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REVIEW

Leprosy in Pregnancy: A Systematic Review of Maternal Characteristics, Complications, and Neonatal Outcomes

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Background: Nearly 40% of leprosy cases occur in females. Although relatively uncommon in pregnancy, immunological changes during gestation and the puerperium may exacerbate leprosy and influence maternal and neonatal outcomes. To date, no systematic review has synthesized the evidence on leprosy in pregnancy.

Purpose: To describe maternal clinical and obstetric characteristics, pregnancy complications, neonatal outcomes, and laboratory findings in pregnant women with leprosy.

Methods: We systematically searched electronic databases from inception to August 2025 for studies reporting leprosy in pregnancy or puerperium using specific terms and inclusion/exclusion criteria. Data were extracted and presented descriptively.

Results: Eighteen case reports and case series described a total of 21 individual patients, while an additional eight original articles were included. Most patients were aged 20–39 years (85.7%), with multibacillary leprosy predominating (80.9%) of cases. Erythema nodosum leprosum was more frequent during pregnancy (14.3%), while reversal reactions predominated in the puerperium (9.5%). Leprosy in pregnancy was most frequently reported in the third trimester (52.4%). Cesarean section was slightly more frequent than vaginal delivery (33.3% vs 23.8%). Overall, maternal and neonatal outcomes were generally favorable, although complications included intrauterine growth restriction, hematologic abnormalities, preterm delivery, neonatal skin discoloration, and hyperbilirubinemia. One infant was diagnosed with leprosy at six months. Slit-skin smears were occasionally positive with a high bacteriological index, and acid-fast bacilli were detected in the placentas of mothers with leprosy.

Conclusion: Leprosy in pregnancy most often affects women of reproductive age with the multibacillary type, typically presenting in the third trimester. While maternal and neonatal outcomes are generally favorable, complications may occur, underscoring the importance of continuing multidrug therapy with close monitoring. Further multicenter studies are required to better define transmission risks and optimize management strategies.

Keywords: erythema nodosum leprosum, gestation, immunological changes, puerperium, reversal reaction

Introduction

Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae* (*M. leprae*) that primarily affects the skin and peripheral nerves.[1] The disease remains endemic in several countries, with Indonesia ranking third globally in incidence after India and Brazil.[2] According to the World Health Organization (WHO) in 2023, 182,815 new cases were reported across 118 countries, of which 39.8% occurred among females.[3] In India, the prevalence of leprosy during pregnancy has been estimated at two to three cases per 2,000 leprosy patients per year, most commonly during the third trimester.[4]

Pregnancy and the puerperium are associated with biphasic alterations in both humoral and cell-mediated immunity (CMI).[5] Several studies have reported that pregnancy may exacerbate leprosy, often leading to progression toward the lepromatous end of the spectrum, characterized by new skin lesions and progressive nerve impairment, likely due to suppression of maternal CMI.[5,6]

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During gestation, the increased mycobacterial antigen load and the shift from a T helper (Th)1 to Th2 response may predispose patients to erythema nodosum leprosum (ENL).[7] Conversely, in the puerperium, the recovery of CMI can trigger reversal reactions (RR), possibly through the release of sensitized lymphocytes sequestered during pregnancy.[5]

Duncan et al (1980) reported that placental weight and neonatal birth weight were lower in mothers with leprosy compared with healthy controls.[8] These neonates also demonstrated slower growth, particularly when mothers had lepromatous leprosy (LL). Furthermore, the prevalence of leprosy in children under two years of age born to mothers with active LL was 5%.[8]

Despite extensive literature on the effects of leprosy on male gonadal function, there is a notable paucity of data on its effect on the female reproductive system.[5] Leprosy in pregnancy is considered uncommon, likely due to underreporting and the absence of large-scale studies. Consequently, leprosy in pregnancy remains poorly documented, with evidence largely limited to isolated case reports and small-scale studies. To date, no systematic review has synthesized the evidence on leprosy in pregnancy. The objective of this systematic review is to describe maternal clinical characteristics, obstetric characteristics, pregnancy complications, neonatal outcomes, and relevant laboratory findings in pregnant women with leprosy. By integrating scattered and fragmented reports, this systematic review provides an overview that may inform clinical practice for antenatal management and neonatal monitoring, thereby guiding future research.

Materials and Methods

A systematic review of the literature was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines in order to study leprosy in pregnancy (Figure 1).

