Review Article

A REVIEW OF THE OPHTHALMIC MANIFESTATIONS OF LEPROSY

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ABSTRACT

Aim: To review the ocular and adnexal manifestations of leprosy.

Methods: Literature was obtained from internet search of various sites including Entrez PubMed, Google Scholar, Hinari, African Journals Online etc. Information was also obtained from textbooks, leprosy registries and dissertations of the Faculty of Ophthalmology, National Postgraduate Medical College of Nigeria.

Results: Leprosy is a chronic granulomatous disease of low grade infectivity in man caused by *Mycobacterium leprae.* It primarily affects the peripheral nervous system and secondarily involves the skin, eye, upper respiratory tract and testes. The causative organism is slow growing and has preference for cool temperature less than 37°C. Therefore its ophthalmic manifestations are more in the cooler ocular adnexa and anterior segment of the eye. Tuberculoid leprosy was the most common form of leprosy in Nigeria. Leprosy has the highest incidence of ocular involvement than any other infectious disease of man, usually leading to low vision or blindness. Sight threatening complications usually are cataract, lagophthalmos, corneal anaesthesia, exposure keratitis, corneal opacities, neuroparalytic keratitis and chronic uveitis. Ophthalmic manifestations include mdarosis, lagophthalmos which can be complicated by recurrent corneal ulceration, resulting in corneal scarring, perforation and panophthalmitis, others are conjunctivitis, episcleritis and scleritis which can cause staphyloma. Keratitis, loss of protective corneal reflex and damage to superficial nerves, chronic uveitis, loss of accommodation, low or elevated intraocular pressure, secondary glaucoma and phthisis bulbi are more of the ocular complications seen.

Conclusion: Leprosy affects the adnexal and anterior segment structures of the eye and may result in several sight threatening complications, which can lead to blindness that will further worsen the quality of life of the person suffering from leprosy.

Keywords: Leprosy, Granulomatous disease, Cataract, Uveitis, Lagophthalmos, Madarosis.

INTRODUCTION

Leprosy is a chronic granulomatous disease of low grade infectivity in man caused by *Mycobacterium leprae.*[1,2] The earliest accurate description of the disease came from an Indian manuscript dated 600 BC.[1,2] In 1873, Gerhard Amauer Hansen, a Norwegian physician isolated leprosy bacilli from lepromatous nodules.[1,3] The disease is therefore sometimes called Hansen's disease. Albert Neisser of Germany in 1879 stained the organism with fuchsin and gentian violet.[4] This was before Robert Koch demonstrated the tuberculosis bacilli.

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Leprosy primarily affects the peripheral nervous system and secondarily involves the skin, eye, upper respiratory tract and testes.[3,5] The causative organism is slow growing and has preference for cool temperature less than 37°C. These organisms are engulfed but not killed by macrophages. Within the eye it is usually found in the slightly cooler anterior segment. It does not kill the affected individual but cripples and blinds him.[4] The stigma attached to the disease causes the patients to hide their ailment and also live in seclusion.

Leprosy is endemic in some tropical countries of Africa, South East Asia, Central and South America.[3,5] Some of the countries with high number of registered patients include India, Brazil, Bangladesh, and Nigeria, with India accounting for 80% of cases worldwide.[5] The *Mycobacterium leprae* (*M leprae*) is an obligate intracellular acid- fast bacillus (AFB) multiplying mainly inside the macrophages of the skin (histiocytes) and nerve cells (Schwann cells).[2,3,5] It closely resembles the bacillus causing tuberculosis. However, *Mycobacterium leprae* is less acid- fast than *mycobacterium tuberculosis.*[2,4] The organism can thrive at temperatures below the normal body temperature. In the eye they are present in the iris, cornea, sclera and eyelids.[3]

