SURABAYA MEDICAL JOURNAL
(SMJ IDI SURABAYA)

Volume 3, Issue 1, May 2025, p.41-48

e-ISSN: 2986-7584
p-ISSN: 2986-2469

CASE REPORT

An infant with biliary atresia: a case report

Syania Mega Octariyandra[1*], Anggraini Dian Prameswari[1], Bagus Setyoboedi[2,3], Sjamsul Arief[2,3]

[1]Perak Timur Community Health Center, Surabaya, Indonesia
[2]Department of Child Health, Dr. Soetomo General Academic Hospital, Surabaya
[3]Department of Child Health, Faculty of Medicine - UNIVERSITAS AIRLANGGA, Surabaya

Article Info

Article history:
Received : 22-10-2024
Revised: 01-02-2025
Accepted: 23-03-2025
Published : 30-05-2025

Keywords:
biliary atresia;
cholestasis;
infant;
jaundice

ORCID ID:
Syania Mega Octariyandra
https://orcid.org/0000-0002-0802-7537

Bagus Setyoboedi
https://orcid.org/0000-0002-3923-6913

Sjamsul Arief
https://orcid.org/0000-0002-6372-2460

ABSTRACT

Background: In children, biliary atresia (BA) is the most frequent cause of liver transplantation. Cholestasis, an early sign of biliary atresia, is rarely identified by **primary** care physicians as it is often mistaken for physiological jaundice. Early detection of biliary atresia is necessary to optimize outcomes and prevent end-stage liver disease. **Case Presentation**: A 2-month-old baby girl presented to the hospital with complaints of persistent jaundice since birth, accompanied by pale-colored stools. Physical examination revealed sclera icteric, hepatomegaly, splenomegaly, and umbilical hernia were found. Laboratory results showed a direct bilirubin level of 5.98 mg/dL, total bilirubin 7.22 mg/dL, albumin 3.31 g/dL, ALP 223 U/L, GGT 360.4 U/L, AST 307 U/L, ALT 313 U/L, APTT 34.2 seconds, and PT 12.4 seconds, Toxoplasma IgG 3.3 (reactive), CMV IgG 90.4 (reactive), and CMV IgM 3.09 (reactive). Thyroid function tests showed FT4 level of 360.4 ng/dL and TSH 307 µIU/mL suggesting congenital hypothyroidism. A 2-phase abdominal ultrasound examination showed impaired gallbladder contractility. Liver biopsy showed extrahepatic cholestasis and fibrosis of the portal tract (F1 stage). Contrast-enhanced MRCP was performed and showed biliary atresia with hepatosplenomegaly. The patient was diagnosed with biliary atresia, CMV infection, and congenital hypothyroidism. The patient was referred to pediatric surgery for Kasai portoenterostomy (KPE) surgery. **Conclusion**: The diagnosis of biliary atresia requires a combination of several laboratory modalities as well as radiologic and histopathologic studies. Early detection of cholestasis is necessary; if the infant is found to be jaundice at 2 weeks of age, serum bilirubin levels must be evaluated to avoid delays in BA management.

Citation:
Octariyandra, S.M., Prameswari, A.D., Setyoboedi, B., Arief, S. (2025). An infant with biliary atresia: a case report, Surabaya. Surabaya Medical Journal, 3(1):41-48. doi: 10.59747/smjidisurabaya.v3i1.99

Corresponding Author:
Bagus Setyoboedi, Department of Child Health, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
Email: bagus.setyoboedi@fk.unair.ac.id

Highlights

1. Cholestatic jaundice should be identify as a physiologic jaundice as hepatobiliary dysfunction.
2. Biliary atresia (BA) is a pathologic condition with devastating consequences and needs Kasai portoentherostomy.

