OPHTHALMIC PRESENTATIONS IN LEPROSY PATIENTS IN (SOUTH – EASTERN) NIGERIA

By

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SUMMARY

Objective: To determine the magnitude and pattern of ocular disorders and blindness among leprosy patients, presenting at three leprosy clinics in South –Eastern Nigeria.

Methodology: All the in- patients, as well as the out- patients that presented to the 3 leprosy clinics during the 2- month period of the study were examined. Altogether, 171 patients were studied. All data were entered into the computer and analyzed using the SPSS software package.

Results: Ocular examinations revealed that 60.2% of the patients had leprotic lesions. Other findings were cataract 24.6%; pterygium 24.6%; refractive errors 21.6%; glaucoma 12.3%; age-related macular degeneration 4.6%; presumed toxoplasmosis 1.2%; optic atrophy 1.2% and squint 0.6%.A total of 10.5% of patients were blind and 39.8% visually impaired. Cataract accounted for 55.6% blindness.

Conclusion: It is concluded that non- leprotic lesions, particularly cataract were responsible for most of the blindness. We recommend that ophthalmic surgeons should organize regular and periodic surgical outreaches to leprosy centers with the aim of dealing with non- leprotic causes of avoidable blindness in such centres.

***Key Words**: Leprosy, Leprotic eye lesions, non-leprotic eye lesions, surgical outreaches.*

INTRODUCTION

The world health Organization (WHO) has made commendable progress towards the 'elimination of leprosy' (meaning reduction of prevalence to I per 10, 000 population). It has now embarked on the 'FINAL PUSH' towards leprosy elimination by the year 2005[1]. This has been made possible by the use of multidrug therapy (MDT), which WHO introduced in 1982. Thus the estimated number of leprosy patients has reduced from 10- 12 million (with 5.4 million registered patients) worldwide in the 1980s to 1.3 million active cases by 1998 [2,3]. Currently there are approximately 0.8 million registered patients worldwide.

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In Nigeria, the prevalence of leprosy has also reduced from 17.3 / 10, 000 population (250, 000 registered patients) in1989 to 0.6/10,000 population (7000 registered patients) 1999 [4].

However the incidence of leprosy has not reduced but is said to be increasing instead[1, 3, 4].Although over 10 million leprosy patients have been released from treatment (RFT), there is the new problem of many 'cured' leprosy patients who are living with disability, particularly deformities of the extremities and ocular disease [5] (See Plates 1& 2).

In 1998 WHO estimated that approximately 250, 000 leprosy patients were

blind worldwide [6]. Almost a decade later in 1997, Courtright and Lewallen [5] estimated that 350, 000 to 400, 000 leprosy patients were blind worldwide. We expect an increase in the eye care needs of leprosy patients as a result of successful cure and increased longevity[6].

Ffytche[7]has compiled results of surveys of ocular disease pooled from various parts of the world. There are variations in the prevalence of ocular disorders among the leprosy patients ranging from zero to 83.3%. Studies done in various parts of Nigeria [8-10] also show variations ranging from 21.27%[10] to 70% [11]. The present study was conducted to determine the magnitude and pattern of ocular disorders and blindness among leprosy patients drawn from three leprosy clinics in South- Eastern Nigeria. It is hoped that the results of this study will stimulate policy makers on the need for the formulation of a more effective policy on prevention of avoidable blindness among leprosy patients in this area.

MATERIALS AND METHODS

We examined the eyes of 171 active and cured leprosy patients drawn for three referral leprosy centers in South – Eastern Nigeria as follows:

1. The Oji River Leprosy services, Enugu State
2. The leprosy Referral Research Centre Uzuakoli, Abia State
3. Mile 4 hospital Abakaliki, Ebonyi State

These three referral clinics were purposively chosen for this study because of the relatively larger number of patients using their facilities. Each of these clinics had adjoining settlements where many 'cured' leprosy patients who have been 'released from treatment' (RFT) still cluster and so were accessible for inclusion into the study.