It is not clear how the leprosy organism is contracted by humans. It is reported that in some cases it follows close contact with an active open case, who may excrete large numbers of organism from nasal discharge.[3,4] These may enter directly through the skin or inhaled as droplet in the air, or possibly by mosquitoes and biting flies.[3,5] The incubation period ranges from 3 months to 40 years, the average being 5 years.[2] The youngest recorded case of leprosy was in an approximately 3 months old Japanese female.[2,4] The clinical appearance in the individual varies according to the individual's cell mediated immunity. The prevalence of leprosy has reduced drastically since the introduction of the multidrug therapy (MDT).[4,5]

The World Health Organization (WHO) in 1982 estimated that the number of leprosy patients reduced from 12 to 10 million worldwide.[5] The number of registered cases fell worldwide from 5.4 million in 1985 to 750,000 by the year 2002. In May 2002, the WHO announced that leprosy has been eliminated as a public health problem at a global level, while individual countries were expected to achieve this by the end of 2005.[5] New cases diagnosed globally each year rose from 550,000 in 1985 to 759,000 (approximately equal to the global prevalence) in 1999.[5] The number of new cases by the year 2000, was 719,330 with children comprising 15% cases, which indicates that the transmission of the disease is dropping.[2,5] This is believed to be as a result of introduction of multidrug therapy.[2,5]

In Nigeria, the prevalence of leprosy reduced from 250,000 registered cases in 1989 to 7,000 registered cases by 19995. The prevalence is higher in the Northern part of the country with a few pockets in the South.[2,4,5] It is associated with a considerable social stigma, fear and ostracization[2], of which most sufferers seek solace in settlement built by missionaries and voluntary organizations.[6] It is estimated that three fourth of one million of the world leprosy population are blind.[7] The prevalence of blindness among these patients depends on several factors which include:

i. The climate: leprosy eye lesions tend to be more severe in colder climates.
ii. The predominant disease type; it is generally observed that most eye complications occur in advanced lepromatous disease.
iii. The quality of case finding and follow up; eye complications in the first 4 or 5 years of lepromatous disease are common. Most at risk in this period are the immunologically unstable patients who may suffer sudden nerve damage. A good programme of case finding, identifying those most at risk will further reduce the incidence of leprosy.
iv. The prevalence of other diseases causing blindness such as trachoma, onchocerciasis and nutritional deficiencies, since some of these diseases may coexist with leprosy.[8]

Eye complications in leprosy could result from some factors, which include; invasion of anterior segment structures by *Mycobacterium leprae*, inflammatory reaction of the anterior segment tissue and sequaelae, impaired sensation of the cornea and conjunctiva, paresis of the orbicularis oculi, damage to the extraocular structures, skin, lacrimal system and secondary infection by other types of bacteria or viruses. The problems

of leprosy include physical disabilities, economic loss, psychological trauma and social ostracism.[5] In most countries, especially the developing countries of Africa, leprosy cases with eye problems are treated under the leprosy services where little or no ophthalmic services are available. A leprosy patient requires good eye sight to guard against injury to his anaesthetic hands and feet. An already physically, economically, socially and mentally disabled leprosy patient with visual disability provides a most unhappy picture of severe handicap.[9] It is important that leprosy patients have as much sight as possible because their hands and feet are anaesthetic.[10] Sight will help to protect them from accidents which will further incapacitate them.

EPIDEMIOLOGY

The prevalence of leprosy has reduced drastically since the introduction of multidrug therapy (MDT) by the World Health Organization (WHO) in 2010.[11] The WHO estimated that the prevalence of leprosy in the world in 1966 and 1976 were 10.8 and 12 million respectively.[5,11] In 1966, registered cases were about 2.8 million.[2,11]

By the end of 2000, leprosy was a public health problem in only 15 countries (prevalence rate greater than 1/10,000) mainly in Africa, Asia and Latin America. These include Algeria, Brazil, Nepal, Paraguay, Liberia, Congo, Côte de Ivoire, Tanzania, Guinea, Niger, Mozambique and Madagascar. At the beginning of 2010, the global registered and reported point prevalence was 211,903 cases; in 2009, 244,796 new cases were detected.[11]

