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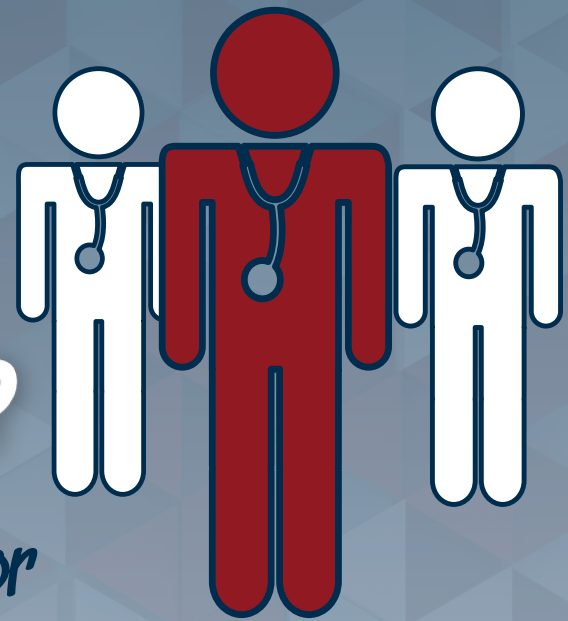
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Progressive Respiratory Failure with Pulmonary Hypoplasia and Persistent Pulmonary Hypertension Associated with Congenital Cytomegalovirus Infection

Abstract

Congenital cytomegalovirus is one of the most common intrauterine infections of human birth, and while most infected infants are asymptomatic, those infants that are symptomatic are often premature infants that may develop one or more deficits. In this case, we present an infant that developed progressive respiratory failure with pulmonary hypoplasia, which has rarely been associated with congenital cytomegalovirus infection and that ultimately led to demise. Evidence pertaining to prenatal and postnatal treatment options for this condition is limited, and more data is needed to guide the management of infants with overwhelming congenital cytomegalovirus infection.

delays, seizures, sepsis-like syndrome, or chorioretinitis.^{1,3} However, lung complications have also been reported as a rare manifestation of symptomatic CMV infections in infants.⁴

Lung complications have mostly been described in the literature as interstitial pneumonitis, bronchopulmonary dysplasia, cystic interstitial emphysema, and interstitial pneumonia; but there have also been a few reports of pulmonary hypoplasia, with fetal ascites associated with congenital CMV infections.⁴⁻⁸ The diagnosis of pulmonary hypoplasia has been associated with significant neonatal morbidity and up to 70% mortality.⁹ While there are only a few reports of lung disease, particularly

The growth restriction had prompted referral to maternal-fetal medicine specialists who sent prenatal testing, which identified elevated immunoglobulin titers and avidity for CMV. The infant delivered early and by caesarean section due to non-reassuring fetal heart tones detected during monitoring, which was performed due to the growth restriction. She required resuscitation at birth including bag-mask ventilation, but transitioned to nasal continuous positive airway pressure (CPAP) then weaned off respiratory support over the next few days. She did not receive surfactant.

An echocardiogram shortly after birth revealed a structurally normal heart with a



Figure 1. Cranial sonography images revealing periventricular calcifications and a right posterior occipital cyst.

Introduction

Congenital cytomegalovirus (CMV) is one of the most common intrauterine infections of human birth, with a reported prevalence ranging from 0.2% to 2.2%.¹ At birth, almost 90% of infected infants are asymptomatic. The 10% of infected infants who are symptomatic are often premature infants born to seropositive mothers.^{2,3} Most infants with symptomatic congenital CMV infection develop one or more deficits including sensorineural hearing loss, mental disability, motor

pulmonary hypoplasia in congenital CMV infections, symptomatic cases in immunocompromised or preterm infants have resulted in devastating outcomes. As such, we present the case of a six-week-old infant who died from progressive respiratory failure with pulmonary hypoplasia attributable to congenital cytomegalovirus infection.

Case Report

A 1520-gram baby girl was born to a 20-year-old mother following a 35-week, four-day gestation, which was complicated by intrauterine growth restriction.

flattened interventricular septum consistent with mild pulmonary hypertension. Cranial sonography revealed periventricular calcifications and a right occipital cyst (Figure 1). Liver enzymes were elevated, thrombocytopenia was present, and CMV was detected via polymerase chain reaction in the blood, urine, and cerebrospinal fluid. Retinal examination did not reveal signs of retinitis. Bilateral hearing screens were failed. The infant was transferred to a level IV neonatal intensive care unit on day of life four for pediatric infectious disease consultation. After discussion of the risks and benefits of antiviral treatment, the

