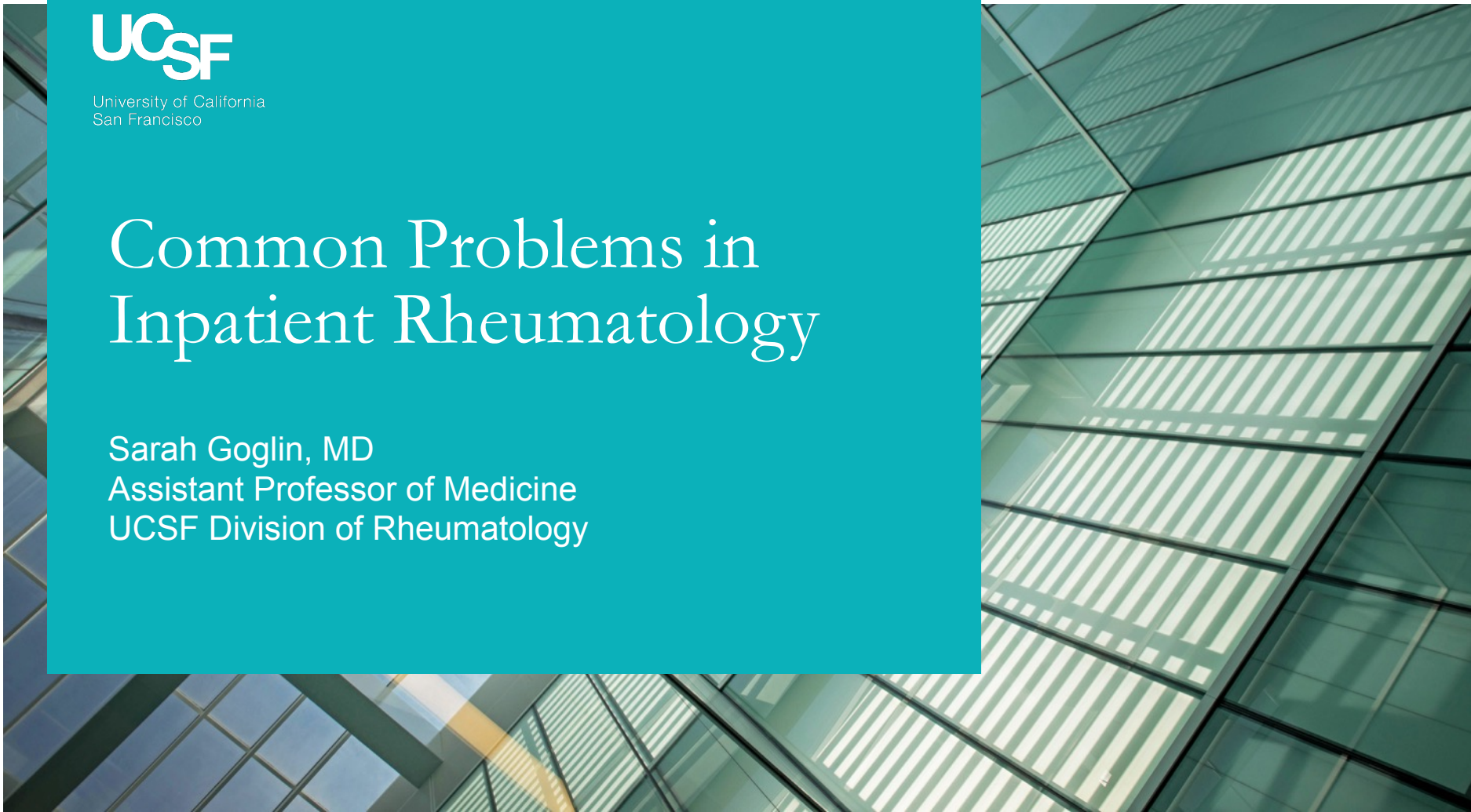
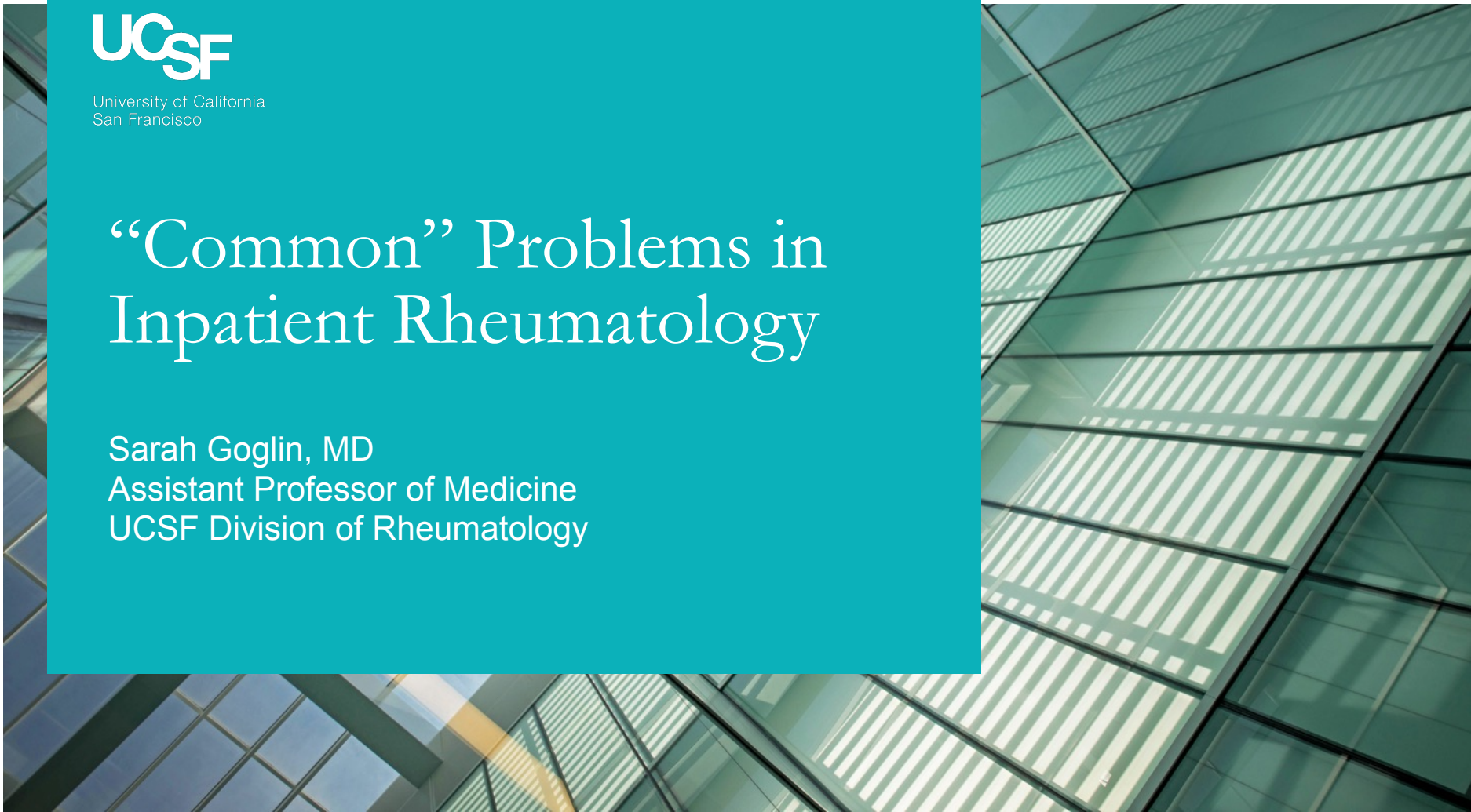


University of California
San Francisco

Common Problems in Inpatient Rheumatology

Sarah Goglin, MD
Assistant Professor of Medicine
UCSF Division of Rheumatology



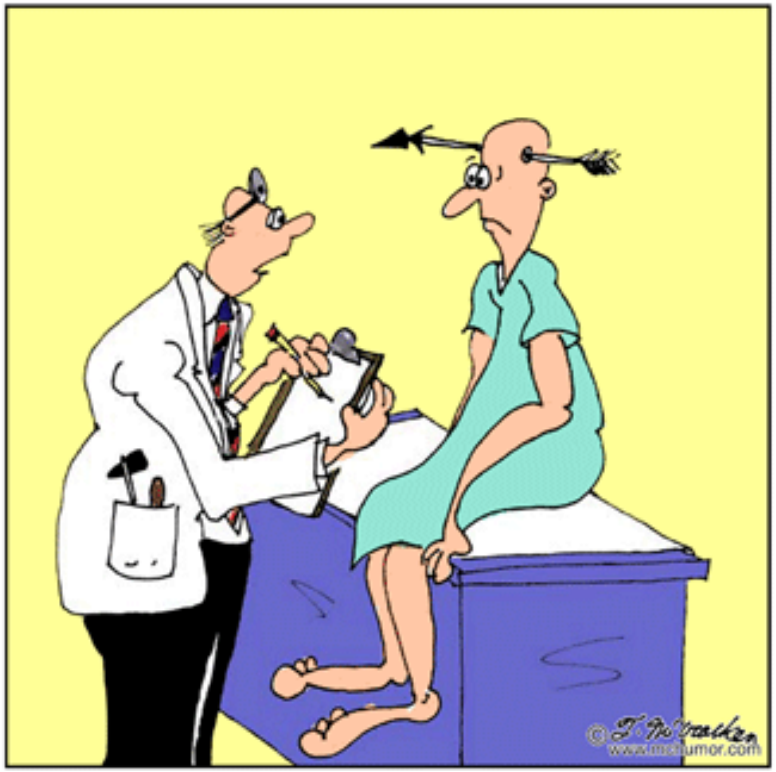
University of California
San Francisco

“Common” Problems in Inpatient Rheumatology

Sarah Goglin, MD
Assistant Professor of Medicine
UCSF Division of Rheumatology

Disclosures

- I have no relevant disclosures
- I will discuss off-label use of medications



"Off hand, I'd say you're suffering from an arrow through your head, but just to play it safe, I'm ordering a bunch of tests."

Objectives

By the end of session, you will:

- Hone your evaluation and treatment of the hospitalized patient with acute inflammatory arthritis
- Develop an approach to the evaluation of a patient with possible vasculitis
- Differentiate lupus flare from non-lupus complications
- Understand complications and adverse effects of some medications commonly used in rheumatology

Case 1: The Swollen Knee

Initial Presentation

72 yo woman admitted 3 days prior with HF exacerbation. Dyspnea has improved with diuresis. Now reports 1 day of left knee pain and swelling.