PREVALENCE OF LEPROSY IN NIGERIA

The incidence of leprosy in Nigeria has also reduced from 17.3/10,000 population (250,000 registered patients) in 1989 to 0.6/10,000 population (7,000 registered patients) by 1999.[5] Ross in 1956 in a survey in the Northern Nigeria recorded a range of 1.6 to 9.8 per 1000.[12] Ayanru[10] reported that tuberculoid leprosy was the most common form of leprosy in Nigeria accounting for 75.4% of his patients. Malu[13] estimated 5 per 1000 population for Kaduna State of Nigeria and 3.4% for Borno State of Nigeria in1995. At Uzoakoli Leprosy Centre in Abia State of Nigeria, the number of registered new cases in 2007, 2008, 2009 and 2010 were 28, 46, 29 and 26 patients respectively. These figures were obtained from the hospital's leprosy registry.[14]

PATHOGENESIS OF LEPROSY

The leprosy bacillus measures about 0.3-0.4 microns by 4-7 microns and thrives very well in temperatures lower than 37^{0}C. This explains why the 9 banded armadillo specie, which is a cold blooded animal with body temperatures between 33^{o}C and 34^{o}C is a natural host of the organism. It can also be cultured in the mouse foot pad.[5] The incubation period before the commencement of the symptoms seems to be 2-7 years.[3] Leprosy bacillus multiplies very slowly and takes about 2 weeks to double their number when growing in an ideal situation. By contrast most bacteria will double their number every 20 minutes.[4] It is likely that infected monocytes from the mucous membrane or broken skin transfer the organism into the nerve during movement of macrophages through the nervous system.[3,5] Factors that help the growth of the organism include nerve density, pH of the tissue and presence of 3,4-dihydroxyphenylalanine (DOPA) which acts as a metabolic substrate for the *M*. *leprae*.[4,5]

CLASSIFICATION

There are three main classifications of leprosy.[4,11]

1. The Madrid classification classifies leprosy into:
   a. Polar tuberculoid leprosy
   b. Borderline tuberculoid leprosy
   c. Lepromatous leprosy
2. The Ridley and Jopling classification (otherwise known as WHO classification).[4,5]
   a. Polar tuberculoid leprosy
   b. Borderline tuberculoid leprosy
   c. Borderline borderline leprosy

d. Borderline lepromatous leprosy
e. Polar lepromatous leprosy
3. An alternative classification also includes:
a. Paucibacillary (PB) leprosy; this includes polar tuberculoid and borderline tuberculoid grouped together.
b. Multibacillary (MB) leprosy; which includes borderline borderline leprosy, borderline lepromatous leprosy and polar lepromatous leprosy.

This 3rd classification was introduced for the multidrug therapy (MDT) programme and was further simplified by 6th Expert Committee on Leprosy in 1988.[5,11] It is a clinical classification based on skin smears (bacteria index) and skin lesions. The polar tuberculoid (PT) and Borderline tuberculoid (BT) of the Ridley and Jopling classification are now grouped as PB leprosy while the borderline borderline (BB), borderline lepromatous (BL) and polar lepromatous leprosy (PL) are grouped as multibacillary leprosy.[4] The stage of the disease compares with the level of the cell mediated immunity of the patient. A leprosy patient with low immunity tends to shift towards multibacillary leprosy. On the other hand, if the immunity is high, the disease tends towards paucibacillary leprosy or may even heal spontaneously. Various receptor mediated mechanisms may play a role in the invasion of human Schwann cells by *Mycobacterium leprae* especially the FC receptor on the immunoglobulin molecules, compliment receptors, fibronectin binding protein and It has also been observed that *M*. *leprae* has special affinity for the G domain of the alpha (α) chain of laminin-2 (laminin 2 alpha) and extracellular matrix protein present in basal lamina of Schwann cells. This *M*. *leprae* laminin 2 alpha complexes then binds to α/β dystroglycan complexes which is expressed only on the peripheral nerves. This may explain why *M*. *leprae* does not infect the central nervous system.[4,5] The cell mediated immunity developed in response to the infecting organism leads to the formation of granulomatous lesions around the nerve tissue, causing irreversible damage to the nerve.[4] The lesions are similar to those seen in tuberculosis with epithelioid and Langerhans giant cells.

