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PREVALENCE, PATTERN AND RISK FACTORS ASSOCIATED WITH PERIPHERAL  
NEUROPATHY AMONG LEPROSY PATIENTS: A RETROSPECTIVE STUDY

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## DECLARATION

Assurance of principal investigator

I, ENDRIS SEID hereby declare that this thesis titled “**Prevalence, Pattern And Risk Factors Associated With Peripheral Neuropathy Among Leprosy Patients, Boru Meda General Hospital, Dessie, Ethiopia: A Retrospective Study**” is my original work and has not been submitted partially or in full for any degree in any other university, and all sources of materials used for the thesis have been duly acknowledged.

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## List of abbreviations

AAU:	Addis Ababa University
AORs:	Adjusted Odds Ratios
BGH:	Boru Meda General Hospital
CORs:	Crude Odds Ratios
CNS:	Central Nervous System
DM:	Diabetes Melitus
HIV:	Human Immunodeficiency Virus
HMIS:	Health Management Information System
IRB:	Institutional Review Board
MB:	Multibacillary
MDT:	Multi Drug Treatment
M.liprae:	Mycobacterium liprae
MRN:	Medical Record Number
NFI:	Nerve Function Impairment
ORs:	Odds Ratios
PB:	Paucibacillary
PI:	Principal Investigator
PN:	Peripheral Neuropathy
PNS:	Peripheral Nervous System
SSA:	Sub Saharan Africa

## Abstract

**Background:** Leprosy, also known as Hansen disease, is a bacterial disease caused by *Mycobacterium leprae*. The disease affected mankind for over 4000 years. Leprosy is believed to be originated initially in Eastern African regions and spread worldwide by means of migration of people. Leprosy causes skin and nerve infections. It was investigated that the Hansen's bacilli, *Mycobacterium leprae*, predominantly targeted peripheral nerves.

**Objectives:** To assess the prevalence, pattern, and risk factors associated with peripheral neuropathy among leprosy patients in Boru Meda General Hospital, Dessie, Ethiopia.

**Methods and materials:** The study was conducted in Boru Meda General Hospital, Dessie, Ethiopia. Institution based retrospective cross-sectional study design was employed. The study was conducted from February 2022– May 2022. A total of 380 charts were selected for the retrospective document review. A pre-test was performed in 5% of the total sample size of patients at Dessie referral hospital. Data were collected using a well-designed open data kit form. Data analysis was performed using SPSS version 25 statistical software. Data were presented as mean  $\pm$  SD. Both bivariate and multivariate binary logistic regressions were employed to identify factors associated with leprosy peripheral neuropathy. A variable with a p-value of  $< 0.2$  in the bivariate model was subjected to multivariate analysis to avoid the effect of confounding variables. Adjusted odds ratios were calculated at 95% confidence interval and considered significant with a p-value of  $\leq 0.05$ .

**Results:** The prevalence of leprosy peripheral neuropathy was found to be 60%. According to the multivariate logistic regression, sex (AOR = 2.218; 95% CI: 1.230-3.998), age (AOR = 5.287; 95% CI: 2.473-11.301), leprosy reaction (AOR = 2.509; 95% CI: 1.269- 4.962), number of skin lesions, (AOR = 3.642; 95% CI: 1.411-9.403), duration of the disease (AOR = 4.866; 95% CI: 1.130-20.958) and WHO classification of leprosy (AOR =3.636; 95% CI: 1.431- 9.243) were significantly associated with the occurrence of peripheral neuropathy in leprosy.

**Conclusion:** More than half of the study participants had leprosy peripheral neuropathy. Male sex, advanced age, presence of leprosy reactions, presence of more than four skin lesions, longer duration of the disease, and multibacillary leprosy were the risk factors associated with leprosy peripheral neuropathy.

**Key words:** Leprosy Peripheral Neuropathy, Prevalence, Associated factors, Ethiopia

## 1. Introduction

### 1.1 Background of the study

Leprosy is a bacterial disease caused by *Mycobacterium leprae*. This bacterium causes skin and nerve infections. Leprosy is otherwise termed as Hansen's disease- so named after a Norwegian physician Gerhard Henrik Armauer Hansen recognized the causative agent in 1873(1). It has been known that leprosy affected manhood for more than 4000 years. Leprosy is supposed to be firstly originated in Eastern African regions and blowout globally by means of movement of people(2). Leprosy is recognized by its predominant inflammatory response to the mucous membranes covering the nose, eyes, upper respiratory tracts, and extremity nerves. When *M. leprae* damages nerves, it causes loss of sensation. This, combined with gradual tissue deterioration, leads to deformity of the extremities. (3). The cause of paralysis in leprosy patients was unclear until the late 1940s. Subsequently, significant progress was made by leprologist and hand surgeon Paul Brand (1914–2003), who discovered that *Mycobacterium leprae* primarily targets peripheral nerves(4).

## 1.2 Statement of the problem

One of the neglected tropical diseases, leprosy still poses a serious threat to public health in many developing countries (5). Approximately 250 000 new people are registered worldwide each year, indicating a high rate of leprosy case detection (6). A WHO estimate states that every year, between 500,000 and 750,000 new cases of leprosy are discovered worldwide. In one instance, more than 500,000 new cases were identified in 2005, which equates to more than 1400 instances each day or about 60 cases every hour(7). Despite the fortunate 55% global decline in leprosy prevalence due to multidrug treatment and WHO surveillance measures, leprosy continues to be a major cause of peripheral neuropathy. Leprosy prevalence in 2012 was highest in south-east Asia (116 per 100,000), followed by Africa (53 per 100,000) and Central and South America (46 per 100,000). Relatively, the illness primarily affects immigrants in Europe and North America(8). Africa appears to be one of the six epidemiological leprosy regions of the world zoned by WHO(5). In terms of impairment, it is expected that leprosy will disable 3 million individuals globally(9). Ethiopia, after the Democratic Republic of the Congo, is the most impacted nation in Sub-Saharan Africa (SSA). Between 2004 and 2010, 4000–4500 new cases were detected annually at medical facilities(7). Of the eighteen nations that reported 93% of all new cases found worldwide in 2009, Ethiopia came up at number seven. The average number of new cases stayed steady between 2001 and 2011, despite a decline in prevalence from 5081 to 4516(10). Nevertheless, Ethiopia reached the leprosy elimination target of WHO which is 1 case per 10,000 population in 1999, and the occurrence of new cases remains a challenge(11). Peripheral nerve damage due to leprosy causes loss of sensation and tissue damage leading to infectious disabilities. This can gradually lead to self-amputation of the hands and feet. Blindness is also part of the complication(12). The disease is curable and physical disabilities that impact on the individual's social and working life can be prevented with early treatment(13). The typical late-stage clinical features of leprosy comprise sensory and motor loss in the face and limbs. It's these aspects of the disease that leads to the severe social consequences of the disease that are so well known(14).

Due to significant peripheral nerve involvement, leprosy grew to become a stigmatizing public health problem of sumptuous dimension(15). The specific nerves affected by leprosy are the facial nerve, the ulnar, median and the radial cutaneous in the arm and hand, and the posterior tibial and the sural in the leg and foot(16).

Among the most prevalent neurological conditions are peripheral neuropathies, which have an annual incidence of 77 cases per 100,000 individuals, a prevalence of 1-20% across all age

categories, and as high as 30% in the elderly.(17, 18). As estimated through clinical case series, about 4%–8% of all leprosy is limited to only peripheral nerves resulting in diagnostic challenge(19, 20). Globally, earlier to diabetic neuropathy, leprosy used to be the commonest peripheral nerve disorder. Even though leprosy affects both CNS and PNS, peripheral nerves are typically involved(21). Although being a chronic infectious disease of public health importance and one of the leading causes of permanent physical disability, the drop in prevalence following MDT has resulted in the neglect of leprosy(22). Despite imposing such a significant societal burden, studies on the prevalence, pattern, and risk factors associated with leprosy-related peripheral neuropathy are limited. Consequently, decision-makers, especially those in the study area, will find useful information from this study regarding the prevalence, pattern, and contributing variables of leprosy-related peripheral neuropathy.