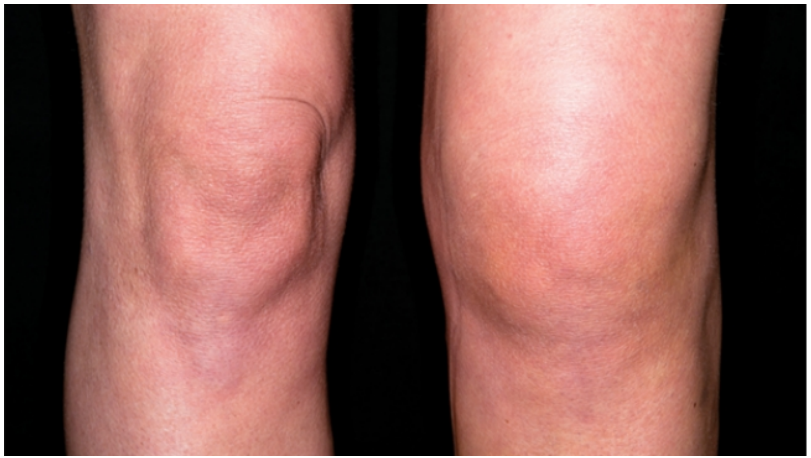
Past Medical History

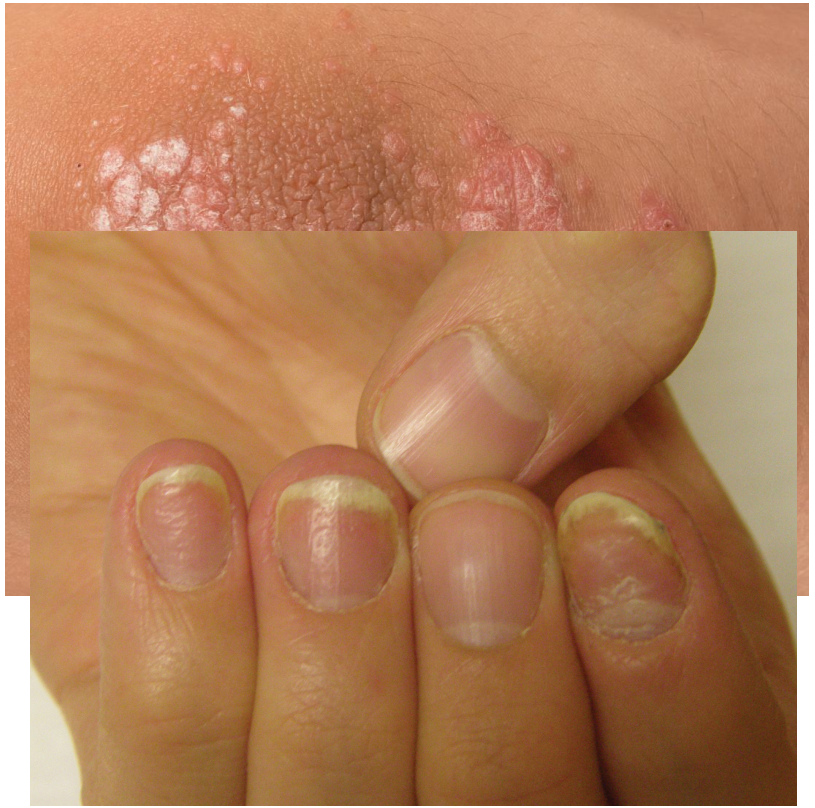
HTN, CAD, HFpEF, CKD (stage 3), Psoriatic arthritis (PsA)

Medications

ACEi, Beta blocker, ASA, IL17Ai (monoclonal antibody biologic for PsA)

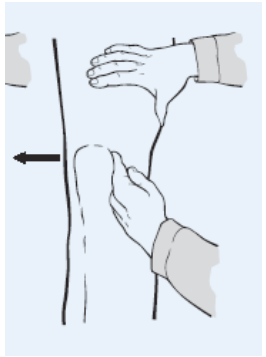
Case

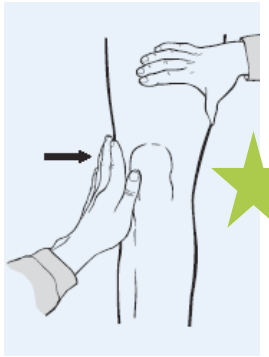




How to identify the inflamed knee

- Temperature: Joints should be cooler than the rest of extremity
- Effusion: Often it's obvious, but if not…Bulge test
  - Squeeze fluid out of the suprapatellar pouch
  - Medial compartment emptied by pressing medial aspect of joint
  - Lift hand away
  - Lateral side sharply compressed
- Ripple seen on the flattened medial surface



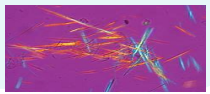


What would you consider in this patient?

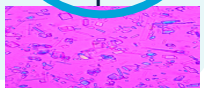
Aspiration of Swollen Joint Narrows ddx

Crystals (+)

Gout
birefringent
-



Acute CPPD /
Pseudo-gout
birefringent
+



Septic Joint

Bacterial
Staph, GC

Atypical
Mycobacteria, spirochete

Fungal

Crystals (-)

RA

Psoriatic Arthritis

Reactive Arthritis

Trauma
Hemarthrosis, meniscal derangement

Osteoarthritis

Patients can have both acute gout/CPPD and a septic joint

DDxof the swollen joint in the hospital

Rule out infection; look for crystals

- **Septic Joint**
  - Immunosuppressed
  - Damaged joint
  - Psoriasis portal of entry
- **Psoriatic Arthritis**
  - Holding meds?
  - Is this typical joint for this patient?
  - Diagnosis of exclusion
- **Gout**
  - Psoriasis
  - Diuresis & low dose ASA
  - CKD
- **Acute CPPD (Pseudogout)**
  - The knee is a good joint
  - CKD (via hyperparathyroidism)

The hot joint: Crystals and Pearls

	Crystals	WBCs/mm^3	Pearls
Septic joint	No	>20,000 (usually >50,000)	Early in process, counts can be lower
Gout	Yes	10,000 - >50,000	
Acute CPPD (Pseudogout)	Sometimes!	10,000 - >50,000	Lab can miss crystals
Rheumatoid Arthritis	No	3,000 – 30,000	Not usually acute
Spondyloarthritis Reactive Arthritis Psoriatic Arthritis	No	 15- > 50,000 3,000 – 30,000	Not usually acute
Non-inflammatory (Osteoarthritis)	No	<2000	Not usually warm

Bacterial Septic Joint

- Prevalence: in adults presenting with >= 1 acutely swollen joint estimated 10%

Feature	Sensitivity, % (95%CI)
Joint pain	85 (78-90)
Joint swelling	78 (71-85)
Fever	57 (52-62)
Night sweats	27 (20-34)
Rigors	19 (15-24)

WBC/microL	LR; 95%CI
<25,000	0.32 (0.23-0.43)
≥25,000	2.9 (2.5-3.4)
>50,000	7.7 (5.7-11.0)
>100,000	28.0 (12.0-66.0)

PMN, %	LR; 95%CI
≥ 90	3.4 (2.8-4.2)
<90	0.34 (0.25-0.47)

Risk factors
Age
IDU
DM, RA, HIV
Joint surgery; prostheses
Skin infection

Margaretten ME, Kohlwes J, Moore D, Bent S; JAMA. 2007;297(13):1478.

Final Diagnosis

- Synovial fluid shows 45,000 WBC, 90% PMNs, +MSU crystals
- Cultures remained negative
- Knee was injected with triamcinolone acetate 40mg

- Synovial fluid shows 45,000 WBC, 90% PMNs, +MSU crystals
- Cultures grew MSSA
- Antibiotics started
- Knee was washed out

Decision to wait on cultures to treat vs. injecting and watching cultures
Generally, if cultures cooking, ok to inject unless signs/risk factors high for infection

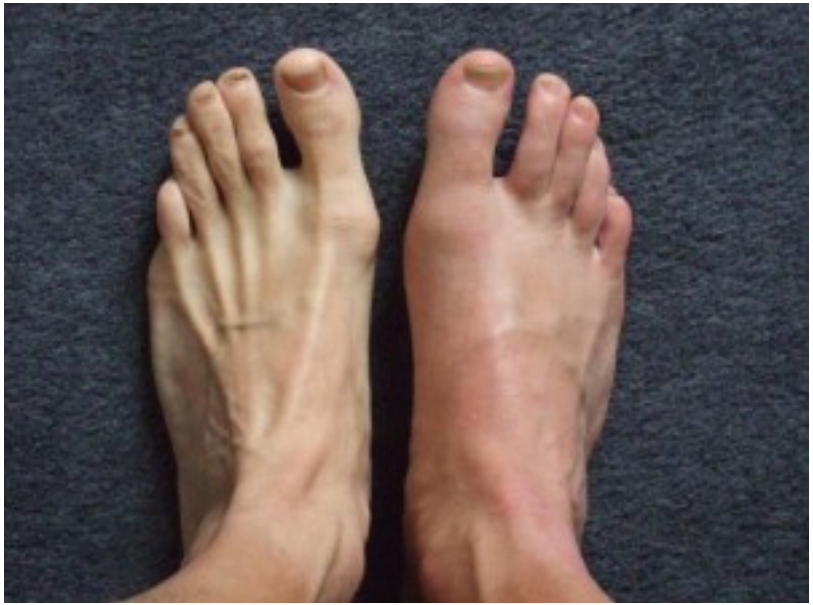
Gout

- Most common inflammatory arthritis in US
  - 8.3 million adults
  - Increased hospitalization rates
  - Increased economic burden
- High rates of cardiovascular disease & metabolic syndrome

Zhu Y et al. Arth Rheum 2011; 63:3136-41; Lim SY et al. Ann Rheum Dis 2012; 71:1765-70; Kuo CF Nat Rev Rheumatol 2015; 11:649-62; Choi HK et al. Arth Rheum 2007;57:109-15; Zhu Y et al. Am J Med 2012:125(7) 679-87

Case, continued

- Knee improves with triamcinolone injection.
- Then develops right foot pain and swelling.
- Creatinine has been rising over past 48 hours.



What treatment do you recommend?

