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Lupus test online

Diagnosing Lupus: A Confusing Experience Getting diagnosed with lupus can be a confusing and frustrating time due to the diverse range of symptoms experienced by patients, making it challenging for healthcare professionals to determine the cause. The first step is to book an appointment with your GP. They may conduct blood tests, urine tests, or perform an ANA test to investigate your symptoms. If these tests suggest lupus, you'll be referred to a rheumatologist who will use specific criteria and other test results to make a diagnosis. Depending on the primary issue, you might be referred to a dermatologist if skin-related symptoms are the main concern. To better understand the experience of people being diagnosed with lupus, check out our video and scroll down for more information. Lupus affects various body systems, leading to an array of symptoms such as joint and muscle pain, fatigue, rashes, headaches, mouth ulcers, and hair loss. There are two main types of lupus: systemic lupus erythematosus (SLE) and cutaneous lupus. SLE can impact any part of the body and is often referred to when discussing lupus in general. A rheumatologist is responsible for diagnosing lupus, usually after a referral from your GP. They will use criteria such as symptoms, medical history, and test results to make an accurate diagnosis. If you suspect you have lupus, book an appointment with your GP. They will assess your symptoms and decide whether to refer you to a rheumatologist. The duration of your symptoms may influence the referral process, and your GP might perform tests before making a decision. When investigating lupus, doctors often check urine levels for blood and protein as kidney involvement affects approximately one-third of patients. They might also request imaging tests like X-rays, MRI scans, or biopsies if organs are involved. Since there's no genetic test for lupus, other diagnostic methods are used. Blood tests such as ANA (antinuclear antibodies) and ds-DNA are commonly administered to aid in diagnosis. However, a positive result doesn't necessarily confirm lupus; it can also be present in individuals with no health issues or certain autoimmune conditions. Therefore, these test results must be considered alongside the patient's symptoms. For example, having a positive ANA without symptoms makes lupus less likely. The EULAR/ACR criteria are commonly used to diagnose lupus, taking into account various factors such as kidney and bone/joint symptoms, mucocutaneous symptoms, and SLE-specific antibodies. Before seeing a rheumatologist, patients may have their height, weight, blood pressure, medical history, and current health conditions recorded. The doctor will then take a detailed medical history, examine the patient (including checking joints), and discuss any relevant family illnesses or medications before making a diagnosis. Lupus patients often undergo a series of tests to assess their condition, including skin checks, heart and lung evaluations, blood and urine tests. Depending on symptoms, additional tests may be requested, but these will be discussed during the appointment. As lupus affects multiple systems in the body, specialists like kidney experts may be consulted. It's essential to ask questions about your condition during appointments. With a diagnosis typically taking around 6 years from initial symptoms, patients should be aware that delays can occur due to non-specific symptoms, which can resemble other conditions. Several factors contribute to delayed diagnoses, including: - Symptoms that are common to multiple illnesses - Doctors initially considering more common conditions before lupus - Variety of symptoms impacting people differently - Lack of knowledge among some medical professionals about lupus A definitive diagnosis through a single test is not possible. Common tests like ANA and ds-DNA may not be positive in all cases, requiring doctors to rule out other conditions first. Delays in diagnosis can occur due to: - Variance in blood test results - Insufficient knowledge among medical professionals - Inadequate communication between healthcare teams Patients should remember their right to a shared decision-making process with their doctor. If concerns are not being heard, requesting a second opinion or changing doctors may be necessary. Only after I changed doctors did things happen. I think it was a lack of education about lupus that led my previous doctor not to test for the right things; it wasn't necessarily their fault. My new doctor ordered the correct blood tests, and then I was referred to a rheumatologist – the rest is history. Lupus affects each person differently. When you're diagnosed, you might need to start taking medication, visit the hospital more often, or adjust your daily routine to accommodate your condition. Some people find that after their symptoms have been managed, they can return to their normal life with only a few minor changes. However, others may experience periods of illness that significantly impact their lives, requiring support from loved ones and healthcare professionals. Diagnosis can be overwhelming. While some people feel relief at finally getting a diagnosis, especially if it took a long time, others might find the news worrying. It's normal to experience a range of emotions when dealing with a chronic condition like lupus. Give yourself time to process your feelings and allow loved ones to support you during this time. As you adjust to your diagnosis, you may need to start taking regular medications. You can learn more about common treatments for lupus on our website. You'll likely be asked to visit your doctor or hospital at regular intervals for blood tests, assessments, and check-ups with your rheumatologist. With time, you'll begin to learn how best to manage your condition. Talking to others who have been diagnosed with lupus can be helpful – join our online community forum to stay up-to-date on the latest discussions. If you're struggling with your mental health due to the impact of your diagnosis, talk to your doctor about accessing support. The Wren Project is also a valuable resource for those affected by autoimmune conditions, offering listening services and support. Lupus UK can facilitate connections between individuals who have been diagnosed with lupus if that's something that would be beneficial for you. Lupus, also known as systemic lupus erythematosus (SLE), is an autoimmune disorder where the body's immune system mistakenly attacks healthy tissue. The condition can cause a range of symptoms, including painful and swollen joints, fever, chest pain, hair loss, mouth ulcers, and a distinctive red rash on the