AAVBC

AMERICAN ACADEMY OF VALUE BASED CARE

# Systemic Lupus Erythematosus

## Quick Reference Guide

2026

# Table of Contents

**1. CLINICAL SNAPSHOT** ..... 3
HCC/RAF V28 Mapping ..... 4
**2. RECOGNITION AND DIAGNOSIS** ..... 6
Medicare Screenings and Surveillance (Adult and Geriatric) ..... 6
Subtle Early Signs in Older Adults (>65) ..... 7
Geriatric Risk Factors ..... 7
Diagnostic Thresholds (2019 EULAR/ACR Weighted Criteria) ..... 8
Clues to Dig Deeper ..... 9
Common Oversights ..... 9
Key Differentials in Elderly ..... 10
Comorbidity Screening ..... 10
**3. MEAT DOCUMENTATION ESSENTIALS** ..... 13
Documentation Elements That Document SLE Complexity ..... 13
Reframing Common Documentation Shortcuts ..... 14
**4. TREATMENT AND REFERRAL QUICK GUIDE** ..... 15
Therapy Escalation Criteria ..... 15
2025 ACR/2023 EULAR/2024 KDIGO-Aligned Treatment Escalation ..... 16
Non-Pharmacologic Care and Supportive Interventions ..... 17
Medication Safety and Key Interactions ..... 18
When to Refer ..... 18
Follow-Up Timing ..... 19
Comorbidity Management — Primary Care Role ..... 19
Cost-Smart Options ..... 20
Patient Education and Adherence ..... 21
**5. CODING REMINDERS AND CASE EXAMPLES** ..... 23
Coding Specificity ..... 23
Annual Clinical Review and Confirmation ..... 24
Good Documentation — EHR Tips ..... 24
**REFERENCES** ..... 26

# 1 CLINICAL SNAPSHOT

**Definition: Systemic lupus erythematosus (SLE)** is a chronic, multisystem autoimmune disease driven by **immune-complex deposition, complement activation, and pathogenic autoantibody production** - particularly anti-double-stranded DNA **(anti-dsDNA)** and **anti-Smith antibodies**. These produce inflammatory injury across the kidneys, skin, joints, cardiovascular system, central nervous system, hematologic system, and lungs.[1] SLE is characterized by **heterogeneous** and often **nonspecific** presentations that **fluctuate during flares** and may mimic fibromyalgia, rheumatoid arthritis, and depression; consequently, a positive ANA alone never establishes the diagnosis. There are **no formal diagnostic criteria for SLE**. The diagnosis remains clinical, though the 2019 EULAR/ACR classification criteria are commonly used to support and standardize it.[2]

## ICD-10 Codes:

SLE is classified under category **M32**. Idiopathic SLE subcodes include **M32.10** (organ involvement unspecified), **M32.11** (Libman-Sacks endocarditis), **M32.12** (pericarditis), **M32.13** (lung involvement), **M32.14** (glomerular disease/lupus nephritis), **M32.15** (tubulo-interstitial nephropathy), **M32.19** (other organ involvement), **M32.8** (other forms), and **M32.9** (unspecified). **M32.0** is drug-induced SLE and should not be applied to idiopathic disease. Cutaneous-only presentations — **L93.0** (discoid) and **L93.1** (subacute cutaneous) — should not be coded **M32** unless full SLE classification criteria are met. When lupus nephritis is present, **M32.14** should be paired with the applicable CKD subcode **N18.x** to fully capture renal involvement. Common comorbidities coded alongside **M32** include hypertension (**I10**), thrombocytopenia (**D69.6**), antiphospholipid syndrome (**D68.61**), and Sjögren syndrome with myopathy (**M35.03**); each should be coded separately when documented and clinically managed.

**Prevalence and Burden:** Approximately **204,000** individuals in the United States have SLE, including approximately **184,000 females and 20,000 males**.[3] SLE is roughly **9:1 female-to-male** in reproductive-age populations.[4] Incidence is **2-3 times** higher in Black, Hispanic, and Asian populations; SLE mortality in higher in non-Hispanic Black versus non-Hispanic White individuals, aOR **3.91**. Black, Hispanic, and Asian populations also carry 2-3 times higher incidence.[5] *For patients aged 65 and older, aOR SLE mortality is **3.20** (95% CI 3.10-3.31) compared with younger patients.*[5] **Late-onset SLE** (onset age 50 years or older) comprises approximately **20%** of all SLE case and presents with a distinct phenotype that complicates recognition.[6] Classic malar rash is less frequent **(53.5% vs. 66-76% in younger patients)**[6] and renal involvement is lower, while serositis (**29.6% vs. 11.4% in juvenile-onset)**, thrombotic events, cardiac involvement, and major depression occur more often.[6] Diagnostic delay is markedly longer - a mean of **45.3** months versus 28.1 months in early-onset disease ($p<0.001$) in the RELESSER registry ($n=3,619$) - and mortality is higher (**14.3% vs. 4.7%**, $p<0.001$), driven primarily by infections and comorbidities rather than disease activity itself. Cognitive impairment and brain fog are common and are frequently **misattributed** to age-related decline or dementia.