BACKGROUND

About 1 out of every 2500 term infants are affected by cholestatic jaundice, which primary care physicians infrequently identify in the context of physiologic jaundice. Hepatobiliary dysfunction is always indicated by cholestatic jaundice, which is pathologic. Evaluation of serum studies typically shows cholestasis, with elevated serum conjugated (or direct) bilirubin providing as important markers of easily recognized hepatobiliary dysfunction (Fawaz et al., 2017).

The most commonly known cause of neonatal cholestasis is biliary atresia (BA), that affects roughly 1 in 12,000 live births and, with approximately 350 new cases per year (Mack, 2015). BA causes (obstructive jaundice during the first three months of life is BA. Around the world, the prevalence of BA varies by geography Fawaz et al., 2017). It is more common in Asians, African Americans, and women (Mack, 2015).

There are several contributing factors to the aetiology of BA. If left untreated, BA may rapidly lead to portal hypertension and end-stage liver disease, which can have devastating consequences on the infant's health. Most children will develop biliary cirrhosis as a result of severe bile duct injury that persists even after a Kasai portoenterostomy (Mack, 2015). Prompt referrals to the pediatric hepatologist and early detection by the primary care physician are crucial for the best outcome and successful treatment (Fawaz et al., 2017).

Case Report

A 2-months old female infant, presented to tertiary hospital with a referral from primary health care facility, the primary complaint of persistent jaundice since birth. This complaint accompanied by pale-colored stools, which began when the infant was one week old (Figure 1). There were no associated symptoms such as fever, nausea, or vomiting. Based on previous medical history, the patient was admitted to hospital for three days after birth for jaundice, but no special treatment was given at that time. The patient was subsequently readmitted to the previous hospital due to persistent jaundice, diaper rash, oral patches, and abdominal distension. The patient was treated for four days during that hospital stay. From the family history revealed no other members with similar symptoms.

Figure 1. Pale stool of the patient

The patient was born full-term via normal vaginal delivery, with a birth weight of 2600 grams and a length of 47 cm. The patient cried immediately after birth, had no cyanosis, but was jaundiced. Her immunization history includes Hepatitis B and Polio vaccines. Nutritionally, the infant was breastfed

Surabaya Medical Journal (SMJ IDI Surabaya)
e-ISSN: 2986-7584; p-ISSN: 2986-2469

for the first month of life, then transitioned to formula feeding. In terms of developmental milestones, the patient is appropriately lifting her head, indicating normal gross motor development for her age.

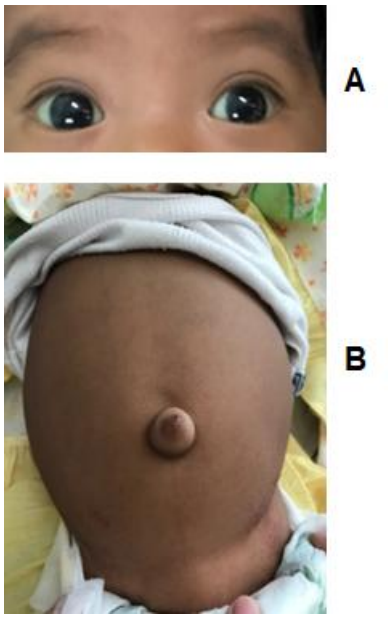

Figure 2. The patient's clinical features were icteric sclera (A) and abdominal distension with umbilical hernia (B).

The patient has a weight of 3.5 kg, a length of 56.5 cm, a head circumference of 37 cm, and an abdominal circumference of 39 cm. Based on abdominal examination, hepatomegaly was found with size of 3.2 x 2 x 1 cm. The liver edge is indistinct, with a smooth surface and soft consistency. Abdominal examination revealed an enlargement of the spleen and umbilical hernia (Figure 2).