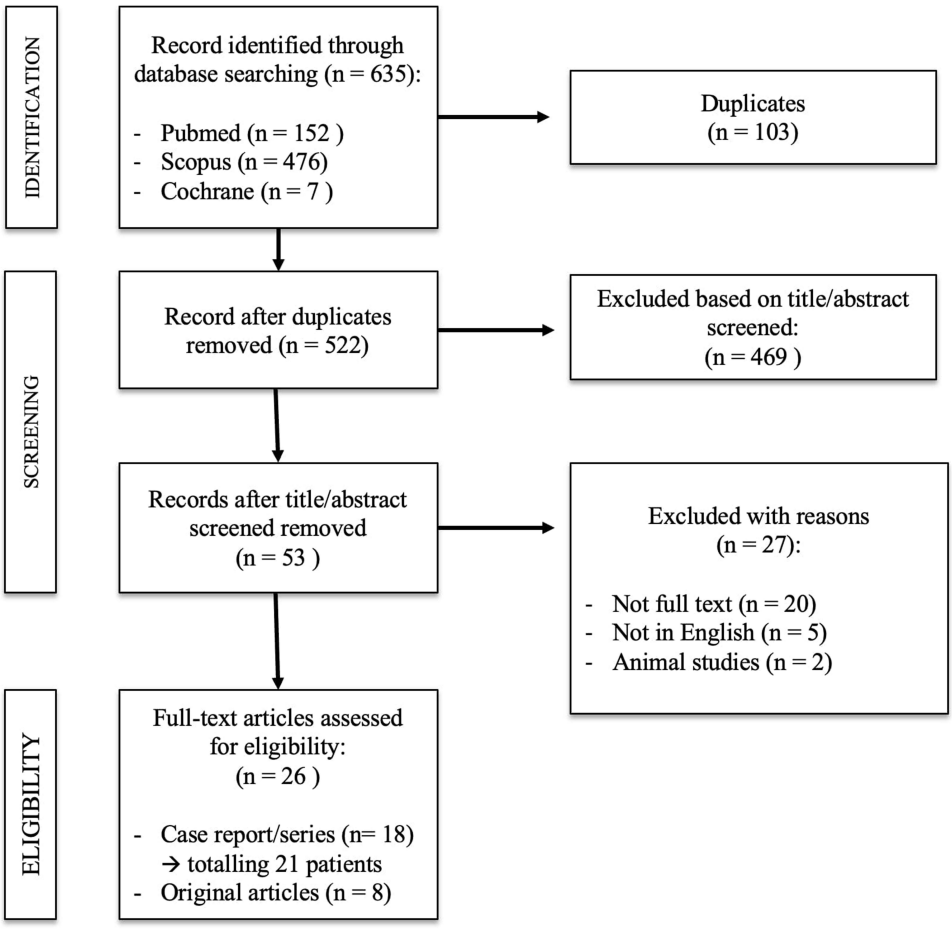

Figure 1 Flow diagram of study selection process.

Literature Search Strategies

Electronic Database

All relevant studies were searched from inception to August 2025 in the following electronic databases: Pubmed, Scopus, and Cochrane databases. No restrictions were applied regarding language or publication type. The following search criteria were used: ("Pregnancy"[MeSH] OR pregnancy OR pregnant OR gestation) AND ("Leprosy"[MeSH] OR leprosy OR Hansen's disease).

Inclusion Criteria

Studies were included if they met the following criteria: (1) pregnant or puerperal women diagnosed with leprosy, (2) diagnosis established by clinical evaluation or slit-skin smear (SSS), and (3) studies reporting maternal, obstetric, neonatal, or laboratory outcomes.

Exclusion Criteria

Studies were excluded if the title or abstract did not align with the study's objective, full text was unavailable, review articles, duplicated, animal studies, or published not in English.

Outcome Measure

The outcome of interest was the identification of maternal clinical characteristics of leprosy, as well as maternal obstetric characteristics and pregnancy complications, neonatal outcomes, and laboratory findings. The following data were extracted from the studies and collected in an electronic database: age, leprosy type, leprosy reaction, gestational age (GA), method of childbirth, pregnancy complications, neonatal outcomes, acid-fast bacilli (AFB) from SSS mothers and AFB from placenta, serology in mother and child, polymerase chain reaction (PCR) from placenta, umbilical cord, amniotic fluid, and breast milk. The search results were analysed using Microsoft Excel for Office Home and Students, 2021.

