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Original research article

Ocular manifestations of leprosy on multidrug therapy

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Abstract

Mycobacterium leprae is the causative agent of leprosy, which is a chronic granulomatous infectious disease. Several visual signs can appear before and during the course of multidrug therapy (MDT), and in individuals who have been bacteriologically healed, they may continue to appear long after the treatment has been finished. Blindness is a devastating condition that may result from a variety of causes at any stage of development. It is necessary for us to become familiar with and detect the early signs and symptoms of ocular problems in order to begin treatment for the patient at an earlier stage. The pattern of ocular manifestation in leprosy patients who are receiving MDT (Multi Drug Therapy) has to be investigated.

Keywords: Leprosy, multidrug therapy, exposure keratitis, lagophthalmos

Introduction

The name "leprosy" comes from the French word "leper" and the Greek word "lepros," which means scaly. The word "scaly" refers to the scales that form on the skin as a result of the disease. Leprosy is referred to as Vata Rakta or Vat Shonita and Kushtha in the most ancient literature of "SUSHRUTA SAMHITA," which were gathered in 600 BC. Leprosy can affect people of any age and can affect both males and females [1].

Daniel Cornelius Danielssen (1815-1894), a Norwegian researcher, and Carl-Wilhelm Boeck (1808-1875), a German physician, are credited with carrying out the first scientific investigation on leprosy [2]. Gerhard Armauer Hansen proposed that leprosy was not an inherited disease but rather a contagious one, and he found the lepra bacillus in the year 1873 [2].

According to the World Health Organisation, there have been 16 million cases of leprosy that have been cured over the course of the last 20 years. The prevalence rate of leprosy fell from 21.1 cases per 10,000 populations in 1985 to 0.25 cases per 10,000 in 2017. This represents a significant improvement. In 2017, the rate of newly discovered cases was 2.77 per 100,000 people in the population. During the course of 2017, a total of around 210 671 new cases were identified. 73% of the worldwide burden was found in the South East Asia Region (SEAR), while India and Indonesia were responsible for around 67.4% of the new leprosy cases both globally and regionally [3].

Mycobacterium leprae is the causative agent of leprosy, which is a chronic granulomatous infectious disease [3]. It grows slowly and has a rod shape that is either straight or slightly bent. It is an intracellular acid-fast bacilli. Because it is held together by a lipid molecule called glia, it has the appearance of agglomerates. The term 'GLOBI' is used to refer to these masses. The 'cigar bundle' appearance is caused by the bacilli in the globi arranging themselves in parallel rows. It most commonly affects the skin and peripheral nerves, but it can also spread to other tissues, including the eyes, the mucosa of the upper respiratory tract, the muscles, the bones, the reticuloendothelial system, and the testes [4]. It is possible for others to become infected by it entering through cracks in the skin, but it is not possible for it to infect undamaged skin. The disease is spread from one person to another by contaminated respiratory droplets. It seems most likely that the infection entered the body through the upper respiratory system.

Lepromatous leprosy and tuberculoid leprosy are the two types of manifestations that systemic illness can take. Lepromatous leprosy is a multisystemic illness, and its manifestations include leonine facies, which is characterised by cutaneous thickening, nasal widening, and thickening of ear lobules; saddle-shaped nasal deformity; peripheral nodules and cutaneous plaques; claw hands; shortening and loss of fingers; and nodules and plaques on the periphery of the skin. Tuberculous leprosy is characterised by symptoms that are localised to the skin and peripheral nerves. These symptoms include annular anaesthesia, hypopigmented skin lesions, and thickening of the peripheral nerves. Direct ocular involvement will be present in the lepromatous variant, which is characterised by a reduced cell-mediated immune response, and iris pearls will include macrophages that are stuffed with bacilli.

