A PRACTICAL APPROACH TO ANA TESTING

JUNE 26, 2026

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Disclosures

- Clinical Trial Support
  - Fate Therapeutics
  - GSK
  - Sanofi
  - Astra Zeneca

LEARNING OBJECTIVES

- Recall history of anti-nuclear antibody tests
- Understand best uses for the anti-nuclear antibody
- Identify other anti-nuclear and anti-cell antibodies and their associated rheumatic conditions

There is a saying about those who forget history. I don't remember what it said, but it was good.

-Stephen Colbert

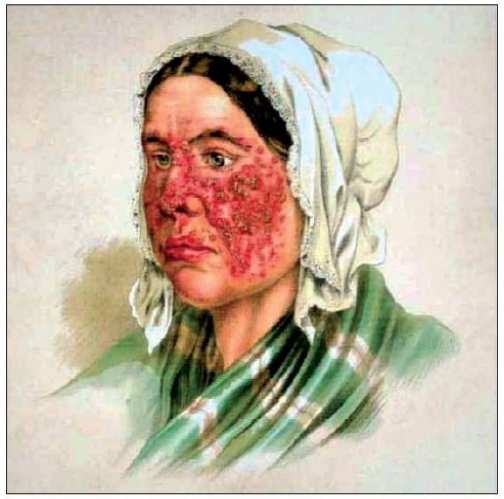


Figura 1 - Lupus eritematoso [tavola di Anton Elfinger, tratta da Atlas der Hautkrankheiten di Ferdinand von Hebra, Wien, 1856 (15)].

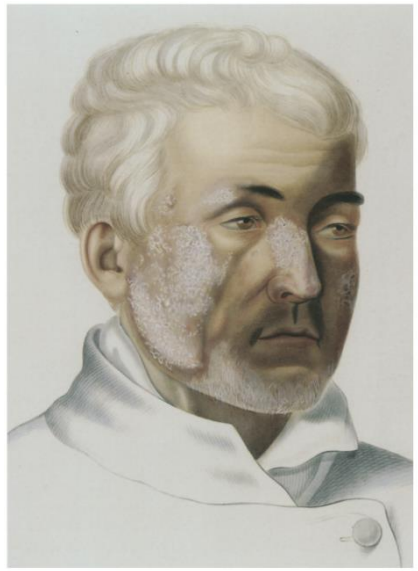


Fig 4. The first modern illustration of cutaneous lupus, labeled "Lupus erythemateux," in Cazenave (Reprinted with permission [15]).

"ERYTHEMA CENTRIFUGUM", "LUPUS ÉRYTHÈMATEUX" (1838, 1850):
LAURENT-THÉODORE BIETT
PIERRE-LOUIS CAZENAVE → LATER MORIZ KAPOSI

Fig 2. Portrait of Pierre Cazenave (Reprinted with permission [14]).

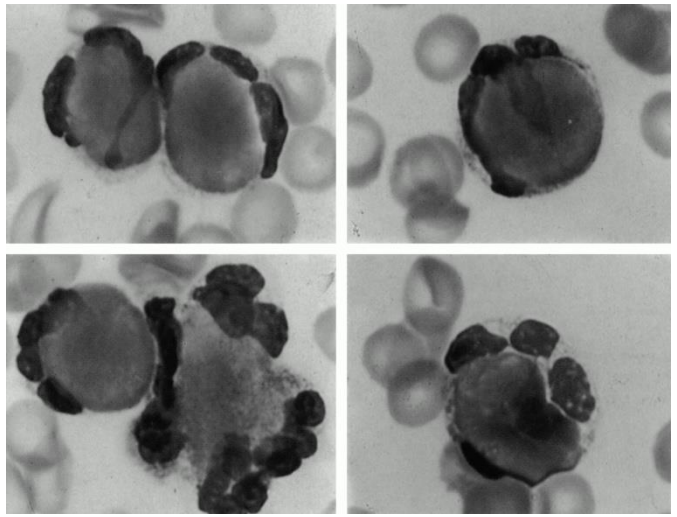
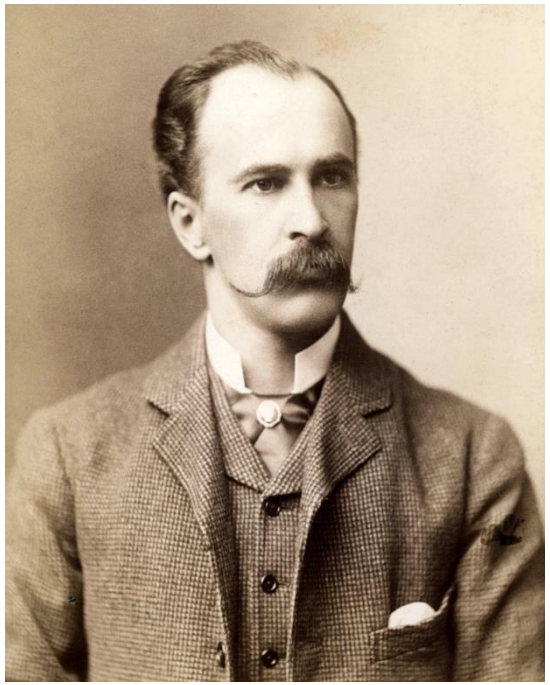


FIG. 1. THE LE CELL. THE LARGE HOMOGENEOUS AREAS ADJACENT TO THESE POLYMORPHS' NUCLEI EACH CONTAIN THE NUCLEUS OF ANOTHER BEING DIGESTED (1948 - HARGRAVES ET AL, MAYO CLINIC)

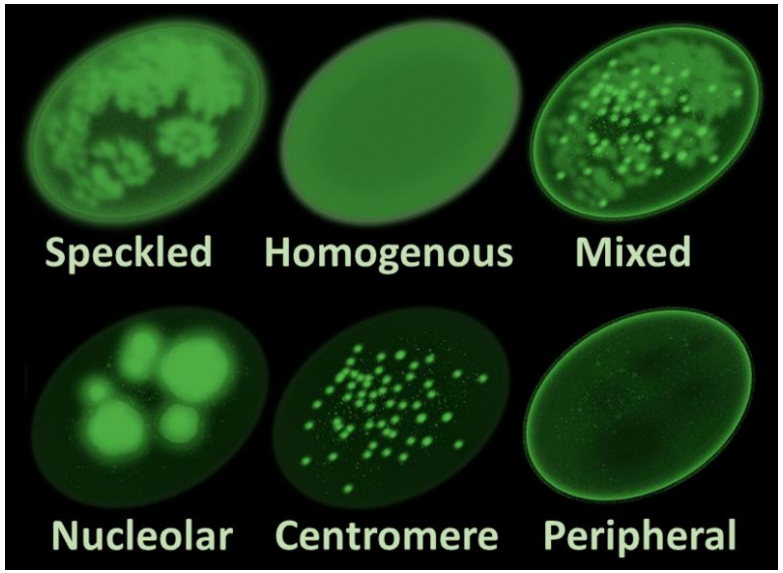
Sir William Osler (1849-1912)



1895-1904: 29 cases of "erythema group of diseases"
In retrospect, cases were likely Henoch Schonlein purpura
Other systemic vasculitis
2 cases of lupus

Felten. The history of lupus through the ages. J Am Acad Dermatol. 2022 Dec;87(6):1361-1369
Scofield, R. The place of William Osler in the description of systemic lupus erythematosus. Am J Med Sci 2009
Wallace, D. Pierre Cazenave and the first detailed modern description of lupus erythematosus. Seminars in Arthritis and Rheumatism. Volume 28, Issue 5, April 1999
Hepburn A. The LE Cell. Rheumatology (Oxford), Volume 40, Issue 7, July 2001

- Henry Kunkel, Rockefeller University, 1960s
  - collaborators
- Sera to detect antibodies:
  - lupus patient Stephanie Smith
    - → antibodies to Sm antigen
- New diagnostic markers – ab to nuclear antigens
  - lupus
  - scleroderma, and drug-induced lupus

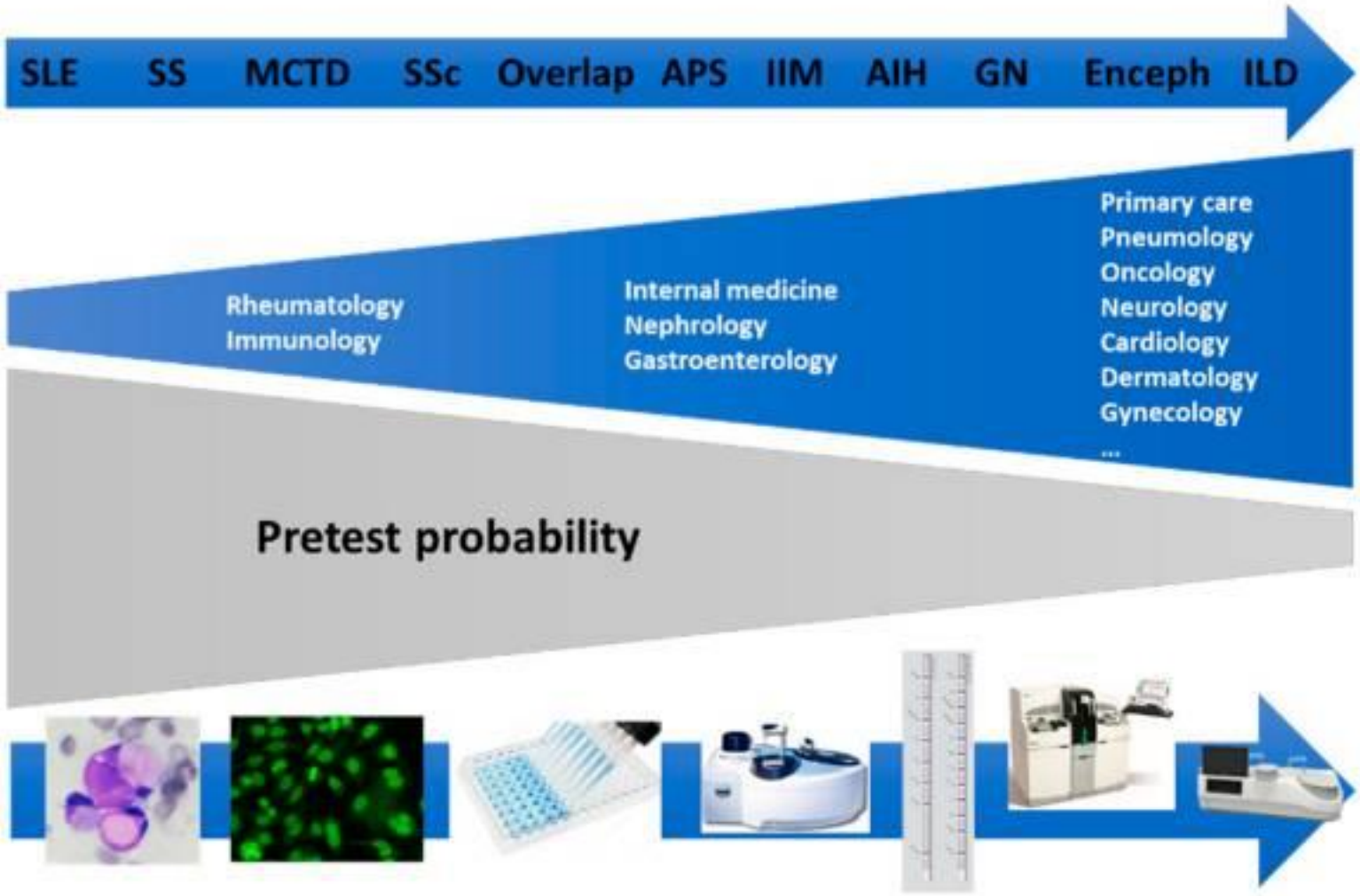


Eng Meng Tan

AUGUST 26, 1926 – MARCH 9, 2024

https://obituaries.tridentsociety.com/obituaries/san-diego-ca/eng-tan-11705130
Henkel, G. He taught us to always go deeper. The-rheumatologist.org. March 2011
Al-Mughales JA. "Anti-Nuclear Antibodies Patterns in Patients With Systemic Lupus Erythematosus and Their Correlation With Other Diagnostic Immunological Parameters.". *Front Immunol* 2022