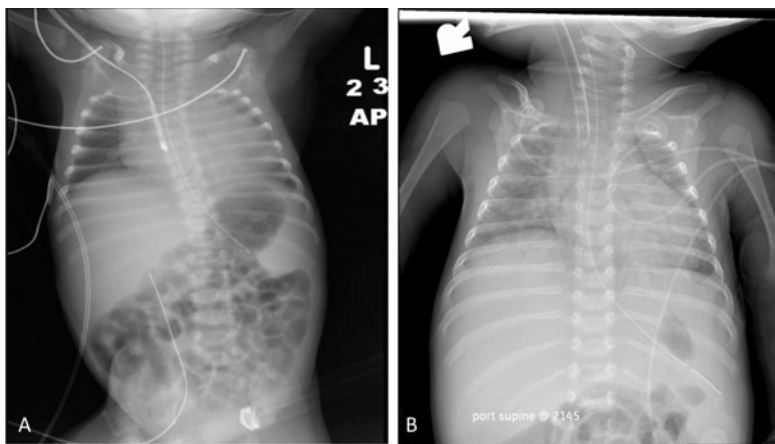


Figure 2. Chest x-ray (A) done at delivery revealing lower-than-expected lung volumes and Chest x-ray (B) done at six weeks of life with progressive pulmonary disease.

parents elected not to start ganciclovir or valganciclovir at that time.

Although the infant initially weaned off all respiratory support by nine days of life, she remained off support for only two days. Lung size was subtly smaller than expected on chest radiographs done shortly after birth (Figure 2). There was a slow escalation in nasal cannula flow and oxygen requirements, and she was placed again on CPAP at a few weeks of life. She had increased work of breathing, oxygen requirements, and carbon dioxide retention, which slowly progressed and required further escalation to nasal intermittent positive pressure ventilation then non-invasive, neurally adjusted ventilatory assist (NAVA). She required intubation for mechanical ventilation at six weeks of life. The infant's respiratory status continued to worsen on conventional ventilation over the next week. The infant developed severe pulmonary hypertension with hypoxemia on 100% fraction of inspired oxygen. She was placed on inhaled nitric oxide. Chest radiographs done at this time showed poorly-expanded lung fields occupying less of the thorax than expected despite high ventilator pressures (Figure 2).

CMV viral load was 410 337 IU/mL (increased from 286 IU/mL on day of life one), and ganciclovir was started at this time. Broad spectrum antibiotics were administered due to clinical decompensation with negative blood cultures and a tracheal aspirate that was positive for *Serratia marcescens* and *Acinetobacter* species.

fraction of inspired oxygen, and 20 parts per million of inhaled nitric oxide. The infant required dopamine and norepinephrine infusions to maintain an adequate blood pressure. After maintaining marginal oxygenation for one week on this support, the infant became progressively edematous and acidotic. Eventually, the infant was unable to achieve oxygen saturations above 60-70% despite increasing ventilator support. After discussion with the parents, the infant was removed from the ventilator and died within a few minutes. Autopsy was declined.

Conclusion

This case illustrates the findings of pulmonary hypoplasia with progressive development of respiratory failure and pulmonary hypertension, which was ultimately fatal in an infant with congenital CMV infection. Evidence pertaining to prenatal and postnatal treatment options for this condition are limited and more data is needed to guide the management of infants with overwhelming congenital CMV infection.

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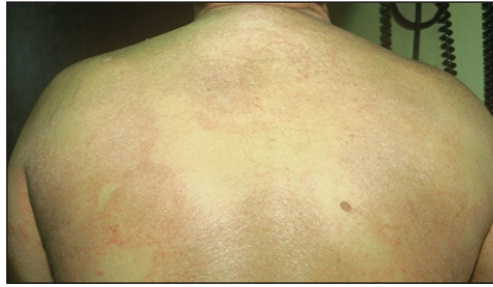
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High-frequency ventilation was attempted on multiple occasions; however, the neonate did not tolerate this, requiring chest compressions for bradycardia twice during these trials. The infant was sustained on pressure-control ventilation with mean-airway pressures of 22, 100%

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For additional references, email ams@arkmed.org.

A 45-year-old man presents to his primary care physician with a widespread rash across the back, present and slowly spreading for several months. No other areas of the body are involved, and the patient reports associated modest itching and flaking. Examination demonstrates a papulosquamous eruption characterized by erythematous, scaly plaques with crisply demarcated borders and central clearing. Small papulopustules are noted on some of the raised borders.



What is the most likely diagnosis and appropriate intervention/treatment?

- A. A potassium hydroxide mount should be prepared from skin scrapings, and if indicative of tinea corporis, a topical azole antifungal prescribed.
- B. The rash represents nummular eczema, and a mid-potency topical corticosteroid cream such as triamcinolone acetonide is recommended.
- C. The patient has psoriasis, which can be effectively managed with a topical vitamin D analog, such as calcipotriene ointment.
- D. The patient has seborrheic dermatitis, and a mixture of topical hydrocortisone 2.5% and ketoconazole 2% cream would be expected to result in improvement.
- E. The eruption represents impetigo, likely due to resident staphylococci, and should be treated with topical mupirocin ointment.