Naproxen 500 mg BID

Colchicine 1.2 mg PO, followed by 0.6 mg every hour x 3

Prednisone 40 mg PO daily

Intra-articular injection of triamcinolone into right ankle

I don't really like any of these options

Acute Gout Treatment

- NSAIDs: often contraindicated in hospitalized patients
- GC Injection: great for a single joint
- Prednisone: slower taper to lower risk of "rebound" flare
- Colchicine: As effective as other agents if taken within 24 hours of flare. Do not exceed 1.8 mg on day one.
  - 1.2 mg for the first dose followed by 0.6 mg an hour later
  - On subsequent days, 0.6mg 1-2x/day until flare resolution

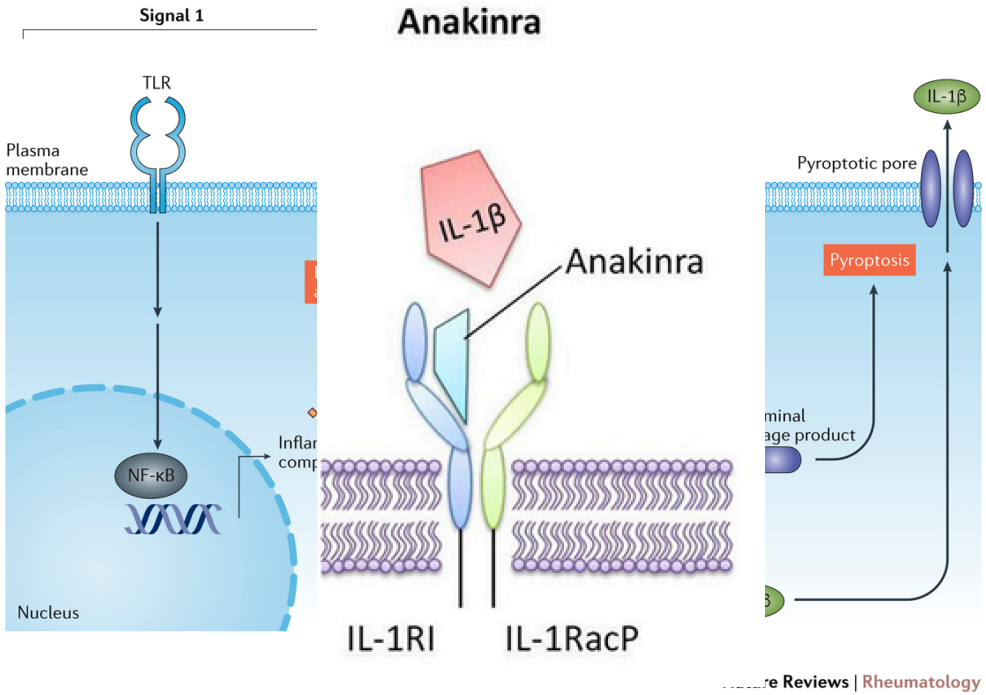
Acute Gout Treatment

- Complete treatment cessation for gout flare usually done within 2-3 days of flare resolution (except prednisone)

Time since symptoms	Typical treatment course
within hours	1-2 days
12-36 hours	5-7 days
4-5 days	1-4 weeks

Biologic options for acute gout

- Interleukin-1 is a major player in gout inflammation
- Anakinra is an IL-1β antagonist - effective in acute gout



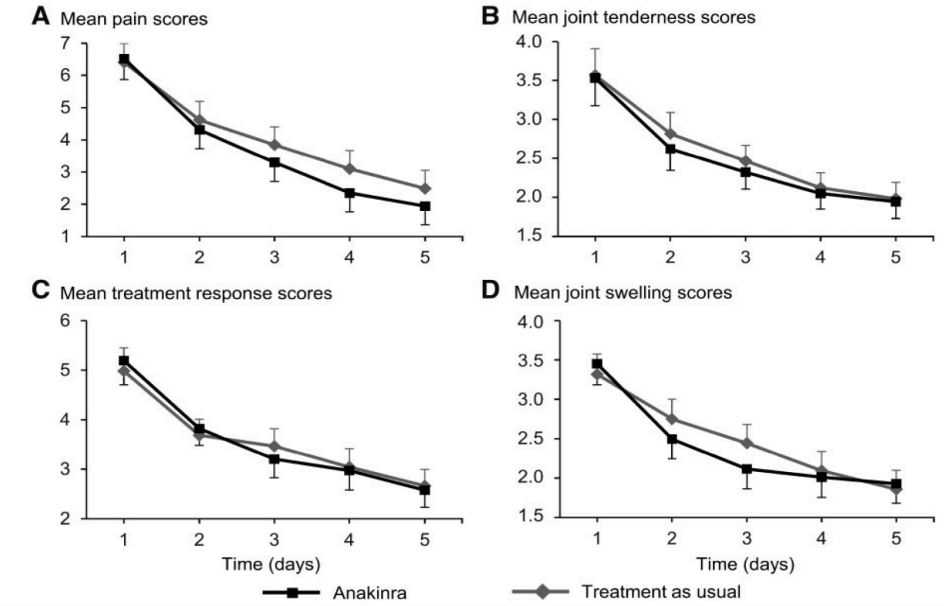
Anakinra is not FDA approved for gout

Original article

Anakinra for the treatment of acute gout flares: a randomized, double-blind, placebo-controlled, active-comparator, non-inferiority trial

- 88 patients with acute gout were randomized 1:1 to treatment with anakinra (100 mg SQ daily x 5 days) plus oral placebo vs. standard care (colchicine, naproxen or prednisone) plus placebo injection
- Primary endpoint was change in mean score; anakinra was non-inferior
- No serious adverse events

FIG. 3 Mean scores on days 1–5 (bars represent one-sided 95% CI) of the secondary outcomes (panel **A**–**D**)



Anakinra for the treatment of acute gout

- For now, consider in the following circumstances:

  1. For hospitalized patients with (relative) contraindications to standard therapies
     - Diabetes
     - CKD
     - Osteoporosis
     - Delirium

  2. For hospitalized patients refractory to standard therapies

- Currently is not FDA-approved so very difficult to get for patients in the ambulatory setting

Our patient is started on allopurinol 100 mg daily and colchicine after discharge. A month later, you are called by the ED physician to admit her for fever, diffuse rash and transaminitis. What are you concerned about?

Allopurinol Hypersensitivity Syndrome

- Incidence ~1/1000 in US
- Spectrum of disease including not only Stevens-Johnson Syndrome and toxic epidermal necrolysis but also systemic disease with eosinophilia, vasculitis, hepatitis, and renal failure
- Reported mortality rate 20-25%
- Risk factors
  - Renal impairment
  - Recent initiation of allopurinol
  - Starting dose of allopurinol: should be no greater than **100 mg/day** for any patient, and start at **50 mg/day in stage 4 or worse CKD.** “Start low and go slow”
  - Ethnicity (related to pharmacogenetic factors)

Allopurinol and HLA-B*5801

- Associated with increased risk for allopurinol hypersensitivity reaction
  - Risk is 0.1-0.4% in general population
  - OR in one Thai series 348.33 (95% CI 19.15 – 6336.88)
- Allopurinol is contraindicated in patients who are carriers for this allele
- Most commonly found in patients of Korean, Chinese, or Thai descent (6-12%)
- Allele frequency in the US: 7.4%, 4%, 1% and 1% among Asians, Blacks, Whites, and Hispanics, respectively

Which of the following is true?

Allopurinol can cause AKI

When allopurinol dose is increased beyond 300 mg/day, the risk of AHS is increased

Febuxostat does not cause a hypersensitivity reaction

Colchicine can cause myelosuppression

Adverse Reactions with Febuxostat

- Transaminitis 5-7%
- Risk of hypersensitivity reaction increased in those with past allopurinol hypersensitivity
- Possible increased vascular and all-cause mortality (compared to allopurinol)

Ridker PM et al. N Engl J Med 2017;377:1119-31

Gout medication pearls

- Allopurinol
  - does not cause AKI
  - is renally cleared but there is no maximum dose in CKD

- Colchicine
  - does not cause AKI
  - is renally cleared and needs to be renally dosed

Singh JA Ann Rheum Dis 2017; 76:113-139 Khanna D et al. 2012 Arth Care Res (Hoboken) 64, 1447-61 Vargas-Santos AB et al. Arthritis Rheumatol. 2016; 68 (suppl 10)

What to do with gout medications in the admitted patient

Allopurinol (or febuxostat)

- Do not stop unless absolutely indicated
  - Hypersensitivity reaction
  - Significant transaminitis
- Consider dose reduction with GFR < 30

Colchicine

- Stop in the setting of
  - AKI
  - Unexplained myopathy or neuropathy
  - Unexplained severe cytopenias

Urate flux => flare

- Stopping/restarting urate lowering therapy increases risk of flare
- In a flare, continue urate lowering therapy

Singh JA Ann Rheum Dis 2017; 76:113-139; Khanna D et al. 2012 Arth Care Res (Hoboken) 64, 1447-61; Vargas-Santos AB et al. Arthritis Rheumatol. 2016; 68 (suppl 10)

Case

- 31 yo man presented with subacute dyspnea and cough. He felt well until two months ago, when he developed fatigue and migratory arthralgias and myalgias. He also noted right eye pain and redness and significant bloody nasal crusting.

Vital Signs

- T 36.8 BP 103/63 P: 90-110 RR 18 Sp02 90% RA

Constitutional

- thin but otherwise well appearing

HEENT

- Poor dentition. No oral ulcers
- + scleral injection. No cervical adenopathy

Cardiac

- Irregularly irregular. No murmurs
- 2+ edema right leg

Pulmonary

- Inspiratory bibasilar crackles

Gastrointestinal

- Soft, NT, no hepatosplenomegaly

Musculoskeletal

- No synovitis, including no joint tenderness or swelling

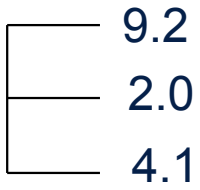
Neuro

- A&O x 3. Motor and sensory exam intact
- Motor strength 5/5 upper/lower extremities

Skin

- No rashes, nodules or purpura

Labs

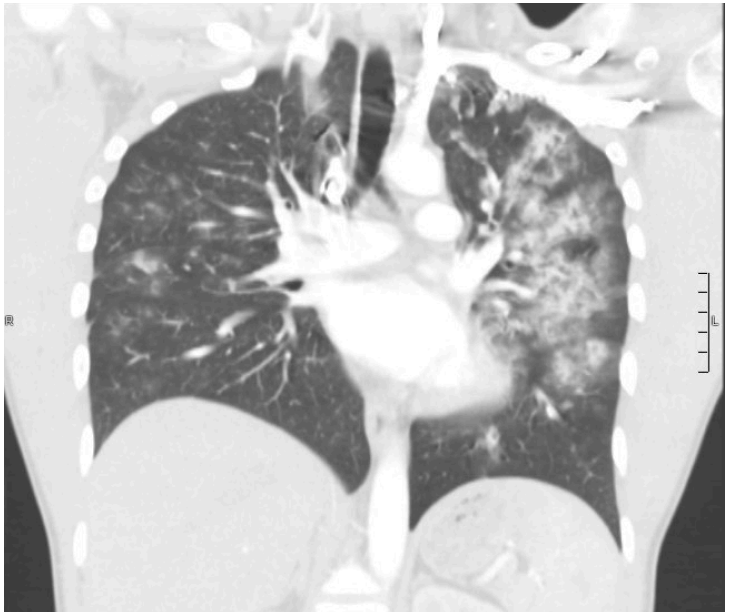


Micro:
Blood cx: negative
HIV negative
HBV and HCV negative
QTF negative

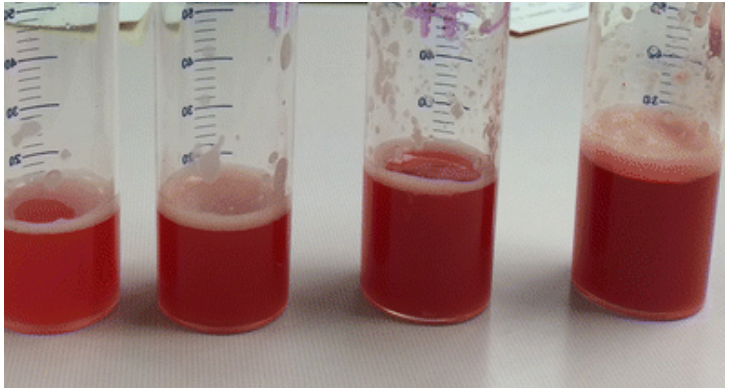
ESR >100
CRP 50

Albumin 3.2
total protein 7.9

Case



- Hb continued to decline, hypoxia worsens
- Pulm consulted and bronch done



What labs would you order?

