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Review Article

Challenges in Management of Neonatal Lupus Erythematosus: A Comprehensive Review

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ABSTRACT

Neonatal lupus erythematosus (NLE) is a rare autoimmune condition which is caused by the passage of maternal autoantibodies through the placenta to the fetus. Manifestations of NLE in different organs are reversible and some are irreversible. Given the diverse and potentially life-threatening nature of NLE, the management of this condition requires a multidisciplinary approach. Research and clinical strategies in the treatment of neonatal lupus focus on managing symptoms and improving outcomes, especially in severe cases such as congenital heart block. Prenatal Therapies including fluorinated corticosteroid, intravenous immunoglobulin, terbutaline, plasmapheresis and hydroxychloroquine were suggested. Moreover, potential postnatal Therapies may include topical corticosteroids, blood or platelets transfusion, systemic corticosteroids, pacemaker insertion and anti-TNF drugs. Research should continue to explore novel therapies and improve our understanding of the disease mechanisms, aiming to further enhance treatment strategies in the future. The aim of this review is to provide an overview of the challenges in the diagnosis and management of NLE, typically including pathophysiology, clinical presentation, and therapeutic strategies.

Keywords: Clinical presentation, Intravenous immunoglobulin (IVIG), Neonatal lupus erythematosus, Pathophysiology

Introduction

Neonatal lupus erythematosus (NLE) is unequivocally caused by the transplacental passage of anti-Ro/SS-A and anti-La/SS-B. This is less commonly associated with U1-ribonucleoprotein (U1-RNP). NLE incidence in mothers with this serological profile is about 2% (95% CI: 1.5–2.5%) and with a probability of 20% (95% CI: 15–25%), it may be repeated in subsequent pregnancies (1, 2).

One of the primary mechanisms by which these maternal autoantibodies contribute to the pathogenesis of NLE is through their ability to induce apoptosis, or programmed cell death, in cells expressing the target antigens. The binding of the autoantibodies to the Ro/SSA and La/SSB antigens on the surface of cells can trigger a cascade of intracellular signaling events, ultimately leading to the activation of apoptotic pathways. This increased apoptosis rate in targeted tissues, such as the skin and heart, can result in the development of the characteristic skin lesions and cardiac abnormalities seen in NLE (3).

Furthermore, the presence of these maternal autoantibodies can also elicit an inflammatory response within the affected tissues. The binding of the autoantibodies to their target antigens can activate the complement system, a complex cascade of proteins involved in immune defense. This activation of the complement system can lead to the recruitment of inflammatory cells, such as neutrophils and macrophages, which can

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further contribute to the tissue damage observed in NLE (4).

In addition to the direct effects of the maternal autoantibodies, the timing and duration of their exposure to the fetus or neonate can also play a crucial role in the pathogenesis of NLE. The critical window for the development of NLE is typically during the first few months of life, as the maternal autoantibodies gradually decline and the infant's immune system matures (5). The severity and clinical manifestations of NLE can also be influenced by the specific autoantibody profiles and titers present in the maternal circulation. Higher levels of certain autoantibodies, such as those targeting the SSA/Ro and SSB/La antigens, have been associated with an increased risk of more severe forms of NLE, including complete heart block and neonatal lupus syndrome (5). The aim of this review is to provide an overview of the challenges in the diagnosis and management of NLE, typically including pathophysiology, clinical presentation, and therapeutic options. For this study, we used data from established databases such as PubMed, Cochrane Library, Scopus, or Web of Science to ensure a broad and relevant literature search. We reviewed as many studies as possible on Neonatal Lupus Erythematosus.

Pathogenesis

Cellular Apoptosis and Autoantibody Complexes

One proposed mechanism for the development of AV heart block in NLE involves the role of cellular apoptosis and the formation of complexes between fetal autoantigens and maternal autoantibodies. This theory suggests that during the disease process, the fetal autoantigens SSA/Ro and/or SSB/La are translocated to the surface of cardiomyocytes, likely due to cellular apoptosis. These autoantigens then form complexes with maternal autoantibodies, resulting in the opsonization of the cardiomyocytes. The opsonized cardiomyocytes are then phagocytosed by macrophages, which subsequently release pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α) and transforming growth factor-β (TGF-β). These inflammatory mediators are believed to play a crucial role in inducing inflammation-based injuries to the atrioventricular node and the surrounding tissue, ultimately leading to the development of AV heart block (6).

