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Full Length Research Article

CURED LEPROSY: EYES ARE STILL IN DARK!

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ABSTRACT

Some Leprotic eye lesions are reported to be progressive in nature even after mycobacterial cure. During last 3 years 980 cured leprosy patients were examined of which 120 patients were followed up every year (2011,2012,2013) Results: 1% per increase in leprosy related eye lesion (Lagophthalmos, keratitis, posterior synechia). 2.5% increase in significant cataract (VA<6/18) 1.5% patients developed ocular morbidity and 0.6%cases became blind. Nonleprotic lesions were however more pronounced (5%) e.g., refractive error, posterior capsular opacification, secondary glaucoma, Keratomalacia etc. Conclusion: Nonleprotic ocular lesions were progressing as in normal population and some Leprotic lesions due to neural damage were progressive and needs special attention from the time of release from treatment.

INTRODUCTION

MDT has changed the scenario of leprosy by curing them bacteriologically very fast. This also has shortened the period of treatment thus improving compliance. This has given us a chance to eradicate this curse. Though global prevalence was 0.32 per 10000 populations but annual new case detection remained around 2.25 lakhs half of them having grade 2 disability. This reflects lack of community awareness and failure in early detection. Nearly a tenth of new cases are children which indicate continued disease transmission. India has got maximum number of leprosy patients with 58.8% of global leprosy population, prevalence is double that of global data, 9% being children and 4% being G2D (Global leprosy Update, 2014). On this background it is very obvious health issue in India though by definition leprosy is no more a public health problem. Butmillions of people who are mostly neglected and have no access to public transport and hospitals are a big issue. Routine eye problem like presbyopia, cataract, refractive error, amblyopia, Vitamin Adeficiency etc, are poorly addressed which add up to morbidity of already burdened leprosy population. The disease has invariably causes many visually disabling sequel, and it is estimated that 3.2% of all leprosy patients are ultimately blinded by long term ocular complications(Courtright and Lewallen, 1998).Several recent studies have documented the main causes of blindness in leprosy which include iritis, posterior synechia, cataract, Lagophthalmos, corneal ulceration, and all of the complications associated with corneal hypoesthesia and exposure. However, none of the studies has addressed the question of whether or not the sight threatening complications of leprosy continue to develop after the infectious component of the disease has been adequately treated. Ocular morbidity in leprosy has three stages of development: ocular complications at diagnosis, during treatment and after release from treatment. Mycobacterial and neural involvement are the main factors in first two stages whereas chronic neural involvement can cause further ocular damage in cured patients. Only a few studies are there on this progressive ocular morbidity of cured patients. So this study was undertaken at a state where the prevalence of leprosy is around 10 even after two decades of MDT.

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Aim

Leprosy control programme has reached a stage from where we can think of controlling the disease as a whole. But sequel of the disease are very difficult to manage, more so if the morbidity still progresses after cure of the disease. The aim of this study was to analyze the data on progressive ocular morbidity in the bacteriologically cured patients

MATERIALS AND METHODS

This is an on-going prospective study in Bokaro district of Jharkhand where leprosy is still health problem. Prevalence has reduced to less than 1 per 10000 populations during last decade. But annual new case detection rate of more than one lakh implicates that even today; 3-4 new cases are registered daily. In our hospital alone, average one new case is registered daily. We had two groups of patients in this study. Patients released from our hospital after successful completion of MDT were grouped together and those settled at different resettlement villages were grouped under second group. All of them had either treated with MDT alone or added MDT after daps one monotherapy. All the patients were examined by our team every year during January and March .Hospital registered cases were seen at hospital OPD and the rehabilitated cases were examined at the yearly eye camps organized at their doorsteps.Visual acuity was measured with Snellen's E chart. External examination was done with torch light and portable slit lamp.

Undilated fundus examination along with refraction was done in all the cases. Intraocular pressure was seen digitally and if in doubt Schiotztonometer was used. Corneal sensation was seen in all the cases with a cotton wisp introduced from below. Fundusexamination and lenticular opacity were assessed after dilatation with Tropicamide and phenylephrine combination. Ocular findings were recorded under the following headings: Visual acuity uncorrected and corrected, lid: blinking, Lagoph thalmos, margins, trichiasis. Pupil: size, shape, reaction. Cornea: lusture, opacity, keratitis, sensation. Iris: synechia, nodules, atrophic patches. Lens: cataract, aphakia or pseudophakia, after-cataract. Demography along with nonocular clinical data was collected. Statistical analysis: Incidence of ocular pathology was calculated from those patients who did not have the specific finding in 2010. Primary leprosy related ocular findings include Lagophthalmos (either on gentle or forced closure), posterior synechia, or keratitis. We used proportional hazards regression (forward step wise) to analyse occurrence of specific findings according to demographic and clinical characteristics associated ($p<0.05$) with pathology by univariate analysis. Relative risks (95% CI) were generated. Our findings are reported by patient rather than by eye. We excluded patients who had died, moved away, or refused examination from our analyses.