ANA request



Irure-Ventura, J. The Past, Present, and Future in Antinuclear Antobodies (ANA). Diagnostics 2022, 12, 647

ANA – KP LAB EXPLANATION

- In the majority of patients a positive result does not indicate disease. However, ANA is usually positive in patients with SLE and may be positive in a number of conditions some of which include: rheumatoid arthritis (seropositive or seronegative), thyroid disease, autoimmune or viral hepatitis, drug-induced lupus (procainamide, hydralazine, other drugs) and other rheumatological disease, e.g. scleroderma, polymyositis, etc. Once positive, repeating this test is not helpful, and it cannot be used to monitor SLE activity. Generally not useful for patients with a single painful joint or a pattern suggesting osteoarthritis, or nonspecific arthralgias or myalgias. Studies of normal populations indicate that false positive tests may occur as follows:

  1:80 = 13%
  1:160 = 5%
  1:320 = 3%

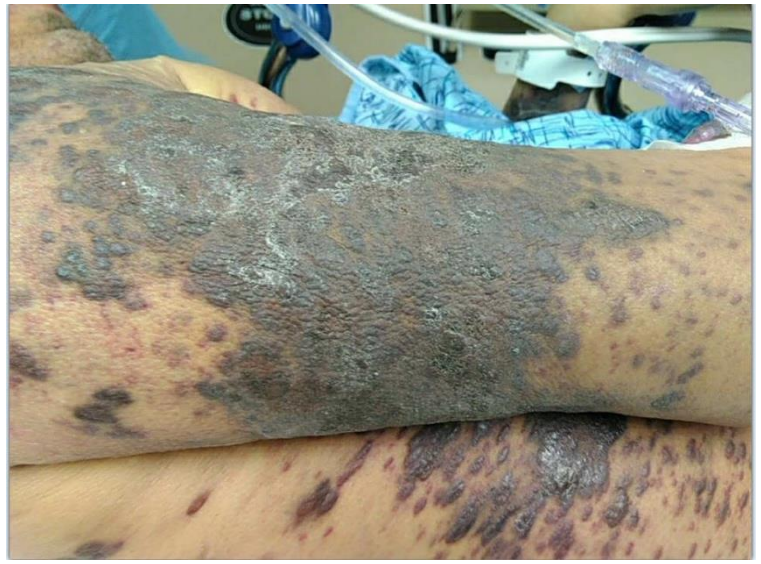
CASE 1

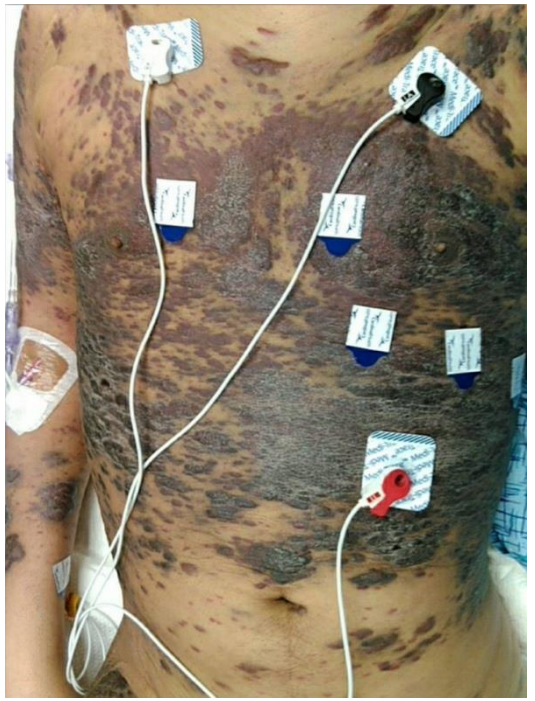
- 48 yo male presents with two months of worsening rash that started on R arm but has gradually spread. Lots of fatigue and low-grade fevers and leg and hand swelling as well as anorexia.
- Labs:

WBC'S AUTO	4.0 - 11.0 x1000/mcL	6.9	4.4
RBC, AUTO	4.50 - 5.90 Mill/mcL	1.33 ⌄	1.31 ⌄
HGB	13.5 - 17.5 g/dL	4.4 !!	4.2 !!
HCT, AUTO	41.0 - 51.0 %	16.1 !!	16.2 !!
MCV	83.0 - 98.0 fL	121.1 ^	123.7 ^
MCH	25.0 - 35.0 pg/cell	33.1	32.1
MCHC	30.0 - 35.0 g/dL	27.3 ⌄	25.9 ⌄
RDW, BLOOD	11.5 - 16.0 %	20.3 ^	20.4 ^
PLATELETS, AUTOMATED COUNT	130 - 400 x1000/mcL	114 ⌄	92 ⌄

ANA, TITER, IF	<1:80	>1:2560 !
ANA PATTERN, SER, IF		

C3	90 - 180 mg/dL	74 ⌄	
C4	10 - 40 mg/dL	16	
C-REACTIVE PROTEIN	<=7.4 mg/L		34.5 ^





QUESTION 1
What is the diagnosis?

1. Lupus
2. Sjogrens
3. Vasculitis
4. Infection

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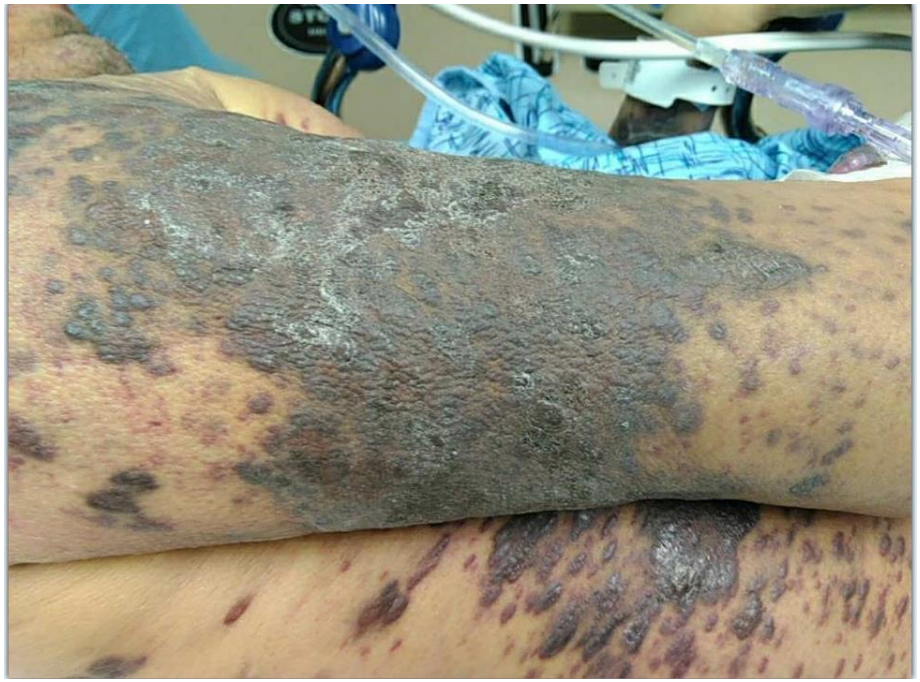
- Kaposi sarcoma

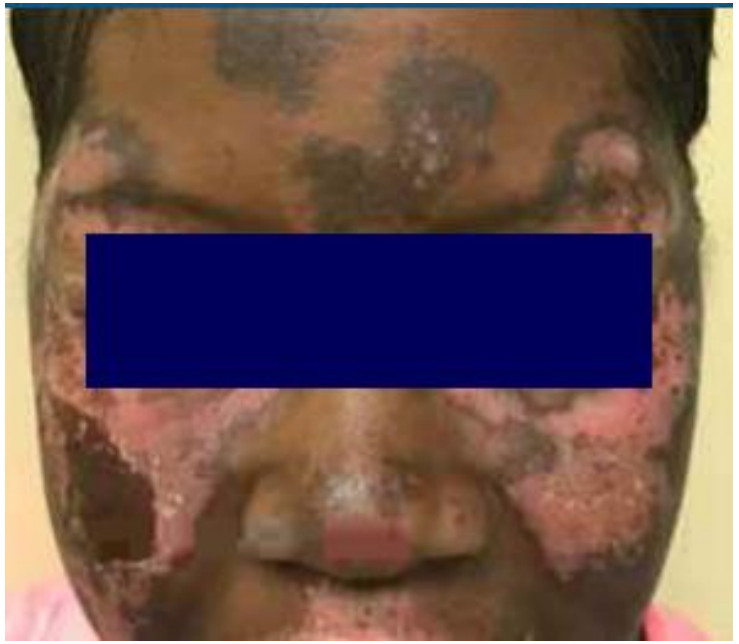


HIV 1+2 CONFIRMATION+DIFFERENTIATION, ICA	HIV Negative	HIV-1 Positive !	
HIV 1+2 AB COMMENT		HIV-1 Ab Reactive/...	
HIV 1 RNA, PCR	<=0 Copies/mL		227,000 ▲
HIV 1 RNA, LOG NUMBER PCR	<=0.00 Log cp/mL		5.36 ▲

CASE #2

- 26 year old married female presents to your clinic with 3 weeks of erythematous rash on cheeks with swelling in metacarpal-phalangeal joints. A friction rub is noted on cardiac auscultation.

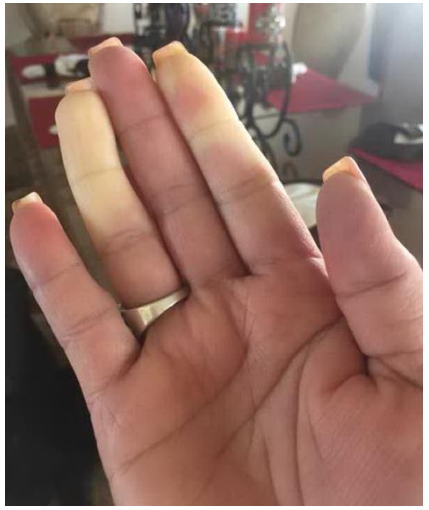
ANA, TITER, IF	<1:80	>1:2560 !
ANA PATTERN, SER, IF		Homogeneous !
C3	90 - 180 mg/dL	32 ⌄
C4	10 - 40 mg/dL	3 ⌄
SMITH EXTRACTABLE NUCLEAR IGG, IA	<=6 U/mL	2
U1 SMALL NUCLEAR RNP IGG, IA	<=4 U/mL	4
DOUBLE STRAND DNA IGG, IA	<=9 IU/mL	257 ^

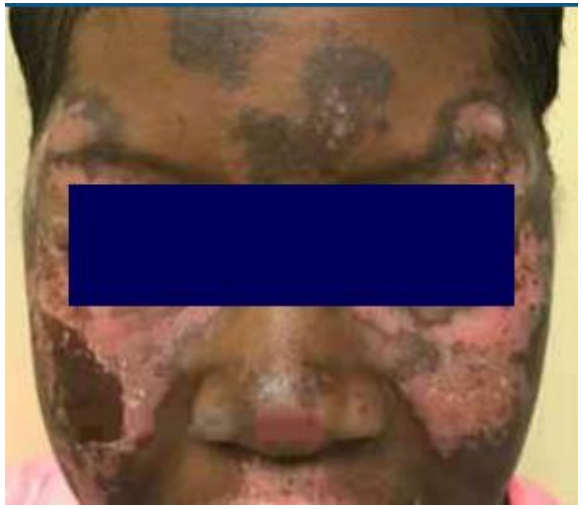


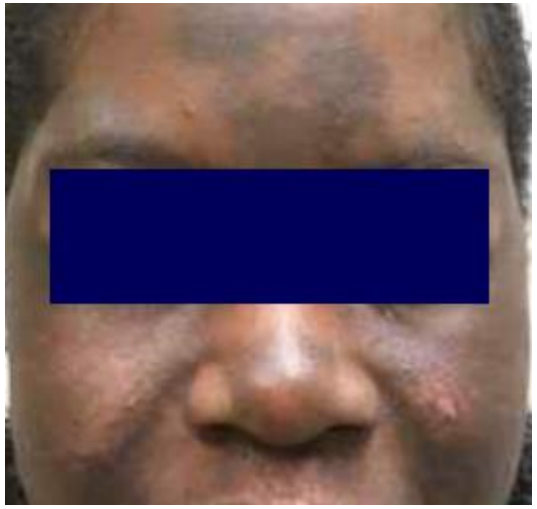
- A prednisone dose pack is prescribed and hydroxychloroquine.