Answer: A. The patient has tinea corporis, colloquially known as “ringworm.” The name is a misnomer, as tinea corporis represents a dermatophyte fungal infection. The organism can be transmitted from direct contact with infected individuals or animals or contaminated fomites.

Dermatophytes require keratin from skin, hair, or nails for sustenance and are comprised of filamentous fungi belonging to the genera *Trichophyton*, *Epidermophyton*, and *Microsporum*. Tinea corporis (infections involving parts of the body other than scalp, face, groin, or feet) can be associated with any of these organisms but is most often caused by *T. rubrum* (approximately 80-90% of cases) and occasionally *M. canis*, when transferred by a cat or dog. Considering that tinea infections are contracted via contact, it is most often transmitted through physical contact with an infected individual, sharing clothing or towels, or participating in a sport with persistent skin-to-skin contact. Highly humid

and warm environments strongly encourage infection.

Tinea corporis is most commonly seen in children, with nearly 50% of cases in the U.S. diagnosed in children <10 years old, though infection in adults is not uncommon. Genetic predisposition may play a role in contracting the infection and persons who are immunocompromised are also at higher risk of developing the disease. The differential diagnosis of tinea corporis includes other annular cutaneous dermatoses, which typically present on the trunk and proximal extremities including erythema annulare centrifugum, subacute cutaneous lupus erythematosus, and granuloma annulare. Other papulosquamous disorders such as psoriasis, nummular dermatitis, and large-plaque parapsoriasis may at times mimic tinea corporis. Tinea corporis classically presents as an annular lesion(s) with a well-defined, raised, scaly border and central clearing. Lesions are often solitary but can be multiple and/or overlapping. Definitive diagnosis is rapidly provided by the observation of segmented, often branching hyphae in skin scrapings utilizing a potassium hydroxide (KOH) preparation. A fungal culture can also be performed, though results will be delayed.

Most instances of tinea corporis can be managed with topical therapy (allylamines or azoles). Extensive infection warrants consideration of an oral agent such as terbinafine, itraconazole, fluconazole, or griseofulvin.

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Primary Psychiatric Disorder Masking the Diagnosis of Neuropsychiatric Syndrome in SLE

Abstract

Systemic lupus erythematosus (SLE) is a widespread, chronic inflammatory autoimmune disorder affecting various organ systems in the body. Neuropsychiatric syndrome of systemic lupus erythematosus (NPSLE) refers to the nervous system involvement of SLE manifesting as neurologic or neuropsychiatric symptoms.¹ Use of older terminology such as “lupus cerebritis” and “lupus sclerosis” is no longer recommended for this condition. NPSLE has a wide range of presentation such as cognitive dysfunction, seizure disorders, movement disorder, psychosis, acute confusional state, myelopathy, or with focal symptoms such as cerebrovascular accident or transient ischemic attacks. NPSLE can present actively during an SLE flare or even precede the SLE flare, but not all cases present during the active disease course.¹ Prompt identification and diagnosis of NPSLE is crucial so that appropriate treatment can be administered and not delayed. The diagnosis of NPSLE remains challenging as there are no confirmatory laboratory or radiologic findings; thus, this condition is a diagnosis of exclusion after ruling out other causes such as infections, electrolyte or metabolic disorders, mass lesions, and primary psychiatric disorders.²

This report discusses a young, female patient without a previous diagnosis of SLE who initially presented with acute confusion, new onset seizures, and acute psychosis in addition to septic shock, bacteremia, severe metabolic acidosis, and acute renal failure requiring hemodialysis. Notably, she had a past medical history of bipolar disorder, IV drug abuse, and multiple previous admissions to the psychiatric unit for acute psychosis, suicide ideation, and aggressive behavior. Her previous psychiatric history made the diagnosis of NPSLE difficult; however, once identified

and treatment with IV methylprednisolone commenced, her neurologic status improved within days.

Case Presentation

A 27-year old African American female with past medical history bipolar disorder (on lithium, risperidone and Depakote) and IV drug abuse presented to the emergency room (ER) with altered mental status, confusion, and bilateral lower extremity edema. Her initial vitals were: blood pressure 100/70 mm Hg, respiratory rate of 25/min, heart rate of 105/min, and an oral temperature of 100.1 oF. The Glasgow Coma Score was 11 (E= 3 V= 3 M= 5). She appeared older than her age, disheveled, cachectic, confused, with slurred speech, and was a poor historian. On general observation, she had bilateral conjunctivitis with green/yellow discharge and discoid rash was noted on her forehead extending to and around the bilateral eyes and there was diffuse thinning of hair and patches of hair loss on scalp. She had bilateral mastitis, with no abscess or nipple discharge. Examination of the chest revealed a normal S1S2 without murmur and decreased breath sounds in bilateral lung fields. She had 3+ bilateral lower extremity edema with palpable pulses. No IV track marks were noted on her body.