The patient's laboratory results show a hemoglobin level of 10.3 g/dL, hematocrit of 31.2%, white blood cell count of 12.81 x 10^3/µl, platelet count of 363 x 10^3/µl, direct bilirubin is 5.98 mg/dL, total bilirubin is 7.22 mg/dL, albumin level of 3.31 g/dL, alkaline phosphatase (ALP) 223 U/L, gamma glutamyl transpeptidase (GGT) 360.4 U/L, aspartate aminotransferase (AST) 307 U/L, and alanine aminotransferase (ALT) 313 U/L. Coagulation measurement showed an Activated Partial Thromboplastin Time (APTT) of 34.2 seconds and prothrombin time (PT) of 12.4 seconds. TORCH serology examination showed the Toxoplasma IgG was reactive (3.3), Toxoplasma IgM was non-reactive (0.11), Cytomegalovirus (CMV) IgG was reactive (90.4), CMV IgM was reactive (3.09), Rubella IgG was non-reactive (1.70), and Rubella IgM was non-reactive (0.07). The thyroid function tests reveal an (Free Thyroxine) FT4 level of 360.4 ng/dL and a Thyroid Stimulating Hormone (TSH) of 307 µIU/mL. HBsAg was non-reactive at <0.10, and C-Reactive Protein (CRP) was 0.85 mg/dL. The extremely elevated TSH with high FT4 points to a congenital hypothyroidism.

The patient underwent a 2-phase ultrasound examination, which showed impaired gallbladder contractility; however, the triangular cord sign, often indicative of BA, was not present (Figure 3). Following this, a liver biopsy was performed, revealing extrahepatic cholestasis and fibrosis in the portal tract (F1 stage), findings that align with a diagnosis of BA. Subsequently, a Magnetic resonance cholangiopancreatography (MRCP) with contrast was conducted, confirming BA, along with hepatosplenomegaly, and no biliary cysts were identified (Figure 4). The patient was subsequently started on treatment, which included ursodeoxycholic acid, vitamin supplementation, nutrition support, and thyroid hormone replacement therapy. The patient was referred to pediatric surgery for Kasai portoenterostomy (KPE) surgery.

