

Case Report

Medicine

A Neonatal Face Serving Diagnosis

Mouhamed AlMakhy Niang^{1*}, Mouhamed Dieng¹, Michel Assane Ndour¹, Matar Ndiaye¹, Oumar Boun Khatab Diouf¹, Boundia Djiba¹, Demba Diédhiou¹, Anna Sarr¹, Maïmouna Ndour Mbaye¹

¹Cheikh Anta Diop University / Abass Ndao Hospital Center / Internal Medicine Department

DOI: <https://doi.org/10.36348/sjmpps.2025.v11i09.001> | Received: 26.06.2025 | Accepted: 30.08.2025 | Published: 02.09.2025

*Corresponding author: Mouhamed AlMakhy Niang

Cheikh Anta Diop University / Abass Ndao Hospital Center / Internal Medicine Department

Abstract

Introduction: Neonatal lupus (NL) is a transient disease linked to the transplacental passage of maternal autoantibodies, mainly anti-SSA/Ro and sometimes anti-SSB/La. It manifests mainly by predominantly cutaneous forms and a prognosis dependent on cardiac involvement. **Observation:** We report the case of a four-week-old girl, born at term (37 weeks + 4 days, weight 2.9 kg), who presented since birth with hypopigmented periorbital macules in a vesperilio pattern. Her 36-year-old mother presented with bilateral inflammatory arthralgias with synovitis of the wrists that had been developing for six months. The infant's clinical examination was normal, the electrocardiogram and echocardiography showed no abnormalities, and neonatal laboratory tests were non-contributory. Maternal ANA was 1/800, anti-SSA/Ro were positive in both mother and child, while anti-SSB/La and anti-U1RNP were negative. The diagnosis was neonatal cutaneous lupus erythematosus in the child and systemic lupus with isolated articular expression in the mother. The treatment combined photoprotection and topical hydrocortisone for the infant, and low-dose prednisone and hydroxychloroquine for the mother. At 1 month, the infant's skin lesions completely regressed without sequelae, the ECG remained normal, and the maternal pain had disappeared. **Conclusion:** This case highlights the diagnostic interest of photosensitive facial eruptions and the importance of joint management of the mother and child, as well as systematic cardiovascular monitoring of newborns exposed to anti-SSA/Ro.

Keywords: neonatal lupus; anti-SSA/Ro; skin lesions; Africa.

Copyright © 2025 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

I. INTRODUCTION

Neonatal Lupus (NL) is a rare, acquired autoimmune disease resulting from the transplacental transmission of maternal antibodies to the fetus. These antibodies, mainly anti-SSA/Ro and anti-SSB/La, but also to a lesser extent anti-U1RNP, bind to fetal tissues, causing various clinical manifestations [1]. Although most symptoms are transient and reversible, cardiac complications, particularly congenital heart block (CHB), are permanent and potentially fatal [1].

In the majority of cases, the disease occurs in children born to asymptomatic or paucisymptomatic mothers, highlighting the importance of systematic screening in exposed infants [2]. Skin manifestations, often the first to appear, can be confused with other benign neonatal eruptions, thus delaying diagnosis [3]. In Africa and more particularly in Senegal, the literature on LN remains limited to isolated observations or small series, with little epidemiological data available, which

complicates the establishment of local recommendations for the management of at-risk infants.

Here we report the case of an infant with cutaneous neonatal lupus associated with paucisymptomatic maternal lupus, illustrating the typical clinical and immunological aspects of this pathology, as well as the importance of early and multidisciplinary follow-up to optimize the prognosis of the child and his mother.

II. CLINICAL OBSERVATION

A 4-week-old female infant, born at term (37 weeks + 4 days), birth weight 2.9 kg, delivered by spontaneous vaginal delivery. The mother, 36 years old, G4P4, all pregnancies carried to term, well-monitored, uneventful, with no known family or personal history of lupus or autoimmune disease. She consulted internal medicine for polyarthralgia evolving for approximately 6 months, inflammatory, bilateral and symmetrical, of

variable intensity, affecting the large joints (wrists, knees, ankles) and small joints (IPP, MCP, MTP), non-deforming, non-ankylosing. Physical examination revealed synovitis of both wrists without cutaneous-mucosal manifestations or other extra-articular signs.