### 1.3 Significance of the study

In order to improve strategies for the management of leprosy peripheral neuropathy, its epidemiology needs to be well studied. In Ethiopia, data is scarce regarding the prevalence, pattern and risk factors associated with leprosy peripheral neuropathy. Hence, this study was intended to assess the prevalence, pattern and risk factors associated with leprosy peripheral neuropathy. The findings of this study could be useful in addressing potential issues related to the disease's diagnosis and therapy. It would also be served as an important input to develop recommendations that inform some clues for future researchers in this area.

## 2. Literature review

### 2.1 Prevalence of leprosy peripheral neuropathy

Even though leprosy is regarded as a disease of skin and nerves, it is the neurological aspect makes leprosy a disease with a high impact. In leprosy, neuropathy can arise gradually or can be a consequence of reactions. New patients often present with some level of nerve function impairment (NFI) with the percentage varies between 2% and 55%, with subordinate figures for PB cases(23). 2.4-29% of the patients develop neuropathy during or after treatment(16) as well; but it's contingent on classification and pre-existing nerve function impairments. Leprosy patients have a significant degree of nerve damage; at the time of diagnosis, up to 60% of multibacillary patients exhibited clinically noticeable nerve damage; 10% of patients incur new nerve damage following medication treatment, and 30% of patients experience further nerve damage during treatment(14). Prevalence of leprosy peripheral neuropathies is estimated to be higher in south east Asia which is 116 per 100,000, followed by Africa, 53 per 100,000 and central and south America, 46 per 100,000(8). Two North American sites, the University of Kansas Medical Center and the University of Texas Southwestern Medical Center, and one South American site, Federal Fluminense University in Brazil, had their neuromuscular databases evaluated for neuropathy diagnoses. There were 1090 cases overall in North America and 1034 cases in South America. In North American, out of 53 leprosy cases, 26(49%) had neuropathy due to leprosy and in South American out of 141 leprosy cases, 39 (28%) were cases of neuropathy due to leprosy(24). A study conducted on 63 patients with Hansen's disease in American, Samoa reported the prevalence of neuropathy to be 54%(25). A study investigated on 221 community-based hospital outpatient leprosy patients in Carville, Long Hansen's Disease Centre, in USA reported the prevalence of neuropathy as 67%(26). According to an institutional based study conducted in Washington, D.C., U.S.A., out of 1721 leprosy patients, 622(36%) had neuropathy(27). An institutionally based cross-sectional study done in Toronto general hospital suggests that among 184 leprosy patients, nerve involvement was 55.5% in the upper extremity and 54.3% in the lower extremity(28). An institutional-based retrospective cross-sectional study of patients referred to the Hospital for Tropical Diseases in London, United Kingdom, showed that 68% of the patients had nerve damage(29). A cross-sectional study conducted in Northern Brazil on 12328 patients showed nerve trunks are affected in 10.4% of patients(30). In a study done in Ecuador on 39 patients to identify patients benefiting from early neurolysis by neurosensory testing the prevalence of leprosy neuropathy determined was found to be 97.4%(31).

A multi-centre prospective cohort study done in North India on 303 patients showed that 115 patients (37.9%) had nerve damage at diagnosis(32). A cohort of 303 was followed for two years and revealed that neuropathy was found to be 20–50%(33). Another institutional based study done on 200 leprosy patients in India revealed the prevalence of neuropathy due to leprosy to be 63%(34). In a Prospective cohort study conducted on 864 patients in Northwest Bangladesh in 2008, NFI occurred in 13%(35). In the Chris Hani Baragwanath Academic Hospital in Johannesburg, South Africa, 80 adult and paediatric patients' clinical records were reviewed backward and 46.2% of people had nerve involvement(36). According to a prospective cross-sectional hospital-based study performed on 70 adult leprosy patients in Sudan, half of the patients had upper limbs sensory nerve dysfunction, 42.86% exhibited sensory nerve dysfunction in the lower limbs and approximately half (48.57%) of the patients had upper limbs motor dysfunction(37). The ALERT MDT Field Evaluation Study (AMFES) which is a cohort study done in Ethiopia showed that 67% of 594 new leprosy cases had NFI(16).

## 2.2 Risk factors associated with leprosy peripheral neuropathy

According to studies, people who come into contact with leprosy sufferers are more likely than the general public to get neuropathy. For leprosy neuropathy, Numerous risk factors have been suggested, including the patient's kind of leprosy, the contact's age and gender, and the contact's physical and genetic proximity to the patient(38). According to a cohort of 6,158 contacts of 1,201 newly-diagnosed leprosy patients treated at the Leprosy Laboratory of Fiocruz from 1987 to 2007, in Brazil, the leprosy-related variables that remained associated with leprosy neuropathy were contacts of male index cases, consanguineous relationship, 4 years of schooling and 4 to 10 years of schooling. Among incident cases, household exposure to leprosy and contacts who were 15 years and older were risk factors for contracting leprosy neuropathy. In another study, in a contact cohort of 192 leprosy patients, consanguineous relationship, intra-household and MB cases were significantly associated with leprosy neuropathy(39). A multi-centre cohort study of 303 leprosy patients in N. India showed that the presence of skin lesions increases the risk of sensory or motor impairment by 3-4 times(40). Drawing on a retrospective institutional-based observational study conducted between 1990 and 2019, 10644 newly diagnosed leprosy patients in Yunnan, China, the following variables were linked to the development of neuropathy, which in turn led to physical handicap in leprosy patients: delayed

diagnosis by 2–5 and 5–10 years, male sex, old age, zero skin lesions, rural occupation, and WHO categorization of PB(41). In a population-based cohort study done in Indonesia, men had a 2.2 times higher risk than women(42). In a case-control study done on 31 leprosy patients and 31 non-lepers in South Sulawesi, Indonesia, Chi-square statistical tests showed that there was a significant relationship between contact history with the incidence of leprosy(43). In a population-based prospective cohort study on 1,037 leprosy patients in northwest Bangladesh, higher age with a bimodal distribution, 15–19 and age >50, contacts of patients with PB leprosy with 2–5 lesions (PB2–5) and those with multibacillary (MB) leprosy, household group and a close genetic relationship when blood-related children, parents, and siblings indicated an increased risk(44). A prospective cross-sectional hospital-based study conducted in Sudan in 2006 showed that 18- 27 years was the most affected common age group(45). The variables that are associated with leprosy neuropathy causing disability in the bivariate analysis, those in the age group 30-50 and those 50 years and above, patients with duration of symptoms of 6 to 12 months and those with greater than 24 months, and age greater than 30 all maintained their association in the multiple logistic regression analysis, according to a cross-sectional institutional based retrospective record review study conducted on 513 patients in ALERT, Ethiopia.(46).

### 2.3 Pattern of leprosy peripheral neuropathy

Typically, sensory nerves are impacted by leprosy before motor nerves. Older histological investigations demonstrated that neuropathy first affects tiny nerve fibers (myelinated A-fibers followed by unmyelinated C-fibers), which transmit pain, heat, and cold sensations and control autonomic function, such as sweating(47). The large fibers (A-fibers), which are in charge of feeling vibration, touch, and pressure, are subsequently affected. However, the INFIR investigation discovered that each patient's initial afflicted modality and nerve fiber varies from one another(48).

### 2.4 Forms of peripheral neuropathy related to leprosy

#### 2.4.1 Pure Neuritic Leprosy

Pure Neuritic Leprosy (PNL) affects only the peripheral nerves and has no associated skin involvement(49). Up to 4%–8% of people have it. According to Jardim et al., paraesthesia was the most prevalent manifestation in 49 patients with PNL, occurring in 55% of cases. This was followed by motor dysfunction in 24% of cases, neural tenderness in 12% of cases, and sensory

loss in 8% of cases. The posterior tibial, common peroneal, ulnar, and median nerves are frequently affected by PNL(50).