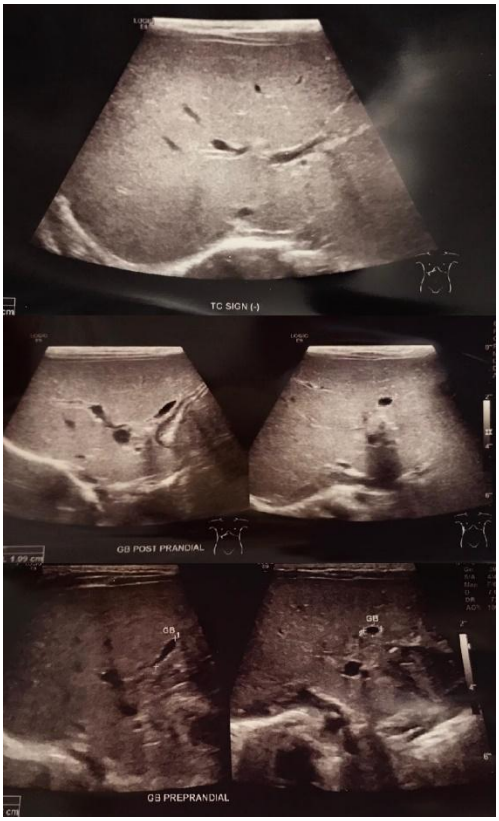

Figure 3. Abdominal ultrasound of the patient

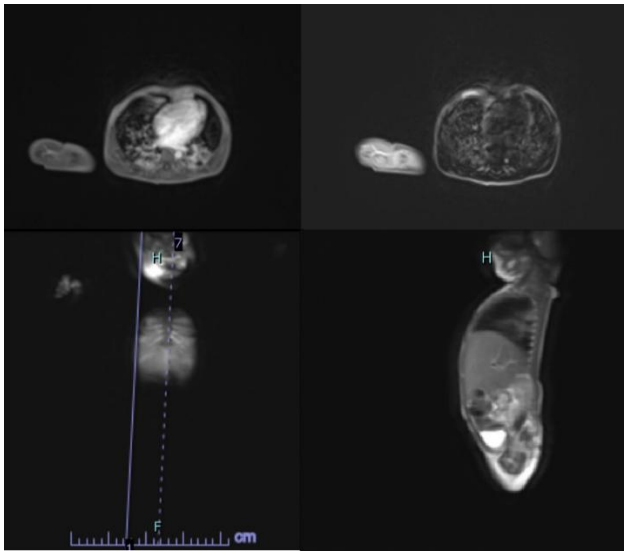

Figure 4. MRCP of the patient

DISCUSSION

Reduced bile production or flow that causes the liver to accumulate biliary is known as cholestasis (Fawaz et al., 2017). Instead of being physiological, neonatal cholestasis is a hallmark of hepatobiliary and/or metabolic disorders, some of which could be fatal if not identified and treated promptly. To promptly identify the causes that can be treated and to provide an accurate prognosis, an systematic and timely evaluation is necessary (Feldman and Sokol, 2019). The most frequent cause of neonatal cholestasis, biliary atresia (BA), should be taken into consideration when the neonate's jaundice continues after two weeks of age (Brahee and Lampl, 2022). Compared to preterm neonates (1:50), term neonates (1:6) had a higher incidence of BA (Ling et al., 2021).

Early diagnosis is crucial for BA because, if left untreated, it can cause liver fibrosis and death within a year (Brahee and Lampl, 2022). Meconium and the first stools are both normal in color in isolated

Surabaya Medical Journal (SMJ IDI Surabaya)
e-ISSN: 2986-7584; p-ISSN: 2986-2469

BA, indicating early duct patency. In this case, the patient had jaundice since birth with normal colored stool, but pale colored stool appeared at 1 week of age. The extrahepatic biliary tree becomes obstructed within the first three months of life, though, and the pathology is consistent with an inflammatory fibrosing cholangiopathy (Mack, 2015). To properly assess the infant who has jaundice, a comprehensive physical examination is essential. Special consideration should be given to hepatomegaly, splenomegaly, and poor appearance. A crucial component of a thorough assessment of the jaundiced infant is the direct visualization of stool pigment (Fawaz et al., 2017). In this case, the jaundice persisted and was followed by abdominal distension. Patient was referred for further examinations.

Biliary injury and obstruction have been caused by several of insults, including toxins, viral infections, genetic variants, immune dysfunction, maternal microchimersim, vascular disturbances, and abnormal morphogenesis (Vij and Rela, 2020). Biliary Atresia: pathology, Etiology and Pathogenesis (Mack, 2015). The pathophysiology of the disease can be inferred from the neonatal presentation of BA. Perinatal viral infections, such as Cytomegalovirus (CMV) infection and other early events that affect the neonatal immune system can change the immune response and encourage the development of a progressive inflammatory or biliary autoimmune disease (Vij and Rela, 2020). According to a study, liver damage can be restored by reducing B cells or preventing antigen presentation in a rhesus-rotavirus-induced BA model (Wang et al., 2020). The etiology of non-BA cholestasis was linked to gestational age ≥ 32 weeks (Ling et al., 2021). This is consistent with the case that the patient had reactive serology test for CMV and was born full term.

It is challenging to diagnose BA because many of its clinical and imaging characteristics are similar to those of other newborn cholestasis causes (Brahee and Lampl, 2022). Numerous biomarkers linked to BA have been observed in recent years, and they have demonstrated significant potential in the three areas of clinical practice of diagnosis, liver fibrosis stage assessment, and native liver survival prediction (Kong et al., 2024). The primary laboratory test carried out is the measurement of serum conjugated (direct) bilirubin (DB). Serum bilirubin levels, both total and conjugated (direct), should be measured in formula-fed infants who exhibit jaundice after two weeks of age in order to assess for cholestasis. If a breastfed baby seems healthy, they should be monitored clinically for three weeks. If they seem icteric, they should then have their total and conjugated (direct) serum bilirubin levels were evaluated (Fawaz et al., 2017). However, the patient was referred to the referral hospital at 2 months of age and a bilirubin level measurement was performed with an elevated direct bilirubin > 1 mg/dL. The patient was found to have a direct bilirubin level of 5.98 mg/dL and a total bilirubin level of 7.22 mg/dL, indicating cholestasis.