Permission to carry out this study was obtained from the management of these centers. A period of one month (four weekly visits) was allocated to each clinic, during which all in-patients were examined. Also RFT patients from the adjoining settlements who presented themselves during the period of the study were examined and included in the study. Some of these RFT patients came for 'care- after- cure'. The patients had already been diagnosed and classified by the attending leprologist using the current WHO classification into either paucibacillary (PB) or multibacillary (MB) leprosy. Data was collected by use of a structured questionnaire. The ocular examination consisted of the following:

-Measurement of visual acuity separately for each eye using illiterate E- chart with multiple optotypes at 6 meters in normal daylight, both unaided and with a pinhole disc.

-Anterior segment examinations were done using torchlight and simple magnifying loupe.

-Eyelid closure was assessed as a test of facial nerve function

-Corneal sensitivity, as an assessment of trigeminal nerve function was tested with a wisp of cotton wool.

-Examination of anterior chamber for flare and cells was done using a portable slit lamp biomicroscope.

-Pupils were examined for size, shape and reaction to light.

-The intraocular pressure (IOP) measurement was done with a schiotz tonometer.

-Fundoscopy was done using a direct ophthalmoscope.

Blindness was defined as best corrected visual acuity of $< {}^{3}/_{60}$ in the better eye while visual impairment was defined as best corrected visual acuity from $< {}^{6}/_{18}$ to ${}^{3}/_{60}$ in the better eye. Refractive errors were considered if the visual acuity improved with a pinhole disc.

RESULTS

In this study 340 eyes of 171 patients were examined. Two eyes were eviscerated presumably due to panophthalmitis. Their ages ranged from 12 to 81 years with a mean age of 47.9 years ± 14.8SD. The age group 41- 60 provided the bulk of the patients (56.7%). There were 112 males (65.5%) and 59 females

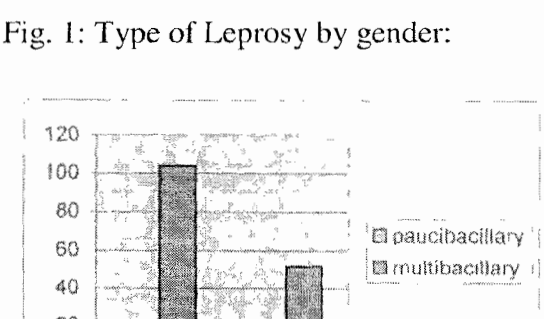
(34.5%). The male: female ratio was 2:1. See Table I.

TABLE 1 AGE AND GENDER DISTRIBUTION OF PATIENTS

AGE RANGE (YEARS)	MALE	FEMALE	TOTAL	%
11 – 20	6	2	8	4.7
21 – 30	14	6	20	11.7
31 – 40	17	8	25	14.6
41 – 50	29	19	48	28.1
51 – 60	33	16	49	28.6
61 – 70	7	5	12	7.0
>70	6	3	9	5.3
TOTAL	112	59	171	100.0
Mean Age = 47.9 years				
SD ± 14.8				
Male: Female = 2: 1				

Most of the patients had multibacillary (MB) leprosy (159 patients or 91.2%) while only 15 patients (8.8%) had paucibacillary (PB) leprosy. More males had MB leprosy than females (M: F=2:1) while about the same number of males had PB leprosy as females (figure 1).

Fig. 1: Type of Leprosy by gender:



All patients who were still on treatment received MDT (Dapsone, Rifampicin and clofazimine). These drugs were donated to these leprosy clinics by the German Leprosy Relief Association (GLRA) and were therefore given free to the patients. However some of the RFT patients who commenced treatment before the MDT era were treated with Dapsone monotherapy but completed with MDT.

Leprotic ocular findings were seen in 60.2% of the patients (table 2). The most common leprotic findings were madarosis, corneal anaesthesia, and uveitis and exposure keratopathy/ corneal opacity (table 3).

