IJTI | INTERNATIONAL JOURNAL OF THERAPEUTIC INNOVATION

Research article

Leprosy in the Modern Age: clinical diagnosis and challenges in the 21st Century

Bhavika Jaiswal*, Praful Rajpurohit, Yuvaraaj Munot, Prashant Mahale, Pavan Gosavi

Ahinsa Institute of Pharmacy, Dondaicha, Maharashtra, India

Corresponding author: Bhavika Jaiswal ✉ bhavikajaiswal26@gmail.com, **Orcid Id**: https://orcid.org/0009-0006-3985-4160

Received - 25-08-2024**, Revised** - 28-08-2024, **Accepted** - 30-08-2024 **(DD-MM-YYYY)**

Refer this article

Bhavika Jaiswal, Praful Rajpurohit, Yuvaraaj Munot, Prashant Mahale, Pavan Gosavi. Leprosy in the Modern Age: clinical diagnosis and challenges in the 21st Century. July-August 2024, V2 – I4, Pages - 0192 – 0202. Doi: https://doi.org/10.55522/ijti.v2i4.0060.

ABSTRACT

The bacterium Mycobacterium leprae is the source of leprosy, also known as Hansen's disease, which is mainly transmitted through person-to-person contact and has a long incubation period of 2–6 years. The disease primarily affects the skin and peripheral nerves, often resulting in long-term disability and severe social stigma. The purpose of this review is to provide an up-to-date analysis of leprosy, focusing on recent discoveries in genetics, susceptibility factors, and carriers of the disease. These results have implications for patient outcomes and management of leprosy. Historical diagnostic and therapeutic efforts have focused primarily on adult leprosy, but pediatric leprosy remains understudied. This review aims to fill this gap by providing updated diagnostic and treatment recommendations for adults and children with leprosy. We summarize clinical, bacteriological, and immunological methods for diagnosing leprosy and also consider contemporary issues facing clinicians and patients, including their impact on migration and disease spread. Directions for future research are also highlighted, emphasizing the need for innovative approaches to diagnosis and treatment. As advances in knowledge continue to progress, renewed focus on the control, prevention, and eradication of leprosy is critical to improving global health outcomes.

Keywords: Mycobacterium lepromatosis, Leprosy classification, Clinical diagnosis of leprosy, Leprosy symptoms and diagnosis.

INTRODUCTION

Calendula, Leprosy, also known as Hansen's disease, is a chronic bacterial infection resulting from Mycobacterium leprae (M. leprae) [1]. M. leprae belongs to the taxonomic order Actinomycetales and the family Mycobacteriaceae. It is an acid-fast, Gram-positive, obligate intracellular bacillus that exhibits a preference for infecting phagocytes in the skin and Schwann cells within peripheral nerves [2]. While the 9-banded armadillo spreads M. leprae in the wild across the southern United States, the bacterium is primarily cultivated in laboratories worldwide using the footpads of mice [3].

Leprosy is a chronic infection caused by the bacterium Mycobacterium leprae, which can affect various parts of the body, including the skin, peripheral nerves, upper respiratory tract, and in men, the testes. It is transmitted primarily through prolonged contact, often via nasal secretions or aerosols carrying bacterial droplets. Interestingly, despite its contagious nature, most people (about 95%) have natural immunity and can resist the infection. There is also a risk of vertical transmission, where the disease can be passed from an infected mother to her baby through the placenta. Understanding the complex modes of

leprosy transmission is important for effective prevention and control of the spread of leprosy.

Nerve damage can have profound, debilitating effects on the body. Loss of sensation in the arms and legs makes these areas especially vulnerable to burns and injuries, which can cause significant physical harm over time. Repeated injuries can exacerbate these issues, eventually leading to the loss of fingers, toes, or even entire limbs—commonly seen in cases of leprosy. Similarly, loss of sensation in the eyes can have serious consequences, as it disrupts the body's natural reflexes that protect the eyes from foreign objects. The absence of these protective mechanisms can lead to vision deterioration or even complete loss of sight, emphasizing the extensive impact of neurological damage on overall health.

Damage to motor nerves may result in muscle weakness and paralysis, leading to deformities such as wrist drop, claw hand, or foot drop. Paralysis of the eye muscles can cause lagophthalmos, a condition in which the individual cannot fully close their eyes. These nerve damage symptoms can severely affect a person's functional abilities, making professional medical care essential for treatment and prevention of further complications.