Results

Study Selection

The study selection process is illustrated in the PRISMA 2020 flow diagram (Figure 1). A total of 635 records were identified through database searches and reference list screening. A total of 53 articles were retrieved for full-text review following the elimination of duplicates and the screening of titles and abstracts. Of these, 27 were excluded for the following reasons: animal studies (n = 2), publication in languages other than English (n = 5), and full text unavailable despite retrieval attempts (n = 20). Finally, 26 studies met the eligibility criteria and were included in this review. These comprised eight original research articles, sixteen case reports, and two case series (reporting two and three patients, respectively).

General Findings

Eighteen case reports and case series described a total of 21 individual patients (Table 1). Most patients (85.7%) were between 20 and 39 years of age, with a mean age of 25.9 years (median 26 years; range 16–36 years). Multibacillary (MB) leprosy was more frequent (80.9%) than paucibacillary (PB) type. Across all cases, three patients developed ENL and three experienced Lucio phenomenon (LP) during pregnancy, whereas in the puerperium, RR and LP were observed in two patients each (Table 2).

Among the 21 patients, 17 described leprosy during pregnancy, most frequently in the third trimester (52.4%). Four patients reported leprosy during puerperium. Method of childbirth was reported in 12 patients: cesarean section (CS) (33.3%) was slightly more frequent than vaginal delivery (23.8%). The most commonly reported pregnancy complications were intrauterine growth restriction (IUGR) and hematologic abnormalities (Table 3). Reported neonatal outcomes included preterm delivery, skin discoloration, and hyperbilirubinemia (Table 4). Laboratory findings such as direct microscopy, serology, and PCR were rarely reported (Table 5).

Eight original articles of leprosy in pregnancy were included. Information regarding the type of leprosy, leprosy reaction, maternal and neonatal outcomes, and laboratory findings were extracted from each article (Table 6). Borderline

Table 1 Reported Cases of Leprosy in Pregnancy

No.	Author, Year	Age	GA	Leprosy Type	Reaction	Method of Childbirth	Pregnancy Complication	Neonatal Condition
1.	Jafari, 1975[27]	16 y.o	3rd	MB	NR	Vaginal Delivery	No Complication	No perinatal problems
2.	Lyde, 1977[28]	29 y.o 27 y.o 25 y.o	N/A N/A N/A	N/A N/A MB	NR 2 NR	N/A N/A N/A	N/A N/A N/A	N/A No perinatal problems No perinatal problems
3.	Hempenstall, 1997[29]	18 y.o	3rd	MB	1	CS	Respiratory distress, anemia, neutropenia, fetal distress	NICU admission
4.	Benard, 2009[30]	28 y.o	Post Partum	MB	LP	N/A	No Complication	N/A
5.	Gutiérrez-de la Peña, 2010[31]	34 y.o	Post Partum	PB	1	N/A	N/A	N/A
6.	Aranegui, 2014[32]	24 y.o	Post Partum	MB	LP	Vaginal Delivery	No Complication	No perinatal problems
7.	Prakoeswa, 2016[26]	29 y.o	3rd	MB	LP	CS	N/A	No perinatal problems
8	Sánchez-Orta, 2016[33]	36 y.o	3rd	MB	NR	N/A	N/A	N/A
9.	Ozturk, 2017[24]	26 y.o	1st	MB	NR	Vaginal Delivery	Gestational diabetes	Skin discoloration due to clofazimine during breastfeeding
10.	Bhatia, 2017[4]	26 y.o	3rd	MB	1	N/A	No Complication	N/A
11.	Chen, 2019[16]	N/A	N/A	MB	NR	CS	No Complication	No perinatal problems
12.	Smoot, 2019[34]	29 y.o	3rd	N/A	NR	Vaginal Delivery	No Complication	Total bilirubin of 10.8 mg/dL
13.	Gimovsk, 2019[15]	27 y.o	2nd	MB	2	CS	IUGR, PROM	No perinatal problems
14.	Nareswari, 2019[22]	25 y.o 21 y.o	3rd 3rd	MB MB	LP LP	CS N/A	IUGR, low amniotic fluid, pansytopenia Anemia, hypoalbuminemia	Preterm delivery, severe asphyxia No perinatal problems
15.	Vijay, 2019[35]	29 y.o	3rd	MB	2	CS	IUGR, fetal distress	No perinatal problems
16.	Noormohammadpour, 2020[23]	27 y.o	3rd	MB	1	Vaginal Delivery	No Complication	Single hypopigmented patch on the back, AFB positive at 6 month old
17.	Schaffenburg, 2023[36]	24 y.o	Post Partum	MB	2	N/A	N/A	N/A
18.	Argentina, 2025[37]	19 y.o	3rd	MB	NR	CS	No Complication	No perinatal problems