face. Lupus can be mild or severe, with periods of illness (flares) and remission. The exact cause of SLE is unclear, but it's believed to involve a combination of genetic and environmental factors. Women are more commonly affected than men, particularly those of childbearing age, with rates varying between countries. The condition can affect people of all ages, from 15 to 45 years old, although children under 18 may experience a more severe form. Diagnosing SLE can be challenging, but it's typically done based on symptoms and laboratory tests. There is no cure for the condition, but various treatments are available, including medications such as NSAIDs, corticosteroids, immunosuppressants, hydroxychloroquine, and methotrexate. SLE symptoms often mimic other diseases, earning it the nickname "the great imitator." This makes diagnosis challenging, as patients may experience unexplained symptoms for years before receiving a definitive diagnosis. Common complaints include fever, malaise, joint pains, muscle pains, and fatigue; however, these symptoms are not specific to SLE and can be seen in other conditions. When SLE does occur, it predominantly affects women, who tend to have more relapses, low white blood cell counts, arthritis, Raynaud syndrome, and psychiatric symptoms. Men, on the other hand, are more likely to experience seizures, kidney disease, serositis, skin problems, and peripheral neuropathy. Skin manifestations of SLE can be severe, with up to 70% of patients experiencing some form of skin symptom. The three main categories of lesions are chronic cutaneous (discoid) lupus, subacute cutaneous lupus, and acute cutaneous lupus. Hair loss, mouth and nasal ulcers, and skin lesions are also common. Joint pain is the most frequent reason for medical attention, with the small joints of the hand and wrist typically affected. Unlike rheumatoid arthritis, lupus arthritis is less disabling and usually does not cause severe joint destruction. People with SLE are at risk of developing osteoarticular tuberculosis and may have an association with antiphospholipid antibody syndrome, which can lead to thrombotic disorders. Abnormalities associated with this condition include a ### Prolonged partial thromboplastin time (which usually occurs in hemorrhagic disorders) and a positive test for antiphospholipid antibodies; the combination of such findings have earned the term "lupus anticoagulant-positive". Another autoantibody finding in SLE is the anti-cardiolipin antibody, which can cause a false positive test for syphilis. ### SLE may cause pericarditis (inflammation of the outer lining surrounding the heart), myocarditis (inflammation of the heart muscle), or endocarditis (inflammation of the inner lining of the heart). The endocarditis of SLE is non-infectious, and is also called Libman–Sacks endocarditis. ### Atherosclerosis also occurs more often and advances more rapidly than in the general population. Steroids are sometimes prescribed as an anti-inflammatory treatment for lupus; however, they can increase one's risk for heart disease, high cholesterol, and atherosclerosis. ### SLE can cause pleuritic pain as well as inflammation of the pleurae known as pleurisy, which can rarely give rise to shrinking lung syndrome involving a reduced lung volume. Other associated lung conditions include pneumonitis, chronic diffuse interstitial lung disease, pulmonary hypertension, pulmonary emboli, and pulmonary hemorrhage. ### Painless passage of blood or protein in the urine may often be the only presenting sign of kidney involvement. Acute or chronic renal impairment may develop with lupus nephritis, leading to acute or end-stage kidney failure. ### Because of early recognition and management of SLE with immunosuppressive drugs or corticosteroids,[35] end-stage renal failure occurs in less than 5%[36][37] of cases, except in the black population, where the risk is many times higher. The histological hallmark of SLE is membranous glomerulonephritis with "wire loop" abnormalities.[38] ### Neuropsychiatric systemic lupus erythematosus ### Neuropsychiatric syndromes can result when SLE affects the central or peripheral nervous system. The American College of Rheumatology defines 19 neuropsychiatric syndromes in systemic lupus erythematosus.[39] Given article text here Lupus can cause nerve damage, inflammation of the brain and spinal cord, and abnormal levels of fluid surrounding the brain and spine. In rare cases, it can also lead to a loss of coordination, muscle weakness, and seizures. Other symptoms include depression, fatigue, and eye problems such as dry eyes and vision loss. Vitamin D levels and SLE treatment: a middle ground proposed due to conflicting studies Studies have yielded mixed results regarding the impact of vitamin D on Systemic Lupus Erythematosus (SLE) activity. Research has shown that patients with SLE often do not have low vitamin D levels, and that vitamin D supplementation may not reduce SLE activity. Moreover, vitamin D levels and responses to treatment vary significantly across different patient populations, depending on factors such as geographic location. A proposed middle ground for using vitamin D in SLE treatment involves administering the supplement to patients with serum levels of 25-hydroxyvitamin D2 plus 25-hydroxyvitamin D3 below 30 ng/ml, with the goal of maintaining levels at or above 30 ng/ml. For patients with major organ involvement, levels should be maintained between 36 and 40 ng/ml. Conversely, patients with serum levels at or above 30 ng/ml do not require vitamin D treatment unless they have significant organ involvement. In such cases, supplementation is recommended to maintain serum levels between 36 and 40 ng/ml. Genetic studies have identified numerous genes associated with SLE, particularly childhood-onset SLE (cSLE). Mutations in these genes can cause cSLE or related disorders, often developing before the age of 18. The most commonly implicated genes include DNASE1L3, TREX1, and IFIH1, among others. These genetic mutations can lead to more severe and potentially life-threatening forms of SLE, characterized by neurological disease, renal failure, and macrophage activation syndrome. Mutations in approximately 40 genes have been linked to cSLE or related disorders, with some genes encoding proteins involved in immune function, such as the complement component 4 protein. The term oligogenic or polygenic inheritance refers to a genetic trait or disorder influenced by the combination of two or more genes. Systemic lupus erythematosus (SLE) is often cited as an example due to its symptom overlap with other autoimmune diseases.[85] Individuals suffering from SLE exhibit higher DNA damage levels than healthy individuals, along with polymorphisms in certain proteins that maintain genomic stability.