When the autonomic nervous system is damaged, sweating decreases, the skin becomes dry and cracked, and the risk of secondary bacterial infections increases. This condition can also lead to hair loss, including loss of eyebrow hair, which can further affect a person's appearance and self-esteem. If there are no neurological complications, leprosy can resemble a common mild infection. It is worth noting that it usually takes 2 to 10 years for the first symptoms of leprosy to appear. Interestingly, once treatment is started and the patient receives the first few doses, they quickly become noninfectious, greatly reducing the risk of spreading the disease. Therefore, isolation is usually not necessary throughout treatment due to the rapid change in the state of infection after starting treatment. Leprosy, which has a long incubation period, most commonly affects children whose immune systems are still developing. People at greater risk for the disease include those who have frequent and close contact with people with leprosy and those who live in areas where leprosy is common, such as South America, Africa, India, Nepal, and Tanzania. To effectively control the spread of leprosy, healthcare providers should educate these populations about prevention methods and early detection strategies.

Leprosy exists in two distinct forms: tuberculoid leprosy, which follows a milder course, and lepromatous leprosy, which is more severe. The World Health Organization (WHO) classifies leprosy into two categories: multibacillary, where larger and more numerous skin nodules are present, and paucibacillary, where skin lesions are less frequently observed. In multibacillary leprosy, patients typically develop multiple skin patches that thicken, lose sensitivity, and become lighter in color, often appearing symmetrically across the body. Symptoms such as nasal congestion and nosebleeds are also common in this form. Conversely, in paucibacillary leprosy, patients may experience similar sensory disturbances across several skin lesions, with symptoms usually lasting for several weeks or months. Each form of leprosy has distinct characteristics and progression patterns, helping to guide clinical diagnosis and treatment.

In addition to the mentioned symptoms, such as enlarged, nodular superficial nerves— especially noticeable near the elbows or knees—people with leprosy may also exhibit other signs, including dry corneas, ulcers on the soles of the feet, and muscle weakness. They may also experience sensitivity to temperature changes when touched, difficulties with fine motor skills, and an increased risk of secondary infections [4].

Ulcers, which are slow to heal, often leave behind atrophic scars or, in more severe cases, cause tissue destruction. These lesions typically develop on the hard and soft palate, beneath the tongue, and on the uvula, lips, and gums. Moreover, the effects of these ulcers can extend to the front of the upper jaw, potentially causing cavities that lead to tooth loss, and further deteriorating overall oral health [5]. Diagnosis is typically based on clinical suspicion and is confirmed through a series of thorough examinations, including bacteriological and histopathological studies, which often reveal characteristic noncaseating granulomas. Verification also involves the use of the lepromin test, a specific intradermal test that usually shows a negative result for lepromatous leprosy and a positive result for tuberculoid leprosy, aiding in the diagnostic process.

When evaluating possible causes of symptoms, it is crucial to consider a comprehensive list of differential

diagnoses, including conditions such as systemic lupus erythematosus, sarcoidosis, cutaneous leishmaniasis, and other similar skin diseases. Tertiary syphilis, cicatricial pemphigoid, fatal midline granuloma, lymphomas, systemic mycoses, traumatic lesions, and malignancies should also be taken into account. Additionally, peripheral nerve disorders, such as syringomyelia, and other neurological diseases must be considered during the diagnostic evaluation [6].

Symptoms and clinical features of leprosy

Headache, Malaise, Chilliness, Mental depression, Numbness of body, Skin Lesions, Oral Health Problems, Eye Problems.

Main diagnostic signs

Skin blemishes or hypopigmented areas are commonly observed in regions affected by leprosy.

Finger amputation.

Toes with claws.

Nerves thicken and hair begins to fall out.

Tumors on the face and ears' skin.

Sweating cessation or hair loss over the skin lesion.

Total or partial loss of sensation (pain, touch, and temperature) is common.

Epidemiology of leprosy

The introduction of multidrug therapy (MDT) worldwide by the World Health Organization (WHO) in 1982 in response to the increasing number of cases of dapsone-resistant leprosy was instrumental in reducing the number of new cases of leprosy worldwide from 5.4 million in the early 1980s to 210,000 cases in 2014[10]. The innovative approach of multimedia therapy (MMT) has proven so effective that the World Health Organization (WHO) has set an ambitious goal of eliminating leprosy as a public health problem by the year 2000. This is defined as reducing the prevalence to less than 1 case per 10,000 people, a milestone that was successfully achieved 20 years ago. Despite this remarkable decline in prevalence, the incidence has not been comparable. The current global incidence of leprosy remains at approximately 200,000 cases per year, with up to 10% of new diagnoses occurring in children under 15 years of age. This discrepancy between declining prevalence and stable incidence highlights the ongoing challenges in controlling leprosy globally and the need to continue to implement comprehensive strategies and interventions to make progress in the control of this ancient disease [7].