Abbreviations: y.o, years old; PB, paucibacillary; MB, multibacillary; LP, lucio phenomenon; NR, no reaction; AFB, acid-fast bacilli; CS, cesarean section; IUGR, intrauterine growth restriction; PROM, premature rupture of membrane; N/A, not available.

lepromatous (BL) and LL types were most frequently associated with adverse outcomes, including increased occurrence of leprosy reactions, ENL during pregnancy and RR during the puerperium, lower birth and placental weights, and fetal distress. Silent neuritis was commonly observed, even in treated patients. Laboratory findings showed negative AFB results from breast milk samples. Consequently, current evidence provides minimal support for vertical or breast milk transmission of *M. leprae*.

The limitation of this review lies in the reliance on isolated case reports and small-sample studies, accompanied by considerable heterogeneity. Important research gaps remain, underscoring the need for future multicenter investigations to strengthen the evidence base and guide clinical management.

Table 2 Maternal Clinical Characteristics of Leprosy

Variables	n = 21 (%)
Age (year)	
<20	3 (14,3%)
20–39	18 (85,7%)
Leprosy Type	
PB	1 (4,8%)
MB	17 (80,9%)
N/A	3 (14,3%)
Leprosy Reaction during Pregnancy	
RR	2 (9,5%)
ENL	3 (14,3%)
LP	3 (14,3%)
No reaction	13 (61,9%)
Leprosy Reaction during Puerperium	
RR	2 (9,5%)
ENL	1 (4,8%)
LP	2 (9,5%)
No reaction	16 (76,1%)

Abbreviations: PB, paucibacillary; MB, multibacillary; RR, reversal reaction; ENL, erythema nodosum leprosum; LP, lucio phenomenon; N/A, not available.

Table 3 Maternal Obstetric Characteristics of Leprosy and Pregnancy Complications

Variables	n = 21 (%)
Gestational Age	
1st trimester	1 (4,8%)
2nd trimester	1 (4,8%)
3rd trimester	11 (52,4%)
N/A	4 (19%)
Puerperium	4 (19%)
Method of Childbirth	
Vaginal delivery	5 (23,8%)
Cesarean section	7 (33,3%)
N/A	9 (42,8%)

(*Continued*)

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Table 3 (Continued).

Variables	n = 21 (%)
Pregnancy Complications	
IUGR, PROM	1 (4,8%)
IUGR, fetal distress	1 (4,8%)
IUGR, oligohydramnios, hematology abnormality	1 (4,8%)
Gestational diabetes	1 (4,8%)
Fetal distress, hematology abnormality	1 (4,8%)
Hematology abnormality, hypoalbuminemia	1 (4,8%)
No complication	8 (38,1%)
N/A	7 (33,3%)

Abbreviations: IUGR, intrauterine growth restriction; PROM, premature rupture of membrane; N/A, not available.

Table 4 Neonatal Outcomes

Variables	n = 21 (%)
Preterm delivery, severe asphyxia	1 (4,8%)
Skin discoloration	1 (4,8%)
Hyperbilirubinemia	1 (4,8%)
Leprosy was diagnosed at 6 months old	1 (4,8%)
No perinatal problem	10 (47,6%)
N/A	7 (33,3%)

Abbreviation:N/A, not available.

Table 5 Laboratory Findings

Variables	n = 21 (%)
SSS	
- Positive AFB	4 (19%)
- Negative AFB	0
- N/A	17 (80,9%)
Placenta	
- Positive AFB	1 (4,8%)
- Negative AFB	2 (9,5%)
- N/A	18 (85,7%)

(*Continued*)

Table 5 (Continued).

Variables	n = 21 (%)
Maternal serology	
- Positive serology	2 (9,5%)
- Negative serology	0
- N/A	19 (90,5%)
Neonatal serology	
- Positive serology	1 (4,8%)
- Negative serology	0
- N/A	20 (95,2%)
PCR	
Placenta	
- Positive	1 (4,8%)
- Negative	2 (9,5%)
- N/A	18 (85,7%)
Umbilical cord	
- Positive	0
- Negative	1 (4,8%)
- N/A	20 (95,2%)
Amniotic fluid	
- Positive	2 (9,5%)
- Negative	0
- N/A	19 (90,5%)
Breast milk	
- Positive	0
- Negative	1 (4,8%)
- N/A	20 (95,2%)

Abbreviations: AFB: acid-fast bacilli; N/A, not available.