Molecular Mimicry

The second proposed mechanism for the pathogenesis of AV heart block in NLE involves the concept of molecular mimicry. This theory suggests that the maternal autoantibodies implicated in NLE cross-react with cardiac L-type calcium channels, which are essential for the proper propagation of electrical signals within the heart. By inhibiting the function of these calcium channels, the autoantibodies can disrupt the normal electrical conduction in the heart, leading to the development of arrhythmias and AV heart block (7).

Cytokine Profile and Clinical Manifestations

Findings of Shimozawa et al. shed further light on the potential role of cytokines in the pathogenesis of NLE. They examined the cytokine profiles of two siblings with NLE, one of whom developed AV heart block and the other presenting with typical skin rash. Despite the vastly different clinical manifestations, both infants exhibited elevated levels of interleukin-6 (IL-6), IL-8, interferon-gamma (IFN-γ), and monocyte chemoattractant protein-1 (MCP-1). Interestingly, the infant with skin lesions also showed increased serum levels of IL-12, IL-13, and IL-17. These findings suggest that the specific cytokine profile may contribute to the development of distinct clinical features in NLE, with the presence or absence of cutaneous abnormalities potentially dependent on the unique cytokine landscape (8).

Clinical Manifestation

NLE can involve different organs including the skin, heart, liver, hematologic and neurologic system. Some of the clinical manifestations of NLE are reversible and some are irreversible (9). Dermatologic manifestations have been reported to occur in 15% to 40% in different studies (1, 10). Many children with NLE (80%) have no skin findings at birth but develop cutaneous manifestations with sun exposure (11). The rash usually appears as macular annular or elliptic erythema, papular or plaque-like lesions on the head, scalp, and trunk or extremities (1).

Anemia, thrombocytopenia, neutropenia, aplastic anemia, hemolytic anemia, immune thrombocytopenic purpura, microangiopathic hemolytic anemia, hypocomplementemia, disseminated intravascular coagulation and thrombosis were hematologic complications observed in some studies (1, 9, 12, 13).

Cholestatic hepatitis, liver failure, conjugated hyperbilirubinemia, transient transaminase elevations were reported in NLE with hepatobiliary involvement (14, 15). Except for

liver failure, other liver complications have a good prognosis and are completely resolve. Hepatic manifestations are associated with a good prognosis and undergo spontaneous resolution with no long-term sequelae (16). The outcome is typically excellent, but cases of death secondary to liver failure have been reported (17).

Neurologic Involvement includes macrocephaly with or without hydrocephalus, hypocalcemic seizures, neuropsychiatric abnormalities, vasculopathy, spastic diplegia, and cerebrovascular accident. Most central nervous system findings are asymptomatic. The most serious complications of NLE involve the cardiac that include first-, second-, and third-degree heart block, QT interval prolongation, ventricular dilation, pericardial effusion, dilated cardiomyopathy, heart failure, AV valve insufficiency, myocarditis, endocardial fibroelastosis, myocardial fibrosis and hydrops (15, 18).

For most children born with neonatal lupus who do not experience severe heart block, there is no direct evidence of long-term neurological impairment. Cognitive development and overall neurological function are generally normal.

The most important factor for neurological prognosis is the management of congenital heart block, which, if severe and untreated, could potentially lead to brain injury due to impaired blood flow. However, with early intervention (like a pacemaker), most children go on to have normal development and neurological outcomes.

According to recent studies, the majority of children with neonatal lupus and congenital heart block do not experience significant neurological deficits once the heart condition is managed. For example, one study suggests that pacemaker therapy in children with CHB associated with neonatal lupus results in normal neurodevelopment in most cases, though close monitoring is needed throughout childhood for both cardiac and neurological health.