In 2016, the World Health Organization (WHO) introduced the Global Leprosy Strategy 2016-2020, an ambitious plan to accelerate progress toward a world without leprosy. This strategic framework aims to strengthen global efforts to eliminate leprosy through comprehensive policies and actions. The strategy's primary goal is to significantly reduce the incidence of stage II leprosy to less than 1 case per million people. Another primary goal is to eliminate leprosy-related disability in children, with a goal of zero reported cases. These efforts are critical to reducing the incidence of leprosy and achieving the goal of reducing the incidence of leprosy to less than 1 case per 10,000 people. Through this strategy, WHO aims to eradicate the persistent burden of leprosy, reduce its impact on individuals and communities, and create strong momentum toward a world without a leprosy epidemic [7]. According to the latest WHO data, 208,613 cases of leprosy were reported in 127 countries in 2018. In 2019, the number of cases decreased slightly by 1.2% to 211,009 cases [8].

Leprosy, a neglected tropical disease, has affected many parts of Africa, Asia, and the Americas, posing a serious challenge to public health systems in these regions. Despite ongoing control efforts, some areas of these endemic countries continue to experience alarmingly high prevalence rates, raising concerns among health professionals and policymakers. The reasons for this persistently high prevalence are unclear, and research is increasing to identify unique factors contributing to this phenomenon. By understanding the epidemiology of leprosy in these specific regions, researchers are working to develop targeted and effective interventions to mitigate the impact of the disease and reduce the burden on affected communities. Studying the complex interplay of social, environmental, and biological factors that influence the spread and persistence of leprosy can lead to the development of more tailored strategies to address this long-standing public health problem [14]. According to WHO data from 2018, the global burden of leprosy is concentrated primarily in two continents and three countries. India reported the most cases, with 120,338, followed by Brazil with 28,660, and Indonesia with 17,017. In comparison, countries such as Nigeria, the Democratic Republic of Congo, Ethiopia, and Tanzania reported between 1,000 and 10,000 cases each. In contrast, Canada, Algeria, and Mongolia reported no cases in that year [9].

Modes of transmission

Contact transmission

The bacteria that cause leprosy can be easily transmitted through direct physical contact with an infected person, such as a family member or patient. Indirect transmission through soil spores is also possible. Maintaining strict hygiene practices is essential to prevent the spread of leprosy.

Droplet infection

Leprosy is a contagious disease caused by the bacteria Mycobacterium leprae, and is mainly spread through the air through coughing or sneezing.

Other routes of transmission

This includes transmission to a child through breast milk, bites from vectors such as fleas or bedbugs, and contaminated tattoo needles [10].

Classification

Leprosy is classified as follows-

Madri classification

Intermediate Leprosy (IL)

Tuberculoid Leprosy (TL

Borderline Lepromatous (BL)

Lepromatous Leprosy (LL)

Indian classification of leprosy

Indeterminate leprosy

Tuberculoid Leprosy

Borderline Leprosy

Lepromatous Leprosy

Pure Neurotic Leprosy

Indian classification of leprosy

The Indian classification, developed in 1981 by the Hind Kusht Nivaran Sangh (Indian Leprosy Society), is a clinical bacterial classification:

Indeterminate leprosy

These early signs of leprosy are not reliable indicators of disease progression. They usually appear as hypopigmented spots with unclear borders that may be smooth or scaly. The number and location of these spots can vary, and some patients may experience loss of sensation in the affected area and thickening of peripheral nerves. Diagnosing leprosy at this stage is particularly difficult, as traditional methods of dermatopathology and molecular biology have limited diagnostic value. Complicating the situation is the fact that it can take up to five years for the disease to fully develop. If the disease is not detected in the early stages, it can progress to more severe forms depending on the individual's immune response, emphasizing the importance of early detection and intervention [11].

Figure 1: Indeterminate leprosy

Tuberculoid leprosy

Leprosy, which occurs in people with a strong immune response, usually presents as asymmetrically distributed plaques and papules on the extremities. Over time, these initial symptoms may develop into erythematous macules with central atrophy and hypopigmentation. In addition to the skin symptoms, some patients may develop secondary complications, such as alopecia, anhidrosis, loss of pain, loss of temperature sensation, and impaired tactile sensation in affected areas. Despite the severity of these symptoms, patients with leprosy are generally not highly contagious due to the microscopic nature of the disease. This emphasizes the importance of early detection and treatment to prevent further complications and potential spread [12].

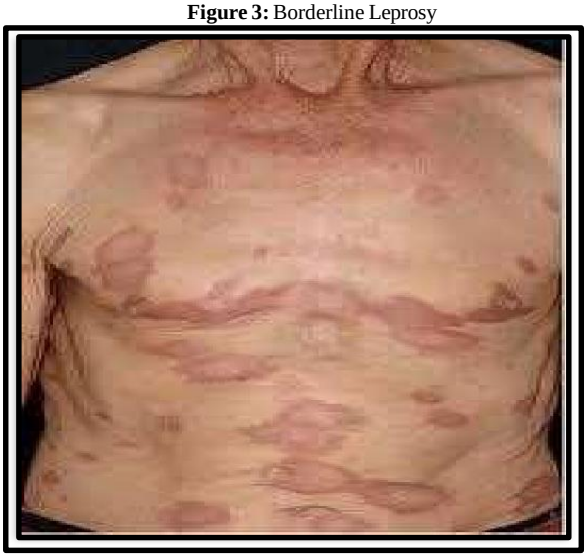
Figure 2: Tuberculoid Leprosy

Borderline leprosy

Leprosy lesions can vary in appearance, consisting of four or more lesions that are flat or raised, have clear or indistinct borders, and exhibit features such as hypopigmentation or erythema. These lesions are often