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Patients suffering from tuberculosis-associated leprosy will have high levels of cell-mediated immunity as well as indirect ocular involvement, such as neurotrophic and neuroparalytic keratopathy. Because of the presence of effective cell-mediated immunity, the tuberculoid type of the disease will exhibit granuloma development and will not contain a significant number of bacilli. Hypopigmentation, erythema nodosum, plaques, and nodules are all examples of systemic symptoms that can appear on the skin. When nerves are involved, the patient will experience facial palsy, anaesthesia of the skin, and thickening of the peripheral nerves. Leprosy can cause a variety of facial and hand deformities, including the saddle nose, leonine facies and claw hand (ulnar nerve palsy). Eye difficulties are a common complication that can be caused by lepromatous leprosy. Indeterminate leprosy, tuberculoid leprosy, borderline tuberculoid leprosy, mid-borderline leprosy, borderline lepromatous leprosy, and lepromatous leprosy were the different stages of leprosy that the World Health Organisation (WHO) recognised. Leprosy is still one of the leading causes of blindness in the world. Patients suffering from leprosy often lose their sight, which is an irrevocable double tragedy. They are unable to see or feel, which places a significant burden not just on themselves but also on their relatives. When it comes to the eyes, the extra ocular structures and the anterior segments are typically damaged. There are two classifications of leprosy that are used when discussing treatment options: multibacillary and paucibacillary [5]. The risk of ocular involvement in leprosy is particularly significant in those who have the multibacillary form of the disease [6]. Lagophthalmos, uveitis, corneal hypoesthesia, secondary glaucoma, and cataract are the leprosy-related eye conditions that can lead to blindness. Conjunctivitis, hypopigmented nodules, thickening of the tarsal plate that can lead to mechanical ptosis, thickening of corneal nerves, diminished corneal sensations, neuroparalytic keratitis, and corneal pannus are some of the other ocular signs that have been observed. It produces acute iritis in the uvea because of a deposit of immune complexes, and it causes chronic iritis because of direct invasion. Formation of mutton fat large keratic precipitates, patchy dense synechia, low grade flare, nodules over iris, hypopigmented patches over the iris, complicated cataract, secondary vitreous degeneration, retinal pearls, choroidal thickening, and choroidal detachment are all symptoms of granulomatous uveitis. There is almost never involvement of the optic nerve. The cranial nerve VII is the one that is affected by leprosy the most frequently, and the other cranial nerves that are affected, in decreasing order, are V, VI, IV, and III. The prevalence of blindness has been estimated to range anywhere from 0.7% to 30% of the population [7].

Materials and Methods

After acquiring the patients' and witnesses' informed agreement for their participation in the study, the research project was initiated. We took a brief ocular history as well as a detailed demographic profile of the patients, which included their ages, genders, occupations, and the length of time they had leprosy. Unaided visual acuity and Best Corrected Visual Acuity (BCVA) utilising Snellen's chart and for near vision utilising Jaeger's chart, anterior segment examination utilising slit lamp biomicroscope. After having the pupil dilated, an assessment of the ocular motility and the posterior segment was carried out using an indirect ophthalmoscope and a 20 dioptre lens. A small piece of cotton was used to test the corneal feeling of the patient. The Schiotz tonometer was used to record the patient's intraocular pressure. During ocular examination we mainly focused on loss of eyebrows, poliosis, trichiasis, lagophthalmos, orbicularis oculi muscle weakness, eyelid abnormalities, corneal ulcer, corneal opacity, clofazimine crystal deposition on conjunctiva and cornea, presence of episcleritis and scleritis, anterior chamber cells and flare, presence of iris atrophy, posterior synechia, pupillary reaction to light and presence of cataract.
When we had reason to suspect that a patient had cataracts or synechia, we used mydriatic drops (a combination of tropicamide and phenylephrine) to dilate their pupils, and then we inspected their eyes using a slit lamp. An experienced examiner utilised a Snellen chart in order to determine the patient's best-corrected visual acuity. After having the pupils dilated, those that had a lower level of visual acuity as well as those that had intraocular problems were examined using an indirect ophthalmoscope.
Dermatologist performed slit skin smear and skin biopsy, and the report produced was positive for Mycobacteria leprae (using the Ziehl-Neelsen technique of staining). Physiotherapy, frequent blinking exercises, lid tape with micropore plaster at night time, and spectacle correction were some of the treatments that were administered to patients who had eye symptoms. Lubricant drops, topical antibiotic with steroid drops, antibiotic eye ointments, and antibiotic eye drops were also used.

Results

Table 1: Distribution

With Ocular Manifestation	Without Ocular Manifestation	Total
11	19	30

Table 2: Manifestations

Ocular Manifestations	No. of Patients N (%)
Lagophthalmos	6
Cataract	7

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Pterygium	2
Exposure keratitis	2
Chalky white deposits on cornea	2
Anterior Synechia	3
Spheroidal degeneration	3
Nystagmus with exotropia	2

Discussion

The cornea receives its nerve fibres from the anterior ciliary nerves, which are extensions of the ophthalmic division of the fifth cranial nerve. Infiltration by leprosy produces a thickening of the nerves, which results in anaesthesia of the cornea. Infiltration of leprosy cells into the 7th cranial nerve, in particular the zygomatic branch, which results in paralysis of the orbicularis oculi muscles. This is due to the breakdown of eyelid function, which leads to corneal ulcers. Patients with diminished corneal sensitivity do not experience any symptoms, therefore their eyes are neglected, which ultimately leads to the perforation of an ulcer, intraocular infection, and blindness. Cornea is a structure that lacks blood vessels. Micronodules are caused by M. leprae when the fungus invades either from a neighbouring structure or along the nerves. These nodules can be easily seen as thick white corneal pearls since the cornea is clear, and they are the source of diffuse superficial punctate keratitis.

