LEPROSY AND THE EYE – A REVIEW OF EPIDEMIOLOGY, PATHOGENESIS, OCULAR COMPLICATIONS AND TREATMENT

*CE OGBONNAYA *MB.BS; FWACS; FMCOph.*, Consultant Ophthalmologist
LU OGBONNAYA *MB.BS; FMCPH*, Consultant Community Physician
Department of Ophthalmology, Ebonyi State University Teaching Hospital, Abakaliki
CM CHUKA-OKOSA *MB.BS; MSc(CEH); FWACS; FICS.* Consultant Ophthalmologist
University of Nigeria Teaching Hospital, Enugu

SUMMARY

Objectives:

1. To update knowledge on the current trends in the epidemiology, pathogenesis, and treatment of leprosy
2. To highlight the ocular complications associated with leprosy

Methodology: Current literature on various aspects of leprosy research obtained from the Internet and supplemented by available journals were reviewed. Findings relevant to our objectives were extracted.

Results: The prevalence of leprosy has reduced from the estimated 10-12 million (with 5.4 million registered) worldwide in the 1980s to about 0.75 million registered patients by 2002. However, the incidence increased from 550,000 by 1985 to approx. 700,000 by 2002. Many *cured* leprosy patients are living with disability, including ocular disability. Many of these are potentially sight threatening. Already, about 350, 000 to 400, 000 leprosy patients are estimated to be blind worldwide.

Conclusion: Though the prevalence of leprosy is reducing, its incidence is increasing. Many cured leprosy patients are, however, living with ocular complications that could lead to blindness. Most of these blinding complications could be prevented with early diagnosis and prompt treatment.

Recommendations:

1. Every health worker, particularly ophthalmologists, should be familiar with the ocular complications of leprosy.
2. Periodic screening and surgical outreach programmes by ophthalmologists should be integrated into leprosy care programmes with a view to treating avoidable causes of blindness, especially cataract, uveitis, and lagophthalmos.
3. Funding for research on leprosy and health care delivery for leprosy should be sustained because available data show that leprosy is still a cause for concern.

Key words: leprosy, ocular complications

INTRODUCTION

Leprosy is a chronic granulomatous disease of man caused by the bacillus *Mycobacterium leprae* (*M. leprae*).[1] This organism is slow-growing and has a preference for cool temperatures – less than 37°C. The skin, the nose, the ear lobes, certain peripheral nerves, the testes and anterior parts of the eye are particularly affected.[2,3] Leprosy includes a spectrum of diseases. At one end of the spectrum is paucibacillary (or tuberculoid) leprosy; while multibacillary (or lepromatous) disease is at the other extreme. Patients with paucibacillary leprosy have relatively intact cellular immune function, and consequently, low bacillary load, while patients with multibacillary leprosy have markedly impaired cellular immunity and very high bacillary load. There is also borderline leprosy, which lies between the two extremes.[1,2,4]

The earliest accurate description of the disease comes from Indian manuscripts dated 600 BC.[1,5] In 1873, Gerhard Amauer Hansen, a Norwegian physician isolated the leprosy bacilli from lepromatous nodules. The disease is, therefore, sometimes called Hansen's disease.[1,2,4] Leprosy is endemic in all tropical countries, especially Africa, South- East Asia, Central and South America.[3,5,6] Some of the countries with a high number of registered patients include India, Brazil, Bangladesh and Nigeria, with India accounting for 80% of cases.[3,6]

* *Author for correspondence*

EPIDEMIOLOGY

The prevalence of leprosy has reduced drastically since the introduction of the multi-drug therapy (MDT) by the World Health Organization (WHO) in 1982.[7,8,9] The estimated number of leprosy patients reduced from 10-12 million worldwide in the 1980s to 1.3 million active cases by 1998.[5,10] Correspondingly, the number of registered cases fell worldwide from 5.4 million in 1985 to 750,000 by the year 2002.[9]

In May 2002, WHO announced that leprosy had been 'eliminated as a public health problem' at a 'global level', while individual countries are expected to achieve this by the end of 2005.[9,11] This 'elimination' (meaning reduction of prevalence to 1/10,000 population within affected communities) is not being matched by a reduction in incidence, which has been increasing.[7,11] The number of new cases diagnosed globally each year rose from 550,000 in 1985 to 795,000 (approximately equal to the global prevalence) in 1999.[7] By the year 2000, there were 719,330 new cases,[9] with children comprising 15% of cases.[9] This indicates that the disease is still being transmitted.