accompanied by sensory loss or other abnormalities that may indicate potential nerve damage or injury. The presence of multiple lesions positive for bacterial infection suggests the presence of a microbial component contributing to the pathology of the disease. Therefore, the wide spectrum of symptoms combined with a potential underlying microbial cause highlights the importance of comprehensive evaluation and investigation to accurately diagnose and treat these lesions. This includes the management of dermatological symptoms as well as careful monitoring for potential systemic complications such as nerve damage or other organ involvement. Effective treatment requires regular monitoring and implementation of appropriate treatment protocols to prevent disease progression and minimize long-term impact on the patient's overall health [13].

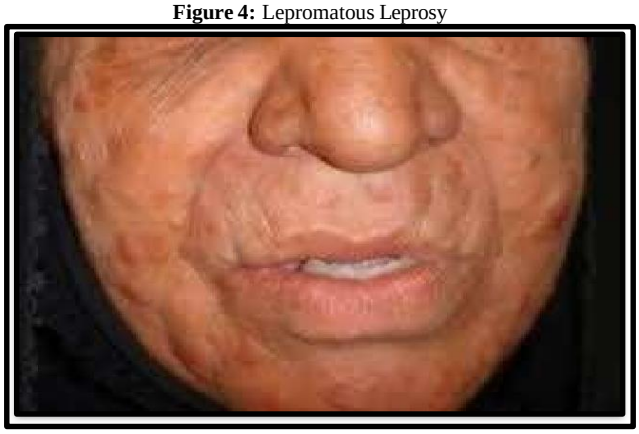
Figure 3: Borderline Leprosy



Lepromatous leprosy

Leprosy is characterized by multiple symmetrical papules and nodules that affect both the skin and mucous membranes. These lesions frequently appear on the lower extremities and face, especially on the earlobes. As the disease progresses, prominent nodules may appear symmetrically across the face, causing the eyebrows to disappear and giving a lion-like appearance. Damage to the nasal mucosa can lead to the destruction of important structures such as the nasal septum and nasopharynx, which can be further complicated by ulcers of the palate and larynx, which pose a serious risk. Among the serious complications of leprosy, loss of vision is a particularly worrisome problem, with up to 10% of patients suffering from complete blindness. Additionally, patients may experience other health problems such as glomerulonephritis, acute orchitis, amyloidosis, hepatitis, and periportal fibrosis, highlighting the wide-ranging impact of this disease on various organ systems [14].

Figure 4: Lepromatous Leprosy



Pure neurotic leprosy

Although there is no visible damage to the skin, this condition affects the nerve endings and disrupts the body's sensory systems in unexpected ways. These neurological effects reveal the complexity of the disease, which may go undetected without typical physical signs. Nerve involvement adds another layer to the medical problem where subtle abnormalities cause a variety of symptoms. This deepens our understanding of human physiology by highlighting the complex relationships between different body systems and the profound impact that seemingly unrelated body parts can have on one another [15].

Figure 5: Pure Neurotic Leprosy

Leprosy reactions

Immune response to M. leprosy plays a significant role in explaining the irreversible neurological damage that occurs in leprosy patients. This interaction between the host immune system and bacteria is important because it highlights the persistent immune response that contributes to persistent neurological damage and disease progression [16]. Leprosy reactions include acute and subacute inflammatory processes that primarily affect the skin and nerves and are a

major cause of morbidity and neurological disorders in patients. These reactions can occur continuously at any stage of the disease, regardless of whether medical intervention is performed. The skin and nerves are particularly vulnerable to these symptoms, which emphasizes the importance of vigilance and monitoring during leprosy treatment [17]. However, effective chemotherapy can sometimes trigger or worsen this reaction, as the bacilli are destroyed during or after treatment, releasing large amounts of antigenic material. This in turn stimulates a strong immune response. The immune system recognizes these antigens and mounts a strong defense, which can sometimes cause inflammatory symptoms as the body attempts to suppress and eliminate any remaining bacilli [18, 19].

Two different types of leprosy reactions have been described, affecting 30–50% of these patients;

Type 1 leprosy reaction also known as reversal reaction.

Type 2 reaction is known also called erythema nodosum leprosum.