#### 2.4.2 Silent Neuropathy

Sensory and motor dysfunction without skin symptoms, nerve soreness, pain, paraesthesia, or numbness are the hallmarks of silent neuropathy (SN). Another name for it is "quiet nerve paralysis"(40). It frequently occurs in both type 1 and type 2 leprosy reactions (erythema nodosum leprosum). At the time of their initial evaluation, up to 7% of newly presenting patients are thought to have silent neuropathy (SN). It has an incidence rate of 4.1 per 100 persons at risk during follow-up visits. Up to 75% of patients develop silent neuropathy within the first year of starting chemotherapy(51). Due to the lack of clinical symptoms reported by SN patients, a regular and thorough nerve function assessment(52).

#### 2.4.3 Leprosy reactions

An immunological event known as a "leprosy reaction" can arise before to, during, or after multidrug therapy (MDT) for leprosy(53). Type 1 responses (T1R) and erythema nodosum leprosum (ENL) are two of the most common consequences of leprosy. These discrete presentations happen independently, but they might appear in the same patient at various points in time. It is crucial to understand that each of these disorders have the potential to cause a permanent loss of nerve function(54). 7.6% type I and 2.9% type II leprosy reactions were seen in an observational prospective study conducted on 722 leprosy patients (77.8% Paucibacillary (PB) and 22.2% Multibacillary (MB)) at six clinics of The Leprosy Mission International Bangladesh (TLMI-B) Dhaka Program. At diagnosis, 7.1% of people experienced a reaction, and 3.4% of people did not(53).

##### 2.4.3.1 Type-1 reactions

Because the immune response initially seems to be waning and then "reverses" to become more severe, type 1 reaction is also known as a reversal reaction. It could be a presenting symptom of leprosy, develop during MDT treatment, or even last for three or four years after treatment is finished(55). A partial change in cell-mediated immunity occurs in type-I response (also known as the Reversal Reaction, or RR or T1 R ). It may happen at any time, but it usually happens right after MDT begins. With a frequency of 30% in these patients, TIR is frequently seen in borderline patients(56). The emergence of acute inflammation in skin lesions, nerves, or both is a T1R's defining feature.(40) T1Rs are often recurring and can cause additional nerve injury(57). Skin lesions that are severely inflammatory, oedematous, and ulcerated may be

observed in T1R. Although systemic symptoms are uncommon, oedema of the hands, feet, and face can also be a sign of a response. If acute neuritis caused by a T1R is not treated promptly and effectively, it might result in NFI, which can permanently impair nerve function and cause peripheral sensory and/or motor neuropathy(58).

#### 2.4.3.2 Type-II reaction

Type-II reaction (Erythema Nodosum Leprosum, ENL) is a type-III hypersensitivity reaction that results in neutrophil infiltration and complement activation when immune complexes are deposited extra vascularily. ENL has an acute onset and affects a variety of organs, but it can also progress to a chronic stage and recur(59). In ENL, systemic symptoms are more noticeable. ENL is characterized by fever and tender, unpleasant red papules or nodules. The face and extensor surfaces of the limbs are frequently affected by the nodules, which develop in crops(56). Wide geographic variation can be seen in the frequency of ENL reactions, with 37% of newly diagnosed borderline and lepromatous cases in Brazil experiencing ENL(60). Statistics in Asia reported that ENL has an acute onset and affects a variety of organs, but it can also progress to a chronic stage and recur(59).

#### 2.4.4 Leprosy late-onset neuropathy

Late-onset neuropathy (LLON) is a type of leprosy neuropathy in which nerve deterioration is manifested years after MDT and cannot be accounted for by relapses or responses. Patients with LLON may have several mononeuropathies or polyneuropathies, which are chronic conditions with a gradual progression.(61)

#### 2.4.5 Leprosy related autonomic neuropathy

The only known bacillus that specifically invades the human Peripheral Nervous System (PNS) is *M. liprae*.(62) Leprosy affects nerve fibres in the sensory, motor, and autonomic systems. Despite the fact that autonomic dysfunction is common in leprosy patients, it is frequently discounted and there is a paucity of research on the subject.(63) Ermakova established in 1936 how the sympathetic chain and vagus nerve are involved in leprosy. H.A. Alnord was the first who studied the anhidrosis in denervated areas of skin in both lepromatous and tuberculoid leprosy in 1949.(64)

## 2.5 Deformities and disabilities due to leprosy neuropathy

Because controlling the process requires rapid diagnosis and therapy, it's crucial to understand neuropathies from the perspective of how disabilities arise. The reason for this is that rapid treatment and early diagnosis are vital for halting the progression of nerve injury and are the most effective measures for preventing and reducing impairments. Nevertheless, the chronic stage of the illness is when deformities and limitations manifest. These come from the wasting and weakening of the muscles that are innervated by the damaged nerves, such as the claw hand from ulnar nerve damage or the foot drop from peroneal nerve damage. Trophic lesions on the hands, fingers, and foot sole may also result from a loss of pain perception. Additionally, fingers and toes might absorb the disease in its later stages.(2)



Figure-1: Pictures showing leprosy with peripheral nerve damage

dropped wrist, clawed fingers with stiff joints due to ulnar and median nerve damage (a), foot ulceration due to loss of sensation (b), facial nerve paralysis (c), and swelling of the great auricular nerve (d)(65).

## 2.6 Conceptual frame work

This is the conceptual framework that shows the proposed relationship between independent variables and leprous neuropathy adopted from literature review.