In Nigeria, the prevalence of leprosy has also reduced from 17.3/10,000 population (250,000 registered patients) in 1989 to 0.6/10,000 population (7000 registered patients) by 1999.[5,8] The prevalence is higher in the northern part of the country, with a few pockets in the South.[6] However, detection is still a problem, due to the failure of medical officers to make the diagnosis.[6] This, not only delays, it also affects treatment.

In spite of the commendable progress made towards the 'elimination of leprosy' by the World Health Organization (WHO), which has now embarked on what it calls the *FINAL PUSH* towards leprosy elimination by 2005, we are still faced with the problem of 'cured' leprosy patients who are living with disabilities, including ocular disabilities.[2,12,13,14] Thus, even though over 10 million people have been released from treatment (RFT), it has been estimated that up to 2 million of these patients still suffer from severe disabilities particularly deformities of the extremities and ocular disease.[2, 4,13,14] Many of such cured leprosy patients are still in need of care-after- cure.[12] Their eye care needs are also increasing as the population of RFT patients is increasing as a result of successful cure; that is, in addition to ageing.[12,15]

CLASSIFICATION

The World Health Organization (WHO) has classified leprosy into *paucibacillary (PB)* and *multibacillary (MB)* types. This classification was introduced for the multi-drug therapy (MDT) introduced in 1982 and further simplified by the Sixth Expert Committee on Leprosy in 1988 to suit field conditions. It is a clinical classification based on skin smears (bacterial index) and skin lesions. The indeterminate (I), tuberculoid tuberculoid (TT) and borderline tuberculoid (BT) of the Ridley-Jopling classification are now grouped as PB leprosy, while the borderline borderline (BB), borderline lepromatous (BL) and lepromatous lepromatous (LL) are grouped as MB leprosy.[2,16]The WHO classification has been widely accepted since it's introduction.

PATHOGENESIS

M. leprae is an obligate intracellular acid-fast bacillus (AFB), which infects mainly macrophages and Schwann cells.[17] It measures 0.3-0.4 microns by 4-7 microns.[18] This organism thrives in temperatures lower than 37^{0}C, and this explains why the 9-banded armadillo, which is a cold-blooded animal with temperatures between 33 and 34^{0}C, is a natural host. It can also be cultured in the mouse footpad.[7] It has a long incubation period, which varies from 3 to 30 years, even for babies born into a family where there is a patient with infective lesions.[7] However, a case has been reported of a 3-month old Japanese female with leprosy.[19,20] Other factors that influence the growth of the organism include nerve density, pH of the tissue, and the presence of 3, 4-dihydrophenyl alanine (DOPA).[18,21,22]

The route of entry into the body is still poorly understood, although evidence points more towards the nasal cavity through droplets infection.[2,7,10,23] Some researchers, however, still believe that skin-to-skin transmission occurs.[24] The older suspicions that insects and soil are sources of infection, have almost been discarded due to lack of evidence.[7,23,26]

Mycobacterium leprae exclusively infects macrophages and Schwann cells.[17] It is likely that infected monocytes from the mucous membranes or broken skin transport the organism into the nerve during normal trafficking of macrophages through the nervous system.[27] This type of trafficking occurs in all individuals as a low, steady-state monocyte exchange between the nerves and the blood.[27] However, in an *M. leprae*-infected individual, infected macrophages might enter a naïve nerve and get trapped there. Various receptor-mediated mechanisms similar to those exploited for invasion of macrophages, may play a role in the invasion of human Schwann cells by mycobacteria, particularly Fc receptors, complement receptors, the fibronectin-binding protein and mannose receptors. These mechanisms are not restricted to Schwann cells; but *M. leprae* has a special affinity for the G–domain of the α-chain of laminin-2 (laminin 2α), an extra cellular matrix protein that is present in the basal lamina of Schwann cells. In turn, the *M. leprae*-laminin 2 α- complexes bind to α/β-dystroglycan complexes expressed on the peripheral nerves and may explain why *M. leprae* does not infect the central nervous system.[7,27]