Additionally, patients with cholestasis should undergo standard biochemical examinations and synthetic liver tests, such as measurement of total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma glutamyl transpeptidase (GGT), prothrombin time (PT) with international normalized ratio (INR), glucose, and albumin, in order to assess the severity of liver disease (Fawaz et al., 2017). In this case, there was an increase in ALP 223 U/L, GGT 360.4 U/L, AST 307 U/L, and ALT 313 U/L. Coagulation and albumin measurements showed normal results.

As newborn screening tools, laboratory tests for cholestasis diagnosis, such as total bilirubin and conjugated bilirubin, have demonstrated enhanced efficacy and earlier diagnosis in BA. Additionally, measures for matrix metalloproteinase-7 (MMP-7) and gamma-glutamyl transpeptidase (GGT) showed potential as useful indicators of disease in BA, particularly when combined with several predictive factors in diagnostic models (Sun et al., 2021). MMP-7 is impractical for general diagnostic use until more evidence is available. Thus, liver function tests combined with ultrasound remain to be the most clinically useful non-invasive diagnostic techniques for BA when cost and operational efficiency are taken into consideration.

Limitations

Due to BA clinically similar with cholestasis, it needs other laboratory investigation such as MMP-7 and GGT that need more cost in limiting sources country.

CONCLUSION

The diagnosis of biliary atresia requires a combination of several laboratory modalities as well as radiologic and histopathologic studies. Early detection of cholestasis is necessary; if the infant is found to be jaundice at 2 weeks of age, serum bilirubin levels must be evaluated to avoid delays in BA management.

Acknowledgment

None.

Conflict of Interest

The author declares that there is no conflict of interest in working on this paper.

Funding

None.

Patient concern for Publication

Informed consent was voluntarily obtained from the patient regarding the dissemination of their case information, upholding their autonomy and privacy rights.

Author Contribution

BS: supervising, conceptual, funding; SMO: data curation, drafting, translating; ADP: data curation, drafting, translating; SA: supervising, editing.

REFERENCES

Alrehaili, J., 2023. Leprosy Classification, Clinical Features, Epidemiology, and Host Immunological Responses: Failure of Eradication in 2023. Cureus 15, 1–19.

Bakker, M.I., Hatta, M., Kwenang, A., Van Mosseveld, P., Faber, W.R., Klatser, P.R., Oskam, L., 2006. Risk factors for developing leprosy - A population-based cohort study in Indonesia. Lepr. Rev. 77, 48–61.

Bhat, R.M., Prakash, C., 2012. Leprosy: An overview of pathophysiology. Interdiscip. Perspect. Infect. Dis. 2012.

Celestino, I.C., Antunes, D.E., Santos, D.F., Gimenes, V.L., de Souza, F.M., Goulart, I.M.B., 2024. Adverse reactions induced by MDT/WHO (Rifampicin+Clofazimine+Dapsone) and ROM (Rifampicin+Ofloxacin+Minocycline) regimens used in the treatment of leprosy: a cohort study in a National Reference Center in Brazil. Front. Pharmacol. 15, 1–9.

Cellona, R. V., Balagon, M.V.F., Dela Cruz, E.C., Burgos, J.A., Abalos, R.M., Walsh, G.P., Topolski, R., Gelber, R.H., Walsh, D.S., 2003. Long-term Efficacy of 2 Year WHO Multiple Drug Therapy (MDT) in Multibacillary (MB) Leprosy Patients. Int. J. Lepr. Other Mycobact. Dis. 71, 308–319.