Type 1

Type 1 responses are characterized by a spontaneous increase in cellular immunity and the development of delayed hypersensitivity to leprosy antigens. In contrast, type2 responses are thought to be caused by an immune complex disorder, although convincing evidence to support this hypothesis is currently lacking. These differences in responses to leprosy shed light on the complex interplay between immune responses and pathogen-specific antigens and the diverse mechanisms underlying the immune manifestations observed in leprosy patients [20]. This phenomenon is also characterized as a counter-reaction, in which a cellular immune response is triggeredin response to mycobacterial antigens. This response involves the activation of immune cells to combat the presence of these microbial antigens in the body [21, 22]. Type 1 reactions usually develop slowly over several weeks. They can also develop suddenly, but in these cases, they are usually accompanied by mild systemic symptoms. If left untreated, these reactions usually gradually disappear over several weeks or months [23, 24]. Clinical manifestations include a variety of symptoms, including erythema and induration around existing lesions, potential skin ulceration,and neuritis that may lead to paralysis, subsequent deformities, and the onset of newsensory loss [25].

Figure 6: Type 1 leprosy reaction also known as reversal reaction

Type 2 reactions

Type 2 reactions usually occur abruptly and are often accompanied by a wide range of systemic symptoms. These reactions usually last for 1 to 2 weeks and frequent relapses are common. The abrupt onset and prevalence of systemic symptoms distinguish type 2 reactions from other types of reactions and make them a serious problem in clinical practice [26, 27]. A type II reaction to erythema nodosum (ENL) is a type III humoral hypersensitivity reaction [28]. The ENL reaction is immunologically characterized by the deposition of immune complexes in tissues, blood, and lymphatics [29]. One of the notable features of this condition is the sudden appearance of numerous painful nodules that appear without any prior lesions. In addition, when signs of the disease appear, one may experience sensory and motor neuropathy, painful lymphadenopathy, orchitis, iridocyclitis, neuritis with myalgia, and arthritis/arthralgia [30].

Figure 7: Type 2 reaction is known also called erythema nodosum leprosum

Diagnosing leprosy

The presence of classic features is important for the diagnosis of leprosy, and additional methods such as skin smears, histopathology, or PCR serve as supporting tools to confirm the diagnosis. It isimportant to consider leprosy if you experience symptoms such as pale or red patches on the skin, decreased or loss of sensation in the affected area, numbness or tingling in the limbs, or weakness inthe extremities. Other important signs include tenderness of the extremities and nerve points, the appearance of nodules on the face or earlobes, and the development of painless sores or burns, especially on the extremities [31].

Leprosy is diagnosed when one or more of the following major signs appear

Definite loss of sensation in areas of skin with less pigmentation or redness.

Thickening or enlargement of peripheral nerves can cause a condition characterized by decreased sensation.

Muscle weakness in specific areas supplied by those nerves.

Presence of acid-fast bacteria in puncture spots [32].

Diagnostic tests

Complete physical examination of the patient and case history.

Histamine test.

Serum examination.

Biopsy examination

Laboratory test of secretions of the nose, throat, and skin of the patient.

Complications

Neurological complications

Leprosy, caused by the bacterium Mycobacterium leprae, primarily affects the peripheral nerves, causing neuritis with tenderness, thickening, and ridges. Denervation of the skin causes dryness, cracking, and ulceration. Secondary infections can cause bone damage and deformities, including saddle nose deformity. The most commonly affected nerves are the facial, trigeminal, ulnar, median, and radial nerves, causing complications such as eyelid closure disorders and dry nose. Early diagnosis and treatment are important to prevent serious complications.

Ophthalmologic involvement

Leprosy can cause eye damage both through the involvement of the branches of the facial nerve innervating the eye or by direct infection of the skin or the eye. Either of these leads to:

Inability to close the eyelids (lagophthalmos).

Keratitis due to exposure, absence of tears, and drying, leading to corneal ulcers.

Iridocyclitis.

Cataract secondary to eye disease.

Hand and foot involvement

Leprosy is a chronic infectious disease caused by the bacterium Mycobacterium leprae and is characterized by nerve damage in the arms and legs. This nerve damage causes a variety of debilitating symptoms, including loss of sensation, muscle paralysis, and deformities such as drooping nails or feet. In addition, the resulting denervation of the skin causes ulcers, making the skin more susceptible to secondary infections, and often results in the amputation of fingers or toes.

Systemic changes

In polybacterial leprosy, systemic damage may occur due to leprosy reactions or bacterial dissemination in the bloodstream. Kidney damage may occur due to type 2 reactions or secondary amyloidosis, leading to conditions such as glomerulonephritis and renal failure. Pulmonary tuberculosis is common and often affects the upper respiratory tract. Peripheral vascular abnormalities, ischemic ulcers, and autonomic dysfunction of the cardiovascular system may occur.

Orchitis is common and can potentially cause infertility. Adrenal cortical lesions may lead to adrenal insufficiency. Other complications include hepatic amyloidosis, pancytopenia due to bone marrow infiltration, bacteremia, and lymphadenopathy [30].

