Multidrug Therapy and After: Changing Visage of Ocular Leprosy

TO THE EDITOR:

The multidrug therapy (MDT) era in consortium with the increasingly efficient leprosy control programs in many parts of the world has apparently caused a decline in the familiar ocular leprosy findings of yesteryear. Gone are the classical chalky-white precipitates of the cornea and the iris pearls that were pointed out to be pathognomonic of leprosy (1). Rare has become the lepromatous pannus, and rarer still the lepromatous nodules of the lids and the globe (1). The adage that iridocyclitis is the most common cause of blindness in leprosy (3, 7) may no longer be true. Low intra-ocular pressure, assumed to be a common phenomenon in leprosy (6), may no longer be tenable.

While it is gratifying to note that several of the well-known manifestations of ocular leprosy have become rare entities, there still exists a sense of apprehension whether well-formulated and -executed, longitudinal, population-based studies would unveil a completely different picture. The shortcomings of methodologies used in the earlier published ocular surveys in leprosy have been well described (2). Since these apprehensions, although compelling, can be laid to rest easily, I would like to share some concerns that have materialized while

working in the ophthalmology department of the Schieffelin Leprosy Research and Training Center, Karigiri, India.

Two well-known complications met with in ocular leprosy are lagophthalmos and iridocyclitis. Although definitive population-based statistics are not easy to come by on the occurrence of these two potentially sight-threatening problems, a disturbing picture is emerging that they can and do occur in patients long after their MDT is over. This situation is alarming for the patient and awkward for the attending leprologist who has announced cure and released the patient from treatment and control.

The etiopathogenesis of facial nerve palsy leading to lagophthalmos in the post-MDT period of a leprosy patient is poorly understood. Does it portend a relapse? Is it associated with a reaction related to leprosy antigens, long dormant but activated now due to whatever reason? These crucial questions need to be addressed. In these groups of patients it is also expedient to rule out other causes of lagophthalmos. The most frequent category of facial paralysis in the general population, regardless of age, sex or ethnic group, is Bell's palsy or idiopathic facial palsy which occurs in about 20 cases per 100,000 persons per year ([5]). Clinically, Bell's palsy occurring in a leprosy patient can be made out by its sudden onset, unilaterality, completeness, and slow improvement over the following 6 months. Facial palsy of leprosy usually would be of gradual onset, either unilateral or bilateral, and the palsy is never complete because the affectation is largely confined to the superficial branches of the facial nerve. Recovery is dependent on early diagnosis and treatment with appropriate steroid regimens. In patients completing MDT, particularly in those with risk factors such as an unstable position in the spectrum of the disease or a face patch, it may be prudent to enlighten the patient and the attending paramedical worker on the possibility of the occurrence of lagophthalmos and to inculcate in them a vigilant attitude.

Inflammatory conditions of the eye, such as episcleritis, scleritis and iridocyclitis, also can occur in the post-MDT period and, again, one is left guessing whether it is a relapse or a reaction, especially when these occur without any skin or nerve reactions elsewhere in the body. Episcleritis, an innocuous condition by itself, may hide an underlying fresh leprosy nodule which is anything but innocuous. New leprosy nodules should always alert suspicion of a relapse unless proved otherwise. Information is almost nonexistent on the exact etiopathogenesis of iridocyclitis that occurs in post-MDT patients. As with lagophthalmos, other causes of iridocyclitis should be searched for in these patients, and although an extensive laboratory workup may be impractical in many of the control area programs, granulomatous diseases that are not uncommon in leprosy-endemic areas such as tuberculosis and syphilis ought to be ruled out.

Decreased corneal sensation is a well known entity of leprosy ([4]). We have noticed that in several of our patients corneal sensation continues to decline long after they have had their full course of recommended MDT. Again, the pathophysiology of this phenomenon is unclear and needs painstaking investigation. A critical thing to be noted here is that patients released from control are seen by the paramedical worker or the leprologist only when they meet with some problem or not at all. This is not a very healthy situation because the post-MDT ocular complications mentioned above justify eye care that should persist until the end of their lives.

Exposure problems and the various ocular inflammations, especially iridocyclitis, that were leading causes of blindness in leprosy may soon, if not already, take a back seat. Senile cataract, as met with in the general population of leprosy-endemic areas, could soon be the foremost reason for blindness among leprosy patients. Intra-ocular lens implantation in leprosy patients, especially of the lepromatous leprosy type, has not been thoroughly explored, and although in some patients this surgery has been done, controlled longitudinal studies are nonexistent. The reluctance to perform this extremely beneficial surgery on leprosy patients has been due, in part, to the cost and the expertise needed in performing the surgery and, in part, to the fear of precipitating a catastrophic uveitic reaction. In our outpatient department we have found the ocular status of six eyes of lepromatous leprosy patients, who had posterior chamber

intraocular lenses implanted in them 5 years ago, to be in very good condition. Although extrapolating from this may not be proper, there is a need to look carefully into this aspect of eye care among leprosy patients since the shifting scenario of ocular leprosy will soon demand it.

—Ebenezer Daniel, M.S., D.O.

Head, Department of Ophthalmology
Schieffelin Leprosy Research
and Training Center
Karigiri, North Arcot District
Tamil Nadu, India 632106

REFERENCES

1. Choyce, D. P. The eyes in leprosy. In: *Leprosy in Theory and Practice.* 2nd edn. Cochrane, R. G. and Davey, T. F., eds. Bristol: Wright, 1964, pp. 310–321.
2. Courtright, P. D. Defining the magnitude of ocular complications from leprosy: problems in methodology. Int. J. Lepr. **56** (1988) 566–573.
3. Kirwan, E. W. O. Ocular leprosy. Proc. R. Soc. Med. **48** (1955) 112–118.
4. Krassi, A. Corneal sensitivity in lepromatous leprosy. Int. J. Lepr. **38** (1970) 422–427.
5. Mellotte, G. Idiopathic paralysis of the facial nerve. Practitioner **187** (1969) 349–353.
6. Slem, G. Clinical studies of ocular leprosy. Am. J. Ophthalmol. **71** (1971) 431–434.
7. Weerekoon, L. Ocular leprosy in Ceylon. Br. J. Ophthalmol. **53** (1969) 457–468.