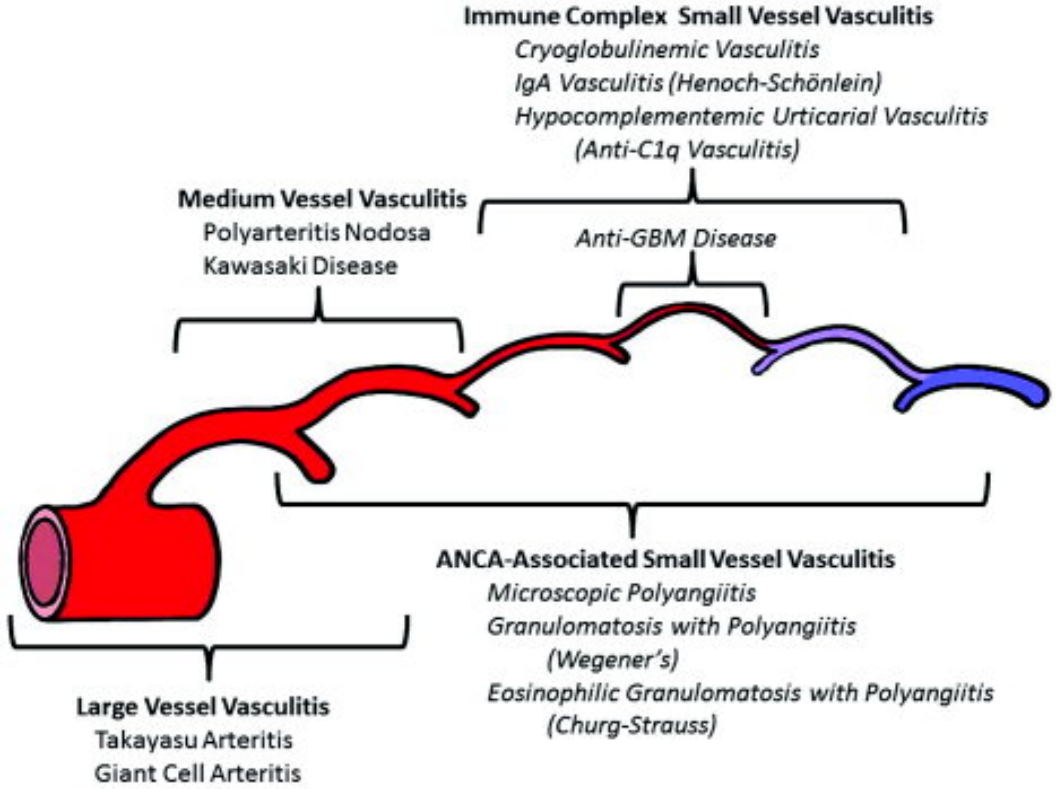

PANIC

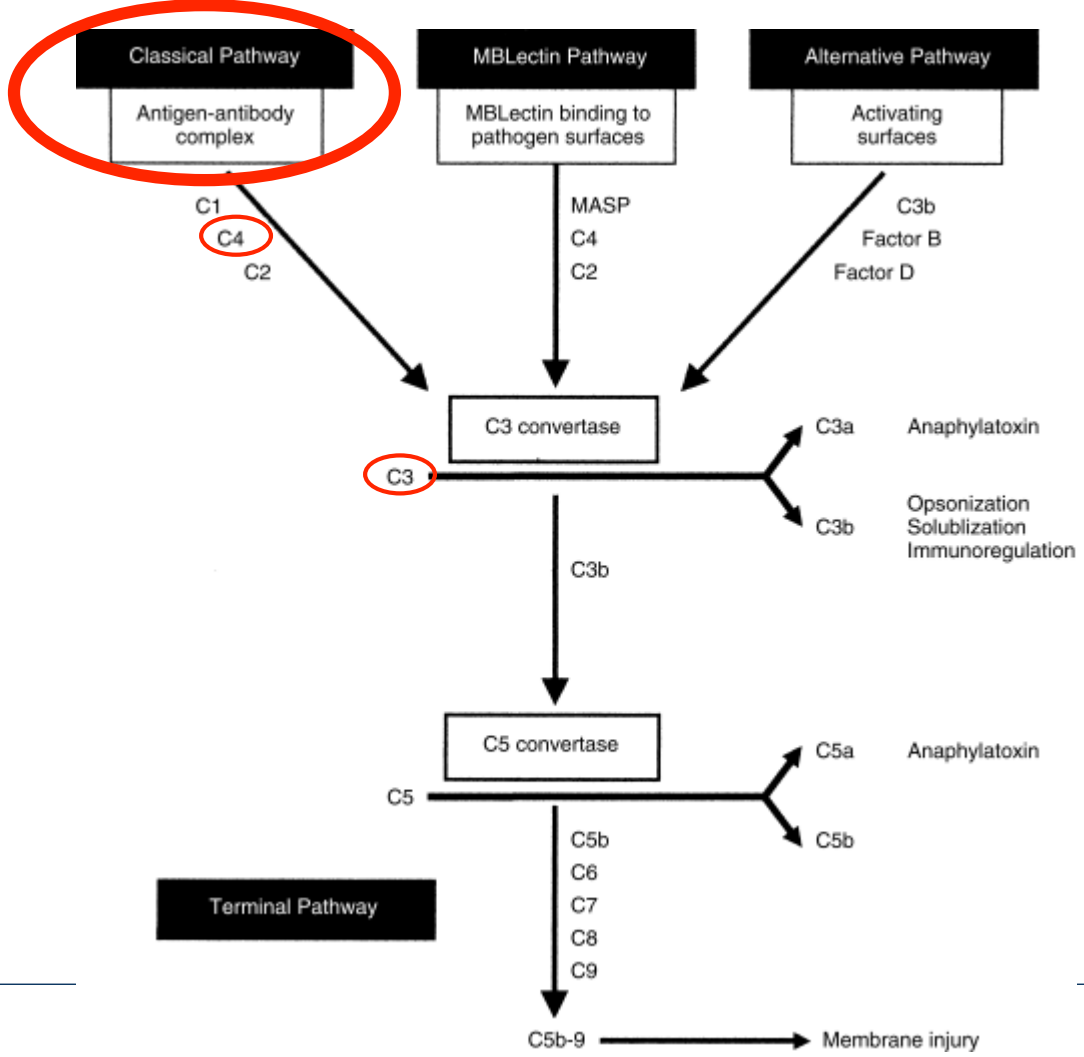

PANIC

Evaluating the patient with possible vasculitis

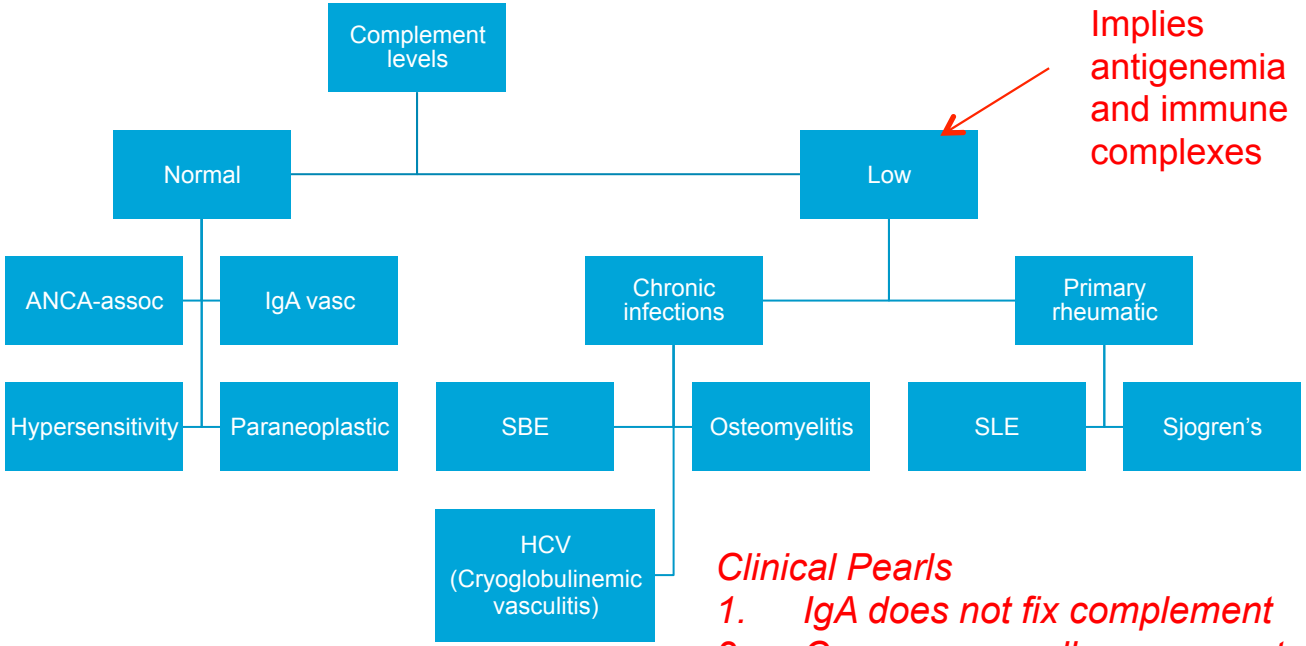
- Decide what size vessel you are concerned about
- Look for evidence of other organ involvement to support diagnosis of vasculitis
- Look for mimics (infection, malignancy, toxins, etc.)
- Send complements to determine if immune complex mediated or not
- Send appropriate serologies
- Involve rheumatology early!