The infant, carried on his mother's back at the time of the consultation, attracted attention due to the presence of periorbital hypochromic lesions. According to the mother, these lesions had been present since birth and were well tolerated; a diagnosis of pityriasis versicolor had been made previously, with treatment having remained ineffective.

Clinical examination of the infant:

The infant was in good general condition, responsive, with age-appropriate tone. Skin and mucosal coloration was normal, hydration was good, breathing was easy, and there was no sign of distress. His spontaneous motor function was harmonious, without asymmetry. There was spontaneous eye opening, pink conjunctivae, white sclera, and age-appropriate visual pursuit. The infant's skin was warm, without cyanosis or jaundice. The hypopigmented macular lesions were painless, arranged in a vespertilio pattern, limited to the face (Figure 1), and were not elsewhere located. The mucous membranes were pink, moist, and non-ulcerated. The B1 and B2 heart sounds were normal, with a regular rhythm and no pathological murmur. The femoral pulses were present and symmetrical, with satisfactory peripheral perfusion. The remainder of the clinical examination revealed no abnormalities. Vital signs were: weight 3.8 kg, temperature 37°C, HR 106/min, RR 31/min, BP 74/52 mmHg, SpO₂ 97% on room air.

Additional examinations:

Biological assessment of the infant:

Transaminases were normal (AST/TG 24 U/L, ALT/TGP 35 U/L), as were total and conjugated bilirubin. Blood ionogram showed: Na⁺ 135 mmol/L, K⁺ 4.3 mmol/L, Cl⁻ 98 mmol/L, with corrected serum

calcium at 2.7 mmol/L. Complete blood count was normal: Hb 14.1 g/dL, leukocytes $10.9 \times 10^9/L$, platelets $350 \times 10^9/L$.

Maternal biological assessment:

Complete blood count, CRP, creatinine, transaminases, and corrected calcium levels were normal. The erythrocyte sedimentation rate was 21 mm/h, and 24-hour proteinuria was normal.

Immunology:

The mother had antinuclear antibodies (ANA) at 1/800 with speckled fluorescence. Anti-SSA/Ro antibodies were positive in both mother and child, while anti-SSB/La and anti-U1RNP were negative.

Cardiology (infant):

The electrocardiogram and echocardiography were normal, with no detectable cardiac abnormalities.

Diagnosis and management:

Based on the maternal and neonatal immunological profile and the cutaneous clinical presentation, the diagnosis of neonatal cutaneous lupus erythematosus was retained for the girl, while the mother presented with systemic lupus with isolated articular manifestation.

The infant was treated with topical 0.5% hydrocortisone, along with photoprotection measures explained to the mother. The mother was given low-dose prednisone (5 mg/day) and hydroxychloroquine 400 mg/day, in addition to appropriate hygiene and dietary measures.

Evolution:

At the one-month follow-up, the mother no longer had joint pain. In the infant, we had regression of the skin lesions, without scars (Figure 2). The general, cardiological, and digestive examination remained normal. No new dermatological lesions or oral ulcerations were observed.

The follow-up electrocardiogram was normal.



Figure 1: Hypopigmentary macules in vespertilio at the time of diagnosis



Figure 2: Evolution of lesions after 1 month of treatment

III. DISCUSSION

Epidemiological data:

Neonatal lupus (NL) is a rare condition, with an estimated incidence of approximately 1/20,000 live births per year in the United States and 0.6/100,000 births worldwide [4,5]. Despite the relatively high frequency of maternal anti-SSA/Ro and/or anti-SSB/La antibodies, only 1 to 2% of exposed children develop the disease [4,5]. The mechanism is based on the transplacental passage of maternal immunoglobulin G, which begins around the 12th week of gestation [6]. The presence of these autoantibodies is essential, but insufficient on its own; fetal and environmental factors also modulate the risk [1].