REVIEW OF MUTIDRUG THERAPY

Multidrug therapy (MDT): this was recommended by WHO in 1981 for the treatment of leprosy.[11]

Treatment of paucibacillary leprosy

- Rifampicin 600mg for patients weighing over 35kg, 450mg for 35kg once a month for 6 months.
- (Administration of Rifampicin should be under supervision). Dapsone 100mg daily (unsupervised).
- Treatment should be continued for 6months

Treatment of multibacillary leprosy

- Rifampicin 600mg once a month (supervised).
- Dapsone 100mg daily (unsupervised).
- Clofazimine 300mg once a month (supervised) and 50mg daily (unsupervised)
- Treatment should be continued up to 1 year and whenever possible until skin smear is negative.

GENETIC FACTORS

Although leprosy is not a hereditary disease, there is increasing evidence that genetic factors may predispose certain individuals to overt disease. Recent gene linkage analysis data revealed association between leprosy susceptibility and genetic markers on chromosomes 10 and 2. There is a significant association between DR2 allele and leprosy in Asia and Africa.[5] Susceptible genes have been observed in certain families in India, although the genetic loci has not been identified.[5]

ENVIRONMENTAL/RACIAL FACTORS

Europeans and Asians appear to have more of lepromatous lesions while tuberculoid type is common among Africans. It is also more common

in areas with poor standard of living, poor hygiene and overcrowding.[4]

GENDER

It has been noted that leprosy affects more males than females, the sex difference is greater in adults than in children, but the reason is not known.[4,15] Ayanru[10] in a survey of 179 in-patients carried out in Ossiomo Leprosy Settlement in the Mid-Western State of Nigeria reported that there were 115 males (64.25%), 51 females (28.49%%). On the contrary Wade[2] had 52.3% females and males were 47.70% in her research carried out at Plateau State of Nigeria. While Waziri-Erameh and Omoti[1] reported 65% for males and 35% for females in Eku Leprosy Centre, Delta State of Nigeria.

AGE

Leprosy affects all ages. The peak appears to have a bimodal distribution.[1] The first peak is between 10 and 14 years. The second peak occurs between 40 and 70 years of age.[1,10] Also it varies according to endemicity and type of disease.[6,16] It was found that children are more susceptible to the disease because of their closeness to their infected parents. Adults tend to acquire immunity with increasing age. It has been noted that the tendency to blindness increases with the duration of the disease and age of the patient.[17] Ayanru and Elebesunu[17] reported that 11 patients who were blinded in both eyes by leprosy had had the disease for more than 15 years and were at least over 40 years.

OCULAR INVOLVEMENT

The mechanisms involved in ocular damage include.[4,5]

- Direct invasion of the eye with resultant atrophy of the anterior segment structures which occurs in multibacillary leprosy.
- *Leprae* reactions which may lead to (a) trigeminal and facial nerve damage from type I (reversal) reaction and (b) acute iritis, episcleritis and scleritis from type 2 reaction (erythema nodosum leprosum- ENL).
- The involvement of trigeminal and facial nerves in turn leads to corneal hypoaesthesia and lagophthalmos respectively.
- The corneal exposure associated with lagophthalmos predisposes the patient to corneal injury, ulceration and its sequelae.
- Bacterial superinfection which is always a risk in chronically affected eye.