Discussion

Maternal Age

The most commonly reported age group among pregnant leprosy patients was 20–39 years (85.7%). The patients ranged in age from 16 to 36 years, with a mean age of 25.9 years. Noguera et al[9] reported that most cases of leprosy in pregnant and lactating women were aged 21–34 years, accounting for 22–44.9% of cases, with a mean age of 32.1 years. In addition, Palacio et al[10] observed that 69% of pregnant women with leprosy were aged 20–39 years. Leprosy can occur at any age. Among adults, the peak incidence is in the 20–40 year range.[11] Leprosy is notable for its long incubation period, typically ranging from two to five years but occasionally extending beyond a decade. Although infection is most often

Table 6 Summary of Eight Original Articles of Leprosy in Pregnancy

No	Author, Year	Type of Leprosy	Leprosy Reaction	Maternal and Neonatal Outcomes	Laboratory Findings
1.	Sharma, 1979[14]	35 subjects with leprosy: • TT: 7 subjects • BT: 2 subjects • BB: 2 subjects • BL: 9 subjects • LL: 15 subjects	N/A	• In 60% of patients, there was no correlation onset or exacerbation of the leprosy between the first appearance of symptoms and any gynaecological or obstetrical event. • 5.8% of patients had a definite relationship with puerperium. • 8.6% of patients had a relationship with pregnancy. • Six weeks incubation of the endometrial tissue on Lowenstein-Jensen medium did not grow tubercle bacilli.	N/A
2.	Duncan, 1980[19]	116 subject with leprosy: • TT/BT: 42 subject • BL: 44 subjects • LL: 35 subjects 31 healthy subjects	N/A	• Babies of healthy mothers and those with TT/BT leprosy were heavier than the babies of mothers with BL/LL ($p<0.001$). • The placental weight in mothers with LL was significantly lower ($p<0.001$). • Fetal distress was observed in 20% of the babies born to mothers with BL/LL. • Perinatal and infant mortality were higher in babies of mothers with leprosy compared with healthy controls, although the difference did not reach statistical significance.	N/A
3.	Duncan, 1982[13]	115 subjects with leprosy: • TT/BT: 39 • BL: 44 • LL: 32 31 healthy subjects	N/A	• **Fifty-one** women with leprosy developed neuritis during the study, for a total of **85 episodes** occurring across pregnancy and lactation. • **All patients**, including those considered cured and off treatment, remained at risk. **Silent neuritis**, inflammation without nerve pain or tenderness, often preceded by complaints of "rheumatism" and accompanied by enlarged peripheral nerves on examination, occurred **more often than overt neuritis.**	N/A
4.	Duncan, 1982[7]	114 subjects with leprosy: • TT/BT: 32 subjects • BL: 40 subjects • LL: 32 subjects 33 healthy subjects	• There was an increase in the incidence of ENL throughout pregnancy and during the first six months of lactation. • RR cases decreased during the second and third trimesters, increased sharply after delivery, and gradually decreased within the first year of lactation.	N/A	N/A
5.	Duncan, 1982[8]	113 subjects with leprosy: • TT/BT: 36 subjects • BL: 42 subjects • LL: 35 subjects 27 healthy subjects	N/A	N/A	• All breast milk specimens were negative for *Mycobacterium (M.) leprae.* • The placenta examination results showed scant AFB debris in half of the cases. • Two (5%) babies under 2 years of age were proven to have leprosy, which showed a marked increase in IgM anti-*M. leprae* antibody.

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6.	Duncan, 1984[38]	80 subjects with leprosy: • TT/BT: 26 subjects • BL: 34 subjects • LL: 20 subjects 16 healthy subjects	N/A	N/A	• No granulomas or any evidence of a villitis. • No AFB or AFB granules were seen on the placentae from lepromatous women.
7.	Da Cunha Menezes Palácios, 2013[10]	149 subjects with leprosy: • PB: 84 subjects • MB: 65 subjects	• Seven cases had RR. • Two cases experienced ENL. • 103 cases were reported as having no reaction. • In 37 cases, the reaction status was not reported.	N/A	N/A
8.	Nogueira, 2015[12]	49 subjects with leprosy: • MB: 23–57.5%	N/A	• Mothers and babies were most commonly without complications (33.4%). • The most frequently reported complication was low birth weight in neonates (22.3%).	N/A