Vasculitis schema by vessel size




Classical Pathway
Antigen-antibody complex
MBLectin Pathway
MBLectin binding to pathogen surfaces
Alternative Pathway
Activating surfaces
C1
C4
C2
MASP
C4
C2
C3b
Factor B
Factor D
C3 convertase
C3
C3a
Anaphylatoxin
C3b
Opsonization
Solublization
Immunoregulation
C3b
C5 convertase
C5
C5a
Anaphylatoxin
C5b
C5b
C6
C7
C8
C9
Terminal Pathway
C5b-9
Membrane injury

Diagnostic Schema – Small Vessel Vasculitis



Clinical Pearls

1. *IgA does not fix complement*
2. *Cryo vasc usually causes extremely low C4*

UCSF

Diffuse Alveolar Hemorrhage

H&P and basic labs may provide clues to diagnosis

ENT, skin, MSK signs/symptoms

Thrombosis, pregnancy morbidity hx

CBC (leukopenia vs. leukocytosis, thrombocytopenia vs. thrombocytosis)

Cr

HCV ab

UA with micro

C3 and C4 (low in immune complex mediated diseases**)

Diagnostic work up

ANCA by IFA, MPO/PR3 by ELISA (**AAV**)

Cryoglobulins (**Cryoglobulinemic vasculitis****)

ANA (**SLE****)

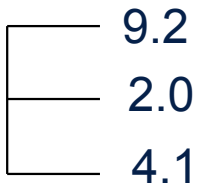
Anti-GBM abs (**Anti-GBM disease**)

APL abs: LAC (dRVVT, HEXA), anticardiolipin IgM and IgG, anti-beta 2 glycoprotein 1 IgM and IgG (**APLS**)

Skin biopsy (**IgA vasculitis**)

Urine tox (**Levamisole vasculopathy**)

Labs



Micro:
Blood cx: negative
HIV negative
HBV and HCV negative
QTF negative

ESR >100
CRP 50

Utox: negative

UA: 11 RBCs, 2 WBC, 100 mg/dl protein, few dysmorphic RBCs
UPCR: 1.89 grams

C3 101
C4 15

ANA 1:320
speckled

PR3>3000

Albumin 3.2
total protein 7.9

IgG 1723 (H)
IgA 508 (H)
IgM 105 (wnl)

SPEP/IFE: no paraprotein

ANA sub-serologies (-)

UCSF

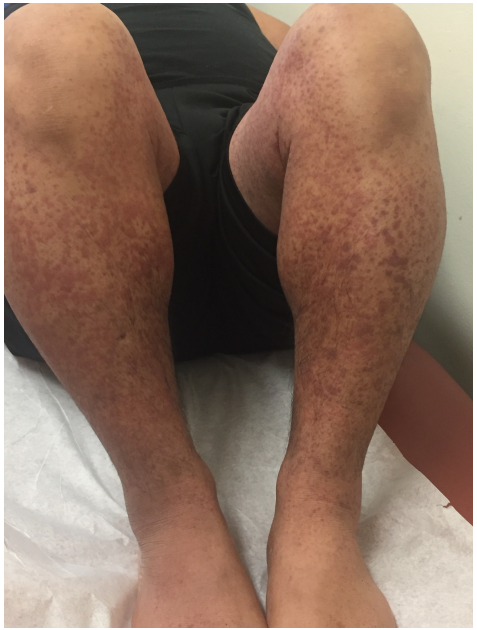
When should you think about vasculitis?

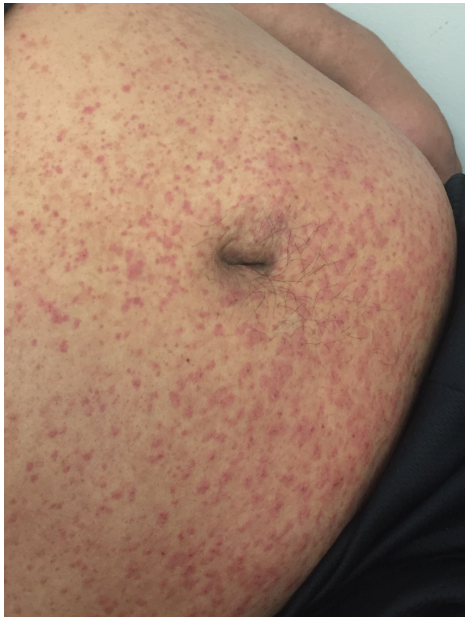
- Constitutional symptoms (ever, weight loss, fatigue)
- Multi-organ involvement

	Large Vessel	Medium Vessel	Small Vessel	Behcet's disease
Pulmonary	-	-	DAH, pulmonary nodules	Pulmonary aneurysms
Renal	-	Infarcts	GN	-
Skin	Scalp necrosis	Ulcers, nodules	Palpable purpura	E nodosum
Neuro	CVA, headache	Mononeuritis multiplex or peripheral neuropathy		Neuro-Behcet's or thrombosis
Ocular	Amaurosis fugax, diplopia	- -	Scleritis, retinal vasculitis	Uveitis

Small vessel vasculitis

Cutaneous manifestations

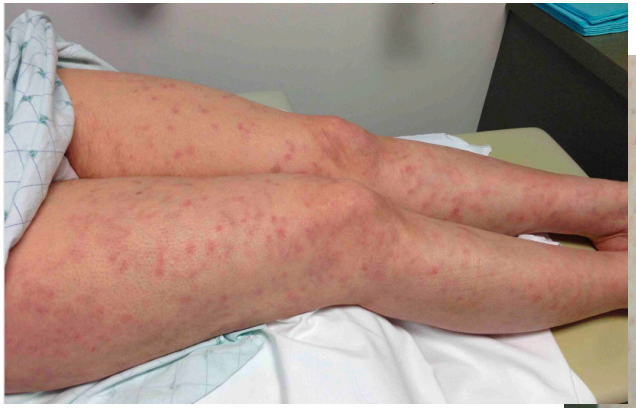




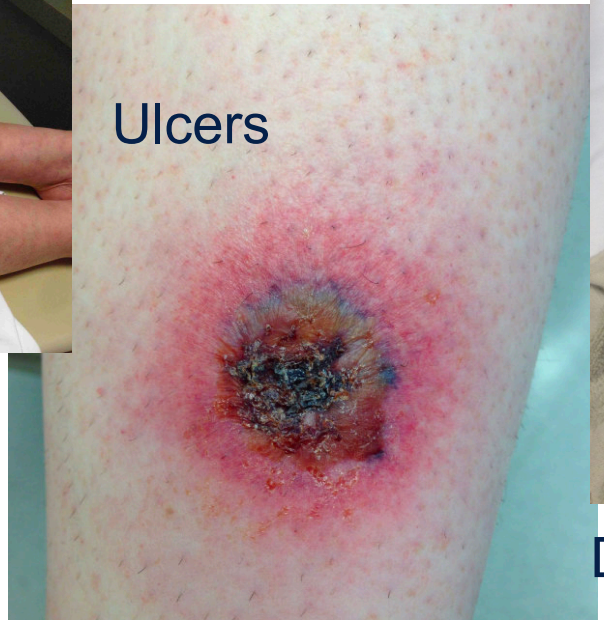
Palpable purpura

Medium vessel vasculitis

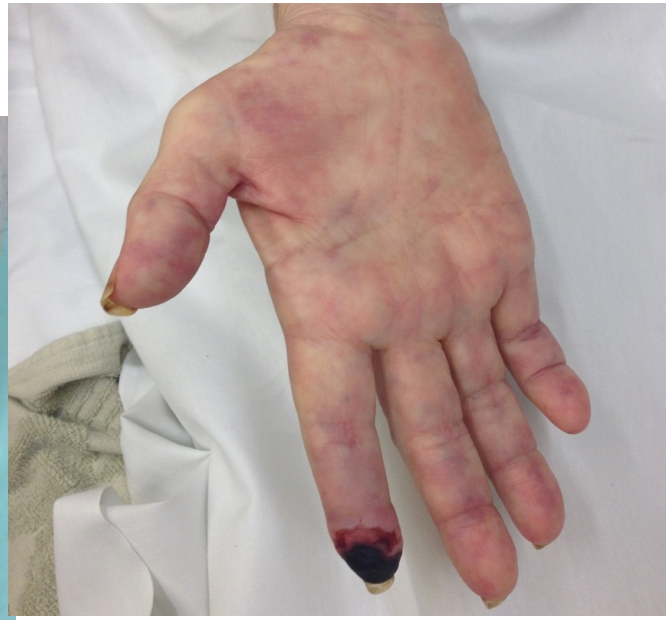
Cutaneous manifestations



Nodules



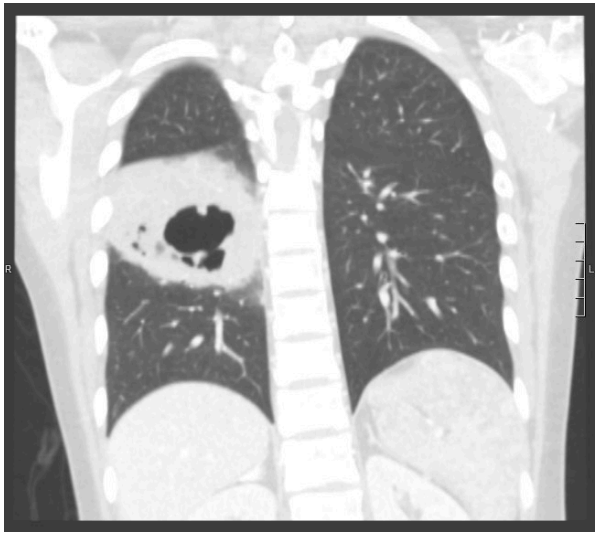
Ulcers



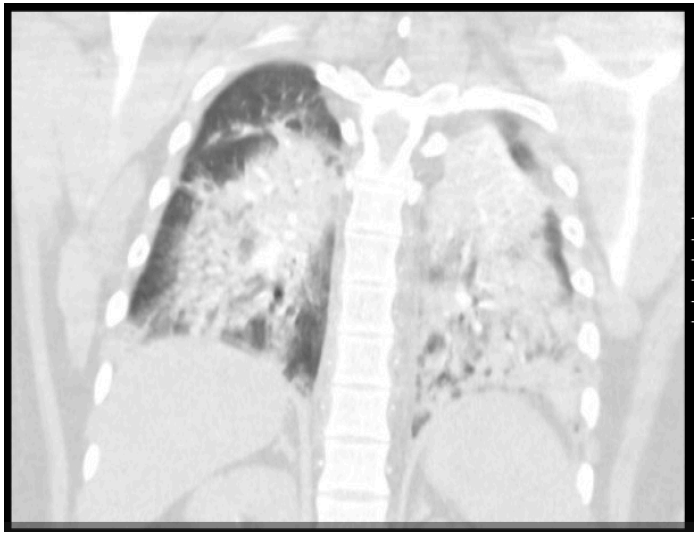
Digital infarcts

Small vessel vasculitis

Pulmonary manifestations



Nodules (GPA)



Diffuse alveolar hemorrhage

Asthma (severe, adult-onset), fleeting infiltrates = EGPA

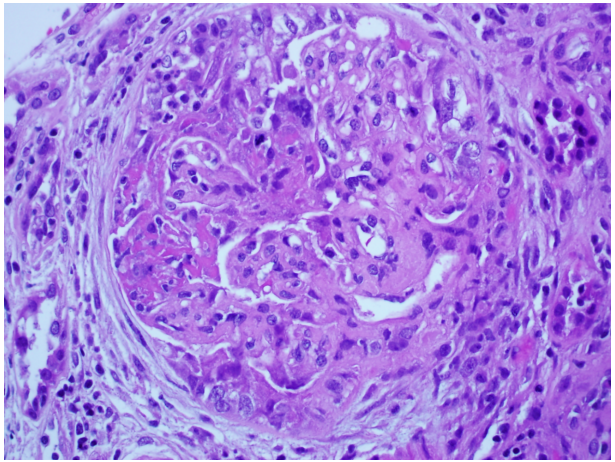
Medium vessel vasculitis

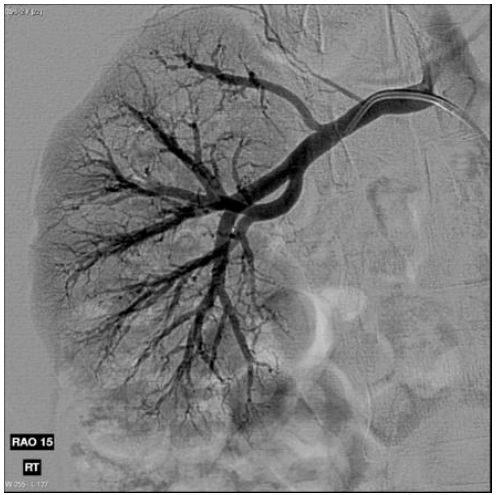
Pulmonary manifestations

- None!