LUPUS

1. Quintessential autoimmune disease
2. Characterized by auto-antibodies
3. 2019 EULAR/ACR classification criteria
4. Early nonspecific symptoms







Entry criterion

Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

↓

If absent, do not classify as SLE
If present, apply additive criteria

↓

Additive criteria

Do not count a criterion if there is a more likely explanation than SLE.
Occurrence of a criterion on at least one occasion is sufficient.
SLE classification requires at least one clinical criterion and ≥10 points.
Criteria need not occur simultaneously.
Within each domain, only the highest weighted criterion is counted toward the total score§.

Clinical domains and criteria	Weight
Constitutional	
Fever	2
Hematologic	
Leukopenia	3
Thrombocytopenia	4
Autoimmune hemolysis	4
Neuropsychiatric	
Delirium	2
Psychosis	3
Seizure	5
Mucocutaneous	
Non-scarring alopecia	2
Oral ulcers	2
Subacute cutaneous OR discoid lupus	4
Acute cutaneous lupus	6
Serosal	
Pleural or pericardial effusion	5
Acute pericarditis	6
Musculoskeletal	
Joint involvement	6
Renal	
Proteinuria >0.5g/24h	4
Renal biopsy Class II or V lupus nephritis	8
Renal biopsy Class III or IV lupus nephritis	10

Immunology domains and criteria	Weight
Antiphospholipid antibodies	
Anti-cardiolipin antibodies OR	
Anti-β2GP1 antibodies OR	
Lupus anticoagulant	2
Complement proteins	
Low C3 OR low C4	3
Low C3 AND low C4	4
SLE-specific antibodies	
Anti-dsDNA antibody* OR	
Anti-Smith antibody	6

Total score:

↓

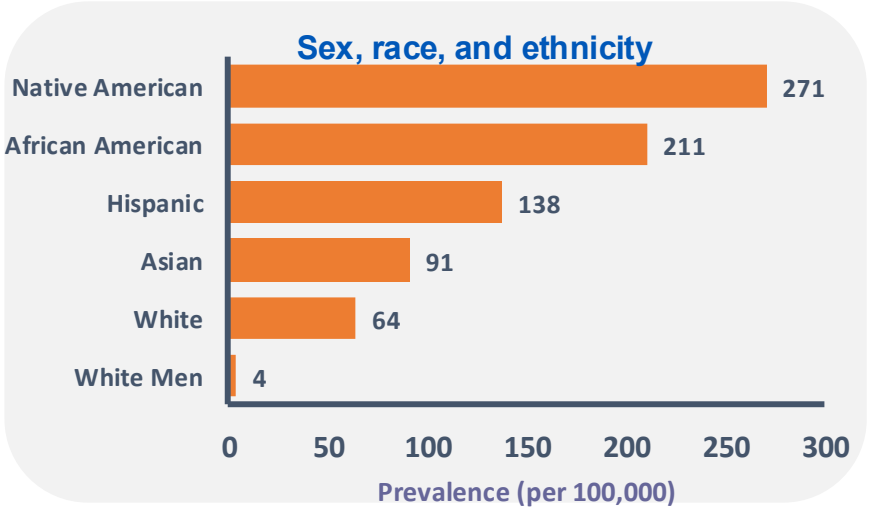
Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.

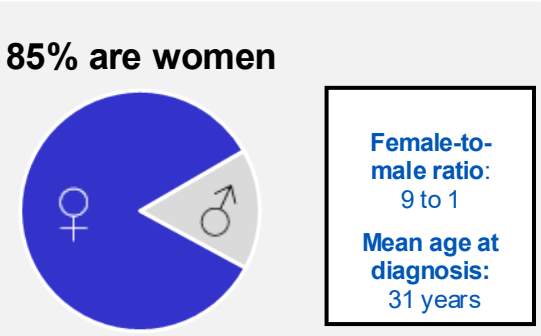
Aringer, M. European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. Ann of Rheum Dis. 2019.

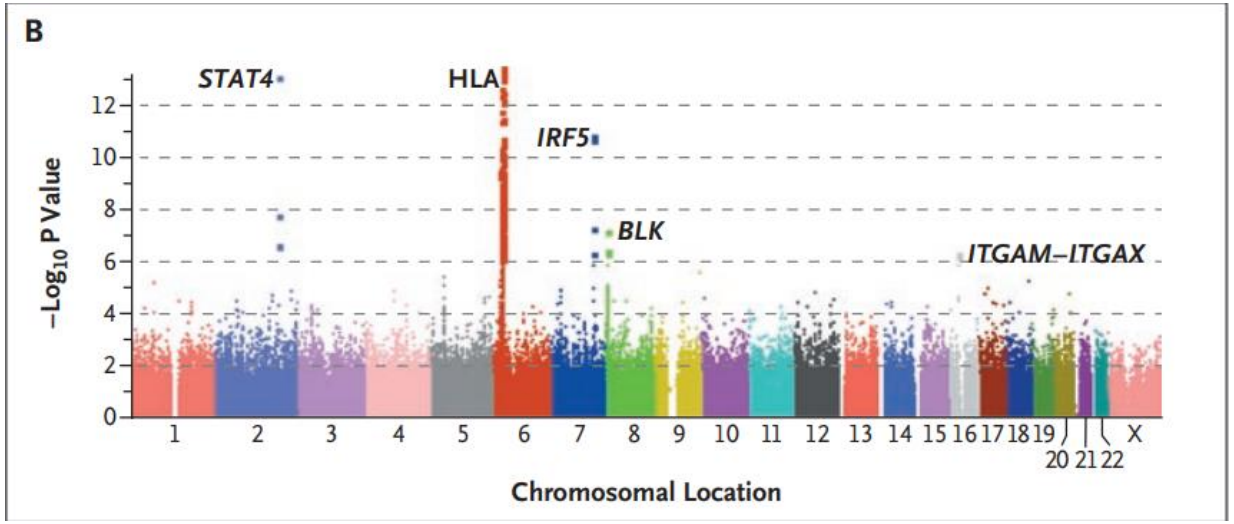
EPIDEMIOLOGY

Age, gender, race and genetics

- Peak incidence 14-45 years
- Female predominance
  - severity is =
- Ethnic predisposition
- Genetics:
  - protein tyrosine phosphatase, non-receptor type 22 (PTPN22)
  - HLA DRB1, ITGAM or ITGAX





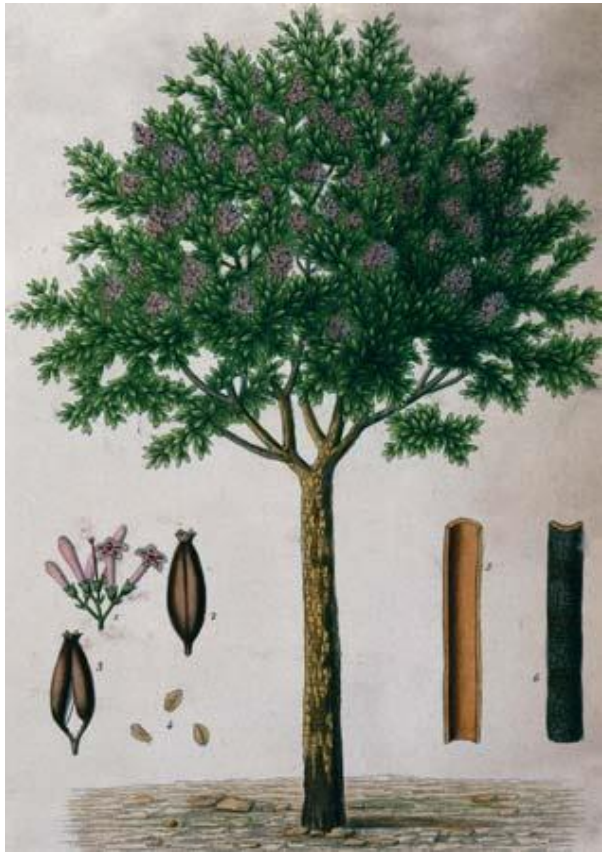


Hom G, Graham RR, Modrek B, Taylor KE, Ortmann W, Garnier S, Lee AT, Chung SA, Ferreira RC, Pant PK, Ballinger DG. Association of systemic lupus erythematosus with C8orf13–BLK and ITGAM–ITGAX. New England Journal of Medicine. 2008 Feb 28;358(9):900-9.

QUESTION:

Historically, South American natives chewed on the bark of the cinchona tree to treat fevers, rashes musculoskeletal pain, as well as to ward of certain infections. The active ingredient in this plant based material is:

- Cannabinoid
- Ibuprofen
- Quinine
- Ozempic



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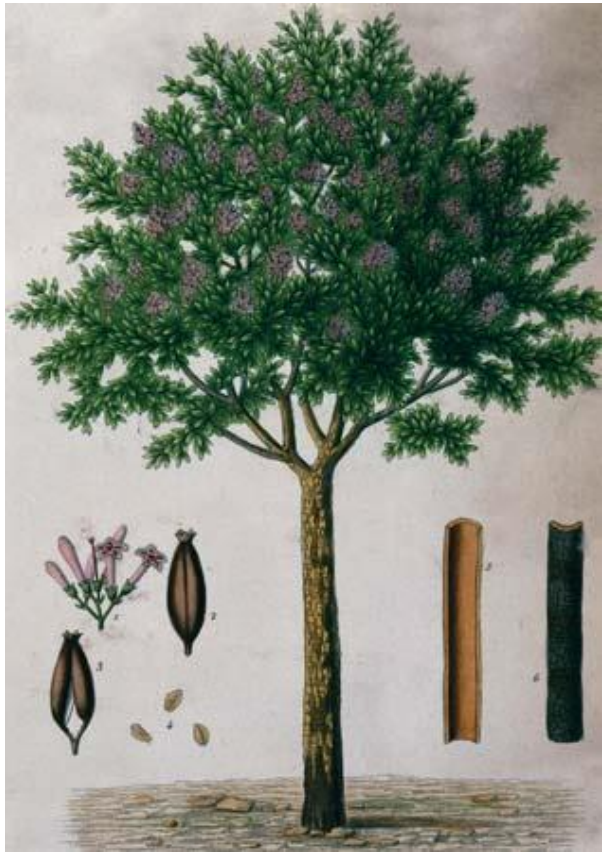


EARLY TREATMENTS

- Quinine
  - Tonic water
  - Reduction in fevers, myalgias/cramp, arthralgias
  - Chloroquine used for malaria in 1800's (precursor to hydroxychloroquine)
- Thomas Payne, British physician noted (1895) effects with lupus
- Cyclophosphamide
  - Chemical warfare: mustard gas
  - 1943: American cargo ship SS John Harvey attacked in Italian port of Bari
    - Autopsies → bone marrow suppression
    - Louis Goodman and Alfred Gilman - stabilized nitrogen mustard to treat lymphoma
  - Subsequent discoveries (methotrexate, azathioprine)

http://www.lupus.org.uk/what-is-lupus/how-it-is-treated/antimalarials
Radis. Plaquenil: From Malaria Treatment to Managing Lupus, RA. The Rheumatologist, May 2015
K. D. Rainsford. Therapy and pharmacological properties of hydroxychloroquine and chloroquine in treatment of systemic lupus erythematosus, rheumatoid arthritis and related diseases. Inflammopharmacology. October 2015, Volume 23, Issue 5, pp 231-269
Helfgott. How Wars Have Shaped Rheumatology. The Rheumatologist, Nov 2014

