

Lupus Treatment and Diagnosis

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Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease in which immune activity can injure the skin, joints, blood cells, kidneys, nervous system, lungs, heart, and blood vessels. Its presentation is rarely identical from one person to another. Symptoms may appear in clusters, improve for months or years, and then return during a flare. That uneven course helps explain why a patient can receive several plausible explanations before lupus is recognized. It also means that no single symptom or laboratory result should be treated as proof of the disease.

Patient B's history illustrates both the difficulty of diagnosis and the importance of reviewing symptoms over time. She developed fatigue, palpitations, and anxiety after the birth of her first child. Later episodes included abdominal pain, pregnancy-related complications, swollen and painful joints, edema, cold-induced discoloration of the hands and feet, anemia, thrombocytopenia, an elevated erythrocyte sedimentation rate, a strongly positive antinuclear antibody test, elevated anti-double-stranded DNA antibodies, and small-vessel vasculitis on skin biopsy. Taken together, these findings are much more informative than any one result viewed in isolation. The original account, however, contains several factual problems. In particular, an ANA titer of 1:640 is not a normal result, platelet levels of $138,000/\text{mm}^3$ are mildly low rather than normal, and modern lupus care is not based on aspirin, acetaminophen, ibuprofen, and corticosteroids alone. A current evaluation must connect the patient's clinical pattern with accepted diagnostic reasoning and contemporary treatment guidance.

Clinical History and Pattern of Illness

Patient B was 20 years old when she first reported fatigue, heart palpitations, and anxiety soon after childbirth. Those symptoms are nonspecific. Sleep deprivation, postpartum anxiety, anemia, thyroid disease, infection, and the physical demands of caring for a newborn can produce a similar picture. The first physician's advice to rest was therefore understandable as an initial response, but the worsening and recurrence of symptoms should have prompted reassessment. A diagnosis based only on the assumption of postpartum stress risks missing a condition that is developing gradually.

Two years later, severe abdominal pain was attributed to gallbladder disease. That explanation may have been correct, unrelated, or only part of the problem. Abdominal pain is not specific enough to identify lupus, and it would be inappropriate to retroactively label every earlier illness as a manifestation of SLE. Even so, the recurrence of unexplained symptoms should have been documented because lupus diagnosis depends heavily on the complete timeline. The National Institute of Arthritis and Musculoskeletal and Skin Diseases notes that clinicians may need to review

symptoms that are no longer present because lupus manifestations often come and go.

Five years after the first episode, Patient B became pregnant again. At six months, she experienced marked fatigue, headaches, premature contractions, and leg swelling. Pregnancy and the postpartum period can alter autoimmune disease activity. Swelling alone may be physiological, but persistent edema accompanied by headache, hypertension, urinary protein, blood-count abnormalities, or impaired kidney function would require investigation for preeclampsia, lupus nephritis, antiphospholipid syndrome, or another medical disorder.

After delivery, Patient B developed swelling in both knees and ankles and had difficulty dancing and climbing stairs. This pattern suggests inflammatory musculoskeletal disease rather than simple fatigue. During winter, painful discoloration of the fingers and toes appeared with cold exposure. That description is compatible with Raynaud phenomenon, in which blood vessels constrict excessively in response to cold or emotional stress. Raynaud phenomenon can occur by itself, but it is also associated with lupus and other connective-tissue diseases. The combination of inflammatory joint symptoms, vascular color changes, edema, and recurrent constitutional complaints made referral to a rheumatologist appropriate.

Interpreting the Diagnostic Evidence

Lupus cannot be diagnosed by one blood test. Clinical history, physical examination, laboratory findings, urinalysis, and sometimes tissue biopsy must be considered together. The 2019 European League Against Rheumatism/American College of Rheumatology classification criteria use a positive ANA at least once as an entry requirement, followed by weighted clinical and immunologic findings. These criteria were created primarily for consistent classification in research; they support, but do not replace, a clinician's diagnosis.

Antinuclear and Disease-Specific Antibodies

The original case reports an ANA "at 1:640," which is best interpreted as a titer of 1:640. Calling this result normal is incorrect. A titer of 1:640 is strongly positive in most laboratory systems, although the meaning still depends on the staining pattern, the testing method, the laboratory reference range, medications, infections, and the patient's symptoms. A positive ANA is sensitive for SLE but not specific. Healthy people and patients with other conditions can also have positive results, so the test should never be used alone to confirm lupus.

Patient B's elevated anti-double-stranded DNA antibody result is more supportive of SLE, especially when it appears with compatible clinical features. Anti-Smith antibodies, antiphospholipid antibodies, and complement levels such as C3 and C4 would also be useful. Low complement can reflect immune-complex activity, while antiphospholipid antibodies matter because they can increase the risk of thrombosis and pregnancy complications. The old lupus erythematosus cell preparation mentioned in

the case is historically important but is not part of modern routine diagnosis; contemporary serologic testing is more informative.