Prevention of complications

Although the response to leprosy is known to be very unpredictable, it is important to emphasize thatdisability due to leprosy is preventable and reversible if detected and treated early. One important aspect that should not be overlooked is the importance of careful patient education on self-care skillsas a primary approach to limiting disease progression. This learning process involves equipping individuals with the necessary skills, including the knowledge and tools necessary to effectively manage their condition and ultimately reduce their risk of potential disability:

Check your wounds and cuts daily and care for them properly.

Avoid plastic shoes and gloves that trap moisture and cause ulcers.

Cover wounds with clean bandages and care for them carefully.

Protect your hands and feet from accidental injuries.

Use the right tools to avoid using too much force, which could injure the patient.

Watch for signs of neuropathy.

Keep skin moist to prevent dryness and cracking, which can lead to ulcers and infections.

Seek treatment for eye infections as soon as possible to preserve vision.

Healthcare providers should also

Look for signs and symptoms of neuropathy and test muscle function and sensation.

Diagnose early.

Provide prompt treatment.

Teach and encourage thorough self-care.

Leprosy control

Nurses play a key role in the effective treatment and control of leprosy and serve as a vital link between healthcare providers and people affected by the disease. Their dedication and expertise are essential to ensuring that all leprosy control programs meet the essential components required for successful implementation and treatment.

Medical Measures: Estimation of the issue, early case discovery, medication therapy with several agents, surveillance, immunoprophylaxis, chemotherapy prophylaxis, deformities, rehabilitation, and others.

Social assistance.

Program administration.

Assessment.

As a community nurse, she is responsible for providing detailed explanations of three key areas of healthcare. This includes emphasizing the importance of cleanliness to prevent infection and the impact of sensory loss on hand, foot, and shoe care procedures. Providing clear advice on these aspects is essential to ensuring optimal health outcomes and quality of life for those in her care.

Treatment

Pharmacological treatment of leprosy typically involves combination drug regimens that include rifampicin, dapsone, and clofazimine. This multi-drug strategy is important for effectively treating the disease because it prevents the development of drug resistance that can occur with single- agent therapies. For example, when dapsone was initially used alone, drug resistance emerged, highlightingthe need to combine multiple drugs to target the leprosy bacteria from multiple angles. This combination approach increases the likelihood of successful treatment and minimizes the risk of resistance [31].

The Indian government has recommended Dapsone, a crucial medication for treating leprosy, as follows

Paucibacillary patients

Dapsone -100mg daily – 6 months Rifampicin - 600mg once a month

After completing the 6-month course of dapsone treatment, continued treatment is determined basedon the patient's condition. It is important to closely monitor patients for at least 2 years after treatment to ensure long-term effects and monitor potential relapses or side effects.

Multibacillary patients

Dapsone 100mg daily - for initial 2 weeks Rifampicin 600 mg daily - for initial 2 weeks. After that the treatment is given for 2 years.

Dapsone 100mg daily. Rifampicin 600mg once a month Clofazamines 50mg daily.

To ensure ongoing monitoring and maintenance of health and treatment effectiveness, it is recommended that daily treatment diagnostic examinations and regular follow-up examinations continue for 5 years.

Duration of treatment

Paucibacillary leprosy for 6 months.

Multibacillary leprosy for 12 months.

Educate the people regarding multidrug therapy.

Educate the importance of regular treatment.

Educate how to take medicine, storage of medicines, side effects of medicines, etc.

Explain about the duration of treatment.

Precaution body

Hygiene

You should wash your hands with soap every day.

Wash all parts of your body daily.

Pay special attention to any arm (or leg) that has no feeling.

Clothing hygiene

Dirty, unwashed clothes can be a breeding ground for a variety of pests, including fleas, lice, and other parasites. These insects not only irritate your skin but alsoopen the door for pathogens to enter your body, causing a variety of health problemsand infections.

Maintaining hygiene in your home

Sweep the house and corners daily and dispose of dust properly. Open the windows to let in some fresh air.

Keep your bathroom spotlessly clean.

It's Crucial to Maintain the Grounds around the House: Keep your lawn trimmed to maintain proper hygiene

Please dispose of kitchen waste in the trash can, not on the premises.

Sensation loss

Skin care

Wash your hands and feet every evening after work. It is recommended to use soap.

Soak your hands (or feet) in water for at least 20 minutes.

Scrape off dead skin cells with a stone (or) finger after bathing. Do not use a razor blade.

Do not use butter to moisturize your skin. Instead, use vegetable oil or petroleum jelly. Soaking is important.

Foot care

After washing your feet, inspect them and do things like press your thumbs into the six points marked with an "x" on your big toe.

If the area is swollen, itchy, or red. These problems are surprising. It can be prevented by not walking.

Take a break from work and rest your feet at home. Do not walk to the hospital if you need a bandage. Rest is the most basic form of treatment.