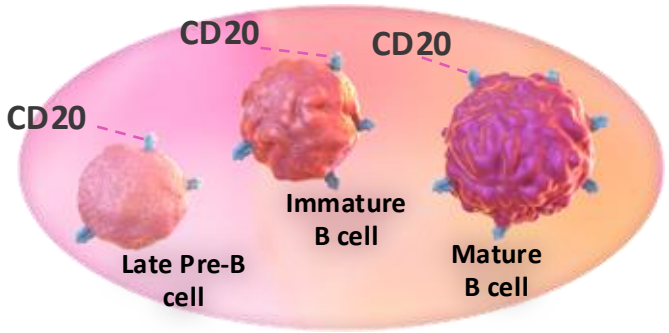
SLE – TREATMENT I

- **Mild severity** (mild skin or joint involvement)
  - NSAID
  - low dose glucocorticoids
  - hydroxychloroquine

- **Intermediate severity** (serositis, cytopenia, marked skin or joint involvement):
  - glucocorticoids (1 mg/kg/day)
  - azathioprine
  - methotrexate, leflunomide
  - mycophenolate mofetil

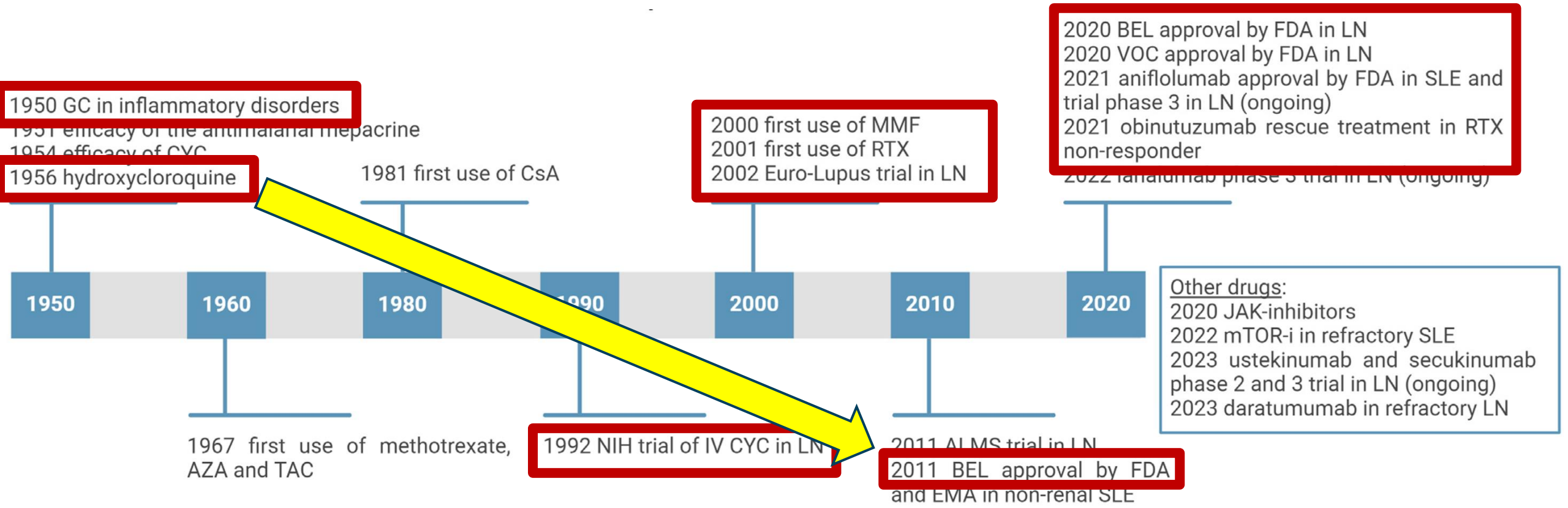
SLE – TREATMENT II

- **Severe life-threatening organ involvements** (pulmonary hemorrhage, pericarditis, nephritis, systemic vasculitis, hematologic, neuropsychiatric manifestations)
  - glucocorticoids (methylprednisolone up to 1000 mg/day x 3 days)
  - cyclophosphamide
  - plasmapheresis
  - IV immunoglobulin
  - mycophenolate mofetil



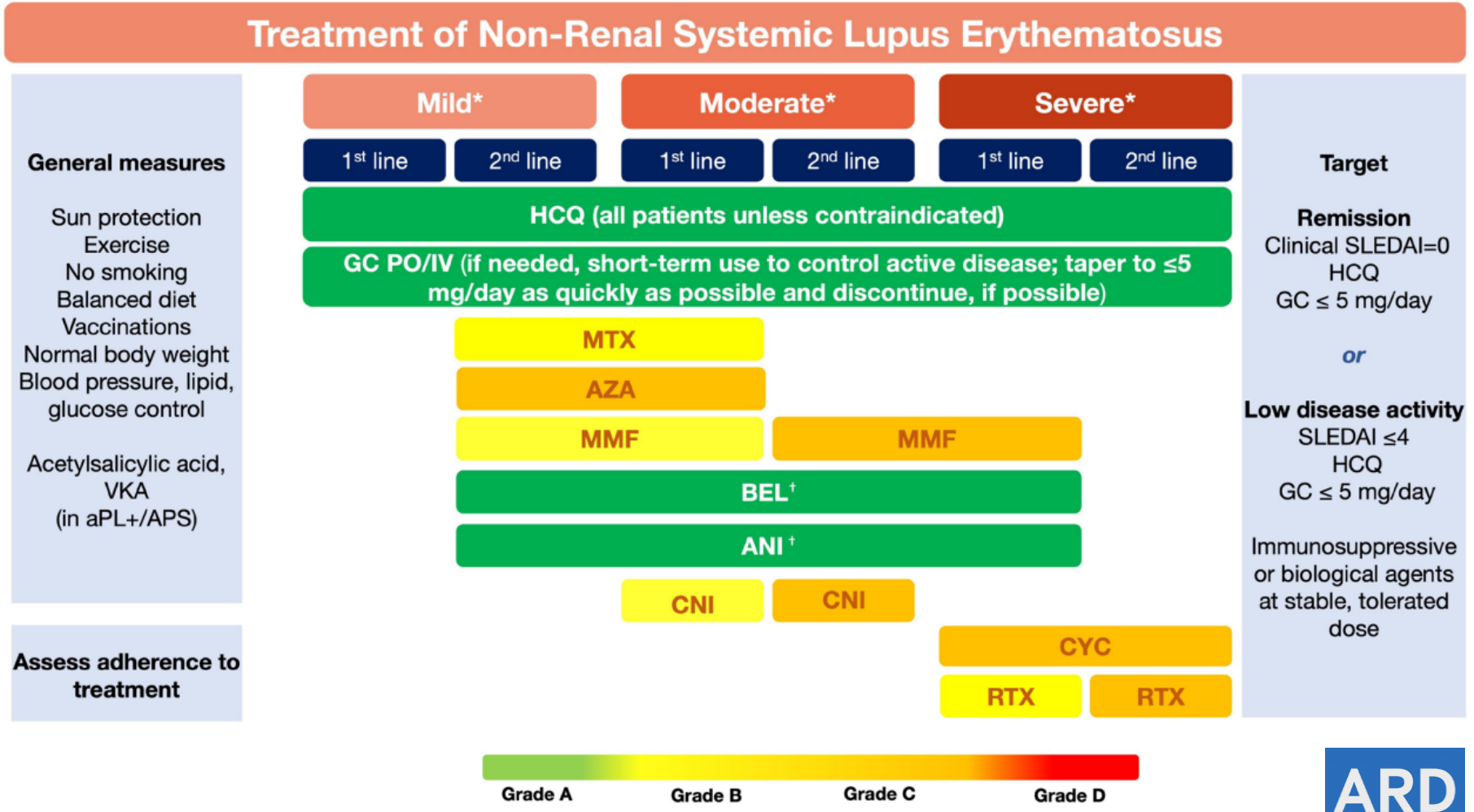
  - belimumab, anti-BlyS, 2011 (FDA→ lupus nephritis (LN): dec 17, 2020)
  - voclosporin, CNI, (FDA→ LN: jan 22, 2021)
  - anifrolumab, anti-Type I interferon (FDA → lupus: aug 2, 2021)
  - obinutuzumab, anti-CD20 (FDA →LN: oct 20, 2025)

THERAPEUTIC ADVANCES IN LUPUS



Roveta, Lupus Nephritis from Pathogenesis to New Therapies: An Update. Int J of Mol Sci. 2024 Aug 18

Treatment of non-renal SLE—recommended drugs with respective grading of recommendation. aPL, antiphospholipid antibodies; AZA, azathioprine; BEL, belimumab; BILAG: British Isles Lupus Assessment Group disease activity index; CNIs, calcineurin inhibitors; CYC, cyclophosphamide; GC, glucocorticoids; HCQ, hydroxychloroquine; IM, intramuscular; MMF, mycophenolate mofetil; MTX, methotrexate; Pre, prednisone; PO, per os; RTX, rituximab; PLTs: Platelets; SLEDAI, Systemic Lupus Erythematosus Disease Activity Index.



Antonis Fanouriakis et al. Ann Rheum Dis 2024

PRACTICAL USE OF THE ANA: LUPUS DIAGNOSIS AND TREATMENT

- Historical recognition of skin domain; then other organ involvement
- Immunopathogenesis (autoimmunity, B cells centrality)
- Updated classification criteria – systemic and organ domains
- Role of B cell targeted and off label options
- Safety of immunomodulatory options

CASE #3

- A 64 year old woman presents with moderate swelling and stiffness in hands and fatigue for the past month, rapidly worsening. Fingers change color – red, purple, white – worse in cold temps. She was recently found to have a spiculated mass on routine mammography.
- SH: married, retired RN
- ROS: mild intermittent dry cough
- PE:
  - firm tight fingers with periungual ulcer and dilated capillary nailfold loops
  - Fine crackles in bases of lungs

QUESTION 2
What antibody is most specific for this condition?