Abbreviations: TT, tuberculoid leprosy; BT, borderline tuberculoid; BL, borderline lepromatous; LL, lepromatous leprosy; PB, paucibacillary; MB, multibacillary; AFB, acid-fast bacilli; RR, reversal reaction; ENL, erythema nodosum leprosum; N/A, not available.

acquired during childhood or adolescence, clinical manifestations frequently remain silent until adulthood. This latency explains why the majority of diagnosed cases are concentrated in the productive age group.[3,12]

Leprosy Type

MB leprosy predominated (80.9%), whereas PB type was reported in only one patient (4.8%). In a study of leprosy during pregnancy, Noguera et al[9] reported that 23–57.5% of pregnant women had MB leprosy. Duncan et al[7,8,13] and Sharma et al[14] also reported that most pregnant patients had MB leprosy. There was a significant association between MB classification and female sex, with women being four times more likely than men to be diagnosed with the MB type.[12] Traditional beliefs and barriers, such as the need for permission, an escort, and financial resources, often delay diagnosis and treatment in women. The predominance of MB leprosy likely reflects delayed diagnosis driven by sociocultural barriers in many low- and middle-income countries. Restricted autonomy, financial dependence, stigma, and limited access to healthcare among women contribute to delayed presentation, allowing disease progression toward the lepromatous pole.[14] In contrast, Palacio et al[10] observed a higher proportion of PB type (56%) than MB (44%) among pregnant women. This discrepancy was thought to be influenced by differences in research methods, population heterogeneity, and variation in measurement tools and quality.[10]

Leprosy Reaction During Pregnancy and Puerperium

Regarding leprosy reactions during pregnancy, ENL and LP were each reported in 14.3% of patients, while RR was observed in 9.5%. In the puerperium, RR and LP were reported in 9.5% of patients, while ENL occurred in 4.8%. Palacios et al[10] reported that seven patients experienced RR and two patients developed ENL during pregnancy. In a prospective study of 76 Ethiopian women with MB leprosy followed through pregnancy and lactation, Duncan et al[8] (1982) reported that 40% of RRs occurred during pregnancy and 60% during lactation; however, the study could not determine whether RR truly decreases during pregnancy and increased during lactation. Pregnancy is associated with a shift from a Th1 to Th2 response, likely driven by elevated estrogen and progesterone levels, with a reversal in the puerperium.[5] Suppression of CMI during pregnancy leads to increased bacillary multiplication and antigen load, thereby raising the risk of ENL and disease progression, which peaks in the third trimester and remains elevated in early lactation.[8] RR are typically seen in patients within the borderline spectrum and tend to occur during the puerperium.[12] This is attributed to the restoration of CMI, possibly triggered by the release of sensitized lymphocytes sequestered during pregnancy. Management of leprosy reactions during pregnancy generally follows standard protocols with continuation of multidrug therapy. Corticosteroids and NSAIDs are commonly used for the treatment of RR and ENL and are generally considered safe during pregnancy when used judiciously and under appropriate clinical monitoring.[5]

Gestational Age

Gestational age at the time of reporting was available for 13 patients, while data were not available for four others (19%). Overall, most cases occurred in the third trimester (≥28 weeks, 52.4%), with one case (4.8%) each in the first (<14 weeks) and second (14–27 weeks) trimesters. Four cases (19%) described leprosy in puerperium. Duncan et al[7] (1982) reported that among 114 pregnant women with leprosy, 7.63% were in the first trimester, 43.22% in the second, and 49.15% in the third. Noguera et al[9] found the highest proportion during the last trimester of pregnancy and the first three months of puerperium. Downgrading was observed particularly during pregnancy, as expected in conditions where CMI is suppressed.[7] The surge in estrogen and progesterone during late pregnancy promotes fetal tolerance by suppressing Th1 cytokine responses and increasing anti-inflammatory cytokines. Consequently, reduced Th1-mediated cellular immunity may increase susceptibility to infections and disease reactivation.[15] The predominance of diagnoses in the third trimester underscores the potential value of targeted prenatal screening and heightened clinical vigilance, particularly in late pregnancy, to facilitate earlier detection, timely intervention, and improved maternal and neonatal outcomes.[5]