If you have lost feeling in your feet, wear appropriate shoes that will protect your feet.

When walking, take small steps. This will not put too much stress on your legs. Avoid uneven terrain.

Eye care

Routine actions such as rubbing your eyelids 20 times three times a day can help in the early stages of thinning eyelids.

Check your eyes daily to see if they are losing sensitivity. Use a small object to check for dust particles in your eyes. If so, remove them with a clean cloth.

Cover your eyes with a clean cloth (or sheet) at night. This will prevent dust from entering your eyes through the roof of the house that we cannot see.

Wash your hands thoroughly and apply fresh oil if your eyes are dry. Dry eyes are harmful. Wear safety glasses when exposed to direct sunlight.

CONCLUSION

In conclusion, although our understanding of leprosy has advanced significantly in the 21st century, there are still challenges to comprehensively addressing this ancient disease. Recent research has expanded our knowledge of the genetic drivers, susceptibility, and potential carriers of leprosy bacillus, revealing the complexity and diverse presentation of leprosy. These ideas have profound implications for disease control strategies and highlight the need for targeted approaches that address both the clinical and social aspects of leprosy. The historical focus on adult leprosy has overlooked important aspects of childhood leprosy that require renewed attention. Children not only suffer from leprosy but also play a critical role in the epidemiology of disease transmission. Therefore, updating diagnostic and treatment protocols that cover both adults and children is essential for a comprehensive approach to leprosy treatment. The integration of clinical, bacteriological, and immunological diagnostic techniques has improved our ability to more effectively detect and treat leprosy, but there are still challenges in applying these advances globally. Contemporary challenges such as international and domestic migration further complicate the fight against leprosy. Population movement affects disease transmission patterns and complicates leprosy case management, highlighting the need for adaptive and flexible strategies that take into account the mobility of affected individuals. As we move forward, several critical areas require targeted research and interventions. This includes developing more sensitive and specific diagnostic tools, optimizing treatment regimens to prevent drug resistance, and establishing comprehensive strategies to combat the stigma associated with leprosy. Additionally, improving global surveillance and response systems is critical to managing outbreaks and ensuring equitable access to health services. In summary, some progress has been made in understanding and eradicating leprosy, but continued research and innovation are needed to overcome the remaining challenges. By expanding our diagnostic capabilities, improving treatment protocols, and addressing the socioeconomic factors that influence disease control, we can move closer to our goal of eradicating leprosy and improving the quality of life for those affected by the disease.

REFERENCES

1. S Ghosh, S Chaudhuri, 2015. Chronicles of Gerhard Henrik Armauer Hansen's life and work. Indian Journal of Dermatology, 60(3) Pages 219. Doi: 10.4103/0019-5154.156310.

2. C F Paige, R W Truman, D T Scholl, 2002. Prevalence and incidence density of Mycobacterium leprae and Trypanosoma cruzi infections within a population of

wild nine- banded armadillose. American Journal of Tropical Medicine and Hygiene. 67(5) Pages 528–532. Doi: 10.4269/AJTMH.2002.67.528.

3. Thomas, Liji, 2023. What is Leprosy? News-Medical. Retrieved on April 08, 2024.

4. Aufderheide AC, Rodríguez-Martín C, 1998. The Cambridge Encyclopedia of Human Paleopathology. Cambridge: Cambridge University Press. Pages 141-154.

5. Fucci da Costa AP, Da Costa Nery JA, Wan-del-Rey de Oliveira ML, et al, 2003. Oral lesions in leprosy. Indian J Dermatol Venereol Leprol. 69, Pages 381-385.

6. Núñez-Martí JM, Bagán JV, Scully C, et al, 2004. Leprosy: dental and periodontal status of the anterior maxilla in 76 patients. Oral Diseases. 10, Pages 19-21. Doi: 10.1046/j.1354-523x.2003.00981.x.

7. Rao P N, 2017. Global leprosy strategy 2016-2020: Issues and concerns. Indian Journal of Dermatology, Venereology, and Leprology. 83, Pages 4-6. Doi: 10.4103/0378-6323.195075.

8. Victor SS, Carlos DFS, Paulo RS, Martin F, and Luis E. Cuevas Leprosy: why does it persist among us? Expert Review of Anti-infective Therapy, 2020. Doi: https://doi.org/10.1080/14787210.2020.1752194.

9. World Health Organisation, Facts Sheet on Leprosy, 2019, (Cited 2020 June 02).

10. World Health Organisation, Neglected Tropical Diseases: News, 2019, (Cited 2020 June 02).

11. Ploemacher T, Faber WR, Menke H, et al, 2020. Reservoirs and transmission routes of leprosy; A systematic review. PLoS Neglected Tropical Diseases. 14(4), Pages 8276. Doi: 10.1371/journal.pntd.0008276.

12. World Health Organisation, Global Health Data Repository: Leprosy- Number of new leprosy cases Data per country (Cited 2020 May 24th).