[86] This raises the likelihood of defective DNA repair contributing to lupus development. In contrast, drug-induced lupus erythematosus is a generally reversible condition triggered by long-term medication use and typically resolves once treatment stops.[87] Although there's no standard diagnosis for drug-induced SLE, researchers agree on criteria including sufficient medication exposure, one symptom compatible with SLE, absence of pre-existing SLE symptoms before treatment, and resolution of symptoms after stopping the medication. Between 1968 and 2017, the VigiBase repository recorded 12,166 cases of drug-induced SLE among 118 causative agents, with five primary classes being most associated: antiarrhythmic, antihypertensive, antimicrobial, interferon inhibitors, and tumor necrosis factor modulators. Discoid lupus, limited to skin symptoms, affects approximately 5% of individuals with this condition and can be diagnosed via skin biopsy. Environmental factors trigger SLE, with the immune system producing antibodies against self-proteins in the cell nucleus, particularly those involved in nuclear protein. This immediate cause leads to chronic inflammation and potentially hypersensitivity responses.[88] People with SLE may exhibit acral pigmentation changes and increased anti-cardiolipin antibody titers or consequences of therapy. Their immune systems display intense polyclonal B-cell activation, favoring immature B cells and memory cells more resistant to immunosuppression, alongside defects in T cell signaling and adhesion.[89] The complement system is linked to SLE, as seen by low C3 levels. Cellular apoptosis is heightened in monocytes and keratinocytes. Fas expression increases on B cells and T cells, while lymphocyte apoptotic rates correlate with disease activity. Necrosis is elevated in T lymphocytes. Tingible body macrophages (TBMs) play a crucial role in germinal centers of secondary lymph nodes. CD68-expressing TBMs engulf apoptotic B cells after somatic hypermutation. In some SLE cases, significantly fewer TBMs are found, and these cells rarely contain material from apoptotic B cells. Uningested apoptotic nuclei can be found outside TBMs, potentially posing a threat to B cell and T cell tolerization. Dendritic cells in germinal centers may endocytose such antigenic material and present it to T cells, activating them. Apoptotic chromatin and nuclei can attach to follicular dendritic cells, making this material available for activating other B cells with self-protein specificity acquired through somatic hypermutation. Necrosis is a pro-inflammatory form of cell death that increases in T lymphocytes due to mitochondrial dysfunction, oxidative stress, and ATP depletion. Clearance deficiency, characterized by deficient phagocytic activity, impaired lysosomal degradation, and scant serum components, contributes to SLE development. Early apoptotic cells express "eat-me" signals, prompting immune cells to engulf them. Apoptotic cells also display "find-me" signals, attracting macrophages and dendritic cells. When apoptotic material is not removed correctly by phagocytes, it can be captured by antigen-presenting cells, leading to the development of antinuclear antibodies. Monocytes isolated from SLE patients show reduced expression of CD44 surface molecules involved in apoptotic cell uptake. TBMs are smaller or scarcer and die earlier in some SLE cases. Serum components like complement factors, CRP, and glycoproteins are crucial for efficient phagocytosis but may be missing, diminished, or inefficient in SLE. Macrophages during SLE fail to mature lysosomes, resulting in impaired degradation of internalized apoptotic debris. This leads to chronic activation of Toll-like receptors and permeabilization of the phagolysosomal membrane, allowing cytosolic sensors activation. Additionally, intact apoptotic debris recycles back to cell membranes and accumulates on cell surfaces. Recent research has found an association between certain SLE patients (especially those with lupus nephritis) and impairment in degrading neutrophil extracellular traps (NETs). DNAse1 inhibiting factors and NET protecting factors play a crucial role in regulating apoptosis, rather than mutations in DNAse1 itself being the primary cause of lupus. The clearance of early apoptotic cells is essential for maintaining immune homeostasis in multicellular organisms. When this process is disrupted, it can lead to an accumulation of apoptotic debris, which stimulates the maturation of dendritic cells and the presentation of autoantigens via MHC molecules. This can result in autoimmune responses as the body's lymphocytes become activated by these autoantigens, leading to inflammation and the production of autoantibodies. In individuals with cutaneous lupus erythematosus (CLE), a deficiency in the clearance of apoptotic cells has been observed in the skin. The germinal centers in people with SLE show abnormalities in the clearance of apoptotic cells, which can lead to an accumulation of debris and stimulate autoreactive B-cells. The tingible body macrophages, responsible for removing apoptotic lymphocytes in healthy conditions, are often ineffective in individuals with SLE. As a result, apoptotic debris accumulates in the germinal centers, attracting follicular dendritic cells that attach antigen material to their surface, potentially serving as a survival signal for autoreactive B-cells. ### Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that may arise from autoreactive B cells, which require additional signals from helper T cells to mature into plasma cells producing autoantibodies and memory cells. In their presence, this may lead to the development of autoimmune diseases. Initially, SLE antibodies targeted specific sequences in nucleoprotein (nRNP), but these targets have since expanded to other areas. Similarities between nRNP and Sm antibodies suggest a common origin for both diseases. Studies have shown elevated levels of high mobility group box 1 (HMGB1) in the blood of people with SLE, a protein involved in chromatin structure and transcriptional regulation. HMGB1 is now recognized as a contributor to the pathogenesis of autoimmune diseases due to its pro-inflammatory properties. Direct immunofluorescence testing, including the lupus band test, is commonly used to detect antinuclear antibodies (ANA) in SLE patients. However, ANA tests are not specific and can be positive in other conditions or normal individuals. Other autoantibodies, such as anti-dsDNA and anti-Smith, are more specific for SLE. Recent research has shown a multianalyte panel (MAP) of antibodies, including ANA, anti-dsDNA, and anti-Smith, to be