TABLE 2 FREQUENCY DISTRIBUTION OF OCULAR DISORDERS

OCULAR DISORDERS	NO. OF PATIENTS	%*
Leprotic lesions	103	60.2
Cataract	42	24.2
Pterygium	42	24.2
Refractive errors	37	21.6
Glaucoma	14	12.6
Age-related Macula degeneration	8	4.6
Presumed toxoplasmosis	2	1.2
Optic atrophy	2	1.2
Squint	1	0.6

*% based on the 171 patients studied

Ocular leprotic findings were relatively higher among females (67.8%) than in males (56.3%). However this difference is not statistically significant ($X^2 = 3.2768$; $P > 0.05$). Low intraocular pressure (IOP) (<10 mmHg) was found in 24.6% of the patients, normal IOP (10-21 mmHg) in 66.1% and high IOP (>21 mmHg) in 3.5% of the patients. IOP could not be measured in 5.8% of patients because they were uncooperative.

TABLE 3 LEPROTIC OCULAR DISORDERS

LEPROTIC OCULAR FINDINGS	NO. OF PATIENTS	%*
Madarosis	52	30.4
Corneal anaesthesia	51	29.8
Lagophthalmos	33	19.3
Uveitis	32	18.7
Corneal opacity/exposure Keratopathy	21	14.6
Anterior staphyloma	2	1.2
Corneal pannus	2	1.2
Episcleritis	2	1.2
Avascular keratitis	1	0.6
Ectropion	1	0.6
Phthisis bulbi	1	0.6

*% based o the 171 patients studied

Almost half of the patients had visual acuity of $^{6}/_{18}$ or better while 68 patients (39.8%) were visually impaired. There were 18 blind patients (10.5% of the population) No blind patient was aged 40years or below. The age range 51-70 provided 72.2% of the blind population. The causes of blindness in this study are listed in table 4. Cataract was the commonest cause of blindness (55.6%) followed by uveitis (22.7%).

TABLE 4 CAUSES OF BLINDNESS IN THE POPULATION STUDIED

CAUSE	NUMBER	PERCENTAGE
CATARACT	10	55.6
UVEITIS	4	22.2
CORNEAL	1	5.5
OPACITY	3	16.7
OTHERS (NON LEPROTIC)		
TOTAL	18	100.0

DISCUSSION

The 60.2% prevalence of leprotic ocular findings in this study compares with Ogundipe's 70% [9], and Nwosu's 63% [10]. It is however higher than Ayanru's 21.27% [8]. This may be explained by the preponderance of MB leprosy in this study. It has been suggested that variations of ocular findings among leprosy patients might be accounted for by methods used in the examination of the anterior chamber, the average duration of disease and the main types of leprosy encountered [8].

The male: female ratio of 2: 1 in this study is in keeping with findings by most other studies [8,9]. Ayanru [8] suggested that this might reflect hospital attendance and admission into non- gynecological clinics in Nigeria. Trautman[11] in his review of epidemiological aspects of leprosy noted that most studies throughout the world indicate that males are twice as likely to contract lepromatous leprosy as female, but with tuberculoid leprosy, the ratio is nearly 1:1. However, sociological research highlights the different influences of stigmatization on the treatment-seeking behaviour of both sexes. In a study of leprosy-affected people who had left or been sent away from home in India, women were found to suffer more severely than men[12]. The qualitative data from this study revealed that girls with leprosy found it more difficult to marry than boys. Research in a different sample of registered patients in Maharashtra shows that family support was more positive for men, leading them to seek treatment sooner than women[13]. However, the exact reason for the male preponderance is said to be unknown [11].

It is also observed in this study that females appeared more susceptible to leprotic ocular complications. This trend raises questions on the possibility of hormones or genetics influencing the sex distribution of the disease. Recent findings show that although leprosy is not a hereditary disease, 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[14]. 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 [14]. 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 [14]. This is an area where further research is suggested. There is hope that the recent breakthroughs in the human genome project will provide the framework for further studies on genetic susceptibility. Other factors influencing susceptibility to the *M. leprae* organism or the likelihood that infection will lead to disease include host immunity, race, environment, socioeconomic status, and age [11].