A striking aspect of NL is that 25 to 60% of mothers whose children are affected are asymptomatic at the time of diagnosis and are unaware of their own autoimmune disease [1,2,6,7]. In fact, in 25 to 80% of cases, NL constitutes the first revealing manifestation of a maternal pathology that was previously silent [8]. Among these initially asymptomatic mothers, approximately half will develop systemic lupus erythematosus (SLE) or Sjögren's syndrome within 3 to 5 years of giving birth [6]. In China, one study even reports that 63.4% of mothers were asymptomatic before pregnancy [3], highlighting the importance of screening for autoantibodies in all women of childbearing age presenting even discreet clinical signs [1].

Regarding the distribution according to sex, if the first observations suggested a female predominance of the cardiac and cutaneous forms, more recent analyses show an equivalent involvement with a M/F ratio close to 1 [7].

Finally, phenotypic variations according to geographical origin have been described. Congenital atrioventricular block (CAVB) is more observed in patients of European (49.4%) and American (35%) ancestry, while Asian cohorts report a higher frequency of cutaneous involvement (45.2%) [1]. In China, skin involvement accounts for over 96% of cases, a rate

significantly higher than that described in the United States where skin involvement is less common [3].

In Africa, knowledge of neonatal lupus is based mainly on isolated clinical cases and small published series, with work by Abiodun *et al* describing a typical presentation in an infant [9]. These observations mainly report cutaneous, hematological and sometimes cardiac involvement, but above all highlight the diagnostic difficulties linked to the delay in clinical recognition and limited access to specialized explorations, such as fetal echocardiography or the dosage of maternal autoantibodies [9]. Despite a few scattered publications in pan-African journals, no large-scale multicenter cohort has yet been carried out, which prevents precise estimation of the prevalence of this condition on the continent.

In Senegal, while several studies have focused on systemic lupus, including its pediatric forms, no specific publication devoted to neonatal lupus has been identified. This absence could reflect underreporting of cases, diagnostic limitations, or even limited dissemination of data from university hospital structures, suggesting the existence of unpublished cases. In this context, the initial diagnostic confusion with pityriasis versicolor illustrates not only the phenotypic variability of neonatal lupus, but also the need to maintain a high level of clinical suspicion in the face of any photosensitive neonatal facial rash.

Clinical data:

The most frequent manifestations are cutaneous, hematological, hepatic and especially cardiac in the form of congenital atrioventricular block (CHB) [10]. Skin involvement is the most common: the lesions, most often erythematous, predominate on the face (as in our observation) and the scalp, but can also affect the trunk and extremities, or even become generalized [11]. The cephalofacial distribution is predominant (up to 95%); the lesions are present immediately in approximately 20% of cases, but most often appear during the first weeks of life (approximately 80%)

[1,3,12]. Exposure to UV rays promotes their occurrence and worsens their development [13–15]. Clinically, they are erythematous rings or maculopapules with raised edges, sometimes oval, with a central hypopigmented or slightly scaly area; the average diameter is about 1 cm, sometimes confluent into large erythematous plaques [16–18]. A small percentage has mucosal involvement, especially oral ulcers [18,19]. Histology reveals granular deposits of IgG at the dermo-epidermal junction, associated with vacuolar alterations of the interface and adnexal damage [20].

The progressive decline of maternal antibodies in the newborn explains the usual favorable evolution of skin lesions. Photoprotection constitutes the first-line treatment [21,22]. Topical corticosteroids are effective but not systematic [18,21,23]. Hydroxychloroquine (HCQ), given to some mothers, has been associated with a decrease in the incidence and severity of rashes in their infants [23]. Skin sequelae may persist: capillary dilation, hyperpigmentation, atrophic scars linked to photodamage, deposition of immune complexes or prolonged use of topical corticosteroids [23–25]. Persistent telangiectasias may benefit from vascular laser treatment.