In paucibacillary (PB) leprosy, the enhanced cellular immune response may be beneficial in terms of 'cleaning' bacteria, as it strengthens the mechanisms by which the organism is killed. However, the

accompanying inflammation in and around the nerve tissues can result in severe irreversible damage within a matter of days. The granulomatous lesions resemble those of tuberculosis, with epitheloid cells of uniform appearance and giant cells of the Langhan or foreign body type. The bacilli are few, if any.[18,27] In multibacillary (MB) leprosy, there is almost complete tolerance of the organism, which replicates in vast quantities within the tissues. The cell type is mononuclear, mainly histiocytes with a foamy appearance. These cells contain masses of intracellular dead or living bacilli, which are referred to as globi or lepra cells. Aggregates of these cells form a leproma, e.g., corneal leproma, iris pearls. Lymphocytes are generally sparse.[27]

Susceptibility to *M. leprae* or the likelihood that infection will lead to disease is influenced by numerous factors as follows:

- *Host immunity:* It is believed that 99% of the population in endemic areas develop adequate protective immunity upon infection, while only a minority of infected individuals develop clinical leprosy.[17] Host immune response also influences the clinical manifestation of the disease, such that patients with impaired cellular immunity have multibacillary leprosy while patients with larger intact cellular immunity have paucibacillary leprosy.[1,4,7]
- *Genetic factors:* Although leprosy is not hereditary, there is increasing evidence that genetic factors may predispose certain individuals to overt disease. There is a highly significant association between HLA-DR2 allele and leprosy in Asia and Africa.[7] There is also an increasing evidence of leprosy susceptibility genes being present in certain families in India, although the genetic loci are yet to be identified.[7] Recent linkage analysis data reveal an association between leprosy susceptibility and genetic markers on chromosome 10. This susceptibility to *M. leprae* is also shared with susceptibility to other infectious diseases.[7] There is hope that the recent breakthroughs in the human genome project will provide the framework for further studies on genetic susceptibility.
- *Race:* The disease affects every race but the type of disease varies with race. Europeans and Asians appear more likely to acquire the lepromatous type while tuberculoid type is more common among Africans.[18,23] The disease has never been reported in native Americans.[23]
- *Environmental/Socioeconomic factors:* It has long been noted that leprosy is more prevalent in the warmer parts of the world. What appears to be the important predisposing factor is the poorer standard of living in these regions; overcrowding, poor sanitation and malnutrition which enhance susceptibility to infection.[4,23]
- *Gender:* Leprosy has been found to affect more males than females.[19,20,23] The reasons are not clear. It has also been observed that this sex difference is greater among adults than children.[19,20]
- *Age:* Leprosy affects all ages, however, the peak age for new cases appears to have bimodal distribution. The first peak is between 10 and 14 years and the second occurs at over 30 years of age. Also the age distribution varies according to endemicity and type of disease.[4] However, some authors believe that children are more susceptible than adults because they are usually more exposed to the disease in an infected parent. It is also stated that adults acquire immunity with increasing age.[20,23]

Leprae reactions are hypersensitivity reactions, which result from sudden changes in the immune response of a patient. There are two types of reactions:

- *Type 1 reaction or 'Reversal Reaction' (RR):* This results from a sudden increase in cellular immunity. It occurs in all forms of borderline leprosy (BT, BB, BL). It can occur before diagnosis, at the start of anti-leprosy treatment or after 'release from treatment' (RFT). There is acute redness and swelling of the affected peripheral nerves. This may cause loss of both sensory and motor functions, leading to anaesthesia and muscle weakness.[2,28] The involvement of the facial and trigeminal nerves leads to lagophthalmos and corneal hypoesthesia respectively.[2,25,27,28] The lagophthalmos leads to exposure keratitis and consequent corneal opacity and blindness. Corneal hypoesthesia causes damage to the corneal surface in several ways, which include; frequent corneal abrasions from foreign bodies and/or misdirected lashes and also by the patient rubbing his itchy eyes (since the patient cannot feel pain). The patient also has reduced tear secretion (reflex tearing), decreased corneal mitosis and, therefore, reduced corneal wound healing. There is also reduced reflex blinking.[25]
- *Type 2 leprae reaction,* erythema nodosum leprosum (*ENL*), occurs only in multibacillary leprosy. This is an antigen- antibody reaction, which occurs only in multibacillary (MB) leprosy. About 20% of MB patients have ENL.[2] It usually appears during treatment or in untreated MB leprosy with a long-standing history of the disease. There is sudden onset of fever, subcutaneous nodules, swelling of the nerves and inflammation of certain organs. Acute iritis and scleritis are ocular manifestations of ENL.[2]