highly effective in distinguishing SLE from other autoimmune connective tissue diseases, with sensitivity and specificity rates of 80% and 86%, respectively. Patients undergoing testing with either the Mixed Autoantibody Profile (MAP) or traditional Antinuclear Antibody (tANA) strategy have shown a significant association with an increased risk of developing Systemic Lupus Erythematosus (SLE). Those who test MAP positive are 6 times more likely to receive a new SLE diagnosis and 3 times more likely to start a new medication regimen compared to those testing positive with tANA. The anti-dsDNA antibody titers also correlate with disease activity, although not in all cases. Other ANA present in patients with SLE include anti-U1 RNP, SS-A (anti-Ro), and SS-B (anti-La), which are more commonly found in Sjögren's syndrome. SS-A and SS-B can increase the risk of heart conduction block in neonatal lupus. Routine tests performed on suspected SLE patients include complement system levels, electrolytes, kidney function, liver enzymes, and complete blood count. Historically, the lupus erythematosus (LE) cell test was used for diagnosis; however, it is now rarely performed due to its limited sensitivity and specificity, as it can be found in individuals with rheumatoid arthritis, scleroderma, and drug sensitivities. The American College of Rheumatology (ACR) classification criteria were established to classify SLE in clinical trials but are not suitable for diagnosing individuals. SLE Diagnostic Criteria SLE is seen in cases of either immune complex-induced inflammation or congenital complement deficiency, predisposing individuals to SLE. Renal disorder: Presence of >0.5 g/day protein in urine or cellular casts; sensitivity = 51%; specificity = 94%. Antinuclear antibody test positive; sensitivity = 99%; specificity = 49%. Immunologic disorder: Positive anti-Smith, anti-ds DNA, antiphospholipid antibodies; sensitivity = 85%; specificity = 93%. Seizures or psychosis present in 20% of cases; sensitivity = 20%; specificity = 98%. Other symptoms include fever >100°F/37.7°C, extreme fatigue, hair loss, and Raynaud syndrome. SLE may also be diagnosed without four of the above criteria and can present with additional features not listed. Recursive partitioning has identified simpler diagnostic criteria: SLE diagnosis requires an immunologic disorder or malar rash; sensitivity = 92%; specificity = 92%. Alternative criteria exist, such as the St. Thomas' Hospital "alternative" criteria in 1998. Treatment involves preventing flares and reducing severity, with corticosteroids, anti-malarial drugs, and cyclophosphamide used to treat specific types of lupus nephritis. Medications like hydroxychloroquine, belimumab (Benlysta), and disease-modifying antirheumatic drugs (DMARDs) are also used. Due to SLE's varied symptoms and organ involvement, individual severity must be assessed for effective treatment. Mild or remittent disease may be left untreated, while nonsteroidal anti-inflammatory drugs and antimalarials can be used as needed. The management of systemic lupus erythematosus (SLE) often involves a combination of treatments to control symptoms and prevent flares. When SLE progresses or worsens, corticosteroids are used to manage the condition. Medications like hydroxychloroquine, an antimalarial drug, are commonly prescribed for their efficacy in reducing symptoms and improving survival rates among people with SLE. However, long-term use of these medications can lead to side effects such as elevated blood pressure and cataracts. In severe cases, immunosuppressive drugs like methotrexate and azathioprine may be used to control the disease. These medications have been linked to an increased risk of serious infections, including those associated with newer biological therapies. Other treatments, such as mycophenolic acid and rituximab, are being actively tested for their effectiveness in managing SLE symptoms. The use of steroids can also lead to Cushing's syndrome, a condition characterized by obesity, facial puffiness, and elevated blood pressure. In some cases, the risk of cataracts and osteoporosis may also be increased. As a result, patients who require long-term steroid treatment must carefully monitor their health and adhere to regular check-ups with their healthcare provider. Given article text here sunlight in SLE is critical as it can worsen the skin symptoms of the disease.[133] People with SLE should avoid activities that cause fatigue since they easily get tired and it can be very debilitating. This can lead to them being housebound for a long time. Exercise has been shown to help improve fatigue in adults with SLE.[133] Certain drugs should not be prescribed unless known not to make the disease worse. Exposure to silica, pesticides, and mercury at work can also worsen the disease.[85] An international task force of clinicians and patients have developed recommendations for evidence-based non-pharmacological treatments for SLE.[133] For people with end-stage kidney disease, which is a complication of lupus nephritis, kidney transplants are often the best option. However, the disease can come back in up to 30% of cases.[134] About 20% of people with SLE have high levels of antiphospholipid antibodies, which are associated with antiphospholipid syndrome.[135] This form of the disease is different from lupus and causes blood clots to form in blood vessels, leading to potential brain damage if they move within the bloodstream.[114] In this case, brain scans may be required for early detection. Treatment usually involves taking anticoagulants like aspirin or warfarin. When it comes to pregnancy, mothers with SLE should stay under medical care until delivery, as the disease can pose a higher risk of complications for their baby.[137] However, with proper treatment, the health of both mother and baby can be maintained. Women with SLE who have anti-Ro or anti-La antibodies often need regular heart checks during pregnancy.[138] It's also recommended that they use reliable forms of contraception to prevent pregnancy, as getting pregnant while actively ill can be harmful.[139] Lupus nephritis, gestational diabetes, and pre-eclampsia are common complications of SLE in pregnancy.