RESULTS

A total of 980 patients were examined in last three years of which 380 were from the hospital and rest were from the resettlement villages in the Bokaro district.45 patients of the hospital and 75 patients from the resettlement villages could be followed up every year for last 3 years. Mean age of the patients is 52.4years; male female ratio was 1:1.5, ratio of multi: paucibacillary disease was 60:40; mean duration of disease was 21.4 years. Those who were totally blind in 2010 had no significant increase in mortality rate. Incidence of Leprotic ocular lesions (Table, 1). Many of these cured patients already had potentially blinding ocular pathology in 2010. Cumulative incidence of leprosy related ocular pathology (Figure 2) ranged as high as 40% (reduced corneal sensation). All posterior synechia cases were among multi bacillary patients; excluding pauci bacillary patients from our analysis reveals a cumulative incidence for posterior synechia of 18% (95% CI: 9.3–22.8%).

Univariate analysis demonstrated that the development of posterior synechia (in the 3 year period) was associated with age, duration of disease, and small pupil size (usually accompanied by poor pupil reaction);regression analysis revealed that only size of pupil were associated with incident cataract. Age and pupil size were independently associated with the development of cataract. (Figure 1) Among the cataract cases 56% had pre-existing posterior synechia; including individuals who developed posterior synechia in the intervening 3 years, this increased to 62%. Lagophthalmos was present in 10% cases (n12) in 2011 which became 12 %(n15) in 2012 and 13% in 2013.Keratitis increased from 12% to 14% in next two years whereas iritis increased from 3% to 5% within 3 years. Iritis was seen only in multibacillary cases. (Figure 2)

Changes in vision

Patients were divided into three groups according to their visual status.20% of the patients examined in 2011 had visual acuity less than 6/18-6/60, 10% had VA <6/60 and the rest had visual acuity >6/18.Over the next two years on an average 3% of cases from each group had lost their vision to some extent and regrouped with the worse level. However, this change was due to lenticular changes. (Figure 4, 5)

Cumulative incidence of cataract

Cataract was the commonest cause of reduced vision.15% had cataract in 2011 which increased to 20% and 25% in next two years. Univariate analysis revealed that age, duration of disease, pupil reaction, pupil size, and posterior synechia were associated with incident cataract .Age and pupil size were independently associated with the development of cataract. Among the cataract cases 56% had pre-existing posterior synechia; including individuals who developed posterior synechia in the intervening 3years.

Table 1. Progressive Leprotic lesion

Year	Lagophthalmos	Exposure keratitis	Cataract	Iritis
2011	10%	12%	15%	3%
2012	12%	14%	20%	4%
2013	13%	14%	25%	5%

DISCUSSION

This prospective clinical study shows that ocular morbidity keeps on progressing even after the cases are cured mycobacteriologically. Although follow up examinations are not possible on all patients those who accepted follow up were not different (demographically or in terms of pre-existing pathology) from those who were not examined.

Pathophysiology of ocular lesions

Neurological damage leads to reduced blinking, Lagophthalmos and corneal hypoesthesia which in turn may contribute to keratitis. Sympathetic neural damage supposedly

leads to chronic uveitis of leprosy; they are the cause of posterior synechia and small nonreactingpupil. Thus all the progressive ocular Leprotic lesions are related to at least in part to chronic nerve damage.

Figure 1. Progressivelagophthalmos, one tenth of patient's presenting sign, mostly affecting young