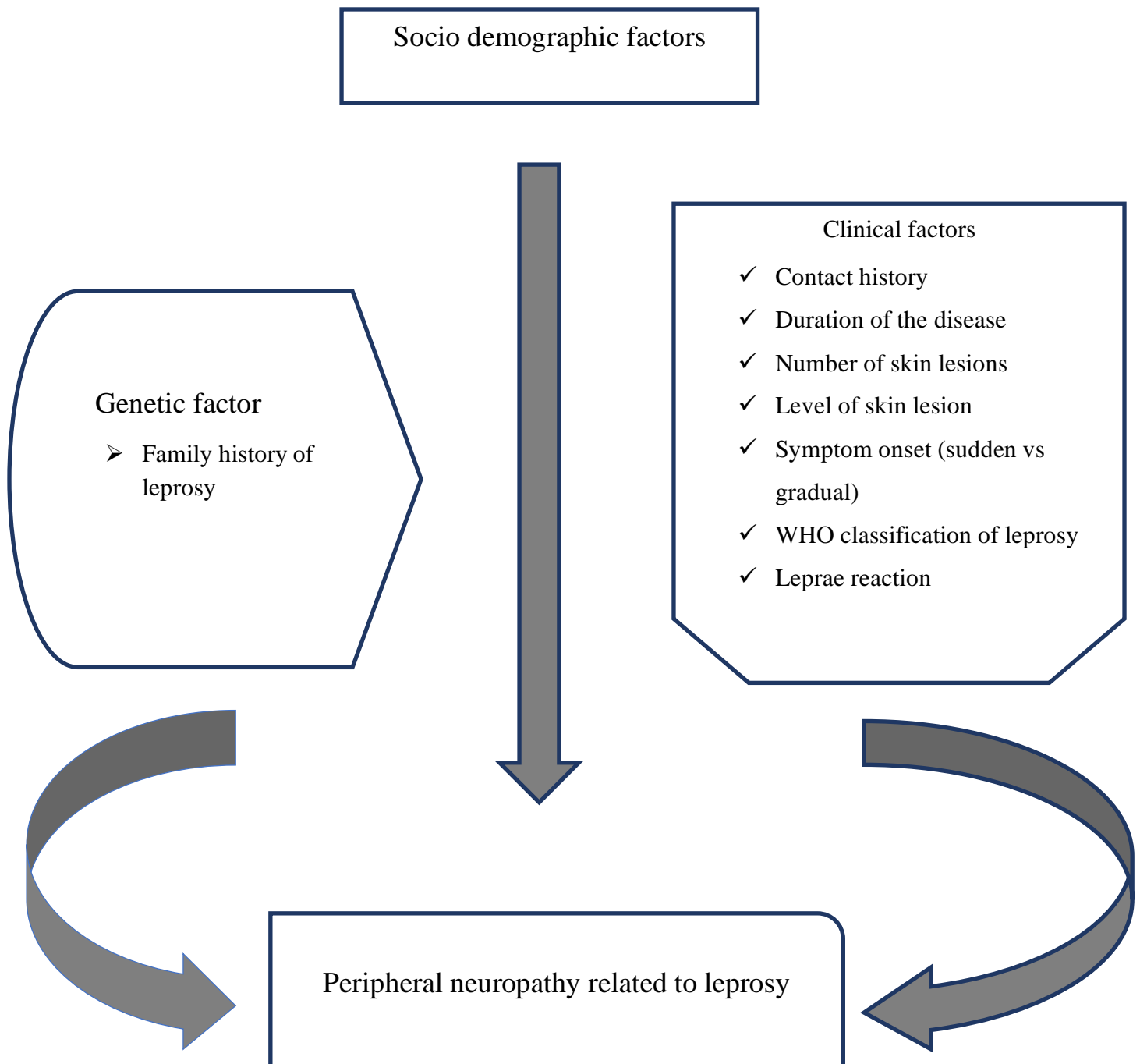


Figure-2: Diagram showing the conceptual framework of the study

### 3. Objectives

#### 3.1 General objective

- To assess the prevalence, pattern and risk factors associated with leprous peripheral neuropathy

#### 3.2 Specific objectives

- To determine the prevalence of leprous peripheral neuropathy
- To assess the pattern of leprous peripheral neuropathy
- To identify risk factors associated with leprous peripheral neuropathy

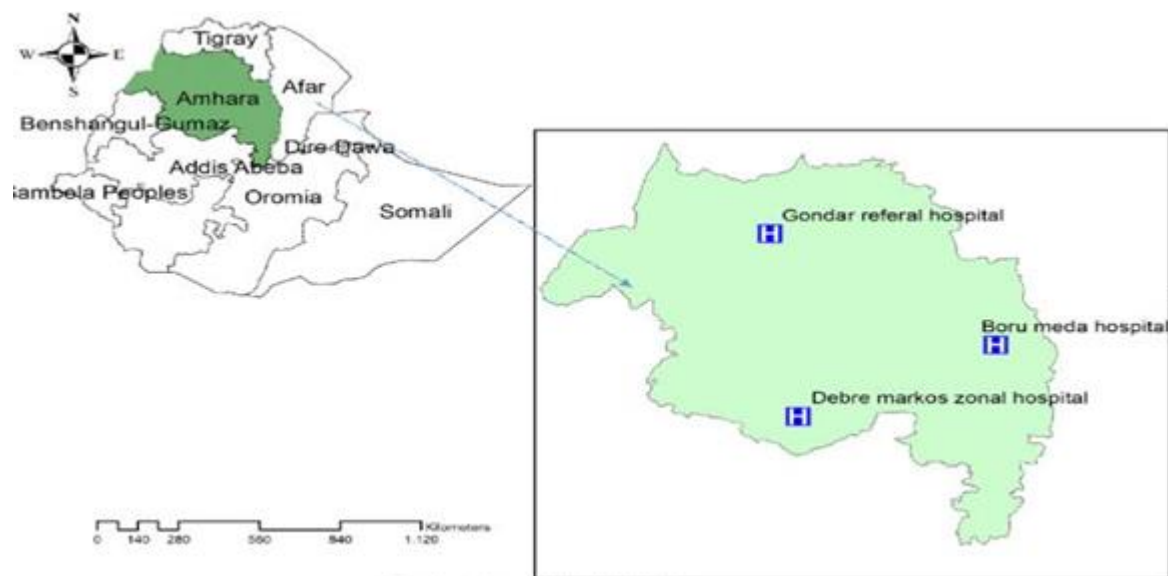
## 4. Materials and methods

### 4.1 Study design

An institution-based cross-sectional retrospective study was conducted to evaluate the prevalence, pattern and risk factors associated with leprosy peripheral neuropathy from Sep, 2020 to 2022.

### 4.2 Study area

The study was conducted at Boru Meda General Hospital, located in the south Wollo zone, Amhara region, Ethiopia. The hospital is among leprosy referral centers distributed across Ethiopia. It was initially established by missionaries in 1954 mainly to provide care for leprosy and related complications. Gradually, the hospital started providing general medical services to the society. The hospital has 40 beds for leprosy and other dermatology cases. In addition, the hospital has three dermatology outpatient offices. On average, approximately 20 leprosy patients are seen per month in the hospital.



*Figure-3: Map showing the study area*

### 4.3 Population

#### 4.3.1 Source population

Charts of all patients diagnosed with leprosy attended Boru Meda General Hospital during the study period.

#### 4.3.2 Study population

Charts of all patients diagnosed with leprosy attended Boru Meda General Hospital during the study period who fulfilled the inclusion criteria.

#### 4.3.3 Study unit

Chart of single participant that is selected based on inclusion criteria and sampling techniques during the study period.

### 4.4 Inclusion and exclusion criteria

#### 4.4.1 Inclusion criteria

Charts of patients diagnosed with leprosy who attended Boru Meda General Hospital during the study period were included in the study.

#### 4.4.2 Exclusion criteria

Patient charts that have a diagnosis of comorbid illness like DM and HIV, documented history of chronic alcohol use and exposure to pesticides at work place were excluded. Accordingly, 10 HIV and 25 DM, a total of 35 patients have been excluded.

### 4.5 Sample size determination and sampling technique

#### 4.5.1 Sample size determination

The actual sample size was determined by using the single population proportion formula, where the following assumptions are considered,  $p = 50\%$  (prevalence of 50% is used), 95% confidence interval, 5% margin of error and 10% non-response. The formula is as follows:

$$n = \frac{(Z_{\alpha/2})^2 * p(1-p)}{d^2}, \text{ where; } p = 0.5$$

$d$  = marginal error between the samples and population  $(0.05)^2$

$Z_{\alpha/2}$  = critical value at 95% certainty  $(1.96)^2$

$n$  = calculated sample size = 384

When 10% non-respondent rate is added, the final sample size was  $n = 422$ .

A random sampling technique was used to select the first chart.

#### 4.5.2 Sampling procedure

The medical record numbers (MRN) of all patients were collected from the dermatology clinic health management information system (HMIS) registration book of the hospital within the study period. While the collection of MRNs of all leprosy patients, they were selected and then assigned by a consecutive number according to the registration number in HMIS. The total prevalence of neuropathy related to leprosy was defined as the number of leprosy neuropathy cases, among total patients who attended Boru Meda General hospital during the study period.

#### 4.6 Study variables

The proposed relationship between independent variables and leprosy neuropathy was adopted from the literature review.

##### 4.6.1 Dependent variable

- Prevalence of leprosy peripheral neuropathy

##### 4.6.2 Independent variables

###### **Sociodemographic factors**

- Age
- Sex
- Education level
- Occupation
- Residence (urban vs rural)

###### **Clinical factors**

- Duration of the disease
- Number of skin lesions
- Level of skin lesion
- Mode of symptom onset
- WHO classification of leprosy
- Contact history with leprosy patient

###### **Genetic factor**

- Family history of leprosy

#### 4.7 Operational definitions

**Neuropathy (peripheral):** Neuropathy (peripheral): functional impairment and/or structural damage to sensory, motor and autonomic nerve fibres within the peripheral nervous system; Pattern of leprosy neuropathy: a form of neuropathy which affects the peripheral nerves. It can be sensory, motor or both(66).