Chen, K.H., Lin, C.Y., Su, S. Bin, Chen, K.T., 2022. Leprosy: A Review of Epidemiology, Clinical Diagnosis, and Management. J. Trop. Med. 2022.

Chen, X., Liu, H.B., Shui, T.J., Zha, S., 2021. Risk factors for physical disability in patients with leprosy disease in yunnan, china: Evidence from a retrospective observational study. PLoS Negl. Trop. Dis. 15, 1–14.

Deps, P.D., Nasser, S., Guerra, P., Simon, M., Birshner, R.D.C., Rodrigues, L.C., 2007. Adverse effects from Multi-drug therapy in leprosy: A Brazilian study. Lepr. Rev. 78, 216–222.

Ebenezer, G.J., Scollard, D.M., 2021. Treatment and Evaluation Advances in Leprosy Neuropathy. Neurotherapeutics 18, 2337–2350.

Edi, G.F.A., Azizah, R., 2023. Analysis of Environmental Risk Factors for Leprosy in Indonesian Society: Meta-Analysis. Poltekita J. Ilmu Kesehat. 17, 105–113.

Fonseca, A.B. de L., Simon, M. do V., Cazzaniga, R.A., de Moura, T.R., de Almeida, R.P., Duthie, M.S., Reed, S.G., de Jesus, A.R., 2017. The influence of innate and adaptative immune responses on the differential clinical outcomes of leprosy. Infect. Dis. Poverty 6, 1–8.

Franco-Paredes, C., Garcia-Creighton, E., Henao-Martínez, A., Kallgren, D.L., Banjade, R., Dyer, J.A., Nelson, T., Zaesim, A., Peluso, M.J., Jain, V., Lee, D.H., Minces, L.R., Wirshup, M., Hoffman, M.S., Katsolis, J., Brust, K., Giron, J., Smiarowski, L., Hoosepian-Mer, P.A., Stryjewska, B., Abstract:, 2022. Novel approaches in the treatment of Hansen's disease (Leprosy): a case series of multidrug therapy of monthly rifampin, moxifloxacin, and minocycline (RMM) in the United States. Ther. Adv. Vaccines 9, 1–10.

Froes, L.A.R., Sotto, M.N., Trindade, M.A.B., 2022. Leprosy: clinical and immunopathological characteristics. An. Bras. Dermatol. 97, 338–347.

Gofur, N.R.P., Gofur, A.R.P., Putri, H.M., Soesilaningtyas, Halimah, A.N., Feriana, D., Rusdiana, E., Wahjuni, S., Rosa,

Surabaya Medical Journal (SMJ IDI Surabaya)
e-ISSN: 2986-7584; p-ISSN: 2986-2469

M.L.L., Yusof, Z.Y.B.M., 2023. Drug choice to lowering risk contiguity with Morbus Hansen disease: A review article. J. Adv. Pharm. Educ. Res. 13, 73–79.

Goncalves, H. de S., Pontes, M.A. de A., Bührer-Sékula, S., Cruz, R., Almeida, P.C., de Moraes, M.E.A., Penna, G.O., 2012. Brazilian clinical trial of uniform multidrug therapy for leprosy patients - The correlation between clinical disease types and adverse effects. Mem. Inst. Oswaldo Cruz 107, 74–78.

Guragain, S., Upadhayay, N., Bhattarai, B.M., 2017. Adverse reactions in leprosy patients who underwent dapsone multidrug therapy: A retrospective study. Clin. Pharmacol. Adv. Appl. 9, 73–78.

Hambridge, T., Chandran, S.L.N., Geluk, A., Saunderson, P., Richardus, J.H., 2021. Mycobacterium leprae transmission characteristics during the declining stages of leprosy incidence: A systematic review. PLoS Negl. Trop. Dis. 15, 1–32.