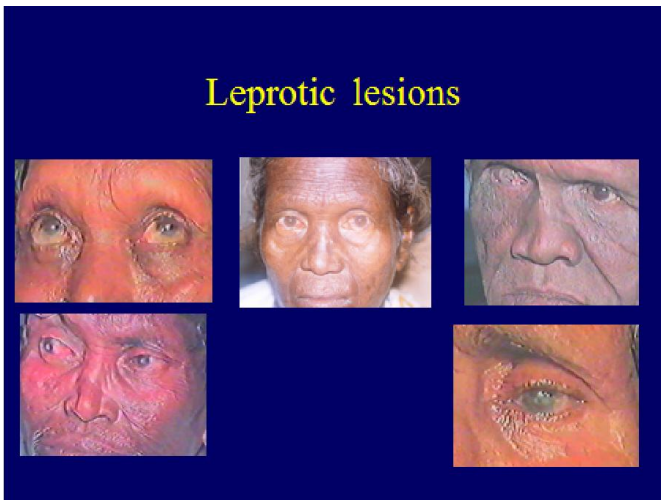

Figure 2. Progressive leprotic lesions in cured leprosy

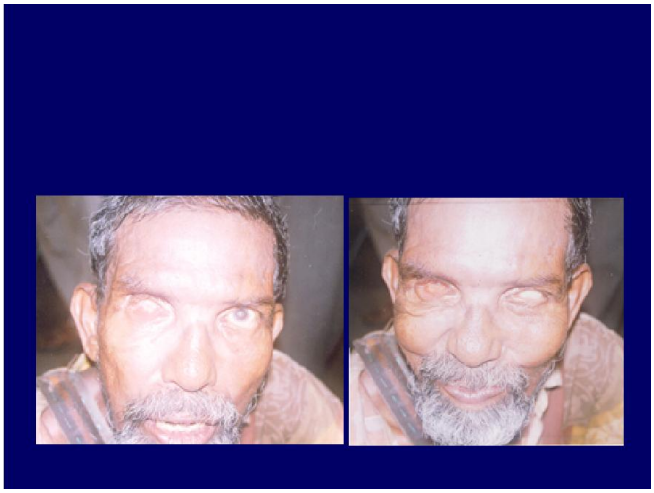

Figure 3. Mixed lesion: progressive bilateral Lagophthalmos (leprotic) and mature cataract (nonleprotic)

Figure 4. Keratomalacia in a leprotic child

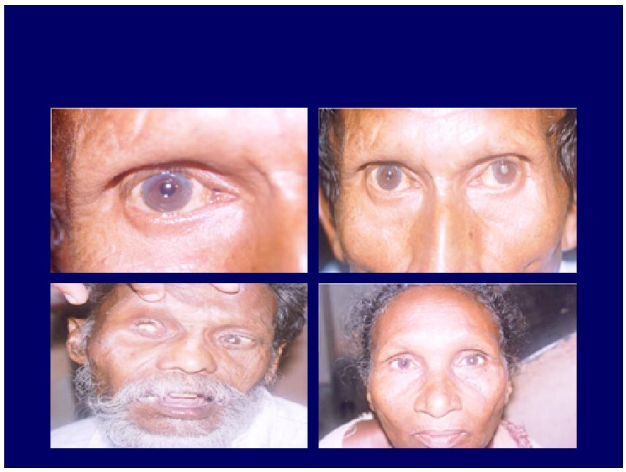

Figure 5. Nonleprotic progressive lesions –refractive error, cataract

Figure 6. Progressive nonleprotic lesions

Programme Implications

These results should lead to further in depth data collection which will have definitive impact in leprosy control programme.40% of all the patients examined in consecutive 3 years (n120) at least one Leprotic ocular lesion was present at the beginning (Lagophthalmos, keratitis or posterior synechia). Among those who did not have any lesion, 1.5% developed

some Leprotic ocular lesions.5% of them developed non Leprotic ocular lesions including age related cataract, tumours or glaucoma. (Figure 4, 5, 6, 7)

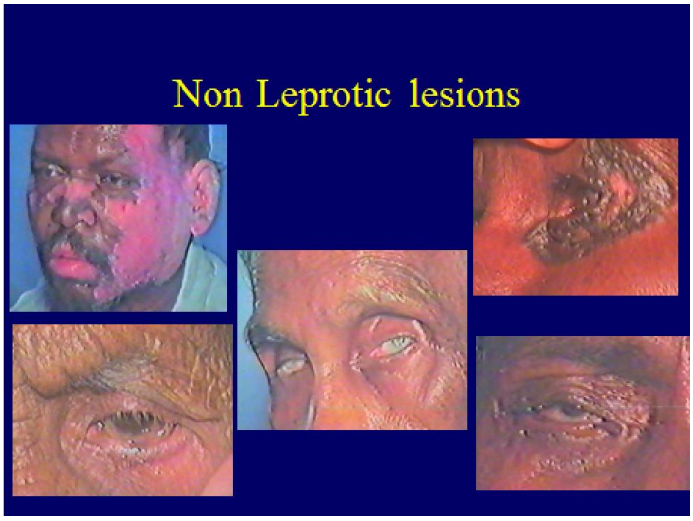

Figure 7. Progressive nonleprotic lesions: chemical burn, ectropion, melanoma, phthisis bulbi

This study was done in one of the most endemic areas of leprosy where access to medical care is very less. Results are in general agreement with the studies done in Korea (Susan *et al*., 2000) and Holland (Hogeweg and Faber, 1991), which reported that Lagophthalmos, keratitis and posterior synechia might progress in cured patients. All the patients in our study are treated with MDT and some are even treated with dapsone in pre MDT era. Only MDT cured patients had less progressive lesions though this separate study is going on.

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