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شبكة المعلومات الجامعية



شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم



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# جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات

لم ترد بالأصل



Faculty of Medicine

# Treatment of Leprosy

*THESIS*

Submitted for Partial Fulfilment for master Degree in  
Dermatology, Venereology and Andrology

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## List of Abbreviations

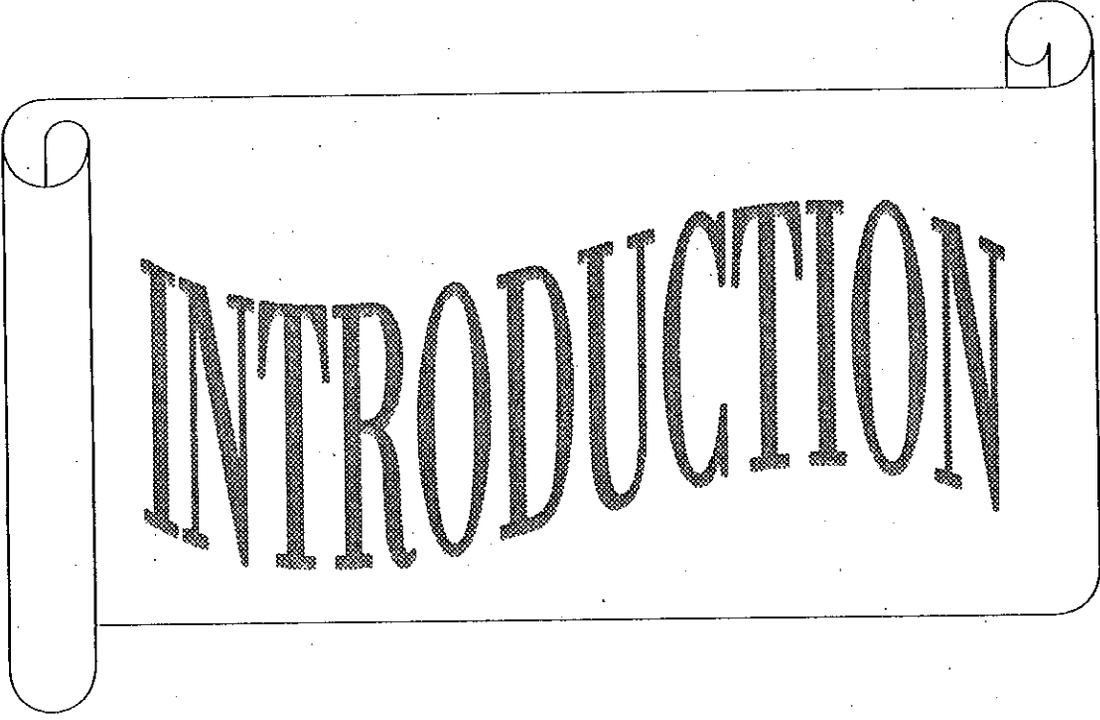
<b>BB</b> =	Borderline Leprosy.
<b>BI</b> =	Bacterial index.
<b>BL</b> =	Borderline Lepromatous Leprosy.
<b>BT</b> =	Borderline tuberculoid leprosy.
<b>CD</b> =	Clusters of differentiation
<b>CD4</b> =	helper-T-cell subsets.
<b>CD8</b> =	suppressor/cytotoxic-T cell subsets.
<b>DTH</b> =	delayed type hypersensitivity.
<b>ENL</b> =	Erythema Nodosum leprosum.
<b>HIV</b> =	Human Immunodeficiency virus.
<b>I</b> =	Indeterminate.
<b>IFN</b> =	Interferon.
<b>IGg</b> =	Immunoglobulin.
<b>IL</b> =	Interleukin.
<b>LL</b> =	Lepromatous Leprosy.
<b>MB</b> =	Multibacillary.
<b>MDT</b> =	Multidrug therapy.
<b>MIC</b> =	Minimal inhibitory concentration.
<b>NFI</b> =	Nerve function impairment.
<b>PB</b> =	Paucibacillary.
<b>PGL</b> =	Phenolic glycolipid.
<b>POP</b> =	Plaster of paris.