Admission laboratories showed a complete blood count with a white cell count of 19,000 mm,³ hemoglobin of 4.9 gm/dl, hematocrit of 16.3% with red cell indices showing a mean corpuscular volume of 93.1 fL, and mean corpuscular hemoglobin concentration of 30.1 gm/dl. Serum chemistries revealed potassium 7.6 mmol/L, bicarbonate 4 mmol/L, blood urea nitrogen 108, creatinine 18.3, lactic acid 2.4 mmol/L, and the glomerular filtration rate (GFR) 3 ml/min. A lithium level of 2.6 mmol/L was also noted. The

urinalysis showed hyaline casts, white blood cell (WBC) clumps, bacteria, red and white blood cells, and proteinuria; a urine drug screen was positive for cannabinoids, barbiturates, and amphetamines. An electrocardiogram (EKG) showed sinus tachycardia, and the chest x-ray demonstrated cardiomegaly and bilateral pleural effusions. A computed tomogram (CT) of the chest and abdomen revealed cardiomegaly with pericardial effusion, bilateral axillary lymphadenopathy, and body wall edema and free fluid in the pelvis. The CT head was unremarkable. An echocardiogram showed the left ventricular ejection fraction 20%, with pericardial effusion but no tamponade. Considering her presentation, examination findings, and laboratory and radiologic results in the context of her previous history of IV drug use, an infectious etiology – bacterial vs. viral – was suspected and cultures obtained, and empiric therapy begun. The patient was admitted to the intensive care unit for initial management with fluid resuscitation, broad spectrum antibiotics, and urgent hemodialysis. Cerebrospinal fluid showed no cells or elevated protein. An erythrocyte sedimentation rate was elevated >150mm/hr and the C reactive protein level was 1.5 mg/dL. Her condition clinically improved and she was transferred from the ICU after 24 hours, but on hospital day four, she had multiple generalized, tonic-clonic seizures and was re-admitted to the ICU and Neurology consultation was obtained. A CT head showed diffuse edema of bilateral cerebral hemispheres, bilateral basal ganglia, bilateral cerebellar hemispheres and brainstem, without hemorrhage or hematoma and no skull fractures. The EEG suggested mild encephalopathy and anti-epileptics were added to her treatment regimen.

The rapid decline in her neurologic status with new onset seizures, deteriorating sen-

Table 1. Neuropsychiatric Syndromes of Lupus Erythematosus ^{3,8,11}

Central Nervous System Related	Peripheral Nervous System Related
Aseptic Meningitis	Acute Inflammatory Demyelinating
Acute Confusional State	Polyradiculoneuroptht
Cerebrovascular Disease	Autonomic Disorders
Demyelinating Syndrome	Cranial Neuropathy
Headache	Mononeuropathy
Movement Disorder	Myasthenia Gravis
Mood Disorders	Plexopathy
Myelopathy	Polyneuropathy
Psychosis	
Seizures	

sorium, and failure to respond to therapy prompted a reconsideration of her provisional diagnosis. An increased suspicion for an underlying autoimmune disorder was entertained considering this patient's age, gender, dermatologic findings of facial rash, elevated serum inflammatory markers, direct Coombs positive anemia, and thrombocytopenia. Multi-organ system involvement further supported this etiology: urinalysis with hematuria and proteinuria and acute renal failure

requiring hemodialysis; heart failure with reduced ejection fraction, pericardial effusion and cardiomegaly; bilateral pleural effusions; and diffuse cerebral edema with new-onset seizures and rapid worsening of mental state and neurologic symptoms.

Autoimmune testing demonstrated a positive ANA screen with ANA titer > 1:1280 with a homogeneous nuclear pattern. Other findings included anti-ds DNA antibodies 725 IU/mL; C3c 50 mg/dL, C4c

10 mg/dL (low levels of C3 and C4), and positive pANCA. The diagnosis of SLE was confirmed on the ninth day of hospitalization, and she was started on high-dose IV methylprednisolone (1000 mg q 24 hr) for three days and placed on hydroxychloroquine (200 mg q24 hr) with subsequent rapid improvement of her neurologic status within days. Since her mental status had improved and she had remained seizure-free with steroid therapy, her anti-epileptic medication was discontinued. Because of her renal failure, a renal biopsy was performed and reported focal crescentic glomerulonephritis, Pauci-immune type; she was subsequently started on treatment with mycophenolate 500 mg orally twice daily.

Following hospital discharge, she was seen in the clinic for a transition of care follow-up visit. She was no longer requiring treatment with anti-psychotics and anti-epileptics, as treatment of underlying SLE had improved her psychiatric and neurologic status. Moreover, her renal failure had resolved, and she no longer required hemodialysis.