The lepromin test involves the intradermal inoculation of heat-killed (autoclaved) extract of armadillo-derived *M. leprae*.[7] The test provides an indication of an individual's capacity to mount a cell-mediated immune

response against *M. leprae* infection. A negative result does not exclude leprosy. It, therefore, has no value for the sub-clinical phase of the disease.[8] Patients with lepromatous leprosy react violently to lepromin. There are two types of positive lepromin reactions:[29]

- The Fernandez reaction, which consists of an area of erythematous swelling, about 13 cm in diameter. It occurs within 2 days of the inoculation and lasts for about 2 days.
- The Mitsuda reaction, which is a delayed reaction. It occurs after 3-4 weeks. This is the classical reaction in tuberculoid leprosy.[8,29]

OCULAR COMPLICATIONS

Ocular complications in leprosy are common and may lead to visual impairment or blindness.[2,4,25,30] A leprosy patient requires good vision in order to avoid objects that could cause injury. He has to visually examine his hands and feet for any ulceration so that these can be detected early and promptly treated. The peripheral neuropathy which such patients have usually predisposes them to injuries as a result of the anaesthesia of their limbs. A leprosy patient who goes blind is therefore in double jeopardy.

The extent of ocular involvement is dependent on the immune status of the patient.[30,31] The mechanisms involved in ocular damage are:

- ⇒ Direct invasion of the eye with the resultant atrophy of the anterior segment structures, which occurs in MB leprosy.
- ⇒ Leprae reactions, which may lead to trigeminal and facial nerve involvement for type I (reversal) reaction; and acute iritis, episcleritis and scleritis, for type 2 reaction (ENL). The involvement of the cranial nerves V and VII, in turn lead to corneal hypoesthesia and lagophthalmos respectively. The corneal exposure associated with lagophthalmos predisposes the patient to corneal injury and ulceration.
- ⇒ Changes in the skin and support tissues of the lids, the tear secretion and drainage systems.
- ⇒ Bacterial superinfecton, which is always a risk in a chronically affected eye.[25,30,32]

Direct invasion and ENL occur in the lepromatous form of the disease, while the reversal reaction occurs in all forms of borderline leprosy (BT, BB, BL). Thus, all forms of the disease may cause ocular complications.[4] Some, however, are more likely than others to give rise to serious visual symptoms and are therefore referred to as 'potentially sight-threatening' (PST).[4,33,34] The important PST lesions include lagophthalmos, corneal anaesthesia, exposure keratopathy, scleritis, staphyloma, acute and chronic iridocyclitis, low intraocular pressure (ocular hypotony), and cataract.[4,31,32,35,36] Other complications such as nodules and madarosis are visually insignificant. They generally reflect a high bacillary load.[4,31,35]

In 1988, WHO estimated that approximately 250,000 leprosy patients were blind worldwide.[4,30] Almost a decade latter, in 1997, Courtright and Lewallen,[4] based on their review of available data, estimated that 350,000 to 400,000 leprosy patients are blind worldwide. They also estimated that 1.5 to 2% of leprosy patients would be blind from the disease itself while an additional 2% would be blind from other causes, particularly age-related cataract. They noted that the three major causes of visual disability among leprosy patients are corneal disease (most often secondary to lagophthalmos and ectropion), uveal disease (in particular, chronic uveitis) and cataract.

Taking the lesions in anatomical sequence, the following eye complications may be seen in leprosy.[2,4,21,30,32]

1. **Eyebrow**

   Superciliary madarosis (or loss of eyebrow) occurs through direct invasion by *M. leprae* in MB leprosy. It begins from the outer third of the eyebrow and spreads medially. It is usually symmetrical. Treatment is usually by the use of eyebrow pencils. Sometimes surgical eyebrow transplants are done.[2,3]

2. **Eye lids**

   Ciliary madarosis (or loss of eye lashes) is common in MB leprosy because of the destruction of hair follicles.[2,4,18]

   Thickening of the eyelids occurs in all forms of the disease.[2,18]