Method of Childbirth

The method of childbirth was reported in 12 patients, while data were not available for nine patients (42.8%). Among those with available data, CS was performed in seven patients (33.3%) and vaginal delivery in five patients (23.8%). In most cases, the indication for CS was not specified. Although CS have occasionally been performed in mothers with leprosy,[16,17] obstetricians do not regard the infection itself as an indication for cesarean delivery. CS to prevent vertical transmission is reserved for a limited set of infections, primarily herpes simplex virus and human immunodeficiency virus.[18] Given the negligible risk of intrapartum transmission of leprosy, CS for this reason is not justified.[5]

Pregnancy Complication

Of the total 18 case reports/series, pregnancy complications were documented in six patients (28.6%). IUGR, with or without additional conditions, and hematologic abnormalities were the most frequently reported. Less common complications included fetal distress (4.8%), premature rupture of membrane (PROM) (4.8%), oligohydramnios (4.8%), and other findings such as gestational diabetes (4.8%) and hypoalbuminemia (4.8%). All pregnant women with leprosy, including those considered cured and no longer undergoing treatment, remain at risk of neuritis. Silent neuritis (neuritis without nerve pain or tenderness), often accompanied by enlarged peripheral nerves on examination, being more common than overt neuritis.[13] Duncan et al[19] (1980) also reported fetal distress in 20% of neonates born to mothers with BL/LL.

Placental insufficiency arising from an inadequate CMI response during pregnancy, particularly in mothers with LL,[7] may underlie complications such as IUGR, oligohydramnios, and fetal distress. Intrauterine infection and inflammation are recognized risk factors for PROM.[20] In active leprosy, especially when systemic inflammatory reactions occur, this condition could plausibly contribute to membrane rupture.

Hematologic complications in pregnancy associated with leprosy likely reflect multiple mechanisms, including leprosy-related inflammation, medication effects, and pregnancy-specific factors such as nutritional deficiencies and physiologic hemodilution.[21] Dapsone is well known to cause serious hematologic adverse drug reactions, such as agranulocytosis and hemolytic anemia.[22] Anemia of chronic disease, particularly in MB leprosy and during ENL, can further lower hemoglobin levels. In addition, iron, folate, and vitamin B12 deficiencies, common in pregnancy, together with second and third trimester hemodilution may exacerbate drug or disease related cytopenias.[23] To date, gestational diabetes and hypoalbuminemia have not been associated with leprosy and most likely represent coincidental comorbidities. Preventive measures and early interventions for pregnancy-related complications in women with leprosy include regular antenatal monitoring, serial fetal growth assessments, optimization of maternal nutrition, and routine screening for anemia and hematologic abnormalities, which may help mitigate adverse maternal and neonatal outcomes.[5]

Neonatal Outcomes

Among all reported cases, complications were described in five infants, including preterm delivery, severe asphyxia, skin discoloration, hyperbilirubinemia, and leprosy diagnosed at six months of age. Nareswari et al[23] reported severe asphyxia in neonatal born to lepromatous mother with pregnancy complications. Noormohammadpour et al[24] and Duncan et al[8] (1983) highlighted leprosy in three infants born to mothers with MB leprosy, presenting with hypopigmented macules at the age of 6–17 months. Both studies emphasized the possibility of exposure to an unusually heavy *M. leprae* inoculum. Ozturk et al[25] reported skin discoloration in newborn due to clofazimine exposure during breastfeeding, which resolved after breastfeeding was discontinued for three months. Meanwhile, Duncan et al[19] (1980) reported that perinatal and infant mortality was increased in babies of mothers with leprosy when compared with healthy controls.

Existing evidence indicates that neonates born to mothers with leprosy have lower birth weight, smaller placentae, slower growth, more frequent infections, and higher infant mortality than those of non-leprous mothers.[12] It has been suggested that mothers with LL have an inadequate CMI response to pregnancy, and this state of tolerance or partial tolerance might be associated with reduced placental weight and lower birth weight.[19] Reports have also noted that clofazimine use during breastfeeding may cause skin discoloration in breastfed infants. The transfer of clofazimine to

breast milk has been estimated at 22% of the maternal dose. A small proportion of rifampicin, about 5% of the therapeutic dose, also passes into breast milk, while dapsone can enter breast milk at approximately 15% of the maternal dose.[25]

Laboratory Findings

Laboratory data were very limited (Table 7). AFB testing from SSS was documented in four cases, all positive, with bacteriological index (BI) values ranging from 4.7+ to 6+. Direct microscopic examination of placental tissue was positive in one case, while two others had negative AFB findings. Maternal serology, immunoglobulin (Ig) G and IgM against *M. leprae* was reported in two patients, both positive. Infant serology was reported in one case, showing IgG