Ostium secundum type atrial septal defect, ventricular septal defect, patent foramen ovale, persistent patent ductus arteriosus, fusion of the chordae tendineae of the tricuspid valve, pulmonary stenosis and pulmonary valvular dysplasia are structural abnormalities that have also been observed in numerous patients (1, 13, 19–21). Other manifestations of NLE are stippling of the epiphyses, proteinuria, raised serum creatinine with decreased creatinine clearance rate has also been reported with NLE (22).

ACR and EULAR do not have separate, detailed guidelines specifically for neonatal lupus, but they provide valuable recommendations within their broader guidelines on SLE management. Fetal monitoring with echocardiograms is emphasized in both sets of guidelines for women with anti-SSA/Ro antibodies, especially in those with a previous child affected by NLE. The aim is to detect early signs of cardiac involvement, such as AV block, to allow for prompt intervention.

Management

Prenatal Diagnosis and Monitoring

Prenatal diagnosis and monitoring are critical in managing pregnancies at risk for NLE. Regular fetal echocardiography is recommended for women with anti-SSA/Ro and anti-SSB/La antibodies, particularly between 16 and 26 weeks of gestation, to detect early signs of cardiac involvement (23). Early identification of heart block allows for timely intervention, potentially preventing progression to more severe forms. In addition to echocardiography, maternal autoantibody testing is essential for risk stratification (Figure 1) (24).

Prenatal Therapies

Fluorinated Corticosteroids

Fluorinated corticosteroids, such as dexamethasone and betamethasone, have been used to treat fetal heart block in NLE. These agents can cross the placenta and reduce inflammation in the fetal heart. Studies have shown that early administration may prevent the progression of first- or second-degree heart block to complete heart block (25). However, the efficacy in reversing complete heart block is limited, and potential maternal and fetal side effects, including growth restriction and oligohydramnios, must be considered (26).

Intravenous Immunoglobulin (IVIG)

IVIG has been investigated as a preventive therapy for congenital heart block in high-risk pregnancies. It modulates the immune response and reduces the transplacental transfer of autoantibodies. Clinical trials have shown mixed results; while some studies suggest a reduction in the incidence of heart block, others report no significant benefit (27). IVIG is generally well-tolerated but requires further research to establish its role definitively (28).

Terbutaline

Terbutaline, a beta-agonist, has been used to increase fetal heart rate in cases of bradycardia associated with heart block. It provides