   Nodules: Small nodules may be found on the eyelids in MB leprosy. The lower lid and skin fold of the upper lids are usually spared and are known as the 'immune areas'. Treatment is by supervised MDT.[18]

   Blepharochalasis may occur in MB leprosy following massive infiltration and secondary atrophy of the eyelid tissues. There is loss of tone and the eyelid becomes 'baggy'. Treatment is by surgical correction.[32,33]

   Trichiasis (or misdirection of the eyelashes) may also occur. This can traumatize the already anaesthetic cornea. Treatment is often by manual epilation, however, where a whole line of lashes is touching the cornea, surgical correction is necessary.[25,31,32]

   Facial nerve involvement: This occurs in all forms of leprosy. It is more severe in PB leprosy and more gradual in MB leprosy.[2,25,32] The results of the facial nerve involvement include the following:

   a. Leprotic stare: This results from loss of facial expression, which is usually bilateral.[4,36]

b. Lagophthalmos, which may lead to exposure keratitis, corneal ulceration and resultant opacification.[2,4,25,29,30,32] Hogeweg[2] believes that lagophthalmos is the most common eye complication in leprosy and suggests that in leprosy-endemic countries leprosy should always be considered in the differential diagnosis of Bell's palsy. Leprosy patients with lagophthalmos usually have a history of swelling of the face (reversal reaction) and may still have visible skin patches. A standard grading for lagophthalmos has been adopted as follows:[37]

Grade 1 = Normal

Grade 2 = Obicularis muscle weakness

Grade 3 = Lid gap with cornea covered in mild closure

Grade 4 = Lid gap with cornea exposed in mild closure

The surgical treatment for lagophthalmos is most commonly by simple lateral tarsorrhaphy.[2, 30, 32] Some plastic surgeons may advise temporalis muscle transfer (TMT) surgery.[2,30] Kuntheseth[38] recently reported a new surgical technique, which involves re-animating eyelids with lagophthalmos by stainless steel weight implantation. This procedure is based on the fact that the upper eyelid provides significantly more of the actual closure in covering the cornea. The weights are implanted into the upper eyelid for the purpose of re-animating the obicularis oculi muscle. The principle for this approach was first described in 1958 by Illing,[39] who used gold weights to achieve the re-animation.

c. Ectropion follows sagging and eversion of the lower lid. Ectropion in turn leads to impaired drainage of tears and therefore epiphora.[36]

3. **Lacrimal system**

Blockage of the lacrimal sac may result from infiltration of the nasal mucosa and collapse of the nose in MB leprosy.[2,31] It is usually characterized by dacryocystitis.[25,32]

Chronic dacryocystitis is the most common type among leprosy patients. It is usually painless and there may be no swelling to attract attention. The condition should be suspected if the eye looks normal except for persistent epiphora sometimes associated with copious exudates which increase if digital pressure is applied over the lacrimal sac. The treatment is usually conservative. It involves daily emptying of the lacrimal sac by asserting pressure, irrigating the lacrimal sac and applying some antibiotic solution, and the twice-daily use of eye drops containing 0.25% zinc sulphate and 5% boric acid. Sometimes surgical treatment (dacryocystectomy) is necessary.[32]

Acute dacryocystitis is usually associated with pain and swelling between the eye and nose; and may also be associated with fever and malaise. Treatment is with systemic antibiotics and local hot compress.[32]

Subacute dacryocystitis is less dramatic, however, it is also associated with swelling of the tear sac and tenderness. Treatment is by use of systemic antibiotics in addition to emptying of the lacrimal sac once daily by applying pressure and also irrigation of the lacrimal sac using a lacrimal canula and syringe.

4. **Conjunctiva**

The conjunctiva may show non-specific conjunctivitis or exposure changes.[36]

5. **Sclera**

Episcleritis and scleritis may be associated with erythema nodosum leprosum (ENL). In episcleritis, there is localized redness of the eye with some eye irritation. There is no associated intraocular pain.[32,36] Scleritis is associated with pain and deep redness. Prolonged recurrent scleritis causes thinning of the sclera and bluish uveal tissue may become visible.[25,32] This may also result in staphyloma.