Small versus medium vessel vasculitis

Renal manifestations





Small vessel vasculitis

- **Glomerulonephritis**
- Clinical features: HTN, proteinuria, hematuria, renal failure (but not always, esp early in course)

Medium vessel vasculitis

- **Renal artery aneurysms**
- Clinical features: HTN, occasionally hematuria, NO PROTEINURIA

Granulomatosis with polyangiitis

- Granulomatous inflammation involving respiratory tract and necrotizing vasculitis affecting small-medium sized vessels
- Classic triad:
  - Necrotizing granulomas of upper/lower respiratory tract
  - Necrotizing/granulomatous vasculitis, usually of small-medium arteries & veins (almost always in the lungs)
  - Focal, segmental necrotizing glomerulonephritis
- Epidemiology
  - Prevalence: 30-60 cases/million
  - Mean age at presentation ~40 years (but also seen in children and elderly)
  - ? More prevalent in those of European descent

Microscopic polyangiitis

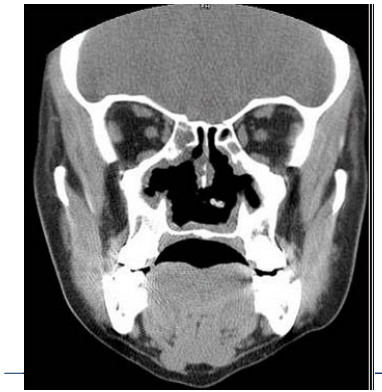
- Non-granulomatous necrotizing vasculitis affecting small-medium sized vessels

- Necrotizing vasculitis, usually of small-medium arteries & veins (almost always in the lungs)
- Focal, segmental necrotizing glomerulonephritis

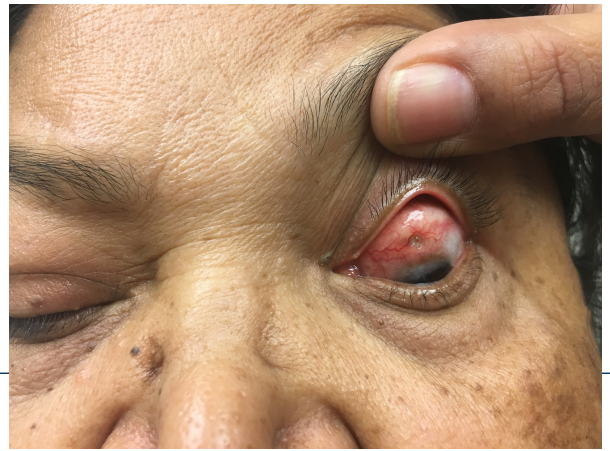
- Epidemiology
  - Prevalence: 10-30 cases/million
  - Men:women 1.8:1
  - Mean age at presentation ~50-60 years

AAV: Widely recognized manifestations

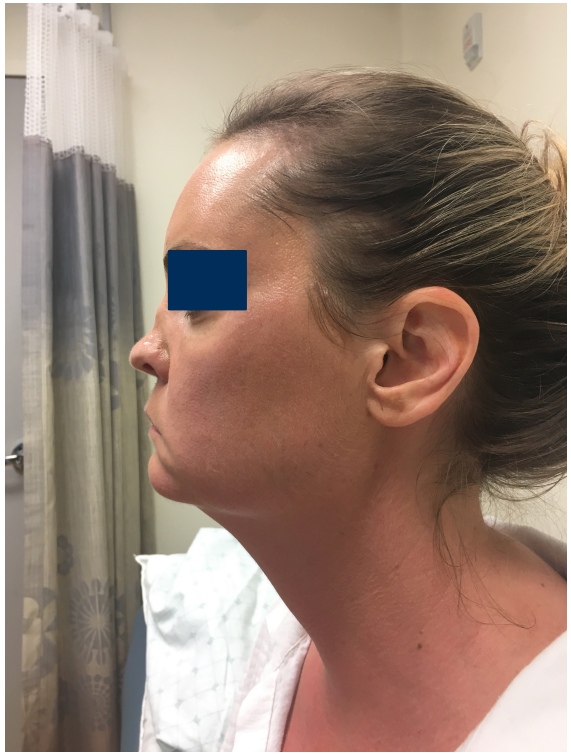
- HEENT (GPA)
  - Sinonasal: destructive sinusitis, epistaxis, nasal crusting, saddle nose
  - Oropharynx: oral ulcers
  - Trachea: subglottic stenosis
  - Ear: otitis media, mastoiditis, hearing loss
  - Ocular: pseudotumor, scleritis



Destruction of the nasal septum and medial walls of the maxillary sinuses



Nodular scleritis with ulceration

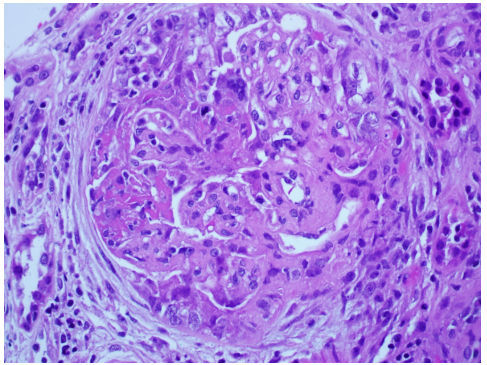


Saddle nose deformity

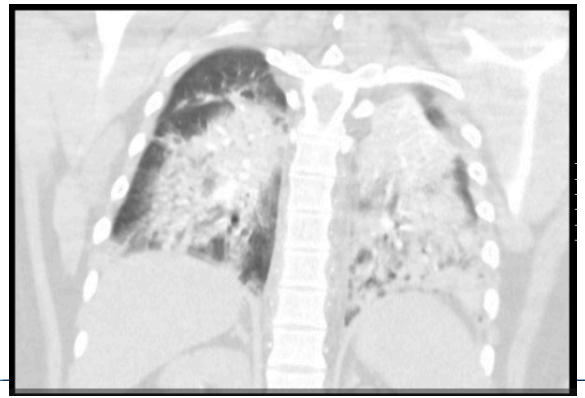
UCSF

AAV: Widely recognized manifestations

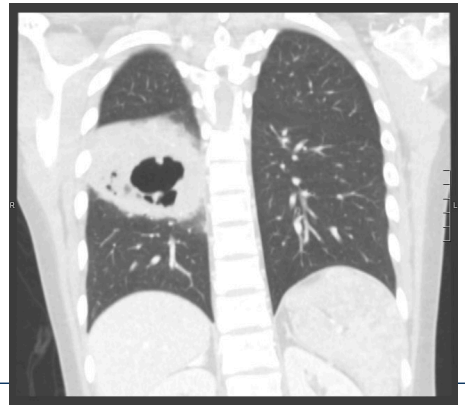
- Lungs: cavitary and non-cavitary nodules (GPA) and pulmonary capillaritis
  - Dyspnea, hemoptysis
- Renal: pauci-immune glomerulonephritis
  - Hematuria, proteinuria, AKI
- Neurologic: mononeuritis multiplex



Pauci-immune GN



Diffuse GGO in patient with DAH



Cavitary pulmonary nodules

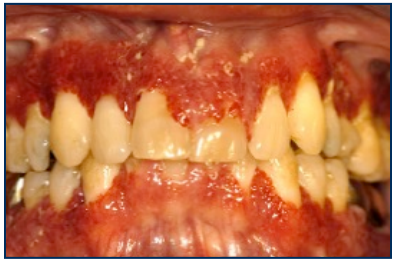
Clinical Pearls

1. *Hemoptysis occurs only in ~50% patients with DAH*
2. *Cr often normal in early GN*

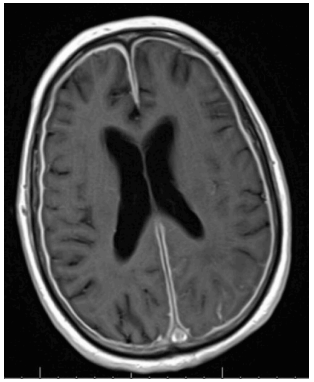
UCSF

AAV: Less commonly recognized manifestations

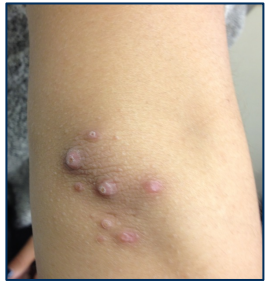
- Oral: hyperplastic gingivitis
- External ear: chondritis
- Ocular: peripheral ulcerative keratitis
- Neurologic: pachymeningitis
- Skin: “Churg-Strauss” granuloma
- Vascular: thrombosis



“strawberry gums”



Pachymeningitis



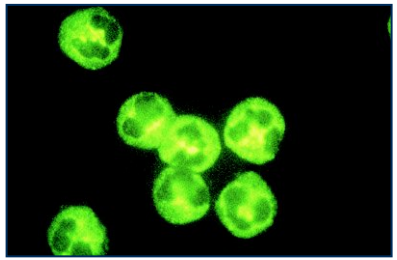
“Churg-Strauss” granuloma

ACR Rheumatology Image Bank; Onyeuku NE et al J Neurol Sci 2014; Merkel PA et al. Ann Intern Med 2009; Personal collection

