitis and 13.5 mmHg in eyes with iridocyclitis. This difference is significant ($p < 0.005$) (data were evaluated by the chi-square test).

Among 423 eyes of lepromatous patients signs of iridocyclitis were seen in 105 (22.3%). The mean intraocular pressure in eyes without iridocyclitis was 14.8 mmHg and 13.4 mmHg in eyes with iridocyclitis. This difference is significant ($p < 0.001$).

The mean intraocular pressure of the 562 eyes of the tuberculoid patients without iridocyclitis was 15.5 mmHg in contrast to 14.8 mmHg in the 318 eyes of the lepromatous patients without iridocyclitis. This difference is significant ($p < 0.005$).

Of the 880 eyes examined in both types of leprosy which showed no signs of iridocyclitis the intraocular pressure was 15.2 mmHg compared with 13.4 mmHg in the 135 eyes with iridocyclitis. This difference is significant ($p < 0.001$).

The average in intraocular pressures in the total of 592 eyes of the tuberculoid type patients was 15.4 mmHg compared with an average of 14.4 mmHg

in the 423 lepromatous type patients. This difference is significant ($p < 0.001$).

A difference of intraocular pressures between both eyes of more than 5 mmHg was found in 11 patients (3.6%) in the tuberculoid group and in 14 patients (6.5%) in the lepromatous type.

Discussion

According to the results of Slem⁴ a tonographic examination of 49 leprosy patients with reduced intraocular pressure showed a clearly diminished drainage in 25% of his patients. (No information as to the type of leprosy or further information on the intraocular condition was given). He also reported that aqueous humour production was significantly diminished. Our results showed a significant decrease in mean intraocular pressure in eyes with iridocyclitis in both types of leprosy as opposed to eyes without chronic plastic iridocyclitis.

If the reduced pressure is primarily caused by reduced production of aqueous humour, then secondary changes in the trabecular meshwork⁵ lead to diminished drainage. It is therefore not surprising that in eyes with normal or low intraocular pressure Slem⁴ found an outflow facility otherwise typically found in eyes with glaucomatous changes of the trabecular meshwork.

The reason that reports of primary glaucoma in leprosy^{2,6,7} are rare is probably that obliterating changes in the ciliary body itself⁶ counteract, through diminished production of the aqueous, primary glaucomatous changes of the trabecular meshwork. On the other hand, when iridocyclitis is present, the excretion of protein leads to an increased viscosity of the aqueous humour, and the protein acts as an additional barrier in the trabecular meshwork.⁸ Obviously the aqueous-reducing components outweigh in most cases the diminished

filtration, so that secondary glaucoma rarely results. When it does occur, it is usually a sign of an acute diffuse iridocyclitis,¹ as it appears in erythema nodosum leprosum.

Because we were able to measure intraocular pressures only once, we cannot state with certainty that the difference of more than 5 mmHg between both eyes is a pathological finding. However, the prevalence of this difference in both types of leprosy indicates that chronic plastic iridocyclitis fundamentally influences intraocular pressure.

We explain the significantly lower intraocular pressure in eyes 'without iridocyclitis' in the lepromatous type than in the tuberculoid type by the fact that our examinations were done without a slit-lamp. Therefore all discrete microscopical signs (such as Tyndall's phenomenon or cells in the anterior chamber) may not have been detected. In many eyes, especially of lepromatous patients, signs of a chronic plastic iridocyclitis can be visualised only by slit-lamp examination.

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