<b>PYAR =</b>	Patient years at risk.
<b>ROM =</b>	Rifampicin, Ofloxacin and Minocycline.
<b>RR =</b>	Reversal reaction.
<b>SLPB =</b>	Single lesion paucibacillary.
<b>TNF =</b>	Tumour Necrosis factor.
<b>TT =</b>	Tuberculoid Leprosy.
<b>VMT =</b>	Voluntary motor testing.
<b>WHO =</b>	World Health Organization.

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# INTRODUCTION

## INTRODUCTION AND AIM OF THIS REVIEW

Leprosy is an infectious disease caused by *Mycobacterium leprae*, which is a slow-growing intracellular bacillus that infiltrates the skin, the peripheral nerves, the nasal and other mucosa, and eyes. The incubation period between infection and appearance of leprosy is normally between two and 10 years but may be as long as 20 years. Leprosy can affect all ages and both sexes.

Humans infected with leprosy are the primary reservoir. Close and prolonged contact within the household is a significant factor in the transmission of disease. Leprosy infection probably occurs when *M. leprae* bacteria are discharged through the nose. Leprosy is transmitted to a previously uninfected person when nasal secretions from a *M. leprae* infected leprosy patient contaminate the nasal mucosa or minor skin abrasions of the previously uninfected person. However, most individuals have considerable natural immunity and many infections are suppressed. Leprosy cases seldom develop in non endemic areas without known close contacts.

Protective immune response in leprosy is based on cellular immune response and leprosy bacilli are killed or eliminated only by this mechanism (Noordeen and Pannikar, 1996).

Leprosy (Hansen's disease) presents a broad spectrum of clinical and histopathological manifestation which reflect the nature of individual's immune response to *mycobacterium leprae*.

This spectrum of clinical manifestation include two polar types of infection, lepromatous leprosy (LL) and tuberculoid leprosy (TT) as well as intermediate borderline forms of disease (Ridley and Jopling, 1966).

To simplify field work, the World Health Organization (WHO) classifies leprosy patients according to the bacillary load as multibacillary (MB) patients, with an elevated bacterial load, and paucibacillary (PB) patients, who presents negative or weak bacilloscopy (Rada et al, 1996). The paucibacillary (PB) group included all polar tuberculoid (TT), borderline tuberculoid (BT) and indeterminate (I) cases. The multibacillary (MB) group consists of polar lepromatous (LL), borderline lepromatous (BL) and mid-borderline (BB) patients.

Tissues infected with mycobacterium leprae contain large amounts of phenolic glycolipid 1 (PGL-1) which is a highly specific antigen of the microorganism. Antibody levels against PGL-1 are frequently used to follow up the therapeutic response and elimination of the bacillary load in patients under treatment (Bach et al, 1988, Chanteau et al, 1989 and Meeker et al, 1990).

Because of the bacterial resistance induced by mono drug therapy, it has been necessary to develop and implement effective multidrug treatment of leprosy patients (Convite et al, 1986; Waters, 1993).

Leprosy produces disability and deformity through nerve damage. Type 1 reactions (reversal reactions) are generally known to be a major cause of nerve damage in borderline leprosy (BL) patients (Job, 1989).

Leprosy can cause damage to the cranial nerves and to many peripheral nerve trunks which leads to visible paralytic deformities (sugumaran, 1996).

Visual impairment or blindness is a frequent complication of leprosy. Blindness results either from mycobacterial infiltration and inflammation of structures in the anterior segment of the eye or from

trophic changes following damage to the trigeminal and facial nerves, resulting in lagophthalmos, deformed or corneal anesthesia (WHO, 1998,a)

**The aim of this review** is to highlighten the various drugs and drug combinations which can be used in the treatment of different types of leprosy and leprae reactions, and deformities to assess their effectiveness and their possible side effects.