Cardiac involvement is a common complication of LN, with a prevalence close to 70% in Europe and America, compared to <30% in Asia [26]. It mainly covers congenital heart block (CHB), arrhythmias and myocarditis. Data also suggest an over-incidence of structural heart malformations compared to healthy newborns [27–29], although the causal link and mechanisms remain uncertain due to a lack of high-level evidence studies. CHB, the most severe form, carries a risk of death from complete block of 15–20% [30,31]. In 60–90% of cases, it results from the transplacental passage of maternal autoantibodies, independently of structural abnormalities [32]. Onset is typical between 18 and 26 weeks. Severe forms are accompanied by fetal bradycardia and, sometimes, myocardial dysfunction related to endocardial fibrosis or hyperplasia [33–35]. Studies report complete recovery from biventricular dysfunction under corticosteroid therapy and immunoglobulins in patients without arrhythmia [36]. A longitudinal follow-up of 239 children with cardiac involvement confirmed progression to heart failure: 2.4% before 1 year, 14.8% between 1 and 17 years and 28.1% after 17 years, with persistence of the disease in 43.8% of cases diagnosed in the first year [37], highlighting the need for prolonged follow-up. The neonatal cardiological assessment of our patient (normal ECG/echocardiography) is therefore a reassuring point; in the absence of cardiac involvement, the prognosis is excellent, but clinical vigilance must be maintained, especially within systems where specialized follow-up can be heterogeneous.

Beyond the heart and skin, there is a spectrum of extra-cutaneous involvement including hepatobiliary

involvement, resulting in hepatomegaly, cholestasis or cytolysis (elevation of transaminases), usually reversible within 6 months; an inaugural food intolerance linked to vasculitis induced by maternal antibodies is possible [12,15,28,38,39]. The frequent hematological involvement combines anemia, then thrombocytopenia and neutropenia, sometimes in the form of transient pancytopenia simulating aplastic anemia; these abnormalities, often spontaneously resolved, may require transfusions and respond to steroids or immunoglobulins [26]. Neurological involvement, probably underestimated, includes convulsions, cerebral hemorrhages, benign macrocephaly or aseptic meningitis; they most often regress with the clearance of antibodies, but neuropsychiatric disorders or growth delays may persist and justify rehabilitation [26]. Endocrine involvement, which is rarer, can affect the pancreas or the thyroid (e.g. hypoglycemic hyperinsulinism, hypothyroidism, hypoadrenocorticism), generally transient but requiring metabolic monitoring [40,41]. In our observation, the initial biological evaluation and clinical examination did not show any significant systemic involvement, which confirms the good prognosis expected for pure cutaneous forms.

Immunological data:

Anti-Ro/SS-A, anti-La/SS-B and, more rarely, anti-U1RNP autoantibodies occupy a central place in the pathophysiology of LN, particularly for cardiac and cutaneous involvement [6,42,43]. Among them, anti-Ro52 is most strongly associated with CHB, detected in 85% of mothers of affected children [6–8]. While CHB is most often linked to anti-Ro/SS-A, some studies report higher titers of anti-La/SS-B in this setting [44], with high specificity but low sensitivity [45]. Anti-Ro/SS-A remains the most prevalent autoantibody (78.4%), often associated with anti-La/SS-B (35.5%); isolated positivity of anti-La/SS-B is exceptional (< 1% of CHB). Finally, skin lesions or even CHB can occur after exclusive exposure to maternal anti-U1RNP, although these situations remain rare [46]. Although our patient does not present with cardiac involvement, this autoantibody signature justifies the screening strategy used and reinforces the education of parents on the warning signs (rhythm disorders, exercise intolerance).

Therapeutic data:

The progressive clearance of maternal antibodies in the newborn explains the generally favorable evolution of skin lesions. Photoprotection remains the first-line treatment [21,22], while topical corticosteroids, although effective, are not systematically necessary [18,21,23].

Hydroxychloroquine (HCQ) given to some mothers was associated with a reduction in the incidence and severity of rashes in their infants [23]. Skin sequelae may persist, such as capillary dilations, hyperpigmentation or atrophic scars, linked to photo-

induced damage, immune complex deposits or prolonged use of topical corticosteroids [23,24]. Residual telangiectasias can be treated with vascular laser.

Prognostic data:

The prognosis of LN depends on the organ involved. Pure cutaneous forms have a favorable outcome, with regression of lesions around 6 months, in parallel with clearance of maternal antibodies. Conversely, cardiac involvement, particularly CHB, is associated with high morbidity due to its irreversible nature, with reported mortality rates of up to 20–30%, mainly related to congestive heart failure. Hematologic and hepatic involvement generally resolve spontaneously in < 6 months, but require close monitoring to detect hemorrhagic events or liver failure early [47,48]. This case allows us to address pre-conception counseling because the risk of recurrence during a subsequent pregnancy is estimated at between 17 and 25%. Information and the organization of specialized fetal monitoring during subsequent pregnancies, particularly during the 18–26 week window, are therefore essential.