1. ANA
2. Anti-RNA polymerase III
3. Smith
4. Anti-thyroperoxidase

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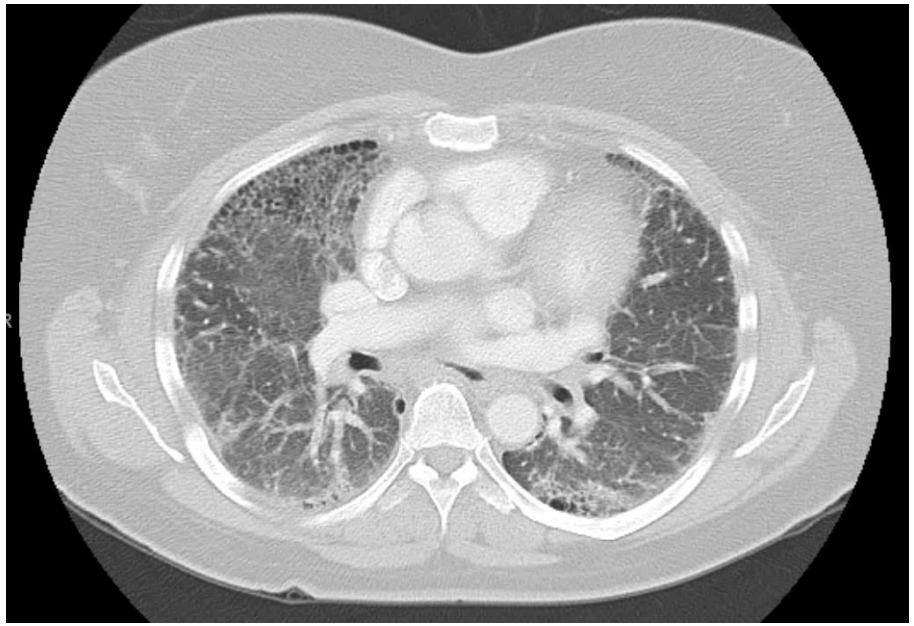
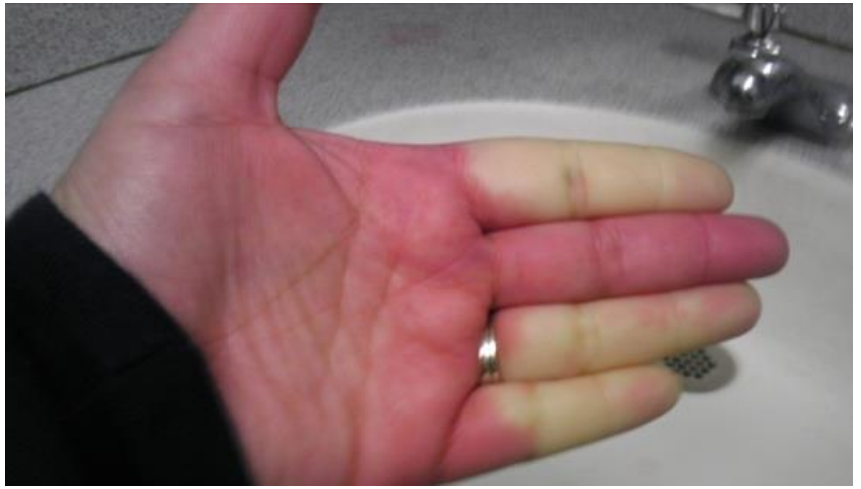
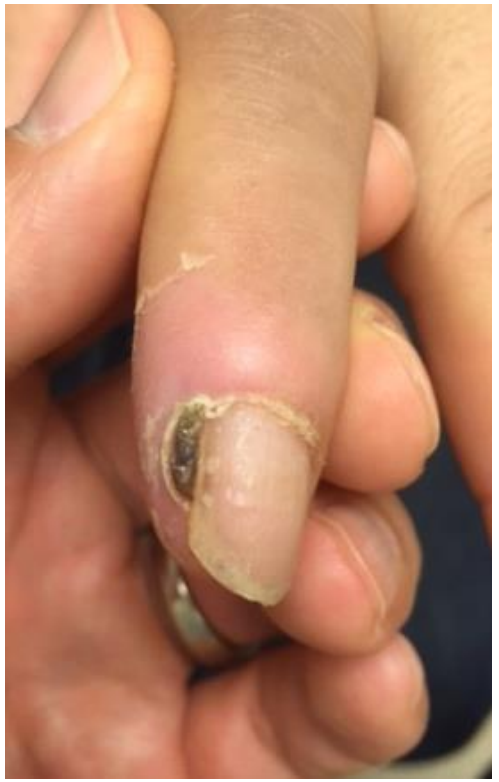
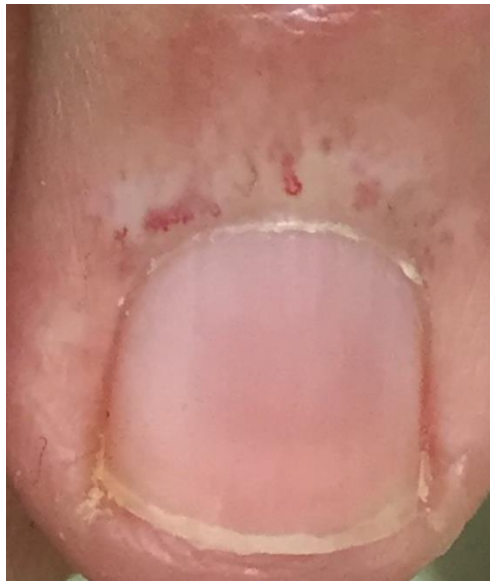
EXAMPLES AND RESULTS

- T=1.4 cm grade 3 invasive ductal with lobular features
  ER: positive, PR: negative, her-2/neu: negative (0+IHC)
- N=7+/17 +extranodal extension

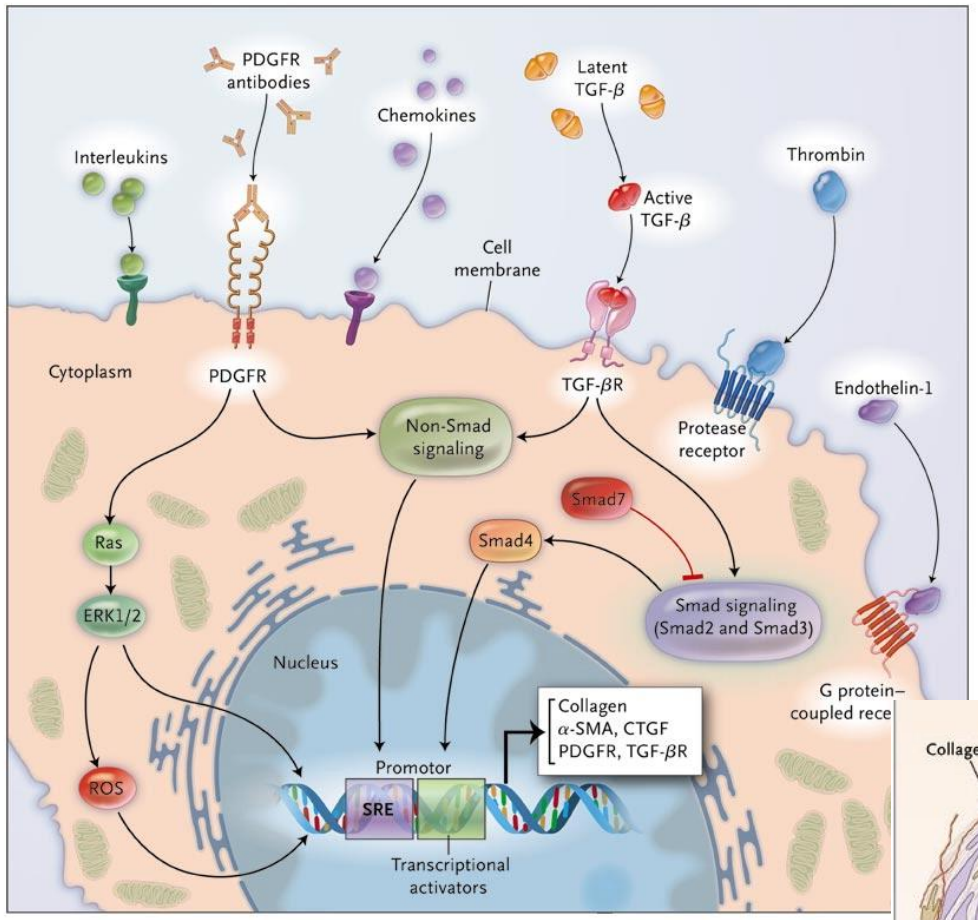


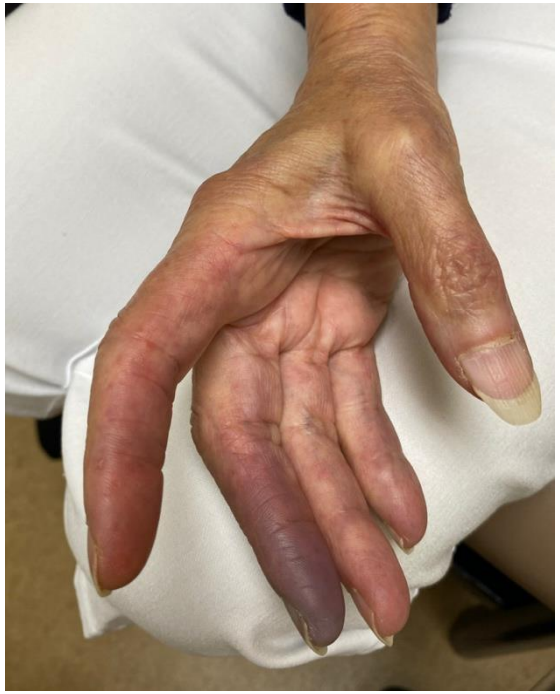


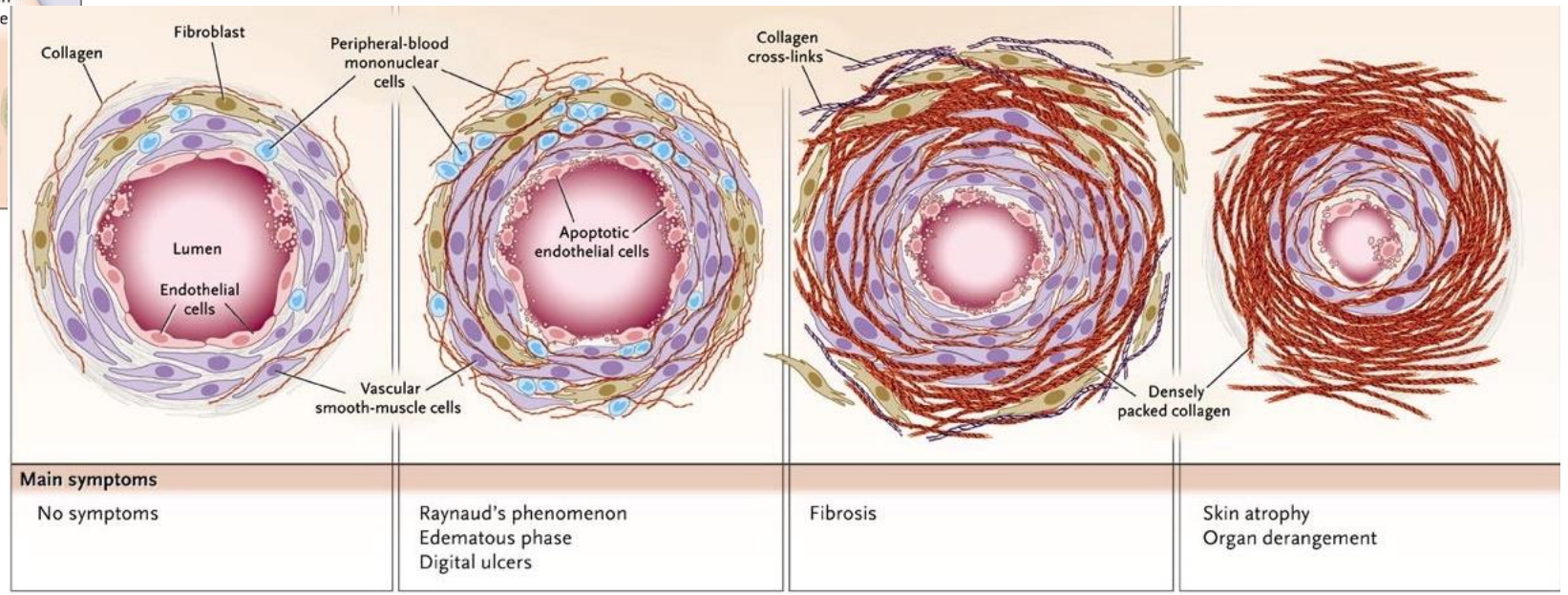




ANA, TITER, IF	<1:80		1:640 !
ANA PATTERN, SER, IF			Nucleolar !
REPORT	<20 Units	RNA Polymerase III Ab: >80	
SCL-70 ANTIBODY, QL	Negative		Positive !







GABRIELLI A ET AL. SCLERODERMA. N ENGL J MED 2009;360:1989-2003.