Leprosy is a systemic disease which often affects the eye.[1,3] Leprosy has the highest incidence of ocular involvement than any other infectious disease of man.[4] These usually lead to low vision or blindness.[18] Swapan et al[16] reported 15% ocular involvement. In another study by the same author in RFT group, they reported 52% of ocular involvement, 7% of which developed from active leprosy while 28% developed after cure.[19] Waziri-Erameh and Omoti[1] reported 9.6% ocular involvement in RFT group in their study of 60 patients in Eku Leprosy Centre (Delta State of Nigeria). Borros[20] reported 64% in lepromatous leprosy while 15% was seen in tuberculoid leprosy among 1279 leprosy patients in his study in India. Rodgers[21] also in India documented 5 – 10% of leprosy patients with ocular involvement. Ayanru[10] in a study at Ossiomo Leprosy Centre, Edo State of Nigeria reported ocular involvement in 21.27% out of 69 leprosy patients seen. Nwosu and Nwosu[22] reported 63% of ocular involvement in their study in 4 out of 5 leprosy clinics in Anambra State of Nigeria. Ogbonnaya et al[5] reported 72% of ocular involvement. Shield et al[23] reported 72% in Brazil while Harley[24] in Panama reported 10.0%. Beldarraín-Chaple[25] reported 10% in Cuba, Shoshamma et al[26] in India reported 24%. The variation of these figures reported in ocular involvement may be due to different sample size and instruments used by the researchers. It shows that ocular involvement rate is a range.

AGE RANGE

Waziri-Erameh and Omoti[1] reported 9-80 years. While Ayanru[10] reported that peak incidence occurred between 41–50 years. Male to female ratio was 2:1. Ogbonnaya et al[4] reported a bimodal distribution. The first peak occurred

between 10 and 14 years while the second peak occurred over 30 years of age.

TYPES OF LEPROSY:

Ayanru[10] reported 75% of tuberculoid leprosy and 25% lepromatous leprosy. Likewise, Swapan et al[16] in West Bengal reported 75% paucibacillary leprosy and 25% multibacillary leprosy. On the other hand, Borros[20] documented 64% lepromatous leprosy, 20% tuberculoid leprosy and 15% mixed (Borderline borderline). The different percentages reported by the above groups could be due to different sampling methods, and the size of the population studied.

VISUAL IMPAIRMENT/ BLINDNESS

Waziri-Erameh and Omoti[1] found that incidence of blindness was high in leprosy patients recently released from treatment. They reported blindness in 15 % of leprosy patients. Ayanru[10] reported that 44% of the lepromatous leprosy patients seen were blind. Nwosu and Nwosu[22] documented 8.7% blindness in Anambra State of Nigeria. Ogbonnaya et al[4] reported a total of 33.0% with blindness in one eye or both eyes. Nguyen[27] in India among 403 patients had 9.9% of blindness and 24.1% of visual impairment. Swapan et al[16] reported 1.5% blindness in their survey in West Bengal while Malu[13] in Kaduna (Northern Nigeria) reported impaired vision or blindness in 7.4% leprosy patients. The difference in their findings could be attributed to their sample size and regional variations.

CAUSES OF BLINDNESS

Malu[13] noted that causes of blindness in leprosy patients included exposure keratitis and chronic uveitis. Exposure keratitis is usually secondary to lagophthalmos. Nwosu and Nwosu[22] attributed the causes of blindness in their patients to cataract, exposure keratitis, and choroidal lesions, while Waziri-Erameh and Omoti[1] documented causes of blindness to be cataract 33.33% (most common), bilateral corneal ulceration with trichiasis in 11.11%, and open angle glaucoma.

SIGHT THREATENING COMPLICATIONS

Waziri-Erameh and Omoti[1] found that the incidence of potentially blinding conditions due to leprosy was seen in 70% of patients examined, while Nwosu and Nwosu[22] reported 43%. Ogbonnaya et al[5] highlighted the sight threatening complications as cataract, lagophthalmos, corneal aneasthesia, exposure keratitis, corneal opacities, neuroparalytic keratitis and chronic uveitis. Ayanru and Elebesunu[17] reported leprotic uveitis in 6.8%. Uveitis was a major sight threatening condition observed and this was only seen in patients with lepromatous leprosy.