**Leprosy related peripheral neuropathy:** is a chronic infectious neuropathy caused by *Mycobacterium leprae*.(67)

**Pauci-bacillary:** five or fewer lesions with no bacteria detected in the skin smear (sample taken from the area); **Multi-bacillary:** more than five lesions or a bacterium is detected in skin smear, or both(68).

**Neuropathic pain:** presence of symptoms (pain, hyperpathy, paresthesias) without progressive loss of neural, sensory and motor function(69).

**Sensory impairment:** a patient is diagnosed as having sensory impairment in any of the following situations: the monofilament threshold is increased by three or more levels (filaments) on any site, OR two levels on one site and at least one level on another site, OR one level on three or more sites for one nerve(70).

**Motor impairment:** a patient is diagnosed as having motor impairment if the Voluntary Motor Test (VMT) score for any muscle is less than four(71).

**Nerve Function Impairment (NFI):** nerves that presented some alteration of sensory and/or motor function

#### 4.8 Data collection tools, procedures and data analysis

Patients were presented to dermatoneurological examination by dermatologists, neurologists and nurses. The diagnosis of leprosy were made according to clinical criteria, histopathology exams and bacilloscopy. Classification of leprosy was made based on the WHO operational classification. Data were collected using a well-designed Open Data Kit (ODK) form. First MRNs were obtained from HMIS registration book to get the main file of the patient from the patient's chart room. Next in the patients' charts room; from the main card, the necessary details were sought in terms of age, sex, and other essential variable data completeness. Finally, based on the inclusion and exclusion criteria of the study, cards which had variables for the study were used. Then, all variables were collected from the main card information. Two nurses with a BSc degree were assigned to collect the data from patients' card room. The data

collectors were supervised by two senior BSc degree nurses during the process of data collection. Timely supervision was undertaken by the principal investigator (PI) during the data collection period.

#### 4.9 Data quality control

A detailed orientation was given to the data collectors and supervisors on techniques of data collection, data collection material and the purpose of the research. Supervision was carried out on a daily basis to check completeness and consistency by the supervisors to assure the quality of data. A pre-test was performed in 5% of the total sample size of patients' charts prior to the actual data collection time and correction was carried out. To increase the data quality, double data entry to Epi data was considered.

#### 4.10 Data analysis and interpretation

After the data was checked for its completeness, it was fed into the Epi data manager, version 4.6.0.0, and then exported to SPSS Version 25 for analysis. For categorical data, descriptive statistics like frequency and percentage were computed and presented by the use of tables, bar graphs. Continuous variables were summarized using means and standard deviation. Logistic regression analysis was employed to identify factors associated with leprous peripheral neuropathy. Each independent variable was first subjected to bivariate logistic regression analysis, and variables with p-values less than 0.2 were identified as potential candidates for multivariable logistic regression, which was used to account for potential confounders. Using multivariable logistic regression, the strength of the connections between the dependant and predictor variables was evaluated at a P-value  $\leq 0.05$  cut-off point using adjusted odds ratios (AORs) and their related confidence intervals (CIs). The Hosmer-Lomeshow goodness of fit test was used to assess the model's fitness. Another test for multicollinearity was performed at a 10% variance inflation factor (VIF).

#### 4.11 Ethical consideration

The Institutional Review Board (IRB) of Addis Ababa University (AAU) College of Health Sciences, Anatomy department, granted ethical approval. To obtain authorization for data collection, a letter requesting collaboration and ethical clearance was also sent to the management of Boru Meda General Hospital. Patient information was kept private by collecting the data in an anonymously. Following data collection, the principal investigator was the only person with access to the raw data, which was maintained.

## 5. Results

### 5.1 Patients' characteristics

#### 5.1.1 Socio-demographic characteristics

Out of 422 reviewed patient charts, 380 (90%) were with complete records fulfilling the inclusion criteria. Of these, 203 (53.4%) were male participants and 177 (46.6%) were females. The mean of age was  $41.25 \pm 9.8$ . Age group of < 30 years accounted for the majority, 128(33.7%) and 30-39 represented the least, 82(21.6%). Majority of participants, 97(25.5%) were unemployed and the least, 33(8.7%) were government employee.

155(40.8%) of the study participants were married. More than half of the patients, 234(61.6%) were rural residents. 119(31.3%) of participants had no formal education and smallest portion, 40(10.5%) were degree holders (Table-1).

#### 5.2 Clinical characteristics

Greater number of patients, 263(69.2%) suffered from leprae reaction. Of which, 140(36.8%) type-1 and 123(32.4%) type-2. Skin lesions of patients ranged 0-7 and the majority, 128(33.7%) had two to three skin lesions. In more than half of patients, 240(67.2%) skin lesions were superficial. The majority, 211(59.1%) of patients started to show symptoms gradually as evidenced by the reported mode of onset of the disease in the history of present illness. The majority of patients, 154(40.5%) had 2-5 years duration with disease and only 25(6.6%) stayed with the disease for >10 years. The mean and SD of duration of the disease of patients were 2.4 and 1.9 respectively. While 263(62.1%) patients had PB leprosy, 144(37.9%) were MB cases.

More than three quarters of patients, 291(76.6%) had no previous history of contact with leprosy patients and only 89(23.4%) had history of contact. Similarly, 285(75%) had not family history of leprosy whereas 95(25%) had familial history (Table-2).

Table-1: Sociodemographic characteristics of patients with leprosy at BMGH 2022, Dessie, Ethiopia.

Variables	Category	Number	Percentage
Sex	Male	203	53.4
	Female	177	46.6
Age	< 30 years	128	33.7
	30-39 years	82	21.6
	40-49 years	85	22.4
	≥ 50 years	85	22.4
Occupation	Unemployed	97	25.5
	Student	42	11.1
	House wife	46	12.1
	Farmer	84	22.1
	Self-employee	78	20.5
	Government- employee	33	8.7
Marital status	Single	105	27.6
	Married	155	40.8
	Divorced	75	19.7
	Widowed	45	11.8
Residency	Urban	146	38.4
	Rural	234	61.6
Level of education	No formal education	119	31.3
	Primary school	74	19.5
	Secondary school	73	19.2
	Diploma	74	19.5
	Degree and above	40	10.5

Table-2: Clinical characteristics of patients with leprosy at BGH 2022, Dessie, Ethiopia.

Variables	Category	Number	Percentage
Lepra reaction	No	117	30.8
	Type-1	140	36.8
	Type-2(ENL)	123	32.4
No of skin lesion/s	0-1	115	30.3
	2-3	128	33.7
	4-5	54	14.2
	6-7	83	21.8
Level of skin lesion	Superficial	240	67.2
	Deep	117	32.8
Mode of symptom onset	Sudden	146	40.9
	Gradual	211	59.1
Duration of the disease (in years)	≤ 1	94	24.7
	2-5	154	40.5
	6-10	107	28.2
	>10	25	6.6
WHO classification of leprosy	PB	236	62.1
	MB	144	37.9
History of contact	No	291	76.6
	Yes	89	23.4
Family history	No	285	75.0
	Yes	95	25.0

Out of 228 infected nerves, the most commonly involved nerves were in the upper limb. Among the specified nerves identified from the patients' charts, ulnar nerve was the most affected nerve with frequency of 25 (10.96%) followed by median nerve, 20 (8.78 %). Whereas radial nerve was the least affected peripheral nerve with frequency of 4(1.75%) (Table-3).

Table-3: Frequency of specific nerves involved in PN among leprosy patients at BGH 2022, Dessie, Ethiopia.