2013 ACR / EULAR Criteria For The Classification Of Systemic Sclerosis (Scleroderma)*

Item	Sub-items(s)	Weight/score †
Skin thickening of the fingers of both hands extending proximal to the metacarpophalangeal joints *(sufficient criterion)*	-	9
Skin thickening of the fingers *(only count the higher score)*	Puffy fingers	2
	Sclerodactyly of the fingers (distal to the metacarpophalangeal joints but proximal to the proximal interphalangeal joints)	4
Fingertip lesions *(only count the higher score)*	Digital tip ulcers	2
	Fingertip pitting scars	3
Telangiectasia	-	2
Abnormal nailfold capillaries	-	2
Pulmonary arterial hypertension and/or interstitial lung disease *(maximum score is 2)*	Pulmonary arterial hypertension	2
	Interstitial lung disease	2
Raynaud's phenomenon	-	3
SSc-related autoantibodies (anticentromere, anti–topoisomerase I [anti–Scl-70], anti–RNA polymerase III) *(maximum score is 3)*	Anticentromere 3 Anti–topoisomerase I Anti–RNA polymerase III	3

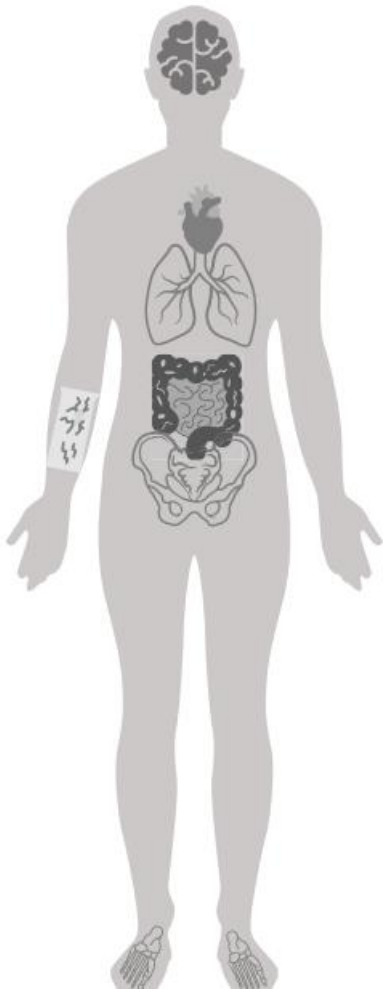
* The criteria are not applicable to patients with skin thickening sparing the fingers or to patients who have a scleroderma-like disorder that better explains their manifestations (e.g., nephrogenic sclerosing fibrosis, generalized morphea, eosinophilic fasciitis, scleredema diabeticorum, scleromyxedema, erythromyalgia, porphyria, lichen sclerosis, graft-versus-host disease, diabetic cheiroarthropathy).

† The total score is determined by adding the maximum weight (score) in each category.
Patients with a total score of ≥ 9 are classified as having definite scleroderma.

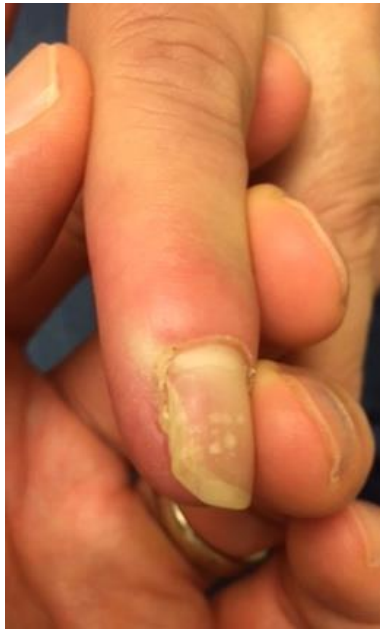
Sensitivity 91% Specificity 92%

Van den Hoogen et al. 2013 Classification Criteria for Systemic Sclerosis. Arthritis and Rheumatism. Vol. 65, No. 11, November 2013, pp 2737–2747

The 2024 British Society for Rheumatology guideline for management of systemic sclerosis



- Scleroderma is multi-system, autoimmune
- Limited scleroderma
  - Anti-centromere
- Diffuse scleroderma
  - Anti-topoisomerase (Scl-70)
  - Anti-RNA polymerase III (neoplasm, cardiopulmonary)
- Newer options for ILD



ILD

MMF is recommended as first line treatment with rituximab or cyclophosphamide i.v. as alternative 1B

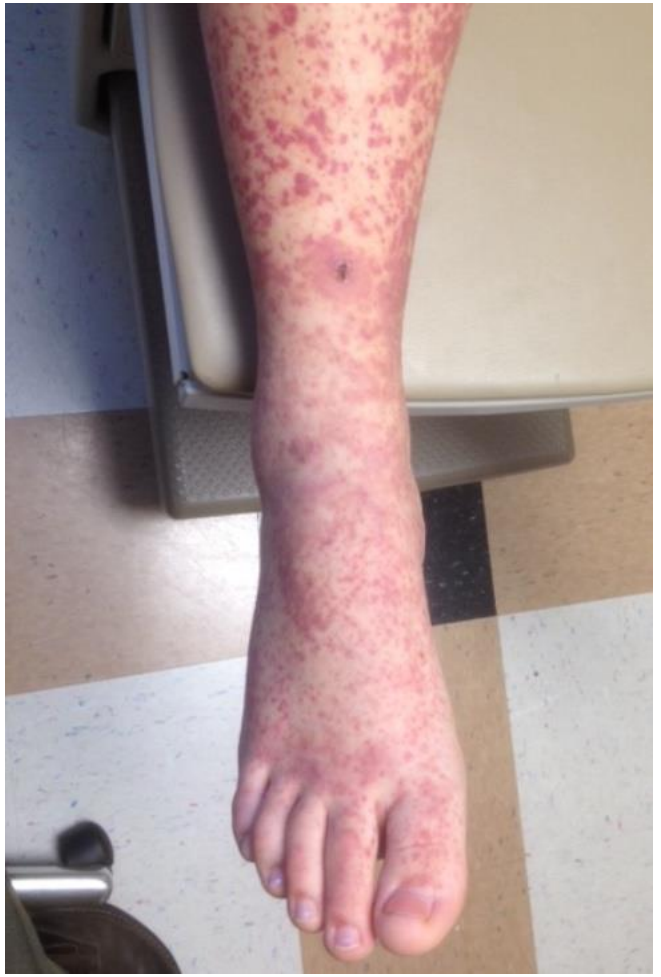
Consider adding rituximab or tocilizumab to MMF for progressive disease 2C

Nintedanib recommended for progressive pulmonary fibrosis 1B

Rheumatology, Volume 63, Issue 11, November 2024, Pages 2956–2975

CASE #4

- A 58 year old woman presents to the emergency department with diffuse skin itch for the past couple months. No skin rashes apparent initially, but in the past week, some scattered red spots on lower legs that alarmed her. Mild aching in PIPs and stiffness and swelling that improves with OTC ibuprofen. Her PCP had been performing some labs tests and she is aware of some 'liver damage', but unsure of lab abnormalities, though recalls a 'POSITIVE ANA'



- SH: divorced, school secretary
  - 2 adult kids, 3 miscarriages
- No tob/etoh
- ROS: mild occasional fevers; periodic gland swelling in neck, under jaw
  - moderate dryness in eyes and mouth
- PE:
  - Mild scleral injection bilaterally, no discharge
  - Scattered submandibular and parotid gland enlargement and xerostomia
  - Slight PIP synovitis in hands
  - Enlarged liver with mild tenderness and subtle ascites detected
- Labs:

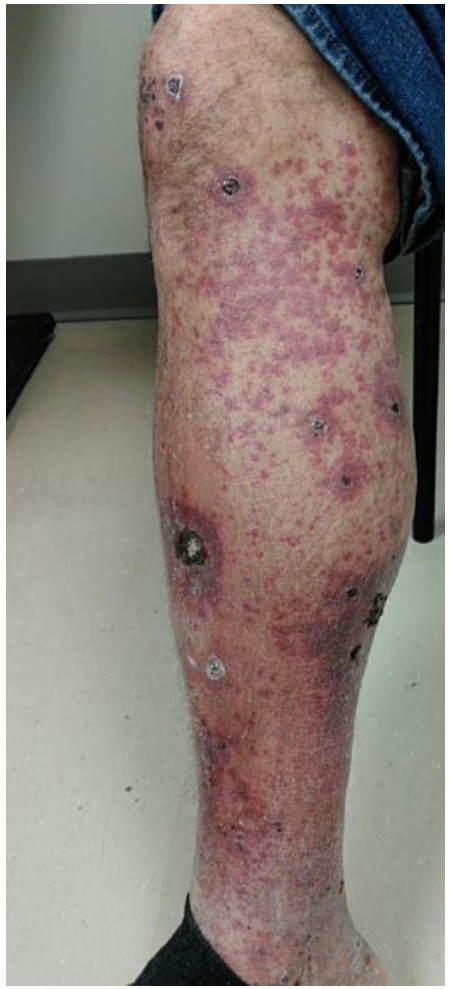
MITOCHONDRIA AB, TITER	<1:20	1:640 !		
SMOOTH MUSCLE AB, TITER,SER,QN	<1:20	<1:20		
SS-A AB, EIA	Negative			Positive !
SS-B AB, EIA	Negative			Negative
ALKALINE PHOSPHATASE	20 - 125 units/L		170 ^	

QUESTION #4

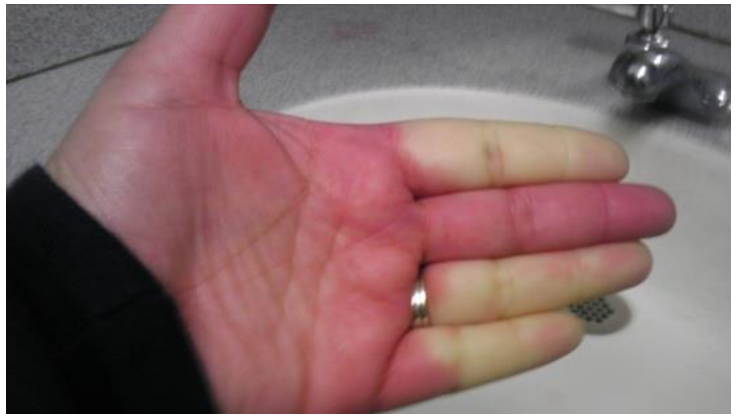
- What is the best explanation for this multi-systemic presentation?
  - Sarcoidosis
  - Lymphoma
  - Sjogren’s syndrome
  - Sjogren’s disease

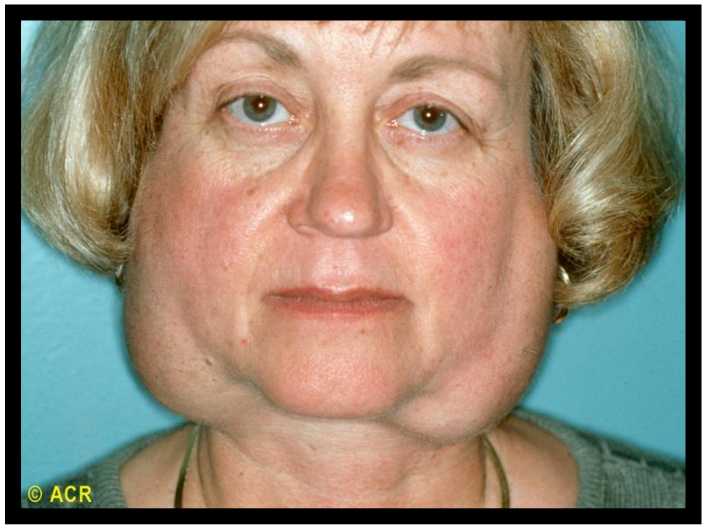
QUESTION #3

- What is the best explanation for this multi-systemic presentation?
  - Sarcoidosis
  - Lymphoma
  - Sjogren’s syndrome
  - Sjogren’s disease

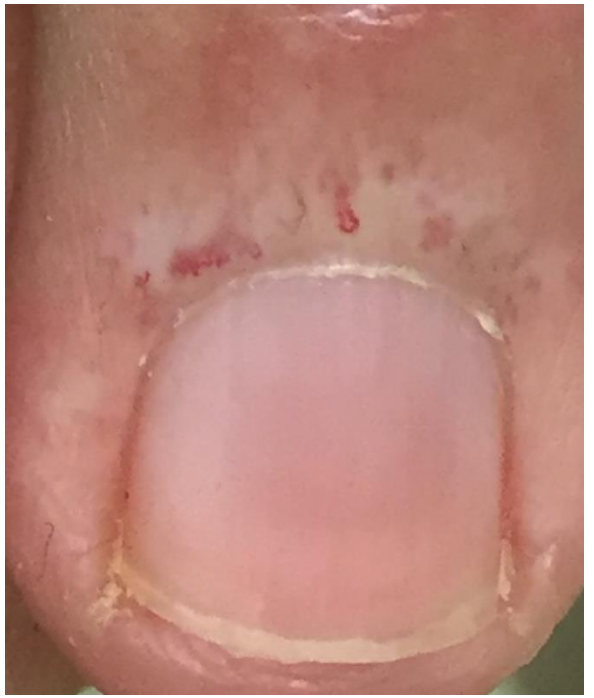


C3	C4
90 - 180 mg/dL	10 - 40 mg/dL
96	14
99	16
100	13
92	3 ↓
89 ↓	3 ↓
103	5 ↓
103	8 ↓
99	19
100	17
117	22
110	18
114	15
104	13
99	13
109	15





Sjögren's Syndrome to Sjögren's Disease: Why the Name Changed



Baer AN and Hammitt KM. Sjögren's Disease, Not Syndrome. *Arthritis Rheumatol.* 2021 Jul;73(7):1347-8. PMID: 33559389.