EYE BROW AND EYE LASHES

The eyelids and anterior segment structures of the eye are the ones most generally affected by leprosy. This is because a link has been demonstrated between cool temperature and the growth and pathogenicity of *M. leprae.*[4,5] In experimental animals, it has been noted that there is a gradient of 6^0C between the cornea and retina.[20] The eye lids act as protective mechanism from excessive light, foreign particles and direct sweat from entering into the eyes. In leprosy, notably but not exclusively in the multibacillary type, the skin and subcutaneous tissues are infiltrated. The brow hair may be lost and there may be elastic tissue atrophy, leaving bald, somewhat sagging skin.[3] A bald brow suggests that the patient has the type and duration of disease consistent with ocular pathology but does not necessarily have leprosy.[3]

Madarosis which is loss of eyebrow and eyelashes is one of the commonest eye changes in lepromatous leprosy. It is usually symmetrical and more common in the lateral part of the eye brow.[8] Shield et al[23] reported bilateral madarosis in 59 cases while loss of cilia occurred in 44%. Daniel et al[28] documented madarosis in 40% of leprosy patients. Madarosis was also reported in 40% by Dana et al.[29] The presence of madarosis was related to the duration of the disease. Richard et al[30] in the United States of America

reported madarosis in only 5% of his study population. Waziri-Erameh and Omoti[1] documented madarosis in 31.67%. All authors seemed consistent except Richard et al.[30] Seghal et al[31] in India found trichiasis and blepharitis ocuring simultaneously in 3 cases among 430 leprosy patients, reflecting very low occurrence in their study.

FACIAL SKIN/FACIAL MUSCLES AND FACIAL PALSY

Lagophthalmos may lead to exposure keratitis, corneal ulceration and resultant opacification.[5] It could even progress to endophthalmitis or panophthalmitis.

A standard grading for lagophthalmos has been adopted as follows[11],

Grade 1 - normal

Grade 2 - orbicularis muscle weakness

Grade 3 - lid gap with cornea covered in mild closure

Grade 4 - lid gap with cornea exposed in mild closure

The facial nerve which is a motor nerve to the orbicularis oculi and other facial muscles may be involved especially in tuberculoid leprosy or borderline leprosy.[3,4] There is inflammatory change in the nerve and this may cause facial palsy. This nerve and other superficial nerves may be thickened and felt under the skin. The anaesthetic or depigmented patch of skin on the face is extremely suspicious of leprosy.[3] The facial palsy which prevents blinking and eyelid closure destroys the motor part of the blink reflex, this causes corneal damage which is usually present when there is corneal anaesthesia and or facial palsy. Keratitis and corneal ulceration if untreated will progress to corneal scar or total destruction of the eye. Seghal et al[31] reported lagophthalmos in 2 cases, hypertrophy of the eyelids in 9 cases and facial skin nodules in 4 cases among 430 leprosy cases studied in India.

Shield et al[23] on the other hand, reported that an isolated palsy of the orbicularis oculi occurred in 13%. Dana et al[29] reported lagophthalmos in 11% while Waziri-Erameh and Omoti reported that one of the most common type of ocular lesions was lagophthalmos which was found in 16.67% of their cases. The sequelae of lagophthalmos include recurrent corneal ulceration, resulting in corneal scarring, perforation and panophthalmitis. All of these can cause visual impairment. Lubber et al[32] examined 57 patients with lagophthalmos and determined the extent of paralysis of the facial muscles. Up to 81% of the patients had involvement of at least one facial muscle group. Generalised weakness of the facial muscle makes the face to lose much of its expression and character. Shield et al[23] reported that thickening of the superciliary ridges, which in combination with weakness of the facial muscles give rise to leonine facie that was seen in 24% of cases. Moreover, the reason for the above variations in their findings could be due to the difference in sample size and type of leprosy seen.