[137] Unfortunately, there is currently no cure for SLE, but many treatments are available. In the 1950s, people with SLE typically lived fewer than five years, but today over 90% survive for more than ten years, and many live relatively healthy lives. Around 80-90% of people with Systemic Lupus Erythematosus (SLE) can live a normal life span, but their mortality rates are higher than those without the condition. Men and children tend to have a poorer prognosis compared to women, but if symptoms appear after age 60, the disease tends to be less severe. Early death from SLE is often caused by organ failure or infections that could be prevented with early diagnosis and treatment. People with SLE are five times more likely to die from cardiovascular disease in late stages due to accelerated atherosclerosis. To reduce this risk, it's essential to manage high blood pressure and cholesterol levels aggressively. To prevent long-term complications, steroids should be used at the lowest dose for the shortest time possible, and other medications that alleviate symptoms should be utilized when necessary. SLE affects approximately 20-70 people per 100,000 worldwide, with a higher incidence among women aged 45-64. The lowest rate of SLE is found in Iceland and Japan, while the highest rates exist in the US and France. However, there isn't enough evidence to determine why SLE is less common in some countries compared to others. It could be due to environmental factors such as varying levels of sunlight exposure affecting dermatological symptoms. Research suggests a possible genetic connection between race and lupus that influences disease prevalence. Studying populations with homogenous racial compositions might help understand the incidence of SLE better. The rate of disease in developing countries is unclear, but it varies significantly between countries, ethnicity, and sex. In the US, estimates range from 53 to over 305 per 100,000 people. SLE occurs more frequently and severely among those of non-European descent. Childhood-onset SLE typically presents between ages 3-15 and is four times more common in girls. Socioeconomic status also plays a significant role, with women from lower socioeconomic backgrounds experiencing higher depression scores, restricted access to medical care, and more self-reported anxiety and depression compared to their counterparts from higher socioeconomic statuses. The connection between race and systemic lupus erythematosus (SLE) may be tenuous. Social support can buffer against SLE-related damage, but studies have not examined whether people from different racial backgrounds receive varying levels of social support, which could confound the relationship between race and SLE. Moreover, self-reported symptom data is susceptible to methodological errors, influenced by factors like social support, helplessness, and abnormal illness behaviors. Socioeconomic status (SES), health insurance, and access to care can also impact disease progression. Studies controlling for SES have found that non-white individuals experience more abrupt disease onset and faster progression, but this may be due to differences in socioeconomic conditions rather than race itself. The severity of symptoms and mortality rates are similar between white and non-white patients. SLE disproportionately affects females, with a ratio of 9:1, potentially due to hormonal mechanisms involving estrogen and androgens. Genetic influences on the X chromosome, such as CD40L mutations or silencing escape, may also contribute to SLE development. A study found an association between Klinefelter syndrome (XY sex chromosomes) and SLE in males with an abnormal X–Y translocation, while research has implicated XIST, a long non-coding RNA involved in X-chromosome inactivation. Further investigation is needed to disentangle the complex relationships between race, socioeconomic factors, hormonal mechanisms, and genetic influences on SLE development and progression. The history of Systemic Lupus Erythematosus (SLE) can be divided into three periods: classical, neoclassical, and modern. The disease was initially considered a non-fatal skin condition in the past, but its classification as an autoimmune disorder in 1851 led to advancements in diagnosis and treatment. Research and documentation progressed throughout each period, ultimately improving life expectancy for those diagnosed with SLE. The term "lupus" originated from Latin, meaning "wolf", due to the red, butterfly-shaped rash that characterizes the disease. The classical period began during the Middle Ages when Italian physician Rogerius Frugard described ulcerating sores on patients' legs. The neoclassical period started in 1851 with Pierre Cazenave's documentation of discoid lupus and subsequent detailed notes to aid diagnosis. Research continued, including observations by Ferdinand von Hebra and Moritz Kaposi, who documented the disease's chronic nature and potential for dormancy. Kaposi's observations noted patients afflicted with the "butterfly rash" often suffered from additional conditions such as tuberculosis, anemia, or chlorisis which frequently proved fatal.[160] Kaposi was among the first to document the remitting and relapsing nature of what is now termed systemic lupus erythematosus, detailing its correlation between skin and systemic manifestations during disease activity.[163] Research into lupus continued in the 19th century with Sir William Osler's work expanding knowledge on internal complications of various diseases including lupus, highlighting that many people with lupus experienced organ damage beyond just skin issues.[160] Notably, Osler added "systemic" to distinguish this type from discoid lupus erythematosus due to its far-reaching impact on numerous bodily organs. His papers noted reoccurrence as a characteristic feature of the disease and described severe afflictions such as arthritis, pneumonia, and central nervous system damage affecting patients diagnosed with SLE.[160] The 1920s saw significant advancements in understanding lupus' cause and treatment, including detailed pathologic descriptions and demonstrations of its effects on kidney, heart, and lung tissue.[165] A major breakthrough occurred in 1948 with the discovery of the LE cell, a white blood cell containing another cell's nucleus due to an antibody-coated invading nucleus. The LE cell test was introduced as one of the first definitive tests for lupus, though its use has since diminished due to variable presence and occurrence in individuals with other autoimmune diseases.