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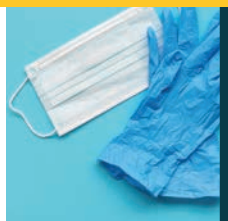
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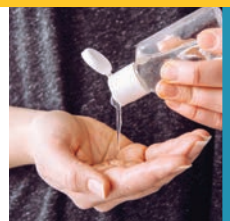


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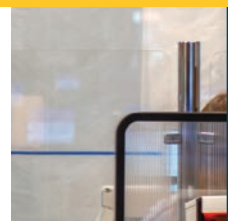
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Discussion

This patient had presented with a septic clinical picture and acute encephalopathy that, coupled with her history of psychiatric co-morbidity and history of IV drug use, masked an acute exacerbation of SLE with neuropsychiatric manifestation. The acute encephalopathic presentation of this patient in the emergency room may have been a reflection of the patient's post-ictal state. The new-onset seizures during the inpatient course were a key factor for considering an alternate diagnosis; they prompted an autoimmune workup and eventually confirmed the diagnosis of SLE.

Systemic lupus erythematosus (SLE) is a multi-organ, chronic, autoimmune disorder with widespread inflammation in affected tissues. It is characterized by production of pathogenic autoantibodies against the nucleic material and binding proteins of one's own cells, thereby resulting in the loss of self-tolerance and over-activation of the immune system.³ Consequently, there is widespread inflammation, which leads to tissue injury and end-organ damage. Clinical presentation can vary depending on what systems are involved in SLE; these range from dermatologic/cutaneous (malar rash, photosensitivity), renal (glomerulonephritis, acute renal failure), hematologic (anemia, thrombocytopenia), cardiac (pericarditis), pulmonary (pleural effusion, pleurisy), or central nervous involvement (aseptic meningitis, cerebritis, seizure).³

For our patient, the clinical presentation of SLE exacerbation was with shock, acute encephalopathy, new-onset seizure disorder, and acute renal failure with metabolic acidosis necessitating management with hemodialysis. Initially an infectious etiology was considered, but failure to improve with therapy prompted expansion of the differential diagnosis to include autoimmune disease, considering her clinical picture in combination with her demographics and laboratory and radiologic evidence of multi-system organ involvement (renal, cardiac, CNS, dermatologic, and hematologic). SLE was confirmed by laboratory testing (elevation of ESR

and CRP, positive pANCA, decreased C3 and C4; positive ANA, and anti-DS-DNA antibodies).

Neuropsychiatric syndrome in SLE (NPSLE) refers to the subgroup of patients presenting with neurologic or psychiatric symptoms of SLE (see Table 1) and is the second leading cause of mortality and morbidity in patients with SLE.⁴ The neuropsychiatric manifestations can be primary (related to the disease activity itself) or secondary (related to treatment [steroid-induced], infections, metabolic, or other systemic manifestations not related to SLE).⁵ Neurologic and/or psychiatric manifestations can precede, occur simultaneously, or follow the diagnosis of SLE; in some patients, it may serve as the only or the initial finding, thereby making diagnosis further difficult. Nonetheless, the presence of neuropsychiatric symptoms identifies those individuals with a higher mortality and a poor prognosis versus those without.⁴

The presenting neuropsychiatric manifestations of NPSLE can be nonspecific (headache, cognitive dysfunction, psychosis, seizure) thus posing a challenge in identifying patients with primary NPSLE as there is no gold standard criteria to support the diagnosis of NPSLE and exclude confounding diagnoses.² As previously noted, this patient's past medical history was significant for bipolar disorder and aggressive behavior since the age of 21 years, IV drug abuse, and prior multiple admissions to the psychiatric unit for acute psychosis. She was on anti-psychotic medication for bipolar mood disorder. Acutely, her neuropsychiatric manifestations were masked by her past medical history of bipolar disorder and IV drug abuse. Appropriate treatment for SLE resulted in control and improvement in neuropsychiatric symptoms, and she no longer required therapy with anti-epileptics and anti-psychotic medications.

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Diagnostic Evaluation in NPSLE

The initial step toward defining a uniform diagnostic criterion for NPSLE was the 1999 American College of Rheumatology guidelines. Since that time, numerous studies applying the ACR criteria have been published to compare cohorts for the presentation of neurologic manifestations of SLE. There are relatively few population-based estimates of the prevalence of NPSLE. A recent attempt to quantify the prevalence of NPSLE evaluated nine studies comprising 5057 SLE patients and estimated the overall prevalence of NPSLE to vary from 11% to 60% of all SLE patients, with the most common disorders being major depression (17%-52%), headaches (38.8%-60%), seizures (26.4%-63%), and cerebrovascular diseases (26%-38.8%).⁶ By design, the 1999 ACR criteria are used for diagnosis and other authors have endorsed modifications to develop a more comprehensive guideline to support clinical decision-making and improve patient care.⁷