CONJUNCTIVA / EPISCLERA /SCLERA CHANGES:

Conjunctiva may show non-specific conjunctivitis or exposure changes.[33] Episcleritis and scleritis have been noted in leprosy. Episcleritis may present with localized redness and irritation while scleritis presents with pain and deep redness.[17] Chronic scleritis may lead to staphyloma formation as a result of thinning of the sclera.[4]

CORNEAL CHANGES

There are two types of corneal involvement in leprosy.[2] The primary change is due to leprosy bacilli invading the cornea. This presents as beading and thickening of the corneal nerves as early sign, punctate keratitis and or interstitial keratitis which is a more extensive inflammatory reaction to the bacilli. The secondary one, result in visual loss due to loss of protective corneal reflex and damage to superficial nerves (these are the seventh and fifth nerves).The fifth nerve which supplies sensation to the cornea could be affected, resulting in corneal anaesthesia. Loss of protective blink reflex is secondary to facial nerve damage. Both nerve palsies (fifth and seventh nerve palsies) produce a situation in which the patient cannot blink, close the lids (causing exposure) and cannot feel if there is inflammation

of conjunctiva or cornea. They may even rub anaesthetic rough hands on anaesthetic cornea with ulcerations (painless) resulting.[3] Choyce[34] reported that 36% of leprosy patients had bilateral decreased corneal sensation.

The keratitis seen could present as pannus, sclerosing keratitis, superficial punctate keratitis, deep keratitis (or interstitial keratitis) or leproma of the cornea.[10] Shield et al[23] reported superficial keratitis in 19% of leprosy patients, which was bilateral in 11% and unilateral in 8%. On the other hand, Sektar et al[35] studied 89 cases of leprosy and found beaded corneal nerves in 5.6%, corneal hypoaesthesia in 28%, and dry eye in 13%. Beaded corneal nerves and stromal infiltration occurred mainly in the lepromatous group. Dana et al[29] found punctate epithelial keratopathy in 28%, corneal hypoaesthesia in 16%, pannus in 10%, prominent beaded corneal nerves in 7%, avascular keratitis in 5%, scleritis in 5% and interstitial keratitis in 3%. Nguyen[27] in his study of 403 patients in Vietnam reported corneal opacity in 15%.

Hierselaar et al[36] compared the corneal sensation in patients with leprosy with age matched controls and found that a significant corrrelation between the loss of power of the orbicularis oculi and the degree of corneal sensation loss could not be established and no significant decrease in corneal sensitivity was found in paucibacillary leprosy patients. Loss of corneal sensation could occur while there is no clinically detectable eye pathology, at least in multibacillary leprosy patients. Daniel et al[28] examined 383 patients and counted unmyelinated corneal nerves. He found that the visibility of these nerves was decreased in the nasal half of the cornea and the reduction in visibility of these nerves were more marked as the disease progressed from tuberculoid to lepromatous pole, the duration of the disease or the duration of anti-leprosy treatment did not alter the visibility of these nerves significantly.

ANTERIOR CHAMBER/ IRIS

Leprosy commonly involves the iris especially the multibacillary leprosy.[3,10] It can present as chronic gradual insidious inflammation or as an acute sudden severe inflammation. It usually affects the iris dilator at first leaving the sphincter action unopposed, thus the pupil becomes progressively constricted.[3] Iris pearls (whitish nodules) may appear on the iris surfaces, this being a diagnostic sign of leprosy.[4] Chronic iridocyclitis may occur with granulomatous mutton fat keratic precipitates on the corneal endothelium. These changes occur slowly and gradually in a quiet, white eye.[4,10] Reaction between systemic antibody and antigen may lead to acute iritis. This reaction is called erythema nodosum lepra (ENL) reaction and usually follows treatment or termination of therapy.[36] Rajak and Sanford-Smith[3] noted that there could be an acute iridocyclitis with a red eye, ciliary vasodilation, protein exudates and inflammatory cells in the anterior chamber. Any of the complications of acute iritis may develop, such as secondary glaucoma, pupil block, iris bombe, diminished secretions of aqueous fluid and phthisis bulbi. Permanent visual loss may result if not treated promptly.[3] Iris and ciliary body are the parts most frequently involved only second to cornea.[34] Ayanru[10] reported leprotic uveitis in 6.8% of his cases. Chronic iritis is usually found more in patients with lepromatous leprosy and is known to be the commonest cause of visual impairment or blindness.[10,36,37]