[168] Given article text here this resulted in more definitive tests for lupus. Building on the knowledge that those with SLE had auto-antibodies that would attach themselves to the nuclei of normal cells, causing the immune system to send white blood cells to fight off these "invaders", a test was developed to look for the anti-nuclear antibody (ANA) rather than the LE cell specifically. This ANA test was easier to perform and led not only to a definitive diagnosis of lupus but also many other related diseases. This discovery led to the understanding of what is now known as autoimmune diseases. To ensure that the person has lupus and not another autoimmune disease, the American College of Rheumatology established a list of clinical and immunologic criteria that, in any combination, point to SLE. The criteria include symptoms that the person can identify and things that a physician can detect through laboratory test results. Over time, medical historians have found that people with porphyria may have shared similar stories of illness, such as folklore about vampires and werewolves. Medication for lupus was first discovered in 1894 when quinine was reported to be effective, followed by the use of salicylates four years later. Later, corticosteroids were found to be more effective treatments until the mid-20th century. Recent studies have tested new drugs like belimumab and genetically engineered immune cells as potential treatments for lupus. In 2022, researchers at the University of Erlangen-Nuremberg reported promising results using CAR T cells to treat severely ill patients with lupus, achieving remission in five patients who had been off medication for several months afterwards. **Systemic Lupus Erythematosus** Lupus, also known as systemic lupus erythematosus (SLE), is a chronic autoimmune disease that affects multiple organs and systems in the body. The exact cause of lupus is unknown, but it is believed to be triggered by a combination of genetic and environmental factors. **Prevalence and Epidemiology** Lupus is more common in women than men, and its prevalence varies depending on the population being studied. According to some estimates, approximately 5 million people worldwide have lupus, with a higher incidence in Africa and Asia. The disease typically affects adults between the ages of 20 and 50, but it can also occur in children. **Symptoms** The symptoms of lupus can vary widely from person to person, but common complaints include fever, fatigue, joint pain, skin rashes, and kidney problems. In some cases, lupus can cause more severe complications, such as seizures, strokes, and organ failure. **Childhood-Onset Systemic Lupus Erythematosus** Research has shown that childhood-onset SLE (cSLE) is a distinct entity from adult-onset SLE. Children with cSLE tend to have more severe disease and are at higher risk of developing complications such as kidney damage. **Treatment and Management** While there is no cure for lupus, treatment options include medications that can help manage symptoms and slow disease progression. These may include immunosuppressants, corticosteroids, and biologics. Researchers have also explored the use of immunotherapy to induce long-lasting remission in some patients. **Impact and Complications** Lupus has a significant impact on quality of life, with many people experiencing chronic fatigue, depression, and anxiety. In addition, lupus can cause severe complications such as kidney damage, heart problems, and even death. Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that affects adults. It can cause joint and muscle pain, osteoarticular tuberculosis, and other complications such as rheumatoid arthritis, vertebral fractures, anemia, and neuropsychiatric symptoms. Research has shown that SLE can increase the risk of developing cardiovascular disease, including accelerated atherosclerosis and pulmonary hypertension. Treatment with steroids is often used to manage symptoms, but the disease can be unpredictable and require ongoing monitoring. One specific complication of SLE is "shrinking lung syndrome," which is characterized by shortness of breath and chest pain. Other respiratory complications include pleuritis and pulmonary embolism. SLE can also affect the nervous system, leading to neuropsychiatric symptoms such as headaches, seizures, and depression. In some cases, patients may experience false-positive serological test results for syphilis or other diseases due to the presence of anti-phospholipid antibodies. Overall, SLE is a complex and multifaceted disease that requires careful management and monitoring to minimize complications and improve patient outcomes. Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that can affect various parts of the body. This review article examines the current understanding of SLE and its neurological manifestations. Research suggests that SLE affects approximately 1 in 1,000 people worldwide, with women being more likely to develop the condition than men. The disease can cause inflammation in various organs, including the kidneys, brain, and nervous system, leading to a range of symptoms such as fatigue, joint pain, and cognitive impairment. A study published in Seminars in Arthritis and Rheumatism found that SLE is associated with an increased risk of developing kidney damage, which can lead to end-stage renal disease. Another study published in The Journal of Rheumatology found that treatment for lupus nephritis (kidney inflammation) has improved over the years, but more research is needed to develop effective therapies. The article also discusses neuropsychiatric manifestations of SLE, including cognitive impairment, seizures, and psychosis. A study published in Arthritis & Rheumatology found that approximately 70% of patients with SLE experience neuropsychiatric symptoms, which can be debilitating and affect daily life. Finally, the review highlights the importance of early diagnosis and treatment of SLE to prevent long-term damage and improve quality of life for affected individuals. A study published in Rheumatology International in May 2012, found that patients with systemic lupus erythematosus (SLE) have a high prevalence of depression and depressive symptoms. The study's findings suggest that SLE patients experience a range of psychological and emotional difficulties. Systemic lupus erythematosus can also affect the eyes, as noted in a review published in Clinical and Experimental Medicine in May 2018. This condition can lead to various eye problems, including dryness, inflammation, and even vision loss. Pregnancy in SLE patients was studied by Clowse ME in Rheumatic Disease Clinics of North America in May 2007. The study highlighted the importance of careful management during pregnancy to prevent complications for both mother and baby. A systematic review and meta-analysis published in Clinical Journal of the American Society of Nephrology in November 2010, analyzed pregnancy outcomes in patients with SLE and lupus nephritis. The findings suggested that these women face increased risks during pregnancy. The Lupus Foundation of America notes that SLE can impact a woman's reproductive health. Women with SLE are at higher risk for preterm birth and other complications during pregnancy. A study published in Rheumatology in June 2002, found that certain clinical factors can predict fetal and maternal outcomes in SLE patients. The researchers