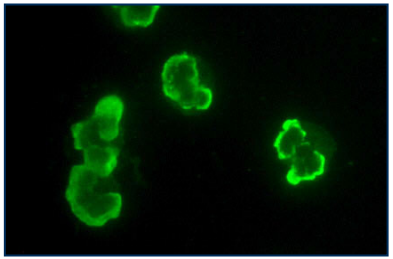
UCSF

AAV: Labs/Diagnostic tests

- ANCA
  - Immunofluorescence: p-ANCA, c-ANCA
  - ELISA: anti-myeloperoxidase (MPO), anti-proteinase 3 (PR3)
- Creatinine, urinalysis: Hematuria with proteinuria, dysmorphic RBCs, RBC casts
- Chest CT: cavitary pulmonary nodules, alveolar hemorrhage
- Biopsy (kidney, lung): document vasculitis prior to therapy; sinonasal biopsies are usually not helpful



c-ANCA



p-ANCA

ANCA testing

Vasculitis type	Formally known as	ANCA +	Typical ANCA Fluorescence	Myeloperoxidase (MPO) & Anti-Proteinase 3 (PR3)
Granulomatosis with Polyangiitis (GPA)	Wegener's Granulomatosis	90% systemic 40% limited	C-ANCA	PR3 > MPO
Microscopic Polyangiitis (MPA)	--		P-ANCA	MPO > PR3
Eosinophilic Granulomatosis with Polyangiitis (EGPA)	Churg-Strauss Syndrome	40%	P-ANCA	MPO > PR3

Other considerations	Typical ANCA Fluorescence	MPO	PR3
Levamisole	P-ANCA	++++	++
IBD	P-ANCA (atypical)		

Vital Signs

- T 36.8 BP 103/63 P: 90-110 RR 18 Sp02 90% RA

Constitutional

- thin but otherwise well appearing

HEENT

- Poor dentition. No oral ulcers
- + scleral injection. No cervical adenopathy

Cardiac

- Irregularly irregular. No murmurs
- 2+ edema right leg

Pulmonary

- Inspiratory bibasilar crackles

Gastrointestinal

- Soft, NT, no hepatosplenomegaly

Musculoskeletal

- No synovitis, including no joint tenderness or swelling

Neuro

- A&O x 3. Motor and sensory exam intact
- Motor strength 5/5 upper/lower extremities

Skin

- No rashes, nodules or purpura

Inflamed Vessels = More thrombosis

Brief Communication: High Incidence of Venous Thrombotic Events among Patients with Wegener Granulomatosis: The Wegener's Clinical Occurrence of Thrombosis (WeCLOT) Study

Condition	Events per 100 person-years
GPA	7
SLE	1
General population	0.3

Merkel PA et al. Ann Intern Med. 2005;142:620-626.

Case

- Your patient achieves remission with rituximab and prednisone
- Two months after rituximab infusion, he develops fevers to 102 and a dry cough and is admitted to your service
- CT chest reveals a 2 cm RUL cavitary pulmonary nodule

This patient is at increased risk for all of the following EXCEPT:

PJP

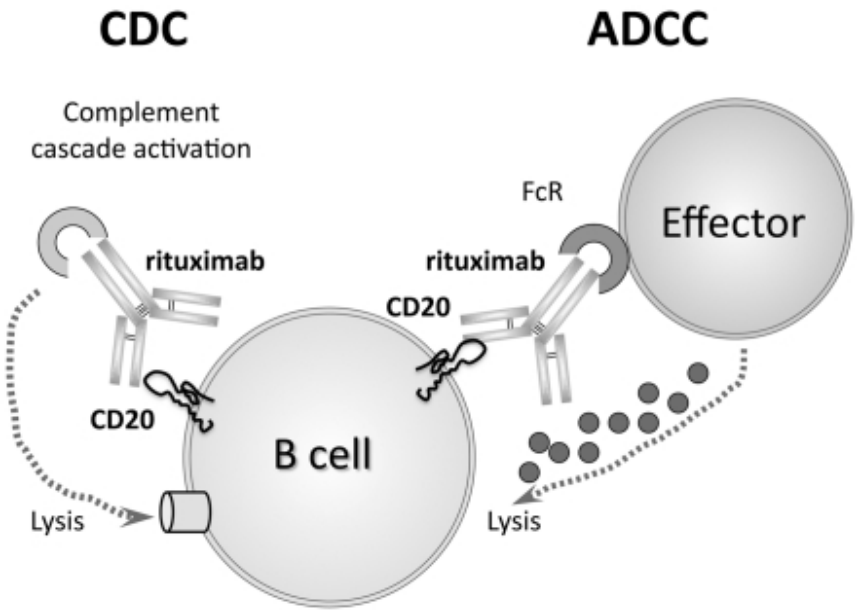
Reactivation of latent TB

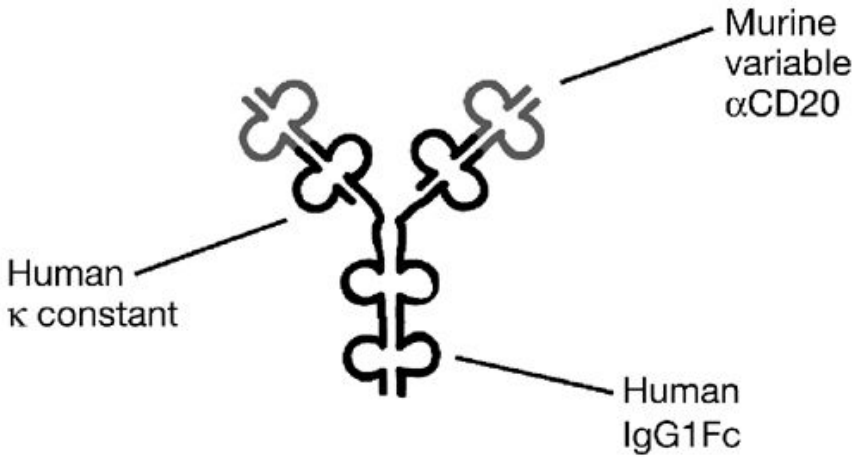
Reactivation of latent HBV

Endemic fungal infections

Rituximab

Chimeric murine/human monoclonal antibody that binds to CD20





Infectious issues and rituximab

- **Hypogammaglobulinemia:** AAV patients treated with rituximab have more prolonged B cell depletion and more significant declines in IgG compared with RA or CTD patients treated with rituximab; may require IVIg replacement and/or change in therapy

- **HBV:** High risk for HBV reactivation. Prophylaxis recommended for patients who are HBcAb+ due to risk of reactivation.

- **PJP:** Prophylaxis important for patients who are receiving induction therapy and concomitantly on moderate to high dose prednisone; less clear for patients are on rituximab maintenance monotherapy

- Does not increase risk of TB reactivation

Case

- Serum IgG 250
- Infectious serologic work up negative
- Bronchoscopy culture + coccidioides immitis
- Diagnosed with pulmonary coccidiomycosis in setting of rituximab-induced hypogammaglobulinemia
- Treated with fluconazole and IVIg

In patients with autoimmune disease on immunosuppression presenting with new symptoms, consider:

1. Is this an infection?
2. Is this a medication side effect?
3. Is this a new/recurrent manifestion of their underlying disease?

28 year old woman with lupus presenting with cough, dyspnea and fever

Initial Presentation

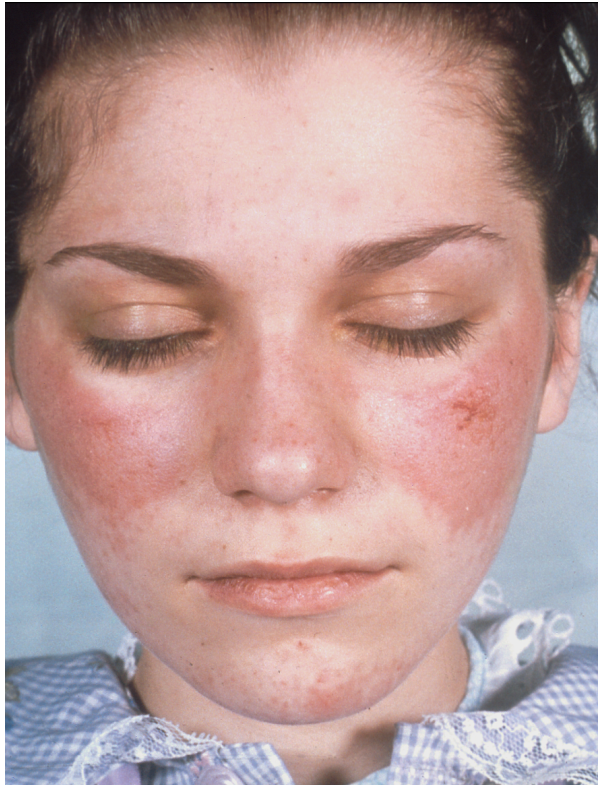
-1 week ago - developed cough & dyspnea.
-Past 6 weeks, lupus has been in a mild-moderate flare, characterized by fatigue, malar rash & increased joint pains
-Reported fevers to 101 & rigors. No night sweats

Past Medical History

SLE: ANA+ dsDNA+ low complements, rash, arthritis

Medications

Hydroxychloroquine
Prednisone 15mg per day
Azathioprine 100mg per day



28 year old woman with cough, dyspnea and fever

Vital Signs

- BP 90/55 , Pulse: 110 , RR 22, O2 sat 89% on RA; Temperature 101.5^{o}F

Constitutional

- Ill appearing, cushingoid
- In moderate respiratory distress

HEENT

- No scleral injection
- No oral ulcers
- Diffuse alopecia

Cardiac

- Tachycardic, regular, no murmurs
- No edema

Pulmonary

- Decreased breath sounds right base
- Crackles at left base

Musculoskeletal

- No synovitis, including no swelling, but +joint laxity and tenderness over the MCPs/PIPs

Skin

- Malar rash, no nodules or purpura.
- +Livedo reticularis

Neuro

- Alert & oriented. Sensation intact
- Motor strength 5/5 U/L extremities

Which of the following findings do not suggest SLE flare?

Elevated CRP

T 101.5

Malar rash

Joint tenderness

What labs would you order?