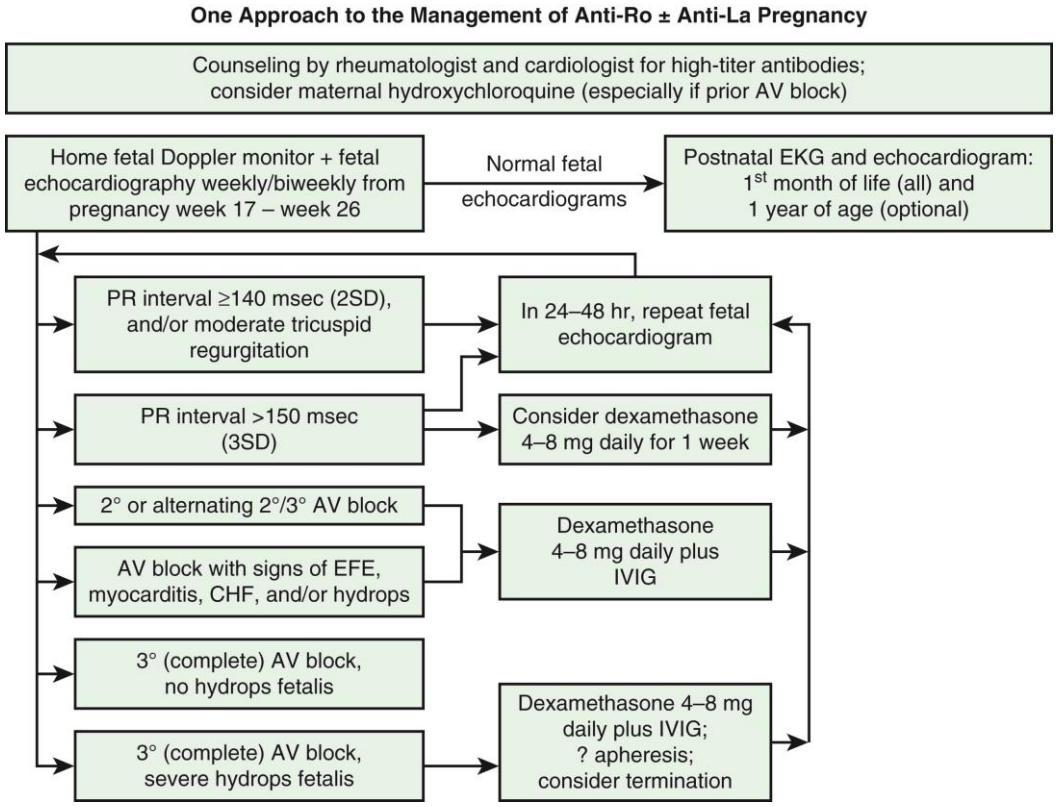

Figure 1. A management for the treatment of cardiac involvement in a fetus with neonatal lupus

symptomatic relief and can be used as a bridge to more definitive therapy, such as pacemaker implantation postnatally. Its use is particularly beneficial in maintaining adequate cardiac output in the fetus (29).

Plasmapheresis

Plasmapheresis aims to remove maternal autoantibodies from the circulation, thereby reducing their transplacental passage. Case reports and small series suggest that plasmapheresis, often combined with IVIG or corticosteroids, may improve outcomes in severe cases of fetal heart block. However, evidence is limited, and larger studies are needed to confirm its efficacy and safety (30).

Hydroxychloroquine

Hydroxychloroquine (HCQ) has shown promise in preventing congenital heart block in pregnancies of women with anti-SSA/Ro antibodies. It modulates the immune response and reduces inflammation. Retrospective studies indicate that maternal HCQ use is associated with a lower risk of fetal heart block, particularly in women with a previous affected pregnancy (31). HCQ is generally safe during pregnancy and is recommended for at-risk women (32).

Postnatal Therapies

Topical Corticosteroids

Cutaneous manifestations of NLE, such as rash, are typically self-limiting but can be treated with topical corticosteroids to alleviate symptoms and promote resolution. Mild to moderate potency steroids are preferred to minimize side effects (33).

Blood or Platelet Transfusion

Hematologic complications, including thrombocytopenia and anemia, may require supportive therapy such as blood or platelet transfusions. These interventions are crucial in severe cases to prevent bleeding and ensure adequate oxygen delivery (34).

Systemic Corticosteroids

Systemic corticosteroids are used for severe or persistent manifestations of NLE, including cutaneous, hepatic, and hematologic involvement. They reduce inflammation and autoantibody effects but should be used judiciously due to

potential side effects (35).

Pacemaker Insertion

Complete heart block in NLE often requires pacemaker insertion, particularly if symptomatic or associated with low ventricular rates. Early implantation can be life-saving and is indicated in cases unresponsive to medical therapy (36).

Anti-TNF Drugs

Anti-TNF drugs have been explored for their anti-inflammatory properties in NLE. While primarily used in maternal autoimmune diseases, their role in treating neonatal manifestations is emerging. Caution is advised due to potential risks in neonates (37).

Challenges and Future Directions

Managing NLE presents several challenges, including early detection, effective prenatal interventions, and long-term monitoring of affected infants. The rarity of the condition limits large-scale studies, making evidence-based guidelines difficult to establish. Future research should focus on identifying biomarkers for risk stratification and developing novel therapies to prevent and treat NLE manifestations. Multidisciplinary collaboration is essential to optimize outcomes for affected families.

Conclusion

Neonatal lupus erythematosus remains a challenging condition with significant implications for affected infants and their families. Advances in prenatal screening and therapeutic interventions have improved outcomes, particularly for cardiac manifestations. However, ongoing research is needed to refine management strategies and develop preventive measures. A comprehensive, multidisciplinary approach is crucial for optimal care.

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Conflicts of interest

The authors declare that they have no conflict of interest.

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