Evaluation of patients with manifestations of NPSLE consists of investigations that first establish the diagnosis of SLE and exclude non-SLE etiologies. The diagnosis is based on clinical picture, cerebrospinal fluid (CSF) analysis, electrophysiological studies, neuroimaging, and neuropsychological testing. Current serologic tests are neither sufficiently accurate for diagnosis of NPSLE nor for as-

sessing disease severity. Analysis of CSF is important to exclude infection, integrity of the blood-brain barrier, presence of autoantibodies (e.g., α -phospholipid antibody, α -ribosomal-P, or α -NR2) or possibly Neuro-inflammatory mediators (e.g., lipocalin 2 or osteopontin). Electroencephalography can assess for seizure disorder. Neuroimaging to evaluate brain structure (e.g., computed tomography or magnetic resonance imaging) and/or brain function (PET) may reveal anatomic or functional abnormality. A variety of screening tools developed to assess neurodegenerative conditions may be used to evaluate cognitive dysfunction, depression, and anxiety in SLE patients. The Montreal Cognitive Assessment Questionnaire is sensitive (0.83-0.94) and moderately specific (0.27-0.46) for detecting cognitive disorder versus normal controls. The Center for Epidemiological Studies-Depression Scale and the Hospital Anxiety and Depression Scale are considered highly reliable for depression and anxiety screening.^{8,9}

Management

Treatment options for patients with NPSLE are not well-standardized since no robust, randomized controlled trials have been performed to validate therapies for specific NPSLE manifestations; therefore, current treatment regimens are based on small, controlled trials, case studies, and expert opinion.³ Therapy should be targeted at the type of neuropsychiatric event (considering the 19 NPSLE syndromes) and individualized based on symptom severity and time from onset, suspected mechanism (detection of specific antibodies or thrombosis), expected morbidity, acute-versus-chronic presentation, whether predominantly inflammatory or vascular involvement, whether disease flare is active and ongoing or resultant from secondary damage, and response to prior therapies.^{3,10}

Symptomatic therapy for mild manifestation of NPSLE such as headache or depression may be sufficient; however, more severe NPSLE manifestations or inadequate response to symptomatic treatment may require aggressive therapy

with immunosuppressive agents to control the underlying autoimmune process and prevent organ damage.^{3,10,11}

For inflammatory NPSLE, systemic glucocorticoids are most often used and result in a beneficial response in 60-75% of patients.³ Differing dosing regimens from prednisolone 0.5 – 1 mg/kg/day to bolus IV methylprednisolone 500 mg to 1 g/day have been used but no strong data support a particular protocol.¹⁰ In severe disease activity, additional immunosuppressive therapy with hydroxychloroquine, methotrexate, cyclophosphamide, and azathioprine have also been used. Rituximab and belimumab have also been used, with high response rates in severe NPSLE. For severe refractory NPSLE – particularly in cases where severe infection, pregnancy, or life-threatening symptoms are present – IV immunoglobulin and plasmapheresis have been used as bridge therapy pending establishment of a maintenance regimen.^{10,11}

For ischemic NPSLE, the decision for secondary thromboprophylaxis is based on definitive presence of anti-phospholipid syndrome (APS).¹¹ The binding of anti-phospholipid (aPL) antibody to endothelial cells starts a cascade that favors platelet clot formation via increasing the expression of glycoprotein IIb/IIIa. Consequently, NPSLE who are APS+ have a higher prevalence of accelerated atherosclerosis and vasculopathy.¹² In addition to statin therapy (target LDL <70mg/dL), NPSLE patients seronegative for aPL (or aPL positive but lacking criteria for APS) may receive anti-aggregation platelet therapy with aspirin (50-325 mg/day) monotherapy, the combination of aspirin plus extended-release dipyridamole, or clopidogrel (75 mg/day) monotherapy. In SLE patients with stroke and fulfilling criteria for APS, the optimal treatment is controversial. Some authorities favor anti-aggregation antiplatelet therapy alone while others advocate for combined anti-platelet therapy and lower intensity anticoagulation with Warfarin (INR 2.0-3.0) or high-intensity oral anticoagulation (INR>3.0).¹¹ Low-molecular weight hep-

arins (e.g., enoxaparin 30mg every 12 hours subcutaneously) are efficacious therapy for SLE patients with vascular thrombosis.⁴

Conclusion

The presence of NPSLE identifies those patients with a higher mortality than those without the manifestations of NPSLE. Therefore, for patients presenting with altered neurological features combined with a fitting clinical picture of SLE such as multi-system involvement, NPSLE should be included in the differential diagnosis to avoid delay in the diagnosis and management of SLE.²