Furthermore, although the exact incidence remains undetermined, these children appear to have an increased susceptibility to later autoimmune diseases.

IV. CONCLUSION

This case of neonatal cutaneous lupus, revealed by vesperilio periorbital macules in an infant exposed to maternal anti-SSA/Ro antibodies, illustrates the diagnostic importance of photosensitive facial eruptions and the frequency of the concomitant discovery of paucisymptomatic maternal lupus.

The favorable evolution of the child, under photoprotection and topical corticosteroids, with a normal neonatal cardiological assessment, confirms the excellent prognosis of isolated cutaneous forms. The maternal response to standard treatment (hydroxychloroquine and low dose of prednisone) underlines the interest of coordinated mother-child management. On a practical level, any newborn exposed to anti-SSA/Ro should benefit from a systematic cardiological evaluation (ECG and echocardiography), clinical and biological monitoring until clearance of maternal Ig (\approx 6 months) and targeted parental education (photoprotection, monitoring of warning signs). This observation also highlights the importance of pre-conception counseling, particularly in preventing the risk of recurrence (\approx 17–25%), with the discussion of hydroxychloroquine in mothers carrying anti-SSA/Ro. Finally, in the African context, it highlights the need to improve access to serology and feto-neonatal echocardiography, as well as the creation of multicenter registries to better characterize the epidemiology and standardize the management of neonatal lupus.

BIBLIOGRAPHICAL REFERENCES

1. Erden A, Fanouriakis A, Kiliç L, Sari A, Armağan B, Bilgin E, et al. Geoepidemiology and clinical characteristics of neonatal lupus erythematosus: a systematic literature review of individual patients' data. *Turk J Med Sci*. 2020;50(1):281-290.
2. Derdulska JM, Rudnicka L, Szykut-Badaczewska A, Mehrholz D, Nowicki RJ, Barańska-Rybak W, et al. Neonatal lupus erythematosus—practical guidelines. *J Perinat Med*. 2021;49(5):529-538.
3. Li YQ, Wang Q, Luo Y, Li XM, Li T, Zhang JM, et al. Neonatal lupus erythematosus: a review of 123 cases in China. *Int J Rheum Dis*. 2015;18(7):761-767. doi:10.1111/1756-185X.12652.
4. Ygberg S, Nilsson A. The developing immune system—from fetus to toddler. *Acta Paediatr*. 2012;101(2):120-127. doi:10.1111/j.1651-2227.2011.02494.x.
5. Holt PG, Jones CA. The development of the immune system during pregnancy and early life. *Allergy*. 2000;55(8):688-697. doi:10.1034/j.1398-9995.2000.00118.x.
6. Vanoni F, Lava SAG, Fossali EF, Cavalli R, Simonetti GD, Bianchetti MG, et al. Neonatal systemic lupus erythematosus syndrome: a comprehensive review. *Clin Rev Allergy Immunol*. 2017;53(3):469-476.
7. Liszewska A, Woźniacka A. Neonatal lupus erythematosus—prevention is better than cure. *Postepy Dermatol Alergol*. 2022;39(6):1021-1026.
8. Systemic Lupus in Children. The Practitioner's Review [Internet]. [cited 13 August 2025]. Available at: <https://www.larevuedupraticien.fr/article/lupus-systemique-de-lenfant>
9. Abiodun M, Adelowo O. Neonatal lupus syndrome in a Nigerian child. *BMJ Case Rep*. 2012;2012:bcr0120125710. doi:10.1136/bcr.01.2012.5710.
10. Gryka-Marton M, Szukiewicz D, Teliga-Czajkowska J, Olesinska M. An overview of neonatal lupus with anti-Ro characteristics. *Int J Mol Sci*. 2021;22(17):9281. doi:10.3390/ijms22179281.
11. Sun W, Fu C, Jin X, Lei C, Zhu X. Neonatal lupus erythematosus: an acquired autoimmune disease to be taken seriously. *Ann Med*. 2025;57(1):e2476049. doi:10.1080/07853890.2025.2476049.
12. Kobayashi R, Mii S, Nakano T, Harada H, Eto H. Neonatal lupus erythematosus in Japan: a review of the literature. *Autoimmun Rev*. 2009;8(6):462-466.
13. Diaz-Frias J, Badri T. Neonatal Lupus Erythematosus. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited August 13, 2025]. Available at: <http://www.ncbi.nlm.nih.gov/books/NBK526061/>
14. Zdraveska N, Kostovski A, Sofijanovska A, Jancevska S, Jovanovska J, Kacarska M, et al. Neonatal lupus erythematosus—a rare syndrome of transient autoimmunity. *Clin Case Rep*. 2022;10(6):e6004.