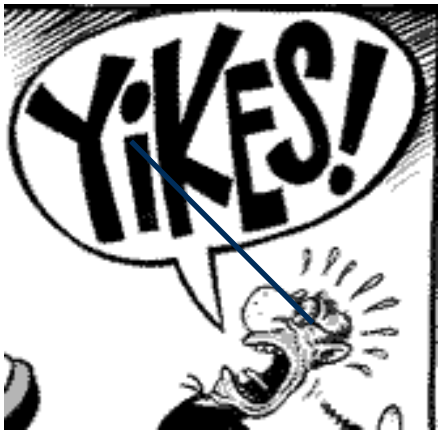
dsDNA

aCL

C4

RVVT

ssA



C3

ssB

ANA

Smith

RNP

RF

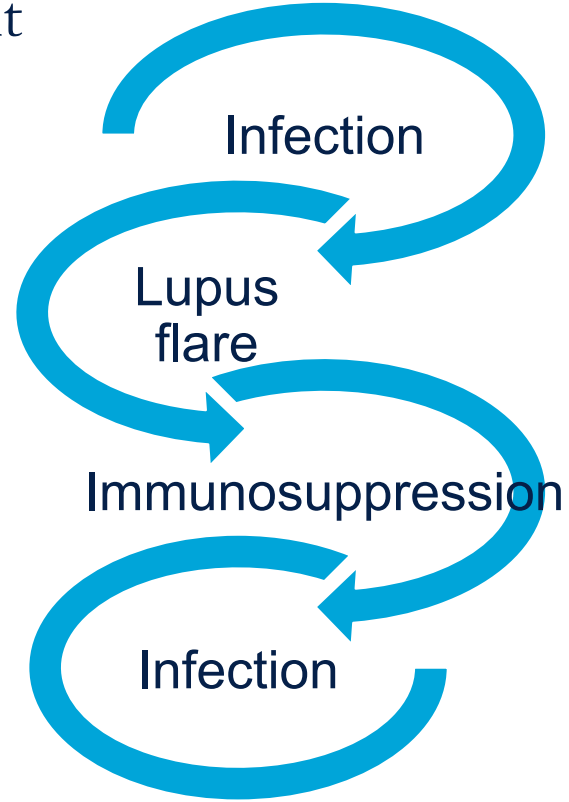
β2 glyp

Lupus Flare	Infection
Leukopenia (lymphopenia), thrombocytopenia	Leukocytosis, thrombocytosis
Normothermic	High fever, shaking chills
Elevated ESR, normal CRP**	Elevated ESR, elevated CRP
Decreased C3 and C4	Unchanged C3 and C4 from prior
Increased dsDNA	Unchanged dsDNA from prior
Specific signs of lupus activity (e.g. arthritis, rash)	Localizing signs of infection

**CRP can be elevated with serositis, severe arthritis

Initial evaluation of hospitalized SLE patient

- WBC with differential
- U/A with micro, Upr/cr
- Anti-dsDNA, complements (C3, C4)
- ESR, CRP
- Infection work up



**Do not send ANA, other subserologies again

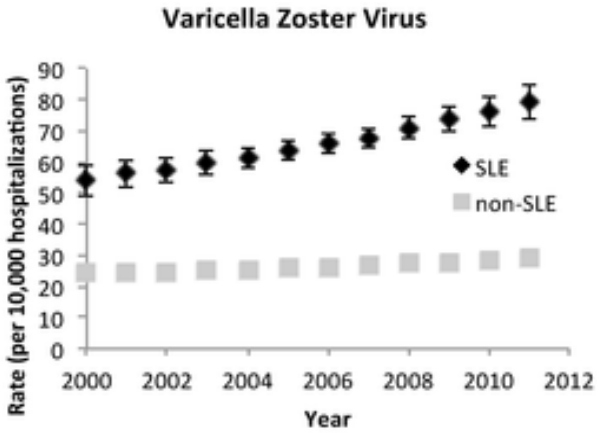
UCSF

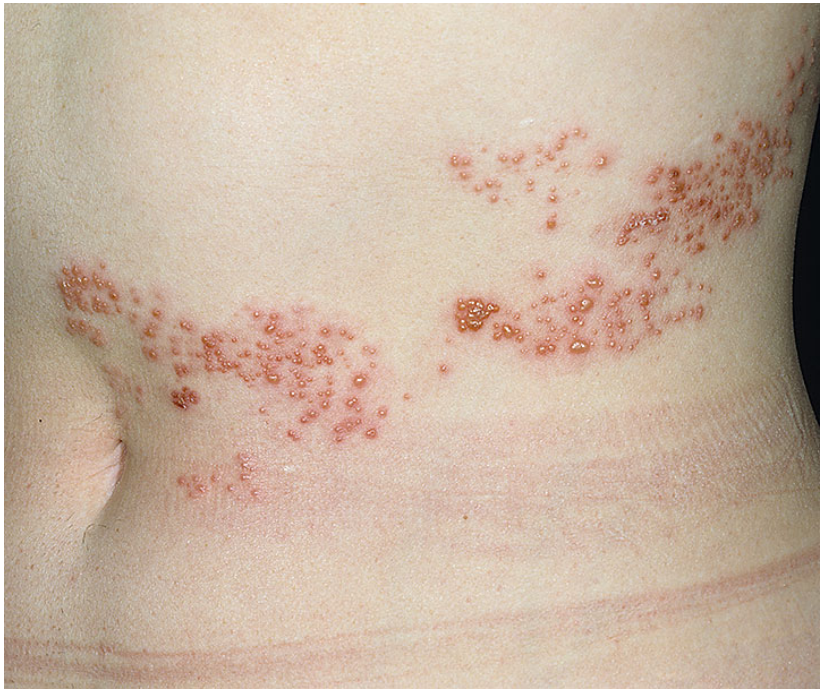
Zoster and Lupus

RESEARCH ARTICLE

National Lupus Hospitalization Trends Reveal Rising Rates of Herpes Zoster and Declines in Pneumocystis Pneumonia

Sara G. Murray[1] *, Gabriela Schmajuk[1,2], Laura Trupin[1], Lianne Gensler[1], Patricia P. Katz[1,3], Edward H. Yelin[1,3], Stuart A. Gansky[4], Jinoos Yazdany[1,3]





Murray, S. PLoS One. 2016 Jan; 11(1).

UCSF

Zoster and Lupus

Table 2 : Incidence rate of herpes zoster per 1000 person years by 10 year age group and auto-immune disease or comparator cohort									
	Cohorts								
	Healthy*	Diabetes	SLE	IBD	RA	PsA	PsO	AS	Gout
	IR	IR	IR	IR	IR	IR	IR	IR	IR
Age group									
21-30	2.7	7.8	24.6	11.6	6.6	N/A	5.9	N/A	2.9
31-40	3.3	5.3	15.2	5.6	8.2	9.8	3.7	8.1	5.2
41-50	3.9	5.3	17.5	10.4	10.0	8.5	6.4	5.1	6.1
51-60	5.8	8.2	20	11.7	14.6	13.2	9.7	8.3	6.9
61-70	8.5 (referent)	11.0	22.7	19.0	17.1	15.9	13.3	14.3	9.5
71-85+	10.6	13.0	20.9	23.8	21.3	19.4	21.2	26.3	13.3

Yun, H. Arthritis and Rheumatology. 2016 Mar 18. [Epub ahead of print]

Infection in SLE

- Infection common in SLE; accounts for 25-50% of overall mortality
- Half of the patients with SLE experience severe infection
- >20% hospitalizations from infections
- Risk factors for infection in SLE: disease activity, prednisone equivalent dose of 7.5 to 10 mg/day, pulse therapy, and cyclophosphamide

Case

Your patient presents to the ED with chest pain radiating to the neck. EKG shows ST depressions in the inferolateral leads. Her troponin is elevated.

What is the most likely cause of her cardiac ischemia?

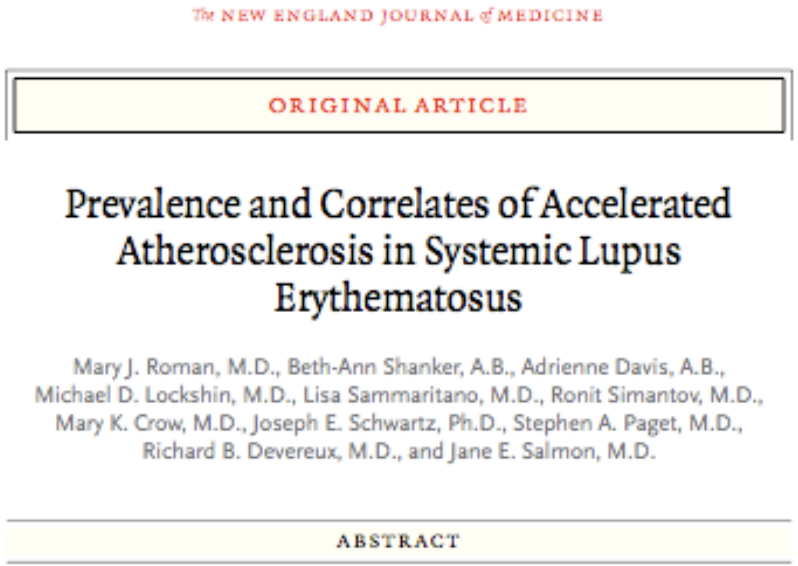
Myocarditis

Coronary artery vasculitis

Pericarditis

Atherosclerotic coronary artery disease

Cardiovascular Disease and Lupus



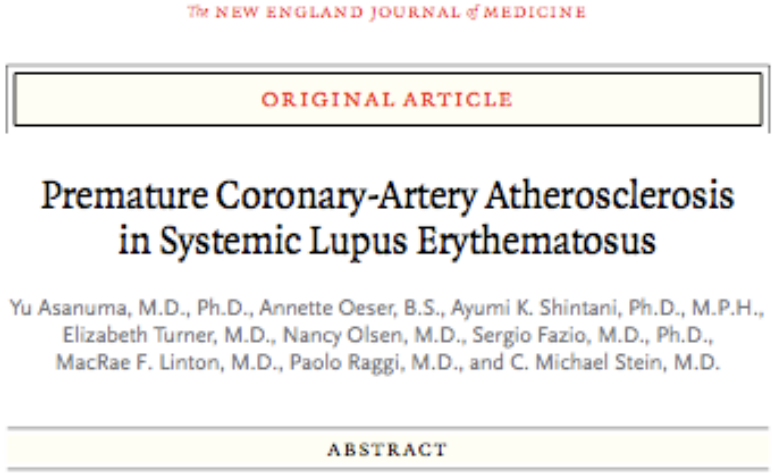
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Prevalence and Correlates of Accelerated Atherosclerosis in Systemic Lupus Erythematosus

Mary J. Roman, M.D., Beth-Ann Shanker, A.B., Adrienne Davis, A.B., Michael D. Lockshin, M.D., Lisa Sammaritano, M.D., Ronit Simantov, M.D., Mary K. Crow, M.D., Joseph E. Schwartz, Ph.D., Stephen A. Paget, M.D., Richard B. Devereux, M.D., and Jane E. Salmon, M.D.

ABSTRACT



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Premature Coronary-Artery Atherosclerosis in Systemic Lupus Erythematosus

Yu Asanuma, M.D., Ph.D., Annette Oeser, B.S., Ayumi K. Shintani, Ph.D., M.P.H., Elizabeth Turner, M.D., Nancy Olsen, M.D., Sergio Fazio, M.D., Ph.D., MacRae F. Linton, M.D., Paolo Raggi, M.D., and C. Michael Stein, M.D.

ABSTRACT

- CVD is a leading cause of mortality in patients with SLE
- All-cause mortality in SLE has declined over the past 20 years, but the risk of death due to CVD remains unchanged

Summary

- When in doubt, tap the joint!
- Rheumatic diseases are typically multi-system
  - Separate signal from noise
  - Call consults early. Don’t wait for the ANCA or GFR to drop
- Infection and flare can occur together
- In vasculitis, leukocytosis, thrombocytosis; high risk of VTE
- In SLE, distinguish disease activity (cytopenias) vs infection (high CRP)