13. Vishal Krishnaji Ghorpade. 2022. A Brief Review on Hansen 's disease (Leprosy). Saudi J Nurs Health Care. 5(7), Pages 158-160. Doi: 10.36348/sjnhc.2022.v05i07.006.

14. Ramesh MB, Chaitra P. 2012. Leprosy: An Overview of Pathophysiology, Interdisciplinary Perspectives on Infectious Disease 2012: 181089. Pages Doi: 10.1155/2012/181089.

15. Fajardo TT, 1973. Indeterminate leprosy: a five-year study, clinical observations. International Journal of Leprosy. 4(1), Pages 576. Doi: 10.12691/ajidm-8-3-1.

16. Marcellus F, 2017. Leprosy - an overview of clinical features, diagnosis, and treatment. Journal of the German Society of Dermatology. 15(08), Pages 801-827. Doi: 10.1111/ddg.13301.

17. Elinav H, Palladas L, Applbaum YH, et al, 2006. Plantar ulcers and eyebrow-hair paucity. Clinical Infectious Diseases. 42(5), Pages 684-685. Doi: 10.1086/502983.

18. D. Pandhi and N. Chhabra, 2013. New insights in the pathogenesis of type 1 and type 2 lepra reaction. Indian Journal of Dermatology, Venereology and Leprology. 79(6) Pages 739. Doi: 10.4103/0378-6323.120719.

19. Ma Victoria F Balagon, Robert H Gelber, Rodolfo M Abalos, et al, 2010. Reactions following completion of 1- and 2-year multidrug therapy (MDT). The American Journal of Tropical Medicine and Hygiene. 83(3) Pages 637– 644. Doi: 10.4269/ajtmh.2010.09-0586.

20. I. P. Kahawita, S L Walker, DN Lockwood, 2008. Leprosy type 1 reaction and erythema nodosum leprosum. Anais Brasileiros de Dermatologia. 83, Pages 75–82. Doi: 10.1590/S0365-05962008000100010.

21. W C S Smith, P G Nicholls, L Das, et al., 2009. Predicting neuropathy and reactions in leprosy at diagnosis and before incident events-results from the INFIR cohort study. PLoS Neglected Tropical Diseases. 3(8) Pages e500. Doi: 10.1371/journal.pntd.0000500.

22. Scollard DM 2006. The continuing challenges of leprosy. Clinical microbiology reviews. 19(2), Pages 338-381. Doi: 10.1128/CMR.19.2.338-381.2006.

23. W. Alemu Belachew, B. Naafs, 2019. Position statement: leprosy: diagnosis, treatment and follow-up," Journal of the European Academy of Dermatology and Venereology. 33(7), Pages 1205–1213, Doi: 10.1111/jdv.15569.

24. E M S´anchez-Mart´ınez, F J Melgosa-Ramos, L. M. Moneva-Leniz, et al, 2021. Erythema nodosum leprosum successfully treated with apremilast: more effective and safer than classic treatments. International Journal of Dermatology. 60(2), Pages E74–E76. Doi: 10.1111/ijd.15205.

25. World Health Organization. Guidelines for the diagnosis, treatment, and prevention of leprosy.

26. Thomas, Liji. 2019. Leprosy Complications. News-Medical, viewed 09 April 2024.

27. Veerabhadrappa, G M, 2015. The Short Textbook of Community Health Nursing. 1st edition, Jaypee brother's medical publishers, New Delhi. Pages 184-189.

28. Teli A R, Ghorapade N, 2018. A Study to Assess the Level of Self-Esteem among Leprosy Patients at Selected Societies of Sangli District. International Journal of Nursing Education. 10(4), Pages 12-14. Doi:10.5958/0974-9357.2018.00091.0.

29. Vishal Krishnaji Ghorpade, 2022. A Brief Review on Hansen's disease (Leprosy). Saudi J Nurs Health Care. 5(7), Pages 158-160. Doi:10.36348/sjnhc.2022.v05i07.006.

30. Imran A Sheikh, Sachin S Sakat, Rakesh S. Dole, et al, 2024. Assessment of the Bio-enhancing Potential of Carum Carvi Extract in Transdermal Drug Delivery Systems for Hypertension. Afr.J.Bio.Sc. Doi:https://doi.org/10.33472/AFJBS.6.Si2.2024.3712-3728.

31. Rakesh S Dhole, Rajesh D Ahire, Purushottam S Marathe, et al, 2024. Impact of Food Nutrient in the IBD Management and Prevention. IJTI. 2(1), Pages 85-88. Doi: 10.55522/ijti.V2I1.0020.

32. Rajesh D Ahire, Rakesh S Dhole, Supplementing with DHA and ARA for infant's brain and vision development. IJTI. 1(2), Pages 4-7 Doi: 10.55522/ijti.V1I2.0002.