15. Ma J, Li Z, Song H, Zhang L. High-risk groups of neonatal lupus erythematosus in term infants: a birth cohort study. *Eur J Pediatr*. 2024;183(1):149-155.
16. Neiman AR, Lee LA, Weston WL, Buyon JP. Cutaneous manifestations of neonatal lupus without heart block: characteristics of mothers and children enrolled in a national registry. *J Pediatr*. 2000;137(5):674-680.
17. Fijałkowska A, Kądziela M, Żebrowska A. The spectrum of cutaneous manifestations in lupus erythematosus: a comprehensive review. *J Clin Med*. 2024;13(8):2419. doi:10.3390/jcm13082419.
18. Drohan A, Snyder A, Plante J, Karlin S, Wine Lee L, Cotton CH. Neonatal lupus erythematosus presenting as orolabial ulcerations: two cases and a review of the literature. *Pediatr Dermatol*. 2021;38(3):643-646.
19. Neel ML, Kern J, Ronis T. A 3-day-old girl referred from her pediatrician for oral ulcerations. *Pediatrics*. 2016;138(3):e20152043.
20. Baltaci M, Fritsch P. Histologic features of cutaneous lupus erythematosus. *Autoimmun Rev*. 2009;8(6):467-473. doi:10.1016/j.autrev.2008.12.014.
21. Okon LG, Werth VP. Cutaneous lupus erythematosus: diagnosis and treatment. *Best Pract Res Clin Rheumatol*. 2013;27(3):391-404.
22. Walling HW, Sontheimer RD. Cutaneous lupus erythematosus: issues in diagnosis and treatment. *Am J Clin Dermatol*. 2009;10(6):365-381. doi:10.2165/11310780-000000000-00000.
23. Barsalou J, Costedoat-Chalumeau N, Berhanu A, Fors-Nieves C, Shah U, Brown P, et al. Effect of in utero hydroxychloroquine exposure on development of cutaneous neonatal lupus erythematosus. *Ann Rheum Dis*. 2018;77(12):1742-1749.
24. Levy R, Briggs L, Silverman E, Pope E, Lara-Corrales I. Cutaneous sequelae in neonatal lupus: a retrospective cohort study. *J Am Acad Dermatol*. 2020;83(2):440-446.
25. Guinovart RM, Vicente A, Rovira C, Suñol M, González-Enseñat MA. Facial telangiectasia: an unusual manifestation of neonatal lupus erythematosus. *Lupus*. 2012;21(5):552-555.
26. Yang X. Clinical features, autoantibodies, and outcome of neonatal lupus erythematosus. *Fetal Pediatr Pathol*. 2022;41(3):436-442.
27. Fu C, Sun W, Peng H, Zhu X. Neonatal lupus erythematosus as a rare trigger of gastrointestinal involvement in neonates. *SciRep*. 2024;14(1):3791. doi:10.1038/s41598-024-54091-z.
28. Ho A, Gordon P, Rosenthal E, Simpson J, Miller O, Sharland G. Isolated complete heart block in the fetus. *Am J Cardiol*. 2015;116(1):142-147.
29. Sheng X, Song X, Xiong Y, Ren T, Chang X, Wu J, et al. Maternal and infant outcomes of pregnancy associated with anti-SSA/Ro antibodies: a systematic review and meta-analysis. *Pediatr Rheumatol*. 2023;21(1):22.
30. Friedman DM, Rupel A, Buyon JP. Epidemiology, etiology, detection, and treatment of autoantibody-associated congenital heart block in neonatal lupus. *Curr Rheumatol Rep*. 2007;9(2):101-108.
31. Brito-Zerón P, Izmirly PM, Ramos-Casals M, Buyon JP, Khamashta MA. The clinical spectrum of autoimmune congenital heart block. *Nat Rev Rheumatol*. 2015;11(5):301-312.