A quiet white eye is the characteristic feature of chronic iritis and a normal visual acuity does not exclude anterior segment activities.[38] Posterior synechiae was reported in all uveitic patients[10]. Various theories have been postulated regarding the cause of this uveitis, that the lepromatous uveitis is not inflammatory but neurotrophic in origin leading to slow atrophy of the iris dilator pupillae muscle, associated with low grade inflammation.[2,10] This chronic plastic iridocyclitis if untreated may lead to lowered intraocular pressure.[10,39] Some authors have indicated the need for vigilance in the quiet white eye in lepromatous leprosy.[10] Ayanru and Elebesunu[17] reported that iritis or iridocyclitis was seen in 23.6% which was exclusively present in lepromatous and borderline leprosy. Synechiae

formation and sometimes plastic exudation and phthisis bulbi were also noted.[34] These were some of the usual complications of anterior uveitis that contribute to blindness. Posterior uveitis is not usually seen.[10] Seghal et al[31] in India reported miliary leproma affecting the iris in 5 patients. Dana et al[29] in America found that 7% of their patients had iridocylitis and Sektar et al[35] also reported uveitis in 7.3% of 89 leprosy patients studied. The variation in the above findings could be due to the type of leprosy predominant in the regions studied.

INTRAOCULAR PRESSURE

The involvement of ciliary body leads to loss of accommodation and low intraocular pressure, mostly in multibacillary leprosy.[5] Atrophy and hyalinization of the ciliary body results in low intraocular pressure which could lead to choroidal detachment and phthisis bulbi[6]. Lewallen et al[39] in South Korea studied 241 leprosy patients and 138 age-matched healthy controls to determine the relationship between ocular autonomic dysfunction and intraocular pressure. Their findings showed that the mean intraocular pressures were significantly lower in the leprosy patient and the mean size of the pupil was significantly smaller in leprosy patients than in controls. There was no correlation between pupil size and intraocular pressure. They concluded that leprosy patients have autonomic dysfunction. On the contrary, Daniel et al[28] reported that the mean intraocular pressures in leprosy patients (13.6mmHg) did not differ significantly from the controls (13.1mmHg) in 166 leprosy patients and healthy controls. Only 1.5% of the leprosy patients had 7mmHg or less and the duration of disease did not affect the intraocular pressure. This was attributed to the use of multidrug therapy resulting in less ocular complications associated with intraocular pressure and that the occurrence of low intraocular pressure may not be as common as it was believed to be. Ayanru and Elebesunu[17] also noted normal intraocular pressure in all their patients except in one that had lepra reaction. Nwosu and Nwosu[22] also reported that some of the patients in their study had glaucoma in soft eyes, in an area where parasitic infections such as trachoma and onchocerciasis are considered to be of public health importance. Ayanru[10] reported 5 cases of secondary glaucoma in his study while Waziri-Erameh and Omoti[1] recorded one case of angle closure glaucoma.

LENS CHANGES: Cataract in leprosy can be age related, steroid induced or complicated. Sektar et al[35] reported cataract in 19% of their cases while Ayanru[10] found cataract in 6 patients out of sixty nine in his study. Treatment of cataract in leprosy patients by extra capsular cataract extraction and insertion of posterior chamber intraocular lens has been done successfully.[3,40,41]

FUNDUS CHANGES: Fundal lesions are not common in leprosy.[2,3] However, peripheral choroidal lesions, retinal vasculitis and papillitis have been reported by some researchers.[4,6,22]

Conclusion: Leprosy affects the adnexal and anterior segment structures of the eye and may result in several sight threatening complications, which can lead to blindness that will further worsen the quality of life of the person suffering from leprosy. Early diagnosis and treatment with multidrug therapy is recommended to reduce ocular complications.

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