Blood Counts, Inflammation, Skin Biopsy, and Kidney Screening

The complete blood count showed a red blood cell count of 3.8 million/mm³, hematocrit of 35%, and hemoglobin of 10.5 g/dL. These findings indicate anemia, but they do not reveal its cause. Iron deficiency after pregnancy, chronic inflammation, kidney disease, vitamin deficiency, medication effects, blood loss, and autoimmune hemolysis are all possibilities. Reticulocyte count, iron studies, bilirubin, lactate dehydrogenase, haptoglobin, and a direct antiglobulin test could help distinguish them.

The platelet count of 138,000/mm³ is mildly below the commonly used lower reference limit of 150,000/mm³; it should not have been described as normal. The white blood cell count of 6,000/mm³ is within a typical adult range. An erythrocyte sedimentation rate of 62 mm/hour is elevated and indicates inflammation, but it is nonspecific. Infection, anemia, pregnancy, autoimmune disease, and other conditions can raise the sedimentation rate. C-reactive protein, clinical examination, and the broader laboratory picture would help interpret it.

The skin biopsy showing small-vessel vasculitis strengthens the case for systemic inflammatory disease, particularly when paired with positive lupus serology. Still, vasculitis has many causes, including infections, medications, other autoimmune disorders, and primary vasculitic syndromes. The biopsy result therefore belongs within a differential diagnosis rather than functioning as independent proof of SLE.

Kidney assessment is a major omission from the original narrative. Bilateral edema may reflect venous, cardiac, hepatic, pregnancy-related, medication-related, or renal causes. In a patient being evaluated for lupus, clinicians should check blood pressure, serum creatinine and estimated glomerular filtration rate, urinalysis, urine sediment, and urine protein. Current American College of Rheumatology guidance recommends periodic proteinuria screening in people with SLE who do not have known kidney disease, with more urgent evaluation when renal abnormalities appear. A kidney biopsy may be needed when proteinuria or otherwise unexplained impaired kidney function suggests lupus nephritis.

Differential Diagnosis and the Reason for Delay

The long path to diagnosis is clinically believable because many of Patient B's early symptoms overlap with common conditions. Postpartum thyroiditis can cause palpitations, anxiety, fatigue, and later sluggishness. Iron-deficiency anemia can cause fatigue, headaches, palpitations, and reduced

exercise tolerance. [Rheumatoid arthritis](#), mixed connective-tissue disease, systemic sclerosis, antiphospholipid syndrome, autoimmune vasculitis, viral illness, and medication reactions can reproduce portions of the picture. Preeclampsia or another hypertensive pregnancy disorder would also need consideration during the second pregnancy.

A careful rheumatology assessment would ask when each symptom began, how long it lasted, and whether it was linked to sun exposure, infection, pregnancy, or medication. Family history, ulcers, hair loss, photosensitive rash, pleuritic pain, neurologic symptoms, pregnancy losses, thrombosis, urinary changes, and fever would be relevant. The aim is not to force every complaint into a lupus diagnosis, but to identify a coherent immune-mediated pattern after reasonable alternatives have been investigated.

In Patient B's case, the combination of a strongly positive ANA, elevated anti-double-stranded DNA antibodies, inflammatory joint symptoms, cold-induced vascular symptoms, mild cytopenias, and biopsy-proven small-vessel vasculitis makes SLE a persuasive diagnosis. The conclusion should nevertheless be stated with appropriate clinical caution: the exact classification score cannot be calculated from the information provided, and renal, complement, antiphospholipid, and other data are missing.

Current Principles of Lupus Treatment

The purpose of treatment is to control active inflammation, prevent flares and irreversible organ damage, minimize medication toxicity, and protect quality of life. Therapy must be matched to disease severity and the organs involved. A person with mild skin and joint disease does not require the same regimen as someone with nephritis, central nervous system disease, severe vasculitis, or life-threatening blood abnormalities.

Foundational and Symptom-Directed Therapy

Hydroxychloroquine is now considered foundational therapy for most people with SLE unless it is contraindicated. The 2025 ACR treatment guideline recommends its routine use and emphasizes a long-term average dose that limits retinal toxicity. Eye screening is necessary, and dosing should be individualized rather than copied from a fixed template. Hydroxychloroquine can reduce disease activity and flares, but it is not an instant pain reliever; benefits may take time to become evident.

Nonsteroidal anti-inflammatory drugs, including naproxen or ibuprofen, may help selected patients with short episodes of musculoskeletal pain or inflammation. They are not appropriate for everyone, particularly when kidney disease, gastrointestinal bleeding risk, cardiovascular risk, anticoagulant use, or pregnancy creates additional concerns. Acetaminophen can reduce pain and fever but does not treat the autoimmune inflammation itself. Aspirin is not a universal lupus treatment. It may be indicated in specific situations, such as certain antiphospholipid-antibody or cardiovascular-risk

scenarios, but routine use without a defined reason can create bleeding and gastrointestinal risks.