Nerve involvement in specific location	Frequency	Percentage (%)
Nerves involved in the upper limb	79	34.6
Nerves involved in the lower limb	55	24.1
Nerves involved both in the upper and lower limbs	24	10.5
Not specified	70	30.7
Total	228	100

### 5.3 Prevalence and patterns of leprosy peripheral neuropathy

From 380 cards selected for analysis, 228 leprosy patients had peripheral neuropathy. This makes the overall prevalence of leprosy peripheral neuropathy 60%. In terms of sex, leprosy peripheral neuropathy was recorded to be 60.1% in males and 39.9% in females.

With respect to age, leprosy peripheral neuropathy was observed 36.7% in <30 years, 58.5% in 30-39, 77.6% in 40-49 and 78.8% in  $\geq 50$ .

The current study intended to present pattern of leprosy peripheral neuropathy in terms of sensory impairment, motor impairment, both sensory and motor impairments, neuropathic pain and nerve thickenings. The most common presentation was sensory impairment which was revealed in 24.5% of patients followed by motor impairment which was encountered in 15.8% of patients. Neuropathic pain and nerve thickening affected 2.4% and 1.3% respectively. Sensory and motor impairments were more common in males and neuropathic pain and nerve thickenings were more prevalent in females.

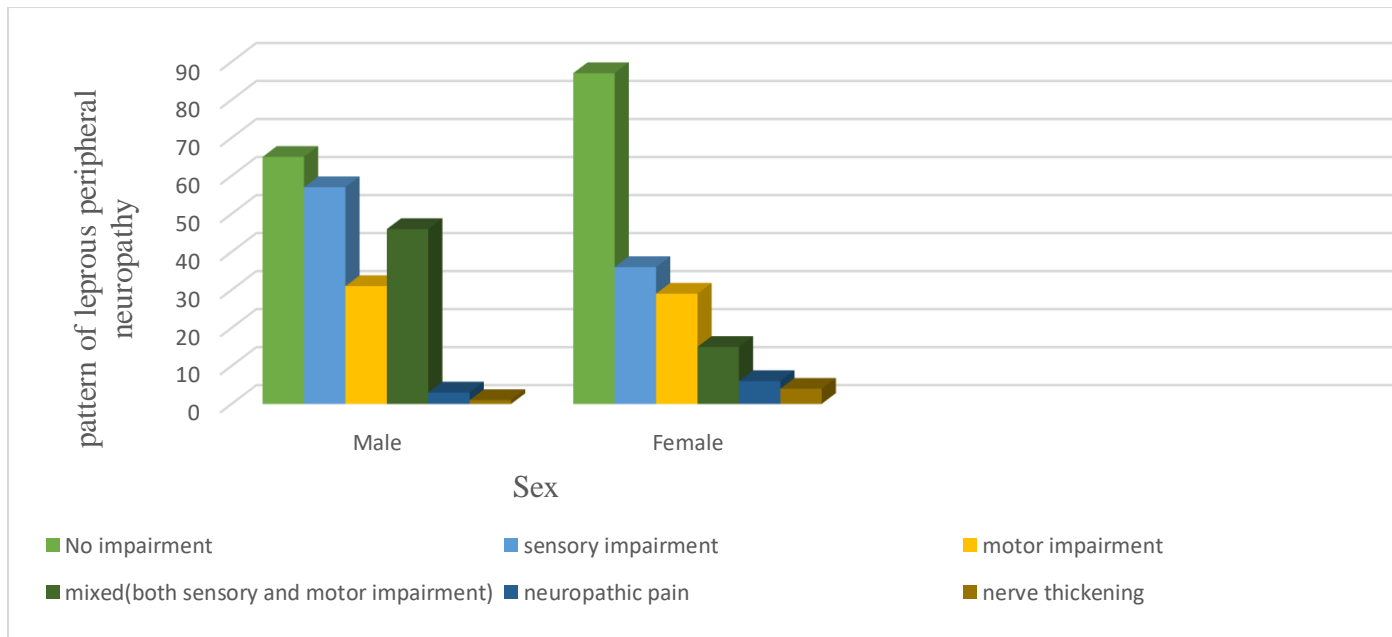


Figure 4: Distribution of pattern of leprosy peripheral neuropathy in terms of sex

#### 5.4 Factors associated with leprosy peripheral neuropathy

In order to determine a crude risk estimate, variables that were anticipated to play a role in the development of leprosy peripheral neuropathy were added to the binary logistic regression analysis. Variables upon bivariate inquiry, which demonstrated evidence of correlation with the outcome variable at a p-value of less than 0.25 were included in the multivariate logistic regression analysis. Accordingly, sex (COR = 1.962; 95% CI: 1.294-2.974, P-value = 0.002), age group 30-39 (COR= 2.433; CI (1.379-4.292), P-value= 0.002), age group 40-49 (COR= 5.987; CI: 3.207-11.175, P-value= 0.000), age >50 (COR = 6.415; 95% CI: 3.408-12.073, P-value = 0.000), unemployed (COR = 2.851; 95% CI: 1.264-6.430, P-value = 0.012), farmer (COR= 2.500; 95% CI: 1.095-5.708, P-value = 0.030), self-employee (COR= 3.077 95% CI: 1.326-7.143, P-value = 0.09), rural residency (COR = 2.056; 95% CI: 1.346-3.139, P-value = 0.001), no formal education (COR= 2.962; 95% CI: 1.416-6.198, P-value = 0.004), primary education (COR = 2.611; 95% CI: 1.185-5.754, P-value = 0.017), secondary education (COR= 2.411; 95% CI: 1.095-5.307, P-value = 0.029), type-1 leprosy reaction (COR= 3.020; 95% CI: 1.806-5.049, P-value = 0.000), type-2 leprosy reaction (COR= 2.324; 95% CI: 1.383-3.905, P-value = 0.001), 2-3 skin lesion (COR= 1.913; 95% CI: 1.148-3.186), P-value = 0.013), 4-5 skin lesion (COR= 3.988; 95% CI: 1.956-8.133, P-value = 0.000), 6-7 skin lesion (COR= 5.419; 95% CI: 2.831-10.373, P-value = 0.000), level of skin lesion (COR= 2.441; 95% CI: 1.494-3.988, P-value = 0.000), mode of symptom onset (COR= 2.235; 95% CI:

1.443-3.461, P – value = 0.000), having the disease for 2-5 years (COR= 2.349; 95% CI: 1.390-3.970, P – value = 0.001), for 6-10 years (COR= 3.777; 95% CI: 2.098-6.799, P – value = 0.000), for more than 10 years (COR= 11.297; 95% CI: 3.156-40.441, P – value = 0.000), WHO classification of leprosy (COR= 3.235; 95% CI: 2.039-5.133, P – value = 0.000) and history of contact (COR= 1.850; 95% CI: 1.109-3.089, P – value = 0.019) were associated with leprous peripheral neuropathy in the crude odds ratio analysis and may be responsible to develop leprous peripheral neuropathy. history of contact (COR= 1.850; 95% CI: 1.109-3.089, P-value = 0.019) were associated with leprous peripheral neuropathy in the crude odds ratio analysis and may be responsible to develop leprous peripheral neuropathy. (Table-4)

Table 4: Bivariate analysis - Predictors of PN among leprosy patients at BMGH 2022, Dessie, Ethiopia