6. **Cornea**

a. *Limbal leproma:* This is a nodule arising in the superficial tissues at the corneo-scleral junction. It usually occurs on the lateral side and may grow to encroach on the cornea. Lepromas are a sign of relapse or drug resistance.[35]Treatment is by supervised MDT.

b. *Beading of the corneal nerves* may be seen in the periphery of the cornea, under slit-lamp examination as focal areas of thickening of the nerves.[35]

c. *Punctuate avascular keratitis* (chalky corneal deposits): This is an early sign of corneal invasion by *M. leprae* bacilli.[30] It may not be associated with changes in corneal sensation.[30] They appear as faint discrete superficial opacities in the upper outer quadrant of the cornea. Histologically they consist of clumps of bacilli-laden cells, which become dense white, resembling specs of chalk dust (corneal pearls).[37] When they coalesce with disease progression, a diffuse haze occurs. This keratitis is usually asymptomatic.[35]

d. *Corneal pannus:* This occurs when the superficial limbal blood vessels grow into the cornea following

repeated or severe avascular keratitis. Typically, this vascularization begins in the upper temporal quadrant of the cornea and extends into the cornea superficially.[17,32,35]

e. *Exposure keratitis:* This is a secondary corneal change resulting from lagophthalmos and ectropion. It may be associated with trauma and infection. It is an important pathway to blindness in leprosy.[35,40] Secondary prevention of blindness is possible by early diagnosis and treatment of lagophthalmos and /or ectropion, use of artificial tears, antibiotic ointments, patching of the eyes especially at night, use of eye shields and timely think-blink habit.[30,32,35]

f. *Absent or reduced corneal sensation* (corneal anaesthesia or hypoesthesia respectively) results from trigeminal nerve involvement, especially in longstanding MB leprosy.[32] It can be simply detected with a cotton wisp applied to the cornea from the side, which should induce reflex blinking. Quantitative measurements are also possible by using the Cochet and Bonnet esthesiometer.[25] There are several ways in which decreased corneal sensation can produce damage to the corneal surface of a leprosy patient:

i. The sensory supply to the cornea is regarded as the sentinel of the eye. Corneal anaesthesia predisposes a patient to corneal injury because of the loss of the warning signal of pain. Thus foreign bodies and misdirected lashes (trichiasis) can easily damage the cornea before the patient becomes aware of their presence.[25]

ii. Intact corneal sensation is important for reflex tearing.[25,41] Thus corneal anaesthesia leads to decreased tear secretion. A dry eye could give rise to epithelial damage and intense itching that could induce excessive rubbing of the eyes by patients. This will further aggravate the existing eye damage.

iii. Corneal anaesthesia is also associated with decreased acetylcholine uptake by the cells in the neurotrophic keratitis.[25,42]

iv. The involuntary blink rate is also reduced in corneal anaesthesia thus encouraging drying of the precorneal tear film.

7. **Anterior chamber**

Anterior chamber examination may reveal signs of anterior uveitis, which include: aqueous flare, cells, or iris pearls, which have dislodged into the lower anterior chamber angle. Secondary closure of the anterior chamber angle by peripheral anterior synechiae (PAS) may follow, and lead to raised intraocular pressure and glaucomatous optic nerve damage.[25,35]

8. **Iris**

Iris pearls: These are rare, but pathognomonic of leprosy. They appear as small white granules extruding from the iris stroma. They are formed of dead calcified leprosy bacilli. The appearance of iris pearls is an evidence of invasion of the anterior uvea by leprosy bacilli. With time these iris pearls dislodge into the lower anterior chamber angle and either get absorbed or cause peripheral anterior synechiae.[30]

Acute iritis is usually associated with ENL. The presentation is similar to acute forms of iridocyclitis with the associated pain, photophobia and ciliary flush. Posterior synechiae may result.[40]

Chronic plastic iritis or neuroparalytic iritis is an insidious form of iritis, which occurs in MB leprosy. It is also an important pathway to blindness in leprosy. The *M. leprae* organism attacks the autonomic nervous supply to the iris especially the sympathetic supply to the dilator pupillae. This leads to a regular pinpoint pupil, which is usually difficult to dilate. The small pupil exaggerates the effects of any small lens opacities. Treatment is first by maximal dilatation of the pupil using 10% phenylepherine. This is sometimes unsuccessful, in which case a broad sector iridectomy will become necessary as well as inferior sphincterotomy at 6 o' clock position to prevent the pupil from becoming updrawn.[17, 30, 32,35,40]