identified several key predictors of outcome. Thefreedictionary.com defines neonatal lupus as a rare condition affecting newborns whose mothers have SLE. A review article published in Lupus Foundation of America highlights the connection between SLE and women's reproductive health. A study on methotrexate, a medication used to treat SLE, notes that it can cause side effects such as nausea, vomiting, and fatigue. Systemic lupus erythematosus is described by D'Cruz DP in BMJ as a complex autoimmune disease with diverse manifestations. The article emphasizes the importance of proper diagnosis and treatment to manage the condition effectively. A study on fatigue in SLE patients published in The Journal of Rheumatology found that it was a common symptom, often associated with depression, pain, and decreased social support. Research on vitamin D's role in SLE suggests that it may influence disease activity and autoimmunity, as reported in various studies including those by Schneider L, Irfan SA, and Ho LJ. A recent review published in The Journal of Rheumatology discusses autoinflammatory diseases, including their causes and effects. Another study published in Current Issues in Molecular Biology found that genetic risk factors play a significant role in the development of childhood-onset systemic lupus erythematosus (SLE). Research published in The Journal of Allergy and Clinical Immunology suggests that genetic variants associated with primary immunodeficiencies also contribute to childhood-onset SLE. Inborn errors of immunity are classified into different categories based on their clinical features, according to the International Union of Immunological Sciences. A study published in Current Opinion in Pediatrics provides an update on new primary immunodeficiencies. Research published in Science Immunology discovered that variants of the UNC93B1 gene can cause TLR7-dependent autoimmunity and lead to conditions like SLE and chilblain lupus. Another study found that gain-of-function human UNC93B1 variants also cause systemic lupus erythematosus and chilblain lupus. A previous study published in Advances in Rheumatology explored the impact of C4, C4A, and C4B gene copy number variation on the susceptibility, phenotype, and progression of SLE. A high-throughput genomic sequence analysis tool called C4Investigator was also developed to analyze complement component 4 genes. Research published in Genes and Immunity found that population differences exist in the genetic risk factors for SLE, with STAT4 and BLK being associated with the condition in Hong Kong Chinese populations. The articles also discuss polygenic inheritance, GWAS (genome-wide association studies), polygenic risk scores, and the search for functional variants. **Systemic Lupus Erythematosus (SLE)** SLE is a chronic autoimmune disease characterized by inflammation and damage to various organs, including the skin, joints, kidneys, and nervous system. The exact mechanisms underlying SLE are not fully understood, but research suggests that DNA damage and repair deficiencies play a crucial role in its pathogenesis. **DNA Damage and Repair** Studies have shown that individuals with SLE often exhibit defects in DNA repair mechanisms, leading to accumulation of genetic mutations and chromosomal instability. This, in turn, can trigger autoimmune responses against self-antigens, contributing to the development of SLE. **Genetic Associations** Recent studies have identified several genetic associations with SLE, including variants in genes involved in complement activation (C8orf13-BLK) and immune regulation (ITGAM-ITGAX). These findings suggest that genetic predisposition plays a significant role in the susceptibility to SLE. **Immunological Aspects** Research has also highlighted the importance of immunological dysregulation in the pathogenesis of SLE. Studies have shown that individuals with SLE often exhibit impaired clearance of apoptotic cells, leading to the accumulation of dying cells and the release of self-antigens. This can trigger autoimmune responses against these antigens, perpetuating the disease. **Complement System** The complement system, which plays a critical role in innate immunity, has been implicated in SLE. Abnormalities in complement activation have been observed in individuals with SLE, suggesting that dysregulation of this system contributes to the disease's pathogenesis. **Clinical Manifestations** SLE is characterized by a wide range of clinical manifestations, including skin rashes (discoid lupus erythematosus), joint inflammation (arthritis), kidney damage (nephritis), and neurological symptoms. The severity and progression of SLE can vary significantly between individuals, making it essential to develop personalized treatment strategies. **Treatment** While there is no cure for SLE, various treatments are available to manage its symptoms and prevent disease progression. These include corticosteroids, immunosuppressive agents (e.g., cyclophosphamide), and biologics (e.g., rituximab). Researchers continue to explore new therapeutic strategies and potential biomarkers for SLE. I tried to preserve the main ideas and findings from the original text while rephrasing it in a more concise and accessible manner. Systemic lupus erythematosus (SLE) studies reveal mitochondrial hyperpolarization and ATP depletion in patients, as well as clearance deficiency and impaired apoptotic cell uptake into tingible body macrophages. Research also highlights defects in lysosomal maturation facilitating the activation of innate sensors in SLE, while apoptotic debris accumulates on hematopoietic cells and promotes disease. Impairment of neutrophil extracellular trap degradation is associated with lupus nephritis, and mutation of DNASE1 in people with SLE has been found. Additionally, studies have identified high mobility group box 1 as a potential therapeutic target for SLE, and T cells play a crucial role in the disease process. The European League Against Rheumatism and the American College of Rheumatology have established updated classification criteria for Systemic Lupus Erythematosus (SLE) in 2019. These criteria aim to improve diagnosis and treatment of the disease. Research on SLE has led to the development of various diagnostic tests, including the lupus band test and the LE cell test. The lupus band test detects the presence of immune complexes that form during SLE attacks, while the LE cell test measures the ability of white blood cells to respond to these complexes. Studies have also explored the role of complement activation products in SLE diagnosis and treatment. Complement activation is a key process involved in SLE pathogenesis, and measuring its levels can help diagnose the disease. Other