The persistent inflammation of the conjunctiva that results from chronic conjunctivitis is what causes the conjunctiva to get involved. Lesions resembling erythema nodosum leprosum can be found on the conjunctiva. It has been noted that some cases of pterygium involve the accumulation of macrophages that contain M. leprae.

Iritis and iridocyclitis are both caused by granulomatous diseases of the iris. Iridocyclitis that persists over time has the potential to cause cataracts. It's possible that the use of steroids to treat lepra responses will speed up the development of subcapsular cataracts. An ulceration in a granulomatous lesion may result in the production of an exudate that is made up of fibrin and polymorphs. Additionally, the pupillary borders may cling to the anterior capsule of the lens, resulting in posterior synechia and a pupil that is fixed, small, and does not react. Destruction of the tissue of the iris and ciliary body will eventually occur.

Body leads to phthisis bulbi, which is characterised by atrophy and a reduction in the size of the globe. Iris Pearls are formed as a result of the collection of lepra bacilli, and they adhere to the pupillary margins and the surface of the iris like a necklace. Scleritis is diagnosed in long-term untreated instances, and it has the potential to weaken the globe as well as form nodules at the junction of the sclera and the cornea. Episcleritis is an extremely uncommon condition. In leprosy, involvement of the posterior section is only observed extremely infrequently. These nodules have a yellowish appearance and are caused by the spread of lesions from the ciliary body to the choroid and retina. A skin biopsy and a skin Test-Lepromin test (also known as a Mitsuda reaction) are both part of the diagnostic process for leprosy. For the purpose of decolorizing culture specimens obtained from skin, ear lobules, nasal mucosa, and smears, the Ziehl-Neelsen stain and 5% hydrogen peroxide were used, respectively.

Acute or subacute inflammation brought on by an immune reaction is what's known as a lepra reaction. Clinical examination is typically what is used to make the diagnosis. Changes in inflammation in skin lesions or the emergence of new skin lesions, patches, or nodules with an immediate beginning. There are two distinct sorts of reactions that can take place: a reversal reaction, also known as type 1, and an erythema nodosum leprosum, also known as type 2. Both types of reactions are possible at any point during or after the MDT treatment, beginning with the beginning of the course. In cases of severe ENL reaction, discomfort can be felt in the eye, with or without redness of the eye; occasionally, there will be a loss of visual acuity [8]. The incidence of leprosy in India is endemic, with a prevalence of 3.8 cases per 10000 people [9]. At the beginning of the year 2005, India was home to around 70 percent of the world's leprosy sufferers who were officially registered [10].

Ocular symptoms may be caused not only by the disease itself but also by reactions to the treatment being administered for the disease [11, 12]. Patients who have finished therapy are regarded cured (since the majority of them are microbiologically negative), yet they still have several disabilities that were present before treatment began. This is despite the fact that patients who have finished treatment are considered cured. In addition to this, individuals may have a preexisting condition that causes nerve damage that cannot be reversed by treatment, which results in a gradual impairment. As a result, the development of a new ocular pathology after leprosy treatment is a distinct possibility [12, 13].

The presence of one or more of the following symptoms, such as lagophthalmos, corneal nerve beading, punctate keratitis, corneal opacity, and the presence of uveal involvement (cells and flare, iris atrophy, and/or keratic precipitate), was used to identify LROP (Leprosy Related Ocular Pathology). PBLROP, which stands for "Potentially Blinding Leprosy Related Ocular Pathology," is an acronym that stands for "Potentially Blinding Leprosy Related Ocular Pathology." This acronym refers to leprosy-related disorders that might cause vision loss owing to uveal involvement and/or lagophthalmos [13].

There has been a gradual shift in the kind of leprosy patients from paucibacillary forms to multibacillary forms, which can be seen in the epidemiology of leprosy cases. Additionally, there has been an increase

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in the case detection rate in older age groups in comparison to younger age groups. There will be a rise in the number of people who survive if socioeconomic conditions and medical services are improved [14]. There was a general agreement that the prevalence of ocular problems can be decreased by beginning ophthalmic inspection at the time of diagnosis and continuing evaluation even after completion of multidrug therapy. This was a recommendation that received widespread support. There is not much of it right now information concerning the extent and character of the incidence of ocular pathology in individuals who are undergoing MDT.

Conclusion

There is a need to create awareness of ocular involvement in leprosy patients to prevent long term visual loss.

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