Glucocorticoids, Immunosuppressive Medicines, and Biologics

Corticosteroids can rapidly suppress active inflammation, but prolonged exposure can cause infection, osteoporosis, diabetes, hypertension, cataracts, weight changes, cardiovascular harm, and other complications. Current ACR guidance favors the lowest effective dose for the shortest practical duration and encourages tapering maintenance prednisone to 5 mg per day or less, ideally to zero, when disease control permits. Steroids should therefore be viewed as a bridge or emergency tool rather than the sole long-term plan.

When lupus remains active or threatens an organ, clinicians may introduce medicines such as methotrexate, azathioprine, mycophenolate, calcineurin inhibitors, or cyclophosphamide, depending on the manifestation. Biologic therapies such as belimumab and anifrolumab are also part of modern treatment for appropriate patients. Rituximab may be considered in selected refractory or organ-threatening situations even though its role differs across guidelines and clinical circumstances. The choice must account for disease severity, pregnancy plans, infection risk, prior treatment, access, monitoring requirements, and the patient's preferences.

If Patient B's vasculitis were limited to mild skin disease, the treatment approach could differ substantially from therapy for systemic vasculitis affecting the kidneys, gastrointestinal tract, lungs, or nervous system. Likewise, if edema were linked to lupus nephritis, renal treatment would require coordinated rheumatology and nephrology care, urine-protein monitoring, and an immunosuppressive regimen appropriate to the biopsy class and severity. Modern lupus-nephritis guidance increasingly uses combination therapy rather than relying on corticosteroids alone.

Nursing Care, Self-Management, and Follow-Up

Nursing care should extend beyond the labels "acute pain" and "impaired skin integrity." Patient B would benefit from repeated assessment of pain, fatigue, mobility, edema, skin lesions, blood pressure, medication adherence, and signs of infection or organ involvement. Practical interventions include pacing activities during flares, preserving joint movement without forcing exercise through severe inflammation, protecting damaged skin, and escalating new chest pain, shortness of breath, neurologic symptoms, reduced urine output, or rapidly worsening swelling.

Sun protection matters because ultraviolet exposure can trigger skin disease and sometimes systemic activity. Cold avoidance and layered gloves or socks may reduce Raynaud symptoms. Smoking cessation, regular physical activity adapted to disease status, cardiovascular-risk management, vaccination planning, and bone protection are also relevant. These measures complement

medication; they do not replace it. Patients should be warned against stopping prescribed therapy suddenly or adding herbal products without checking for interactions.

Because Patient B's major flares appeared around pregnancy and childbirth, future reproductive planning should involve her rheumatologist and obstetric team before conception whenever possible. Disease activity, kidney function, antiphospholipid antibodies, blood pressure, and medication safety should be reviewed. Some lupus medicines are compatible with pregnancy, while others must be changed well in advance. Shared planning can lower avoidable risk without treating pregnancy itself as an illness.

Follow-up should measure more than whether pain has improved. Disease activity and accumulated damage need regular review, along with blood counts, kidney function, urine protein, liver tests when relevant, medication toxicity, cardiovascular health, bone health, and eye screening for hydroxychloroquine. A patient may feel better while silent renal or hematologic abnormalities persist, so symptoms and objective monitoring must be interpreted together.

Conclusion

Patient B's history is consistent with a delayed recognition of systemic lupus erythematosus, but the diagnosis should rest on the complete clinical pattern rather than a single positive test. The most important corrections are that an ANA titer of 1:640 is strongly positive rather than normal, a platelet count of $138,000/\text{mm}^3$ is mildly reduced, an elevated sedimentation rate is nonspecific, and the obsolete LE cell preparation should not guide modern diagnosis. Her anti-double-stranded DNA antibodies, inflammatory joint symptoms, Raynaud-like episodes, anemia, mild thrombocytopenia, and small-vessel vasculitis provide meaningful support for SLE, while missing kidney, complement, and antiphospholipid data limit certainty.

Treatment has also advanced beyond a simple list of pain medicines and corticosteroids. Most patients now receive hydroxychloroquine unless contraindicated, glucocorticoid exposure is deliberately minimized, and conventional or biologic immunosuppressive therapy is introduced according to organ involvement and severity. For Patient B, an effective plan would combine rheumatology-led treatment with renal screening, reproductive counseling, nursing support, medication-safety monitoring, and long-term follow-up. The central lesson is not merely that lupus can be difficult to diagnose. It is that recurring symptoms become clinically meaningful when they are recorded, compared over time, and interpreted with accurate laboratory knowledge.

References

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