32. Gordon PA. Congenital heart block: clinical features and therapeutic approaches. *Lupus*. 2007;16(8):642-646.
33. Martin TA. Congenital heart block: current thoughts on management, morphologic spectrum, and role of intervention. *Cardiol Young*. 2014;24(Suppl 2):41-46. doi:10.1017/S1047951114001358.
34. Jain S, Spadafora R, Maxwell S, Botas C, Nawaytou H, von Scheven E, et al. A case of neonatal lupus presenting with myocardial dysfunction in the absence of congenital heart block. *Pediatr Cardiol*. 2023;44(3):736-739.
35. Saxena A, Izmirly PM, Bomar RP, Golpanian RS, Friedman DM, Eisenberg R, et al. Factors associated with long-term cardiac dysfunction in neonatal lupus. *Ann Rheum Dis*. 2020;79(2):217-224.
36. Zuppa AA, Riccardi R, Frezza S, Gallini F, Luciano RMP, Alighieri G, et al. Neonatal lupus: follow-up in infants with anti-SSA/Ro antibodies and review of the literature. *Autoimmun Rev*. 2017;16(4):427-432.
37. Wang YA, Sibbald C, Moon AT. Retrospective, single-center case series of neonatal lupus. *Pediatr Dermatol*. 2020;37(3):484-489. doi:10.1111/pde.14132.
38. Sun W, Ding L, Li M, Fu C, Yang Z, Zhu X. Neurological and endocrinological involvement in neonatal lupus erythematosus: a retrospective study at a tertiary hospital in Eastern China. *Clin Rheumatol*. 2023;42(9):2461-2468. doi:10.1007/s10067-023-06622-8.
39. Vinet É, Bernatsky S. Outcomes in children born to women with rheumatic diseases. *Rheum Dis Clin North Am*. 2017;43(2):263-273. doi:10.1016/j.rdc.2016.12.006.
40. Didier K, Bolko L, Giusti D, Toquet S, Robbins A, Antonicelli F, et al. Autoantibodies associated with connective tissue diseases: what meaning for clinicians? *Front Immunol*. 2018;9:541. doi:10.3389/fimmu.2018.00541.
41. Ihn H, Yamane K, Yazawa N, Kubo M, Fujimoto M, Sato S, et al. Distribution and antigen specificity of anti-U1RNP antibodies in patients with systemic sclerosis. *Clin Exp Immunol*. 1999;117(2):383-387. doi:10.1046/j.1365-2249.1999.00961.x.
42. Tunks RD, Clowse MEB, Miller SG, Brancazio LR, Barker PCA. Maternal autoantibody levels in congenital heart block and potential prophylaxis with anti-inflammatory agents. *Am J Obstet Gynecol*. 2013;208(1):64.e1-64.e7. doi:10.1016/j.ajog.2012.09.020.

43. Assari R, Ziaee V, Moradinejad MH, Mirmohammadsadeghi A. Neonatal lupus erythematosus following rheumatoid arthritis: case report and literature review. *Iran J Pediatr*. 2014;24(4):445-448.
44. Izmirly PM, Halushka MK, Rosenberg AZ, Whelton S, Rais-Bahrami K, Nath DS, et al. Extending the spectrum of maternal autoantibodies from SSA/Ro-SSB/La to U1RNP in cutaneous and cardiac neonatal lupus: clinical and pathologic implications. *Autoimmun Rev*. 2017;16(9):980-983.
45. Lee LA, Sokol RJ, Buyon JP. Hepatobiliary disease in neonatal lupus: prevalence and clinical characteristics in cases enrolled in a national registry. *Pediatrics*. 2002;109(1):e11.
46. Wisuthsarewong W, Soongswang J, Chantorn R. Neonatal lupus erythematosus: clinical character, investigation, and outcome. *Pediatr Dermatol*. 2011;28(2):115-121. doi:10.1111/j.1525-1470.2011.01300.x.