Hansen, G.A., 1875. The etiology of leprosy. Br Foreign Med Chir Rev 55, 459–489.

Kerr-Pontes, L.R.S., Barreto, M.L., Evangelista, C.M.N., Rodrigues, L.C., Heukelbach, J., Feldmeier, H., 2006. Socioeconomic, environmental, and behavioural risk factors for leprosy in North-east Brazil: Results of a case-control study. Int. J. Epidemiol. 35, 994–1000.

Khadilkar, S. V., Patil, S.B., Shetty, V.P., 2021. Neuropathies of leprosy. J. Neurol. Sci. 420, 117288.

Kinanti, H., Kurniati, Faidati, W., 2024. The Burden of Leprosy Reaction in the Post-Elimination Era: A Study from Gresik City, Indonesia. Berk. Ilmu Kesehat. Kulit dan Kelamin 36, 41–46.

Lazo-Porras, M., Prutsky, G.J., Barrionuevo, P., Tapia, J.C., Ugarte-Gil, C., Ponce, O.J., Acuña-Villaorduña, A., Domecq, J.P., De La Cruz-Luque, C., Prokop, L.J., Málaga, G., 2020. World Health Organization (WHO) antibiotic regimen against other regimens for the treatment of leprosy: A systematic review and meta-analysis. BMC Infect. Dis. 20, 1–14.

Li, Y.Y., Shakya, S., Long, H., Shen, L.F., Kuang, Y.Q., 2021. Factors Influencing Leprosy Incidence: A Comprehensive Analysis of Observations in Wenshan of China, Nepal, and Other Global Epidemic Areas. Front. Public Heal. 9, 1–11.

Lockwood, D.N.J., Penna, G.O., Lambert, S., Pai, V.V., Walker, S.L., 2022. Safer and newer antimicrobial drugs for leprosy – time to test monthly ROM in an adequately powered randomised trial? Lepr. Rev. 93, 96–101.

Lubis, R.D., Darmi, M., Prakoeswa, C.R.S., Agusni, R.I., Kusumaputra, B.H., Alinda, M.D., Listiawan, M.Y., Anum, Q., Argentina, F., Menaldi, S.L., Gunawan, H., Yuniati, R., Mulianto, N.R., Siswati, A.S., Widasmara, D., Rusyati, L.M.M., Mamuaja, E.H., Muchtar, V., 2022. Leprosy Epidemiology According to Leprosy Type in 13 Teaching Hospitals in Indonesia between 2018 and 2020. Open Access Maced. J. Med. Sci. 10, 1812–1817.

Martahi, R.L., Tasman, H., 2021. Model of transmission of three types Leprosy Disease with treatment. J. Phys. Conf. Ser. 1725.

Martoreli Júnior, J.F., Ramos, A.C.V., Alves, J.D., Crispim, J. de A., Alves, L.S., Berra, T.Z., Barbosa, T.P., da Costa, F.B.P., Alves, Y.M., Dos Santos, M.S., Gomes, D., Yamamura, M., Pinto, I.C., Fuentealba-Torres, M.A., Nunes, C., Pieri, F.M., Arcoverde, M.A.M., Dos Santos, F.L., Arcêncio, R.A., 2021. Inequality of gender, age and disabilities due to leprosy and trends in a hyperendemic metropolis: Evidence from an eleven-year time series study in central-west Brazil. PLoS Negl. Trop. Dis. 15, 1–16.

Menaldi, S.L., Harini, M., Nelfidayani, N., Irawati, Y., Setiono, S., Wahyuni, L.K., Rahayu, T., Andayani, G., Friska, D., Nugraha, B., 2022. Functional activity limitation of leprosy cases in an endemic area in Indonesia and recommendations for integrated participation program in society. PLoS Negl. Trop. Dis. 16, 1–13.