The preponderance of MB leprosy in this study agrees with the findings by the National Tuberculosis and Leprosy Control Programme (NTLCP) [4] and other recent Nigerian studies [10] However older studies by Ayanru [8] and Ogundipe [9] report higher prevalence of tuberculoid (or paucibacillary) leprosy for

Nigeria than lepromatous (or multibacillary). This difference may be explained by the change in case- definition. The current WHO definition for MB leprosy is more clinical and includes all cases of leprosy with more than 5 nodules with or without smear- positivity, whereas the older studies based their classifications on bacterial index.

It has been established by various studies that ocular morbidity in leprosy increases with both age of patient and duration of disease [5,6]. The present study also corroborates this finding for both leprotic ocular findings and blindness. No blind patient was aged 40 years or below.

The commonest leprotic ocular finding in this study was madarosis, and this finding is in keeping with the preponderance of MB leprosy in this population. Both superciliary and ciliary madarosis are caused by direct invasion by *mycobacterium leprae* and therefore indicate high bacillary load [5,7].

Corneal anaesthesia, lagophthalmos and uveitis were also common. These are potentially sight – threatening (PST) lesions. Corneal anaesthesia occurs from leprotic involvement of the ophthalmic division of the trigeminal nerve. It has been implicated as the underlying factor for corneal ulceration and consequent scarring in leprosy. 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. The combination of corneal anaesthesia and lagophthalmos was common among patients in this study. Both increase the risk of corneal ulceration and opacity. Most of the patients who had lagophthalmos also had corneal opacity. This indicates poor eye care in these leprosy clinics.

Uveitis was another leprotic finding in this study. Most of the patients who had uveitis had chronic uveitis with quiet white eyes, atrophic irides and miotic, non- reactive, irregular pupils and posterior synechiae. Iris pearls were seen in one patient. Only one patient had acute uveitis with ciliary injection and anterior chamber flare. This pattern agrees with the findings by most other workers [5,8,9,10].

Several studies have reported low intraocular pressures (IOP) among leprosy patients [11,9,15]. This low IOP is said to result from atrophy and hyalinization of the ciliary body leading to low secretion of aqueous humor. This study corroborates this finding. For similar reasons glaucoma is reported to be uncommon in leprosy [9]. In this study, the prevalence of glaucoma (8.2%) was similar to the 8.3% found in the general population in the same area [16].

The major causes of blindness in most studies of leprosy patients have been shown to be cataract, corneal opacity (following lagophthalmos and exposure keratopathy) and uveitis[5]. Cataract, especially age-related cataract, has been shown to be the most common cause of blindness both for leprosy patients and the general population[5, 6]. This agrees with the finding in this study. The causes of cataract among leprosy patients are varied and may or may not be related to leprosy. It may be age-related, or due to repeated inflammations and consequent steroid treatment of the reactions in leprosy [5,6].

In a comparative study, Girma et al [17] found that leprosy patients who came for cataract surgery were younger than the non-leprosy patients who came for the same surgery. This raised the possibility of leprosy being the cause of their cataract. Courtright and lewallen[5], reviewed data pooled from several settings of leprosy patients who have completed MDT and had no evidence of chronic uveitis. They found that lens opacities occurred in 25% of MB and 7.5% of PB patients. The magnitude of this difference suggests that there are factors that place MB leprosy patients at risk of cataract. Although the MB and PB leprosy groups varied in terms of age and duration of disease, however, they observed that differentiating age- related from complicated cataract in patients who may have sub- clinical inflammation is problematic.

Pterygium is believed to represent a response to chronic dryness and exposure to the sun. Daniel et al [18] have suggested that leprosy may contribute to the pathogenesis of pterygium

in various ways including increased exposure to sunlight because of their predominantly outdoor lives (due to social ostracization, lagophthalmos and loss of blink reflex which keep the eyes open). Leprosy- related granulomatous reactions are also suspected based on the histological findings of 93 tissue specimens from leprosy patients who had pterygium excision. However, more studies are needed to confirm this.

Posterior segment lesions observed in this study include age- related macula degeneration and chorioretinal scars. The chorioretinal scars were presumed to be caused by toxoplasmosis since posterior segment lesions are rare in leprosy [9,10]. However, peripheral choroidal lesions, retinal vasculitis and papillitis have been documented in leprosy [9]. Onchocerciasis has to be considered a possible cause for these scars since the study area falls within the endemic zone.