References

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Hayes, Richard L., MD
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Hightower, Michael D., MD
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Hui, Anthony N., MD
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Johnson, Philip H., MD
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Harris, Breanna, MD
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Haynes, Jackson, MD
Hayre, Ann-Marie, MD
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Khatri, Danish, MD
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Lamagna, Matthew, DO
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Lynch, Jeffrey, MD
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Powers, Joseph, DO
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Price, Austin, DO
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Rashed, Anum, MD
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Soto, Ernie, MD
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Stanley, Marc, MD
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Henson, Jeffrey
Herzog, Abigail
Hickey, Sawyer
Hill, Sarah
Hochstein, Erika
Hollaway, Moriah
Howells, Morgan
Dennis, Coleman
Dewan, Mahfuz
Diaz-Cruz, Alexandra
Divino, Katherine
Doderer, Emily
Dominguez, Dylan
Draper, Elizabeth
Dreher, Brian
Dreher, Katie
Bolivar, Karime
D'Spain, Wyatt
Duke, Cameron
Dulaney, Breyanna
Dunn, Kristin
Dupree, Devin
Dwyer, McKenzie
Eaton, Kathryn
Ederle, Amanda
Edington, Maclain
Edwards, Michaela
Ellenburg, Caleb
Eller, Andrew
Embry, Austin
Erwin, Jake
Estes, Tyler
Estes, Joshua
Evridge, Stephen
Fazli, Yama
Fedor, Dylan
Feezell, Haley
Ferguson, Kaley
Fialkowski, Kevin
Fischer, Ashley
Flack, Joseph
Fletchinger, Teresa
Flippo, Brittany
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Frazier, Brett
Friedhoff, Allison
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Glenn, Grayson
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Gocke, Paige
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Gottspomer, Josephine
Gowen, Jared
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Graham, Rachel
Graham, Tyler
Gray, Kori
Gray, Joshua
Green, Anna
Griffin, Brittany
Guha, Anveshi
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Marruffo, John
Martin, Austin
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Massey, Jackson
Massey, Colmon
Matulich, Patrick
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Mayer, Collin
Maynard, Nicolas
Mayo, Rachel
Mazzo, James
McAlexander, Bailey
McClain, Paula
McCormick, Alexa
McCoy, Katie
McCracken, Scarlett
McCray, Ashley
McDaniel, Cynthia
McEver, William
McGill, Jocelyn
McKay, Scotty
McKinnon, Alyson
McKissack, Lillian
McKissock, Shayla
Mears, Anna
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Mendez, Michael
Merten, John
Messersmith, George
Meurer, Logan
Mihalcin, Joshua
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Milstead, Elena
Minor, Erica
Mitchell, Reece
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Moore, Moira
Moore, Benjamin
Morehead, Lauren
Moreira, Rebecca
Morgan, Austin
Morgan, Austin
Morrison, Callie
Morrison, Mary
Mundy, Allison
Munley, Benjamin
Munshi, Kavina
Musser, John
Nallur, Varenya
Naseem, Nasseeruddin
Newhart, Hamilton
Nissen, Caleb
Noyes, Amos
Nunez, Jarrett
O'Brien, Patrick
Okolo, Christina
Okoree-Slaw, Nachia
Olson, Erica
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Otwell, Alexandra
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Parker, De'Jon
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Patel, Anjali
Pavlovic Segal, Emily
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Loiacano, Olivia
Lone, Sarish
Long, Spencer
Long, Brianna
Longing, Joshua
Losada, Nicole
Loy, Hannah
Luckcock, Kierstyn
Lukhi, Pooja
Ly, Sophia
Majagi, Anusha

Preston, Zachary
Purtie-Smith, Ashton
Purvis, Connor
Quiroz, Gabriela
Rago, Cierra
Mahman, Ryan
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Ramos, Patricia
Ramponi, Carl
Rather, Peyton
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Ratliff, Adam
Ratliff, Calvin
Ravikumar, Pratheepa
Ray, Emily
Ray, Olivia
Reber, Lemuel
Reed, Raynie
Renard, Kelsey
Rice, Grace
Rice, Kristen
Richards, Mitchell
Richards, Nicole
Roark, Casey
Robertson, Annie
Roeder, Lauren
Rogers, Sydney
Rogers, Andrew
Rojas, Anapaula
Rosales, Pamela
Rose, Kaitlin
Rose, Tyler
Rose, Ryan
Rosenkrans, Lelia
Roset, Connor
Ross, Jonathon
Rostollan, Mason
Ruelas, Steven
Sabbagh, Rami
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Saleem, Humaira
Salem, Tariq
Savage, Matthew
Scarborough, Catherine
Schlott, Kiley
Schmidt, Emma
Scholl, Lindsey
Scott, Winter
Scott, Hayden
Scott-Kirchen, Logan
Scroggins, Sandra
Seltzer, Michaela
Shah, Dhaval
Shah, Naman
Shah, Dimple
Shahare, Humam
Shaw, Collie
Shaw, Madison
Shelton, Reid
Shi, Weijia
Shumate, John
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Simmons, Neil
Simon, Arlesia
Singleton, Bailey
Sivakumar, Sowmya
Smith, Chelsea
Snelgrove, Jessica
Soliman, Mohammed
Song, Jini
Sparks, Darby
Speed, Olivia
Squires, Austin
St. Columbia, Reed
Stahler, Katie
Steele, Jordan
Stinnett, Corbin
Stirewalt, Breanna
Stover, Alec
Pham, Quy
Phelan, Natalie
Phillips, Chase
Philpott, Rebecca
Pickhardt, Abigail
Pilkington, Collin
Pilkington, Anna
Plumley, Melissa
Pool, Hunter
Porter, Eric
Potter, Genna
Powell, Michael