Table 7 Reported Laboratorium Findings of Leprosy in Pregnancy

No	Author, Year	Direct Microscopy		Serology		PCR			
		SSS	Placenta	Mother	Child	Placenta	Umbilical Cord	Amniotic Fluid	Breast Milk
1.	Jafari, 1975[27]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2.	Lyde, 1977[28]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
3.	Hempenstall, 1997[29]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
4.	Benard, 2009[30]	6+	N/A	N/A	N/A	N/A	N/A	N/A	N/A
5.	Gutiérrez-de la Peña, 2010[31]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
6.	Aranegui, 2014[32]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
7.	Prakoeswa, 2016[26]	6+	N/A	IgG, IgM anti-PGL I positive	N/A	N/A	N/A	Positive	N/A
8	Sánchez-Orta, 2016[33]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
9.	Ozturk, 2017[24]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
10.	Bhatia, 2017[4]	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A	N/A N/A N/A
11.	Chen, 2019[16]	N/A	Positive	IgM NDO-BSA: positif, IgG MMP-II: positive, IgG LID-1: positive	IgM NDO-BSA: negative, IgG MMP-II: positive, IgG LID-1: positive	Positive	N/A	N/A	Negative
12.	Smoot, 2019[34]	N/A	Negative	N/A	N/A	N/A	N/A	N/A	N/A
13.	Gimovsk, 2019[15]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
14.	Nareswari, 2019[22]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		6+	N/A	N/A	N/A	Negative	Negative	Positive	N/A
15.	Vijay, 2019[35]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
16.	Noormohammadpour, 2020[23]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
17.	Schaffenburg, 2023[36]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
18.	Argentina, 2025[37]	4.67+	Negative	N/A	N/A	Negative	N/A	N/A	N/A

Abbreviations: SSS, slit skin smear; Ig, immunoglobulin; PGL-1, phenolic glycolipid-1, NDO: Natural disaccharide–octyl; MMP-1, matrix metalloproteinase-1; LID, leprosy IDRI diagnostic antigen; BSA, bovine serum albumin; CS, cesarean section; IUGR, intrauterine growth restriction; PROM, premature rupture of membrane; N/A, not available.

positivity. Duncan et al (1982)[8] reported marked increases in *anti-M. leprae* IgA and IgM in two infants, both diagnosed with leprosy before age two and born to mothers with MB leprosy. PCR testing of the placenta, umbilical cord, amniotic fluid, and breast milk identified *M. leprae* DNA in one placenta and in two amniotic fluid samples; however, no study reported positive PCR results from the umbilical cord or breast milk.

Downgrading was observed most frequently during pregnancy, consistent with the suppression of CMI during this period.[7] This immunosuppression likely contributed to increased bacillary proliferation. Increased leprosy activity, characterized by new skin lesions and a rise in the BI during the third trimester, puerperium, or lactation, was observed in 44% of women with tuberculoid leprosy (TT) or borderline tuberculoid (BT) leprosy and in 54% of those with BL and LL.[19] The placenta is considered a protective barrier against feto-maternal transmission of *M. leprae* and can effectively prevent vertical transmission.[16,26] The presence of *anti-M. leprae* IgA and IgM in the cord blood of infants born to mothers with leprosy likely reflects intrauterine immune stimulation, possibly triggered by transplacental exposure to *M. leprae* antigens.[8] Although no morphologic evidence of placental infection has usually been observed, a few AFB have been demonstrated in rare cases. *M. leprae* has also been occasionally detected in the breast milk of mothers with MB leprosy, although the viability of these bacilli remains questionable. Furthermore, there is no evidence that oral ingestion of *M. leprae* results in clinical disease.[5]

Conclusion

Leprosy in pregnancy is uncommon but most reported cases involved women of reproductive age with MB leprosy, clustering in the third trimester. ENL was more frequent in pregnancy, while RR predominated in puerperium. Maternal and neonatal outcomes were generally favorable, yet complications, such as IUGR and hematologic abnormalities were observed. In addition, AFB can be detected in the placenta of mother with leprosy. Continuing multidrug therapy with careful maternal and neonatal monitoring is essential in leprosy during pregnancy to early identify and manage potential complications. Further multicenter studies are needed to clarify transmission risks and improve care strategies.

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Disclosure

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