Variables	Category	LPN		Bivariate analysis	
		Yes	No	p-value	COR (95% CI)
Sex	Male	137	66	.002*	1.962 (1.294-2.974)
	Female	91	86		1
Age	< 30	47	81		1
	30-39	48	34	.002*	2.433 (1.379-4.292)
	40-49	66	19	.000*	5.987 (3.207-11.175)
	≥ 50	67	18	.000*	6.415 (3.408-12.073)
Occupation	Unemployed	63	34	.012*	2.851 (1.264-6.430)
	Student	26	16	.055	2.500 (.981-6.372)
	House wife	22	24	.457	1.410 (.569-3.492)
	Farmer	52	32	.030*	2.500 (1.095-5.708)
	Self-employee	52	26	.009*	3.077 (1.326-7.143)
	Government-employee	13	20		1
Residency	Urban	72	74		1
	Rural	156	78	.001*	2.056 (1.346-3.139)
Level of education	No formal education	79	40	.004*	2.962 (1.416-6.198)
	Primary school	47	27	.017*	2.611 (1.185-5.754)
	Secondary school	45	28	.029*	2.411 (1.095-5.307)
	Diploma	41	33	.118	1.864 (.853-4.070)
	Degree and above	16	24		1
Lepra reaction	No	51	66		1
	Type-1	98	42	.000*	3.020 (1.806-5.049)
	Type-2(ENL)	79	44	.001*	2.324 (1.383-3.905)
No of skin lesion/s	0-1	48	67		1
	2-3	74	54	.013*	1.913 (1.148-3.186)
	4-5	40	14	.000*	3.988 (1.956-8.133)
	6-7	66	17	.000*	5.419 (2.831-10.373)
Level of skin lesion	Superficial	133	107		1
	Deep	88	29	.000*	2.441 (1.494-3.988)
Mode of symptom onset	Sudden	74	72		1
	Gradual	147	64	.000*	2.235 (1.443-3.461)
Duration of the disease (in years)	≤1	37	57		1
	2-5	93	61	.001*	2.349 (1.390-3.970)
	6-10	76	31	.000*	3.777 (2.098-6.799)
	>10	22	3	.000*	11.297 (3.156-40.441)
WHO classification of leprosy	PB	118	118		1
	MB	110	34	.000*	3.235 (2.039-5.133)
History of contact	No	165	126		1
	Yes	63	26	.019*	1.850 (1.109-3.089)

In the multivariate analysis, age, sex, duration of the disease, leprae reaction, number of skin lesion/s and WHO classification of leprosy appeared to have association with leprosy peripheral neuropathy at a p-value of  $< 0.05$ . After adjusting for all other model parameters, patients between the ages of 40 and 49 had a 4.2-fold increased risk of developing leprosy peripheral neuropathy compared to those under 30 (AOR = 4.214; 95% CI: 1.973-8.999). Additionally, participants 50 years of age and above had a 5.3-fold increased risk of developing leprosy peripheral neuropathy compared to individuals under 30 years of age (AOR = 5.287; 95% CI: 2.473-11.301), assuming that no other variables changed. The other associated factor was sex. Male participants were 2.2 times more likely to develop leprosy peripheral neuropathy as compared with females (AOR = 2.218; 95% CI: 1.230-3.998). Those participants of greater than 10 years of duration with disease were 4.87 times more likely to develop leprosy peripheral neuropathy as compared with those with shorter leprosy history less than 1 year (AOR = 4.866; 95% CI: 1.130-20.958). Similarly, compared to patients lacking a leprae reaction, leprosy patients with type-1 and type-2 leprosy reactions had 1.9 and 2.5 times higher odds of developing leprosy peripheral neuropathy (AOR = 1.982; 95% CI: 1.050-3.742) and (AOR = 2.509; 95% CI: 1.269-4.962), respectively. Likewise, patients who had 4-5 skin lesions were 3.2 times and those who had 6-7 skin lesions were 3.6 times more likely to develop leprosy peripheral neuropathy as compared with those who had less than or equal to one skin lesion (AOR = 3.187; 95% CI: 1.115- 9.108) and AOR = 3.642; 95% CI: 1.411- 9.403) respectively. Finally, patients who had MB leprosy were 3.6 times more likely to develop leprosy peripheral neuropathy as compared to those who were PB (AOR = 3.636; 95% CI: 1.431-9.243) (Table-5).

Table 5: Multivariate analysis - Predictors of PN among leprosy patients at BMGH 2022, Dessie, Ethiopia.

Variables	Category	LPN		Multivariate analysis	
		Yes	No	p-value	AOR (95% CI)
Sex	Male	137	66	.008*	2.218 (1.230-3.998)
	Female	91	86		1
Age	< 30	47	81		1
	30-39	48	34	.099	1.825 (.892-3.733)
	40-49	66	19	.000*	4.214 (1.973-8.999)
	≥ 50	67	18	.000*	5.287 (2.473-11.301)
Occupation	Unemployed	63	34	.318	1.821 (.562-5.897)
	Student	26	16	.081	3.213 (.868-11.899)
	House wife	22	24	.415	1.749 (.456-6.707)
	Farmer	52	32	.902	.925 (.267-3.205)
	Self-employee	52	26	.146	2.397 (.737-7.796)
	Government-employee	13	20		1
Residency	Urban	72	74		1
	Rural	156	78	.283	1.425 (.746-2.720)
Level of education	No formal education	79	40	.072	2.557 (.921-7.099)
	Primary school	47	27	.064	2.717 (.942-7.837)
	Secondary school	45	28	.387	1.589 (.557-4.539)
	Diploma	41	33	.190	1.969 (.715-5.422)
	Degree and above	16	24		1
Leprae reaction	No	51	66	.024	1
	Type-1	98	42	.035*	1.982 (1.050- 3.742)
	Type-2(ENL)	79	44	.008*	2.509 (1.269- 4.962)
No of skin lesion/s	0-1	48	67		1
	2-3	74	54	.232	1.563 (.751- 3.254)
	4-5	40	14	.031*	3.187 (1.115- 9.108)
	6-7	66	17	.008*	3.642 (1.411- 9.403)
Level of skin lesion	Superficial	133	107		1
	Deep	88	29	.813	.863 (.255-2.924)
Mode of symptom onset	Sudden	74	72		1
	Gradual	147	64	.839	1.072 (.545-2.109)
Duration of the disease (in years)	≤1	37	57		1
	2-5	93	61	.617	1.207 (.577-2.524)
	6-10	76	31	.327	1.534 (.651-3.615)
	>10	22	3	.034*	4.866 (1.130-20.958)
WHO classification of leprosy	PB	118	118		1
	MB	110	34	.007*	3.636 (1.431-9.243)
History of contact	No	165	126		1
	Yes	63	26	.035	.297 (.096-.917)

\* Value statistically significant; AOR: adjusted odds ratio; 1: reference.

## 6. Discussion

### 6.1 Prevalence of leprosy peripheral neuropathy

The purpose of the current study was to ascertain how common peripheral neuropathy is among leprosy patients as well as its correlates. The study findings indicated that 60% of people had leprosy peripheral neuropathy. The prevalence of the current study is nearly similar to those reported from Toronto, where it was determined to be 55%, and India, 63% (28, 72). A study from Carville, USA reported the prevalence of neuropathy as 67% (26). This slight difference may be resulted from variations in the study population, diagnostic standards, and participant selection procedures. Prevalence of leprosy peripheral neuropathy in the current study was slightly less common than the 68% prevalence reported from the United Kingdom (29). The difference in the instruments, sample strategies, and study designs may account for this discrepancy. The prevalence of leprosy peripheral neuropathy in the present study was lower than that of a study done in Ecuador, which was 97.4% (31). The higher prevalence of the Ecuador study may be attributable to variations in the study design, instrument, and study conditions. The region was also extremely endemic.

On the other hand, the prevalence of the present study was higher than studies conducted in India and Northwest Bangladesh which were 37.9% and 13%, respectively (35, 40). This difference might be explained by the difference in genetic susceptibility, health care qualities and also the study design used, which was prospective cohort in both studies.

Inconsistent with the present finding, an institution-based descriptive retrospective study conducted in Colombia on 282 leprosy patients using electronic health records showed that the prevalence of neuropathy to be 13.8% (73). This discrepancy might be due to the fact that in our study most patients were from rural areas, low social level, strenuous roads and inaccessibility to health care centers account for higher prevalence rate.