RATIONALE: WHAT'S IN A NAME CHANGE?

- Several rheumatic conditions have had nomenclature changes in past:
  - Reiter's→ Reactive arthritis
  - Wegener's granulomatosus→ Granulomatosus with polyangiitis (GPA)

- Sjogren's disease acknowledges systemic autoimmune inflammatory processes:
  - Exocrinopathy
  - Vasculitis and peripheral neuropathy
  - Interstitial lung disease
  - Primary biliary cholangitis (anti-mitochondrial antibody)
  - Lymphoma

C3	C4
90 - 180 mg/dL	10 - 40 mg/dL
96	14
99	16
100	13
92	3 ↓
89 ↓	3 ↓
103	5 ↓
103	8 ↓
99	19
100	17
117	22
110	18
114	15
104	13
99	13
109	15

- Mimics can include IGG4 related disease, sarcoidosis, viral infection (HCV, HIV), lymphoma, amyloid

Price E. British Society for Rheumatology guideline on management of adult and juvenile onset Sjögren disease Rheumatology, Volume 64, Issue 2, February 2025, Pages 409–439

Shiboski, C. (2016) 2016 ACR-EULAR Classification Criteria for primary Sjögren's Syndrome: A Consensus and Data-Driven Methodology Involving Three International Patient Cohorts. 2016 ACR-EULAR Classification Criteria for primary Sjögren's Syndrome: Arthritis Rheumatol. 2016 Oct 26;69(1):35–45.

CASE #5

- 54 yo man has changed insurances and presents for new PCP appointment. Feels well but has PMH of lupus – diagnosed at age 49 out of state. History is vague, but he believes he was diagnosed based on some joint pains and ‘abnormal labs’. He has been on hydroxychloroquine since then and has felt well.
- No rashes, mouth sores, alopecia, joint swelling, hematuria, seizures, psychosis, thrombosis, chest or abdominal pain, dyspnea, edema
- PMH:
  - Hypertension
  - Renal stones
- SH:
  - Married, 3 adult kids

ANA IF	<1:80	<1:80
ANA PATTERN, SER, IF		

Component	Latest Ref Rng & Units	9/28/2015	6/10/2016	10/25/2016	2/28/2017
ENA SM+ RNP AB, SER	<=19 unit(s)	8			
ENA AB EIA	<=19 unit(s)	6			
C3	90 - 180 mg/dL		120	124	126
C4	10 - 40 mg/dL		14	17	17
ANA IF	<1:80	<1:80			
ANA PATTERN, SER, IF					
DS DNA AB IF	Negative				
DOUBLE STRANDED DNA AB TITER, IF	<1:10				
DNA DS AB	<=29.9 IU/mL		27.6	16.4	26.5

Component	Latest Ref Rng & Units	9/28/2015	6/10/2016	10/25/2016	2/28/2017	2/20/2019	4/5/2019	5/6/2019
ENA SM+ RNP AB, SER	<=19 unit(s)	8				**30 (H)**		
ENA AB EIA	<=19 unit(s)	6				**23 (H)**		
C3	90 - 180 mg/dL		120	124	126	**74 (L)**	**69 (L)**	**72 (L)**
C4	10 - 40 mg/dL		14	17	17	**2 (L)**	**2 (L)**	**2 (L)**
ANA IF	<1:80	<1:80				**1:640 (A)**		
ANA PATTERN, SER, IF						**Speckled (A)**		
DS DNA AB IF	Negative						**Positive (A)**	**Positive (A)**
DOUBLE STRANDED DNA AB TITER, IF	<1:10						**1:160 (A)**	**1:80 (A)**
DNA DS AB	<=29.9 IU/mL		27.6	16.4	26.5			

TABLE 1: Historical Features Helpful in Distinguishing Raynaud Disease (RD) from Raynaud Phenomenon Due to Connective Tissue Disease (RP-CTD)

Features	*RD*	*RP-CTD*	*Associated Connective Tissue Disease*
migraine headache	+	±	APL
fibromyalgia	+	0	
irritable bowel syndrome	+	0	
depression	+	0	
arthralgias/arthritis	0	+	SLE, SS
pleurisy	0	+	SLE
dry eyes/dry mouth	0	+	SS
proximal muscle weakness	0	+	PM/DM
rash	0	+	DM, SLE
swollen fingers	0	+	SSc
heartburn	0	+	SSc
distal dysphagia for solid foods	0	+	SSc
proximal dysphagia for liquids	0	+	PM/DM

SLE= systemic lupus erythematosus; SS=Sjogren syndrome; SSc=systemic sclerosis; PM/DM= polymyositis/dermatomyositis; APL = antiphospholipid antibody syndrome

www.dept-med.pitt.edu/rheum or www.UPMCPhysicianResources.com

TABLE 2: Physical Examination Findings Suggestive of CTD

Findings	*CTD*
puffy fingers	SSc
sclerodactyly (thickening of digital skin)	SSc
digital pitting scars, ulcers or gangrene	SSc
rash of DM	DM
rash of lupus	SLE
glossitis	SS
pleural or pericardial friction rub	SLE
proximal muscle weakness	PM/DM
grossly abnormal (dilated) nailfold capillaries	SSc, others
subcutaneous/intracutaneous calcinosis	SSc
digital/facial/lip telangiectasias	SSc
palpable tendon/bursal friction rubs	SSc
polyarthritis	SLE, others

SLE= systemic lupus erythematosus; SS=Sjogren syndrome; SSc=systemic sclerosis; PM/DM= polymyositis-dermatomyositis

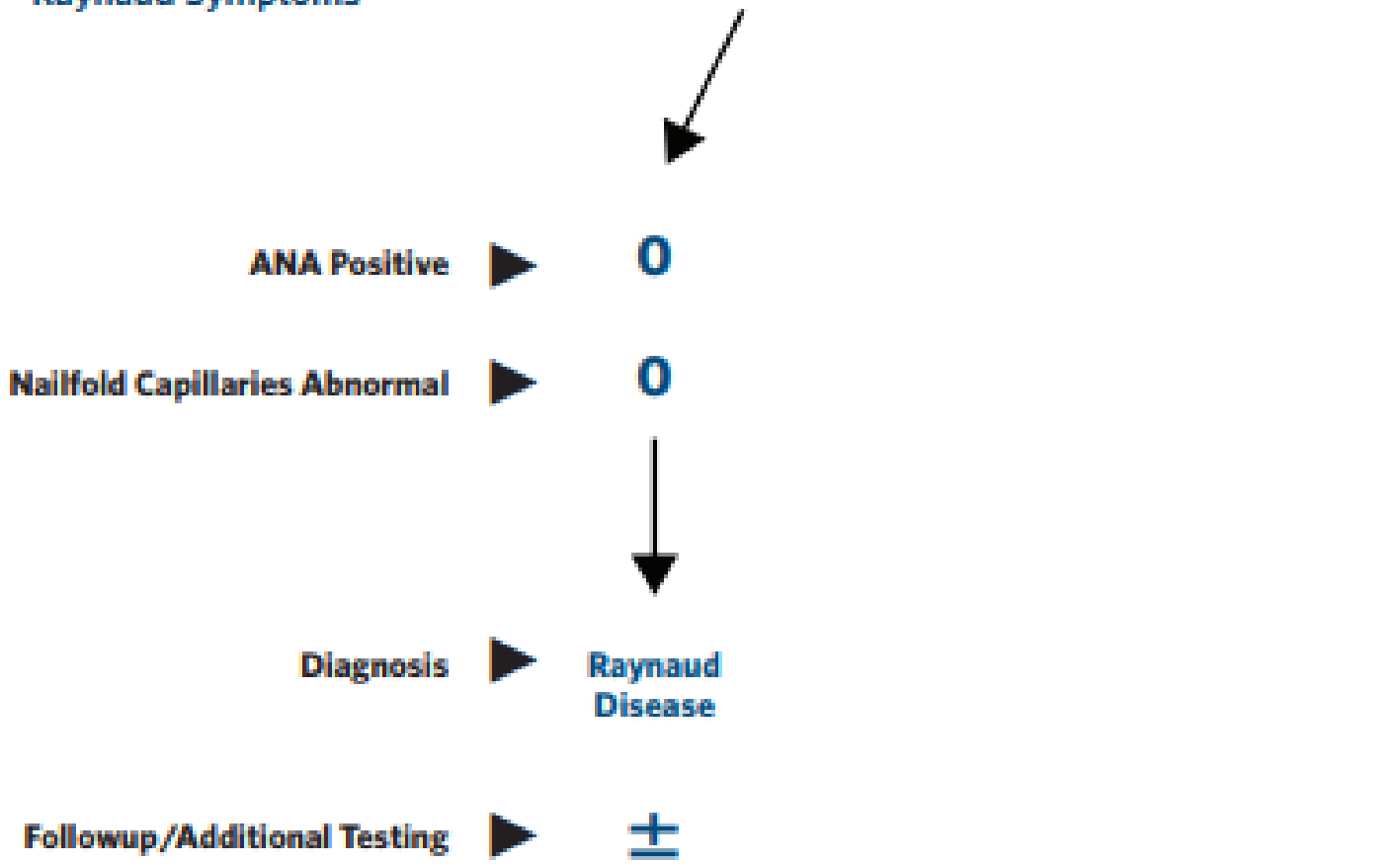
www.dept-med.pitt.edu/rheum or www.UPMCPhysicianResources.com