research has focused on the development of diagnostic criteria for SLE, including the use of patient-specific autoantibodies and serum complement levels. The 2019 classification criteria take into account these factors to provide a more accurate diagnosis of SLE. In addition, studies have investigated the impact of SLE on pregnancy and congenital heart block in newborns. Maternal autoantibodies have been shown to be associated with an increased risk of congenital heart block, highlighting the importance of monitoring and treatment for pregnant women with SLE. The text also mentions various online resources and articles that provide information on SLE diagnosis, classification criteria, and treatment options. These resources aim to educate healthcare professionals and patients about the disease and its management. Overall, the research and updates in this field have aimed to improve our understanding of SLE, its diagnostic challenges, and effective treatment strategies for patients with this complex autoimmune disease. **Systemic Lupus Erythematosus (SLE) Research and Treatment** Several studies have investigated the classification criteria, treatment guidelines, and outcomes of patients with SLE. In 2003, an international consensus statement was published on the classification criteria for catastrophic antiphospholipid syndrome. Research has also explored the association between livedo reticularis (a skin condition) and pregnancy morbidity in patients without antiphospholipid antibodies. Additionally, a study found that seronegative patients with SLE had similar clinical manifestations to those with positive tests for antiphospholipid antibodies. **Treatment Options** Researchers have investigated various treatments for SLE, including: * Cyclophosphamide and methylprednisolone for neuropsychiatric involvement * BAFF inhibitors as a potential therapeutic target * New biological treatments such as rituximab and belimumab, which have shown promise in improving outcomes **Economic and Healthcare Burden** A study estimated the healthcare costs of SLE in the United States, finding that patients with severe disease incur significantly higher costs. Another study found that tacrolimus use in lupus nephritis was associated with improved outcomes. **Safety Concerns** The FDA has issued warnings about the potential for increased infection risk with certain treatments, including mycophenolate mofetil and rituximab. Overall, research into SLE continues to uncover new insights into its classification, treatment, and economic burden. Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that affects millions of people worldwide. Research has shown that certain risk factors are associated with an increased likelihood of developing cataracts in SLE patients. Additionally, studies have investigated the effectiveness of various treatments for managing symptoms of SLE and rheumatoid arthritis. The National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) provides information on SLE, including its causes, symptoms, and treatment options. The NIAMS also offers guidelines for non-pharmacological management of SLE and systemic sclerosis, which include lifestyle changes and alternative therapies. Furthermore, research has explored the impact of pregnancy on women with SLE, as well as the role of antiphospholipid antibodies in the disease. Some studies have found that certain contraceptive methods may be safer for women with SLE than others. Overall, the references provided highlight the complexities of SLE and the need for continued research into its causes, symptoms, and treatment options. **Lupus Erythematosus Overview** * Despite improvements in treatment over the last decade, systemic lupus erythematosus (SLE) remains a serious and potentially life-threatening autoimmune disease. * SLE disproportionately affects women, particularly those between 15-44 years old. * The disease is characterized by inflammation of various organs and tissues, including the skin, joints, kidneys, brain, and other parts of the body. * Research suggests that socioeconomic status may play a role in the development and progression of SLE. **Epidemiology** * According to estimates, approximately 5 million people worldwide have SLE, with women being affected at least 9 times more often than men. * The disease is more common among individuals of African American, Hispanic, and Asian descent. * Studies suggest that genetic factors, particularly those related to the X chromosome, may contribute to the increased risk of developing SLE in women. **History of Lupus** * The term "lupus" comes from Latin, meaning "wolf," due to the skin rash often associated with the disease resembling a wolf's bite. * Historical accounts suggest that lupus erythematosus has been described as far back as ancient Greece and Rome. * In modern times, SLE was first recognized in the early 20th century. **Symptoms and Treatment** * The symptoms of SLE can vary widely depending on the affected organs and tissues. * Common symptoms include fatigue, joint pain, skin rashes, kidney damage, and cognitive impairment. * Treatment options for SLE often involve a combination of medications, lifestyle modifications, and alternative therapies. **References** The text is a compilation of references from various medical journals, articles, and sources, citing research on the epidemiology, symptoms, treatment, and history of systemic lupus erythematosus. This guide provides comprehensive information on lupus, a chronic autoimmune disease that affects millions of people worldwide. It covers historical background, diagnosis, treatment options, and coping strategies for patients and families affected by systemic lupus erythematosus (SLE). The text references various sources including medical journals, books, and online articles. It also touches on the history of lupus, from ancient times to modern-day research and treatments. The guide includes information on immunosuppressants, medications, and alternative therapies that can help alleviate symptoms. Additionally, it highlights notable cases of people living with lupus, including Olympic athlete Shannon Boxx and pop star Selena Gomez, who have used their platforms to raise awareness about the disease. The text also mentions recent breakthroughs in treating lupus, such as genetically engineered immune cells that have shown promising results in mice, and anti-CD19 CAR T cell therapy for refractory SLE. Overall, this guide aims to provide a comprehensive overview of lupus, its effects on patients and families, and the latest research and treatment options available. Lupus is a chronic autoimmune disease that can lead to significant health issues, including the need for a kidney transplant.