CONCLUSION AND RECOMMENDATIONS

In this study, both leprotic and non-leprotic lesions were seen in the patients, and more than half the population were at a risk of blindness because of potentially sight threatening (PST) lesions. Blindness amongst these patients was almost 12 times that of the general population in the area [13]. As in most other studies, cataract was found to be the commonest cause of blindness accounting for 55.6% of blindness. Blindness from cataract, uveitis and exposure keratopathy resulting from lagophthalmos are preventable by early diagnosis and appropriate treatment.

It is therefore recommended that in addition to preventive measures to limit blinding leprotic complications; regular surgical outreaches to leprosy centres should be organized by ophthalmologists to deal with non-leprotic causes of avoidable blindness. Also programmes responsible for leprosy control should incorporate routine eye care, especially surgery for cataract and severe lagophthalmos.

REFERENCES

1. Walgate R "Massive decay" in leprosy genome, massive research too? Walgate @ science analyzed.com February 22nd 2001.
2. Roy 111 C, Sally B, Kelvin C, David R, Samuel SU, Lowell Y. Leprosy in the post genome era. Lepr Rev 2001; 72: 8 –22.
3. Daumerie D. Current World Health Organization sponsored studies in the chemotherapy of leprosy. Lepr Rev 2000(suppl); 71: 88 – 90.
4. Federal Government of Nigeria. Update on the National Leprosy Control Programme. MOH review report 2001.
5. Courtright P, Lewallen S. Ocular manifestations of leprosy. In Johnson G.J, Minnasia DC, Weale R.(eds), The epidemiology of eye disease. Chapman and Hall Medical; 1998, 249 – 264.
6. Hogeweg M. Cataract: the main cause of blindness in leprosy. Lepr Rev 2001; 72:139 – 142.
7. Fftytche T.J, Result of Surveys on ocular Leprosy. In Courtright P, Jonshon G.J (eds). Prevention of blindness in Leprosy, London. ICEH Publication, 1991. P.35-36.
8. Ayanru JO. Ocular leprosy in Nigeria. Nig. Med. J. 1975; 5(3): 258 – 262.
9. Ogundipe OO. The eye in leprosy. National Post-graduate Medical College of Nigeria. Unpublished FMCOph. Dissertation. May 1986. p. 1- 68.
10. Nwosu SN, Nwosu MC. Ocular findings in leprosy patients in Nigeria. E.Afr. Med. J. 1994; 71(7); 441 – 444.
11. Trautman JR. Epidemiological aspects of Hansen's disease. The Star 1989, 49(2): 1 – 5.
12. Vlassof c, khof S, Rhao S. Double jeopardy: women and leprosy in India. In work, health and contraception from women's perspective, 1998 (ed ME khan). Center for operations research and training. Bauda.
13. Vlassof C, Raos, carolev, karundikar N, Azar M, kanade A. The family: Anglected determinant of health in south Asia. Social change, 1996 26:57- 73

14. Roy 111 C, Sally B, Kelvin C, David R, Samuel SU, Lowell Y. Leprosy in the post genome era. Lepr Rev 2001; 72: 8 – 22.
15. Lewallen S, Courtright P, Lee HS. Ocular autonomic dysfunction in Intraocular pressure in leprosy. Br. J. Opththalmol. 1989; 73: 946 – 949.
16. Ezepue UF. Magnitude and causes of blindness and low vision in Anambra State, Nigeria: result of 1992-point prevalence survey. Pub Hlth 1997; 111(5): 305 – 309.
17. Girma T, Mengistu F, Hogeweg M. The pattern of cataract and post-operative outcome of cataract extractions in Ethiopian leprosy patients as compared with non-cataract patients. Lepr Rev 1996; 67: 318 – 324.
18. Daniel E, Thompson K, Abraham S, Job CK. Pterygium in Lepromatous Leprosy. Int J Lepr 1996; 64(4): 428 – 432.

Plate 1: Leprosy Patient With Limb Deformities

Plate 2: Lagophthalmos in A Leprosy Patient