Menaldi, S.L.S.W., 2020. Pedoman Nasional Pelayanan Kedokteran Tatalaksana Kusta. Kementeri. Kesehat. RI 106.

Nascimento, O.J.M., 2013. Leprosy neuropathy: Clinical presentations. Arq. Neuropsiquiatr. 71, 661–666.

Noer, R.R., Kurniawan, S.N., 2023. Leprosy neuropathy: Clinical presentations. J. pain headache vertigo 4, 27–30.

Oliveira, I.V.P. de M., Deps, P.D., Antunes, J.M.A. de P., 2019. Armadillos and leprosy: From infection to biological model. Rev. Inst. Med. Trop. Sao Paulo 61, 1–7.

Ploemacher, T., Faber, W.R., Menke, H., Rutten, V., Pieters, T., 2020. Reservoirs and transmission routes of leprosy; A systematic review. PLoS Negl. Trop. Dis. 14, 1–27.

Prakoeswa, C.R.S., Lubis, R.S., Anum, Q., Argentina, F., Menaldi, S.L., Gunawan, H., Yuniati, R., Mulianto, N.R., Siswati, A.S., Widasmara, D., Rusyati, L.M.M., Mamuaja, E.H., Muchtar, V., Agusni, R.I., Kusumaputra, B.H., Alinda, M.D., Listiawan, M.Y., 2022. Epidemiology of Leprosy in Indonesia: a Retrospective Study. Berk. Ilmu Kesehat. Kulit dan Kelamin 34, 29–35.

Rahmawati, D., Bimanto, H., 2021. Analysis of Factors Affecting Leprosy Cases in East Java Province with Spatial Autoregressive Model (SAR). J. Berk. Kesehat. 7, 59.

Rahmi, A.M., Listiawan, M.Y., Murtiastutik, D., Nurul, A., Ervianti, E., Utomo, B., 2024. Unveiling the Past : Retrospective Exploration of Multidrug Therapy (MDT) Efficacy in Multibacillary Leprosy Patients. Mitig. Lett. 21, 51–58.

Rees, R.J.W., McDougall, A.C., 1977. Airborne infection with Mycobacterium leprae in mice. J. Med. Microbiol. 10, 63–68.

Rosdiana, B., Astari, L., Astindari, A., Prakoeswa, C.R.S., Zulkarnain, I., Damayanti, D., Utomo, B., Listiawan, M.Y., 2021.

Risk factors of type 1 leprosy reaction in leprosy patients attending leprosy division of dermatology and venereology outpatient clinic of dr soetomo general hospital in 2017–2019: A retrospective study. Open Access Maced. J. Med. Sci. 9, 1359–1363.

Sales, A.M., Ponce de Leon, A., Düppre, N.C., Hacker, M.A., Nery, J.A.C., Sarno, E.N., Penna, M.L.F., 2011. Leprosy among patient contacts: A multilevel study of risk factors. PLoS Negl. Trop. Dis. 5, 1–6.

Swift, T.R., Sabin, T.D., 1998. Neurological manifestations of leprosy. Neurologist 4, 21–30.

Urban, C., Blom, A.A., Pfrengle, S., Walker-Meikle, K., Stone, A.C., Inskip, S.A., Schuenemann, V.J., 2021. One Health Approaches to Trace Mycobacterium leprae's Zoonotic Potential Through Time. Front. Microbiol. 12, 1–7.

White, C., Franco-Paredes, C., 2015. Leprosy in the 21st century. Clin. Microbiol. Rev. 28, 80–94.

World Health Organization (WHO), 1969. Classification of disabilities resulting from leprosy, for use in control projects. Bull. World Health Organ. 40, 609–612.

Wulan Dewanti Martamevia, 2024. Epidemiological overview of new cases of leprosy in West Java In 2021-2023, Indonesia. World J. Adv. Res. Rev. 22, 1701–1707.