Taylor, Nathan
Thomas, Kevin
Thompson, Natasha
Thompson, Cody
Tian, Brittany
Tibbs, Molly
Tidd, Savannah
Tierney, Zachary
Totten, Madeline
Townsend, Julia
Tran, Aimee
Tran, Yenchi
Trotter, Timothy
Tupa, Lane
Turner, Natalie
Turner, Tarahn
Tzeng, Olivia
Vanscoy, Joseph
Varghese, Reen
Vaughn, Tiffany
Vellanki, Krishna
Veluvolu, Manasa
Ventran, Victor
Vestal, Claudia
Vutarn, Michael
Wade, Dillon
Walajahi, Amad
Walden, Kaitlyn
Walker, Breanna
Walker, Allyson
Walker, Emily
Walls, Joshua
Wang, Hsin-Ping
Wanjala, Humphrey
Wary, John
Wayland, Hunter
Weatherford, Denim
Webb, Conner
Weidling, Vanessa
Wells, Kendall
West, Danielle
Westlake, Sarah
Westley, Monique
Whaley, Madison
White, Tyler
White, Jessica
Whitt, Jedidiah
Wilcoxson, Joshua
Willis, Whitney
Williams, Monica
Williamson, Jonathan
Willis, Cameron
Willis, Delaney
Wilson, Taylor
Windham, Madison
Winn, Kesley
Wolfe, Jordyn
Woodruff, Margaret
Woods, Bryce
Woods, Forrest
Woodward, Morgan
Woodford, Layton
Yao, Tianyuan
Yarbro, John
Yeates, Aimee
Yee, Addison
Young, Megan
Yusufali, Taher
Zanganeh, Nassim
Zeb, Adam
Zee, Clara
Zehr, Katherine
Zhang, Sairi
Zhao, James

OUR
FIGHT
IS AGAINST
COVID.

MINI-GRANT PROGRAM

The Arkansas Medical Society is excited to be able to assist clinics that want to provide or continue to provide COVID-19 vaccines to their patients through the Mini-Grant Program of AMS's *Our Fight is Against COVID* initiative.

The application submission deadline is November 30, 2021; however, AMS will review applications as they are submitted and you will be notified within 10 business days. Funding will be awarded based on eligibility listed below.

**VISIT [ARKMED.ORG/COVID19](https://arkmed.org/covid19)
FOR MORE INFO OR TO SUBMIT AN APPLICATION.**



■ Eligible Applicants

Any Arkansas physician-led medical practice or clinic that provides or would like to provide COVID immunizations in Arkansas is welcome to apply. You must be enrolled or in the process of enrolling in the Arkansas Department of Health's WebIZ program. (AMS can facilitate education and training for WebIZ at no additional cost if needed). At least one physician in the practice must be a member of the Arkansas Medical Society.

■ Approved Areas of Focus for Grant Funds

- Vaccine refrigerators/freezers (CDC Compliant)
- Vaccine transport coolers
- EMR upgrades or technology needs
- Generators
- Other needs associated with COVID vaccine administration and tracking

■ Items NOT Approved for Grant Funds

- Legislative/lobby activities
- Ongoing operating costs
- Purchase of vaccines

■ Grant Reimbursement Process

The Our Fight Against COVID Mini-Grant is a reimbursement grant that provides funding to recipients *after* expenses have been incurred. AMS will accept applications through November 30, 2021. Upon receipt of the application, the review process will begin, and a decision will be made within 10 business days. Items for reimbursement must be purchased after the grant approval date. Expenses incurred before the grant application deadline will not be eligible. Paid invoices and/or receipts of items must be submitted to AMS for reimbursement of funds. The expected timeline of reimbursement of items is estimated within six weeks.

■ Grant Reporting

Sixty days (60) after reimbursement, recipients must submit a status report to AMS. The final Grant Activity Outcome report is due June 1, 2022.

Got Questions? Email fightcovid@ARKMED.org.



We have your back, so you can move forward.

When a problem occurs, you need a proactive partner that can navigate unforeseen challenges and help you solve the tough legal issues that come with practicing medicine. In our collaborative claims process, our in-house attorneys and our roster of local defense attorneys help you – our valued members – to be ready for what lies ahead.

Move forward with us at
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