The prevalence of leprosy peripheral neuropathy reported from Sudan was 42.9%, which was smaller than the current study (37). The possible reason for this difference might be that the sample size was small and the study design was prospective cross-sectional.

Utilizing the currently available individual-based model SIMCOLEP, a study was carried out to forecast leprosy incidence patterns in the future by simulating the spread of *M. leprae* within a household-structured population. Research was conducted in areas with significant

endemicity. According to this study, almost 75% of patients did not recognize they had ever interacted with an index patient(74). However, compared to high-endemic Bangladesh, Richardus et al. (75) discovered that in low-endemic Thailand, a smaller percentage (one-third) of newly diagnosed leprosy patients had no prior contact history. This study was done in Ethiopia, where there is a low endemicity and a higher percentage of patients did not have a history of contact. PB leprosy cases in this study had a higher rate than MB cases. However, a trend of MB form preponderance has been noted in other research.(76-78). Our population's high prevalence of PB forms could be attributed to comparatively strong immunity.

32.4% of patients in the current study experienced a type 1 reaction, whereas 36.8% experienced a type 2 reaction. Type 1 reaction prevalence rates have been reported in a variety of previous research, with ranges from 19% to 30% (58, 79).

The number of males (53.4%) exceeded that of females (46.6%). In general, leprosy had been more prevalent in males than females(80). Male patients outnumbered female patients in certain Asian studies conducted in the Philippines and India, but no sex preponderance was noted in certain African nations(81-83).

The ulnar nerve was the most damaged nerve in the present investigation. In a comparable fashion, studies conducted in Colombia revealed that the most often afflicted nerves were the ulnar, anterior, and posterior tibial nerves(73).

According to the current study, sensory impairment was the most frequent manifestation, occurring in 24.5% of patients, and motor impairment in 15.8% of patients. When using nylon monofilaments for testing, Brown et al.(84)found that 29% of people had sensory impairment.

Leprosy was usually discovered in patients evaluated in our center after a protracted period of symptomatic sickness. This is in line with a prior analysis of Hansen disease patients in North America(85). The reason for this delay may be related to patients were not seeking care or having limited access to care(86, 87).

## 6.2 Associated factors of leprous peripheral neuropathy

Based on the current study, patients' age above 40 years appeared to be a predictor of leprous peripheral neuropathy. This is consistent with national and international studies. For instance, previous study conducted to measure the prevalence of disability and associated factors among

registered leprosy patients in all Africa TB and Leprosy Rehabilitation and Training centre (ALERT), Addis Ababa, Ethiopia supported this finding(46). A study from Colombia also revealed similar result(73).

Research conducted in Bangladesh and China found a link between leprosy peripheral neuropathy and advanced age (41, 44). Peripheral neuropathy is a chronic leprosy consequence that takes time to develop, which may account for this association's possible explanation in older leprosy patients.

According to the current study, there is a substantial correlation between skin lesions and leprosy peripheral neuropathy. Patients who have skin lesions have a threefold increased risk of developing leprosy peripheral neuropathy. This conclusion is corroborated by a study from northern India that found that the presence of skin lesions was the best predictor of future neuropathy, with skin lesions showing a 3–4 times increased risk for leprosy peripheral neuropathy(88).

Male sex is found to be more likely associated with leprosy peripheral neuropathy in the present study. Similarly, a study from Indonesia(89) also showed the same association. According to WHO, people with leprosy are predominantly male with which this study is consistent.

This could be because more male patients tend to be more mobile, offer more opportunities for interaction, and report to medical facilities more frequently in order to receive treatment. Furthermore, male patients may put off receiving care because of irregular income and extended work hours that make it difficult to schedule appointments. The overrepresentation of men in jobs requiring physical work may have two implications(73). On the other hand, females cover most of their body parts which could lead to decreased detection of symptoms. These could be the factors responsible for higher chance of male to be a victim of the disease.

MB leprosy was found to be associated with the development of leprosy peripheral neuropathy in this study. This is in line with the study done by Moschioni *et al.*(90), which has shown that multibacillary leprosy is more prone to neuropathic manifestations. The Bangladesh Acute Nerve Damage Study (BANDS) carried out at the Danish Bangladesh Leprosy Mission also support this finding, where patients with MB leprosy had a 65% risk of developing nerve damage(91).

In the current study, patients who had leprosy reaction had associated with leprosy peripheral neuropathy. This finding is also consistent with previous studies(92, 93). Similarly, the AMFES study(94) also reported that reversal and ENL reactions were risk factors for the development of leprosy neuropathy. This finding might be justified by the fact that *M. leprae* affect Schwann cells and the infected cells are susceptible to host immune response and are killed by activated T-cells(95, 96). In addition to this, loss of nerve function may also be triggered by the mechanical effect of increased pressure within the neural sheath caused by the inflammatory oedema(97).

In this study, it's confirmed that those patients who stayed with the disease for a longer duration of time are riskier to develop leprosy peripheral neuropathy than does their counterparts. This is consistent with the results of previous studies which showed a higher risk of disability in patients who had symptoms for prolonged duration(46). This could be described by the fact that most of the patients sought medical care very late after the diseases had progressed.

### 6.3 Limitations of the study

- The study is confined to a limited geographical setting and was conducted over a short period of time
- Errors can occur while estimating the prevalence, because it's based on the potential diagnosis, no other tools are used to detect the presence of peripheral neuropathy
- It is challenging to infer casual correlation because the study is cross-sectional

### 6.4 Conclusion

- In conclusion, this study showed high magnitude of leprosy peripheral neuropathy among registered leprosy patients. This implies how the study was significant. Male sex, advanced age, presence of leprosy reactions, presence of more than four skin lesions, longer duration of the disease, and MB leprosy were the risk factors associated with leprosy peripheral neuropathy. Prolonged sickness was one of the risk factors for nerve injury. As such, it is critical to evaluate the case detection process as soon as possible.

## 6.5 Recommendations

- This study shows evidence of high prevalence of leprosy related peripheral neuropathy in the study area. This calls for further attention for researches to determine the underlying reasons and provide ways forward for the national program through employing different study designs
- A larger sample size, with controls, would allow more momentous conclusions
- Larger studies complemented with community-based surveys should be conducted across the country
- Greater awareness on leprosy and its manifestations should be given to decrease delays in diagnosis and potentially reduce the morbidity caused by this disease
- Psychosocial support is recommended for patients lived with the disease for longer duration

## 7. References

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## Annex I

### Questionnaire

<b>Part I- sociodemographic factors</b>			
<b>S.no.</b>	<b>Questions</b>	<b>Response</b>	<b>Skip to</b>
101	What's your gender?	1. Male 2. Female	
102	How old are you?	----- years	
103	What's your job?	1. No job 2. Student 3. Farmer 4. Housewife 5. Self-employee 6. Government Employee	
104	Where do you live?	1. Urban 2. Rural	
105	How long have you attended school?	1. No formal education 2. Primary school 3. Secondary school 4. Certificated and diploma 5. Degree and above	
<b>Presence or absence of Peripheral Neuropathy</b>			
	<b>Question</b>	<b>Response</b>	
106	Is peripheral neuropathy present?	1. yes 2. No	
<b>Part II- Clinical factors</b>			
	<b>Questions</b>	<b>Response</b>	
107	How many nerves are involved?	-----	
108	What type of nerve impairment/s is/are encountered?	1. Sensory impairment 2. Motor impairment 3. Mixed (both sensory and motor) impairment 4. Neuropathic pain 5. Nerve thickening	
109	Which specific nerve/s is/are involved	-----	

110	How many skin lesions are present?	-----	
111	How long you have stayed with the disease?	-----	
112	WHO classification of leprosy	1. MB 2. PB	
113	Do you have previous history of contact with leprosy patient?	1. Yes 2. No	
<b>Part III- Genetic factor</b>			
	<b>Question</b>	<b>Response</b>	
114	Do you have family history of leprosy?	1. Yes 2. No	