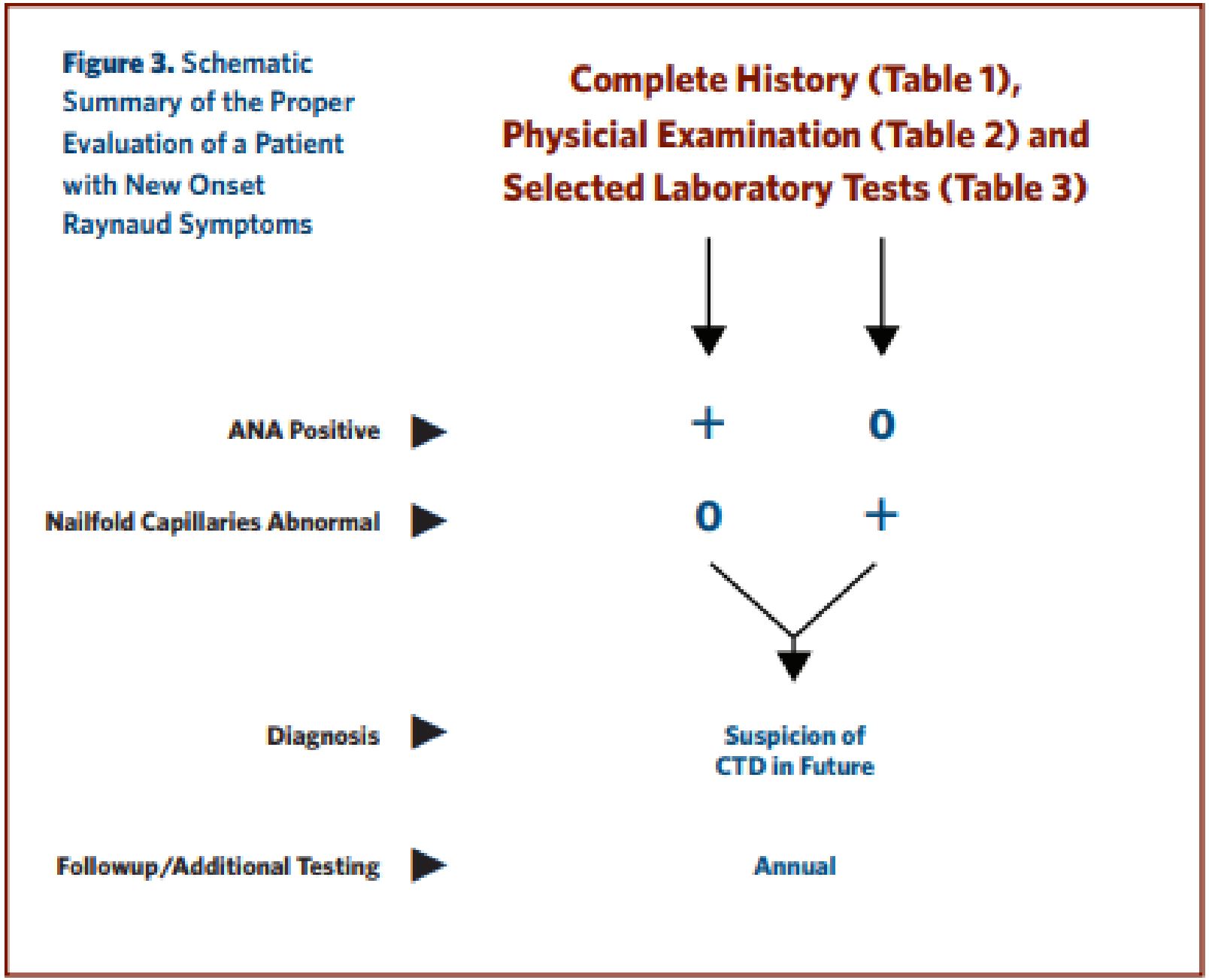
TABLE 3. Laboratory and Special Test Findings Distinguishing RD from RP-CTD

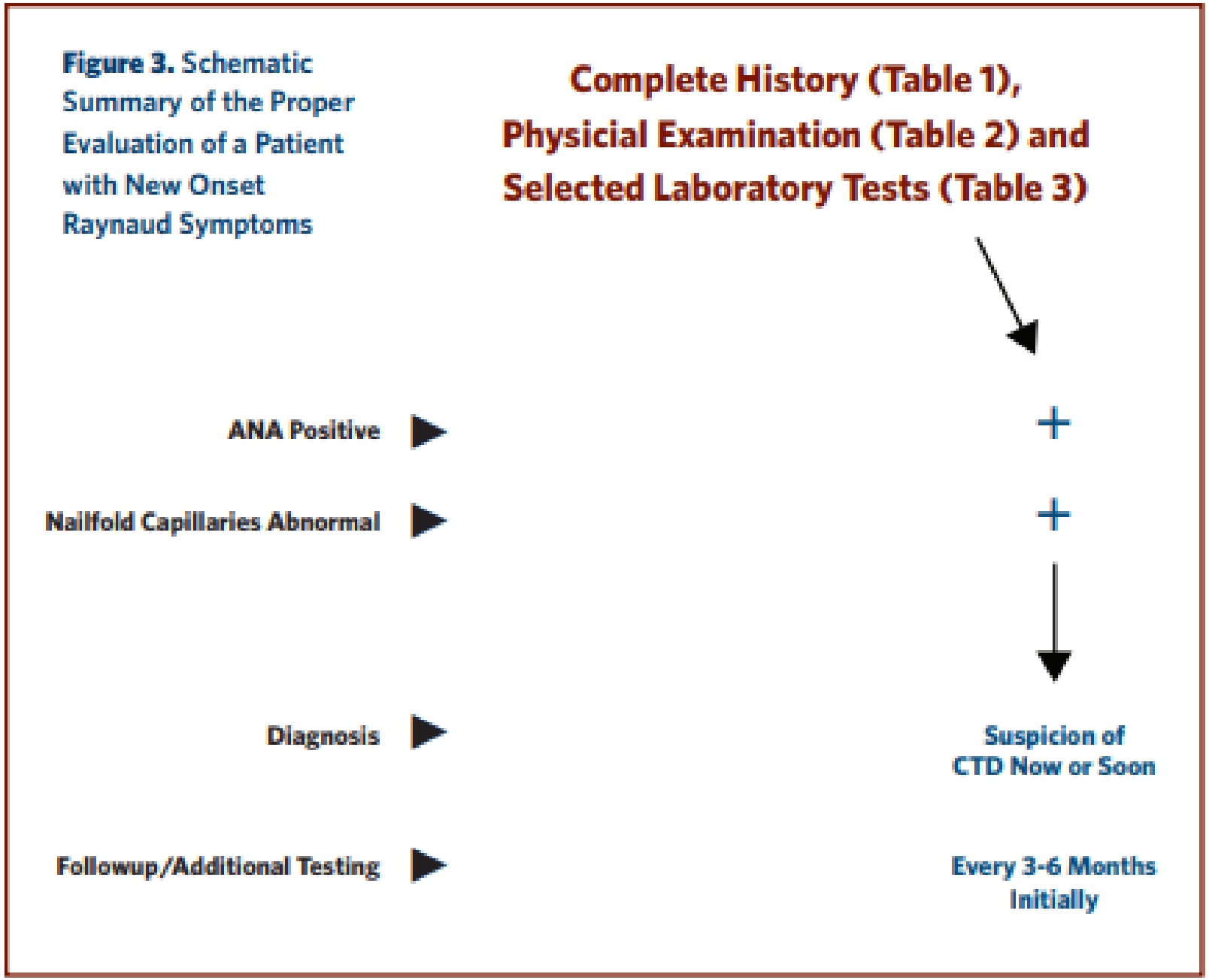
Findings	*RD*	*RP-CTD*	*Associated CTD*
anemia	0	±	all CTDs
leukopenia	0	+	SLE, SS
thrombocytopenia	0	+	SLE
elevated ESR/CRP	0	+	all CTDs
acute renal failure	0	+	SLE, SSc
elevated CPK/aldolase	0	+	PM/DM
positive ANA	0	+	all CTDs
anti-SSA/SSB antibody	0	+	SS
anti-RNA polymerase III antibody	0	+	SSc
anti-centromere antibody	0	+	SSc
anti-Scl 70 antibody	0	+	SSc
anti-Jo1 antibody	0	+	PM/DM
anti-U1RNP antibody	0	+	SSc, SLE, PM/DM
abnormal nailfold capillaries	0	+	all CTDs
abnormal peripheral arterial Dopplers or arteriogram	0	+	all CTDs
distal esophageal hypomotility	0	+	SSc
low C3 and C4 complement levels	0	+	SLE, SS
low C4 complement level only	0	+	SS
abnormal sialogram or lip biopsy	0	+	SS
pulmonary fibrosis	0	+	SSc, PM/DM
microscopic hematuria/proteinuria	0	+	SLE, SSc
abnormal pulmonary function tests	0	+	SSc, PM/DM
pulmonary arterial hypertension	0	+	SSc
cardiomyopathy/pericardial effusion	0	+	SSc, SLE

SLE= systemic lupus erythematosus; SS=Sjogren syndrome; SSc=systemic sclerosis; PM/DM= polymyositis-dermatomyositis

Figure 3. Schematic Summary of the Proper Evaluation of a Patient with New Onset Raynaud Symptoms







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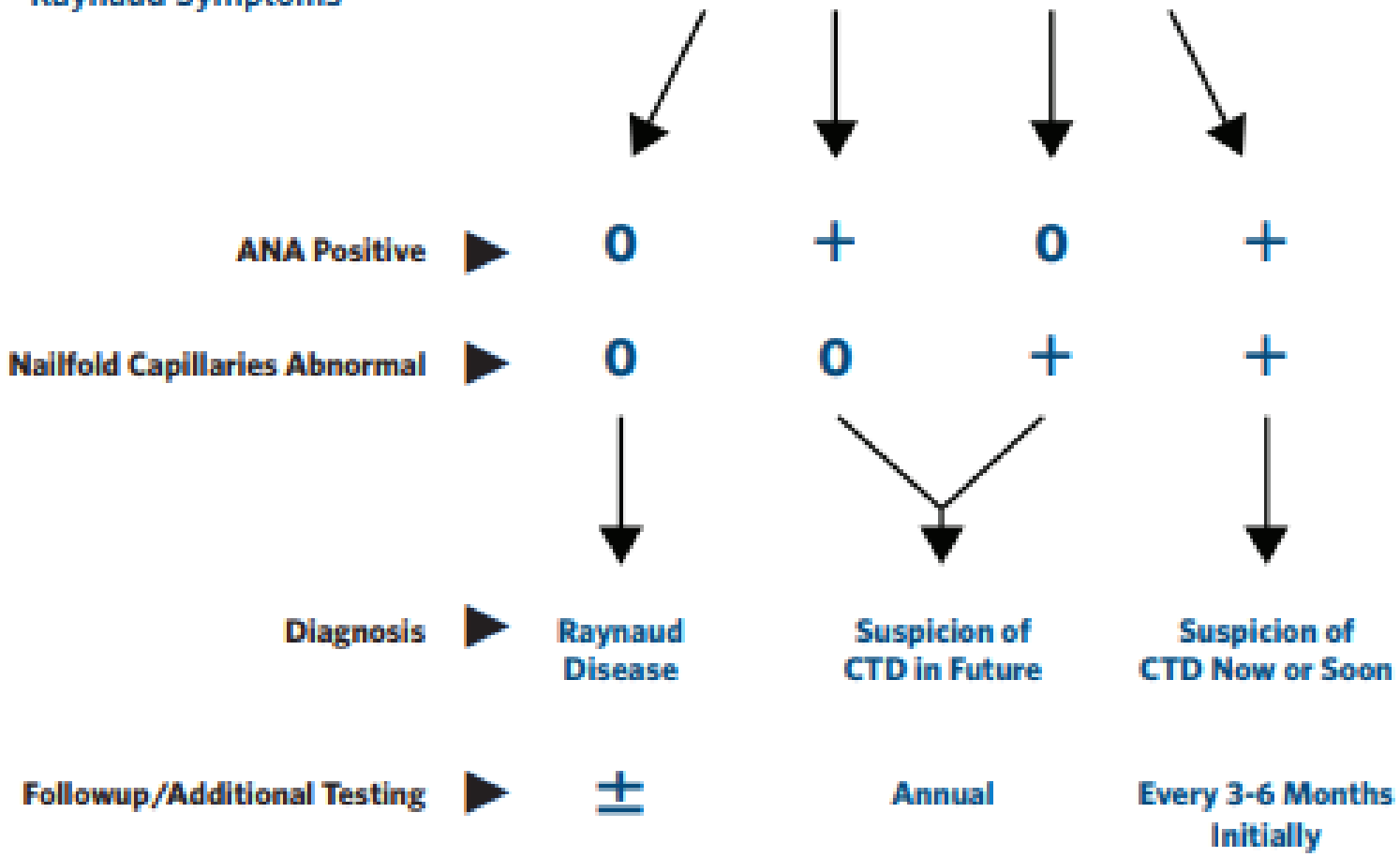
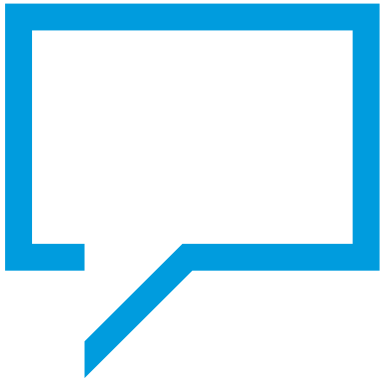


CONCLUSIONS:

- ANA’s are directed against components of nuclei and cells
- ANA tests should be based on:
  - Thorough ROS and exam
  - Family history
  - Demographics
  - Pre-test probability for lupus, scleroderma, sjogrens
- ‘False’ positives: Hashimoto’s, viral infection, normal populations

QUESTIONS & ANSWERS



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