Iris atrophy occurs in long-standing MB leprosy. It is associated with neuroparalytic iritis. The iris may have a 'moth-eaten' appearance as the result of patchy iris atrophy, which may cause full thickness iris holes (polycoria). In addition, the pupil may become decentred, being typically drawn towards the lower temporal limbus. The pupil may also be enlarged due to progressive atrophy of the iris sphincter.[18,30]

9. **Ciliary body**

The ciliary body is probably the port of entry into the eye for the leprosy bacilli.[30] The involvement of the ciliary body may lead to loss of accommodation and low intraocular pressure, especially in the later stages of MB leprosy. Low intraocular pressure results from atrophy and hyalinization of the ciliary body.[18, 30] *Very* low intraocular pressure could lead to choroidal detachment and phthisis bulbi.

10. **Lens**

Cataract in leprosy patients can be age-related; steroid-induced or complicated.[43] Treatment of cataract is by cataract extraction as in the general population. The use of posterior chamber intraocular lens implants, following extracapsular

cataract extractions is now widely accepted. A cautious approach by appropriate use of topical, subconjunctival and systemic corticosteriods is generally advised to limit postoperative inflammation.[44]

11. **Fundus lesions**
Fundus lesions are rare in leprosy.[2,4,30,31,32,35,36] However peripheral choroidal lesions, retinal vasculitis and papillitis have been documented.[18,31]

SYSTEMIC TREATMENT

The systemic treatment of leprosy has undergone a lot of progress. The discovery of 4, 4-diaminodiphenyl sulphone (DDS) or dapsone, revolutionalized the treatment of leprosy. It was introduced into India, Nigeria and Brazil in the late 1940s.[7] Treatment with dapsone monotherapy was for life. With time, resistance to dapsone by *M. leprae* became a problem, leading WHO to adopt the multidrug therapy (MDT) in 1982, with the hope that the use of the three drugs: rifampicin, clofazimine, and dapsone, will preclude the development of resistance to any of the drugs. Initially the MDT was for 2 years for MB leprosy but it has now been shortened to 1 year for MB leprosy and 6 months for PB leprosy.[45]

Newer drugs have been developed for the treatment of leprosy. These include ofloxacin (a fluroquinolone), clarithromycin (a macrolide), minocycline (a tetracycline) and rifapentine (a rifampicin). These newer drugs have the potential for increased effectiveness and shortened duration of antileprosy chemotherapy. In addition they may prove useful against strains of *M. leprae* that are resistant to the drugs currently in use, especially rifampicin.[11] The therapy of these newer drugs, i.e., rifampicin, ofloxacin and minocycline is referred to as the 'ROM' regime.[45]

The current WHO recommended MDT is as follows:[2]

A: Adults

1. PB leprosy (single skin lesion)
- Rifampicin 600mg as a single dose
- Ofloxacin 400mg as a single dose
- Minocycline 100 mg as a single dose

2. PB leprosy (2-5 skin lesions) (period of treatment: 6-9 months)
- Dapsone 100mg, unsupervised, once daily
- Rifampicin 600mg, supervised, once per month; 6 doses

3. MB leprosy (i.e.>5 skin lesions) (period of treatment: 12 to 18 months)
- Dapsone 100mg and clofazimine (lamprene) 500mg unsupervised once daily
- Rifampicin 600mg and clofazimine 300mg, supervised once per month; 12 doses.

B: Children

Adult doses are adjusted according to age and/or body weight.[28] Clinical trails of vaccines have shown good results for BCG vaccination, especially booster doses.[7,46] and mycobacterium W (Mw) vaccine; but not for killed *M. leprae* vaccines.[46,47]

CONCLUSION AND RECOMMENDATIONS

Though the prevalence of leprosy is reducing, its incidence is increasing. Many 'cured' leprosy patients are living with ocular complications that could lead to blindness. Most of these blinding complications are preventable by early diagnosis and prompt treatment. It is therefore recommended as follows:

1. Every health worker, particularly ophthalmologists, should be familiar with the ocular complications of leprosy.
2. Periodic screening and surgical outreach programmes by ophthalmologists should work in collaboration with leprosy care programmes with a view to treating avoidable causes of blindness especially cataract, uveitis, and lagophthalmos.
3. Funding for research and service delivery for leprosy should not cease because available data show that leprosy is still a cause for concern.

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