Rheumatology
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Elena Myasoedova, MD, PhD
Assistant Professor of Medicine
Mayo Clinic Rochester, Minnesota
myasoedova.elena@mayo.edu
Disclosures

• None
Learning objectives

1) Diagnosis and approach to management of:
   • Autoimmune inflammatory arthritis and crystalline arthritis
   • Connective tissue disease
   • Vasculitis
2) Rheumatologic emergencies – overview
Inflammatory Arthritis vs DJD

**Inflammatory**
- Sudden onset
- Bilateral symmetric symptoms
- Constitutional symptoms
- Elevated inflammatory markers
- Morning stiffness > 30 minutes
- MCP/ MTP squeeze tenderness

**Degenerative**
- Gradual onset of scattered symptoms in joints of fingers, knees, spine
- No constitutional symptoms
- Normal inflammatory markers
- Use related pain with minimal stiffness
- Heberden nodes (DIP), Bouchard nodes (PIP)
Osteoarthritis

Management:

- Tylenol, NSAIDs (topical NSAIDs for patients >75 yo)
- Intra-articular steroid injections
- Viscous supplementation
- Physical therapy
- Joint replacement
- Emerging treatments: Tanezumab, a humanized monoclonal antibody that blocks nerve growth factor

Hochberg et al. Arthritis Care & Research 2012, 64: 465–74
Case 1

- A 36 year old woman presents with a 2-month history of morning stiffness in her hands, wrists, knees, feet for 1.5 hours. Her only medication is ibuprofen which is helpful.

- Physical exam: normal vital signs. Tenderness and swelling of the 2nd, 3rd and 5th MCPs bilaterally, 2-4th PIPs bilaterally, right wrist, left knee and 2-5th MTPs.
What is the most appropriate diagnostic test to perform next?

• A) Anti-cyclic citrullinated peptide antibodies
• B) HLA-B27
• C) Parvovirus IgG antibodies
• D) Serum urate
• E) TSH
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- A) Anti-cyclic citrullinated peptide antibodies
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Rheumatoid arthritis
Risk factors

• Genetic (60%): HLA-DRB1 and other HLA and non-HLA susceptibility genes
• Environmental (40%): smoking, air pollution
• Other: periodontitis (Porphyromonas gingivalis), hormonal (?)

Viatte S et al. Arthritis Rheumatol 2016
van Beers-Tas MH et al. Best Pract Clin Rheumatol 2015
Rheumatoid Mimics Early Synovitis Patients

- Viral Arthritis
  - Rubella
  - Parvovirus B19
- Reactive Arthritis Syndromes
- Seronegative Arthritis Syndromes/PMR in the Elderly
- Lupus
- Atypical crystalline arthritis – CPPD, gout
## RA laboratory panel

<table>
<thead>
<tr>
<th></th>
<th>Anti-CCP</th>
<th>RF</th>
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</thead>
<tbody>
<tr>
<td><strong>Sensitivity</strong></td>
<td>70%</td>
<td>72%</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>95%</td>
<td>80%</td>
</tr>
</tbody>
</table>
| **Utility**          | - Identifying early inflammatory arthritis patients at risk for erosive disease  
                       - Evaluating RF negative inflammatory arthritis patients  
                       - Evaluating a positive RF in a person who doesn’t seem to have RA  
                       - In high titers uniquely specific for potentially erosive rheumatoid arthritis  
|                      |                   | - Higher likelihood of detection and higher titers in established disease  
                       - High titers correlate with more severe disease |
| **Impact of other factors** | Smoking         | Smoking, age  |
Case 1, continued

- Patient returns with the following results of additional work-up:
  - CRP 36.0 mg/dl
  - Positive CCP antibody >250 U
  - Positive Rheumatoid factor 120 IU
  - Hand x-rays reveal periarticular osteopenia

Magnified view of the left 5th MTP
Which of the following is the most appropriate treatment at this time?

- A) Continue ibuprofen
- B) Initiate mycophenolate mofetil
- C) Initiate methotrexate
- D) Initiate monotherapy with prednisone
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RA management: Methotrexate

- Methotrexate is the anchor DMARD
- Prevents radiologic progression (disease-modifying!)
- Weekly PO or SQ dosing
- Potential up-titration to max 25 mg/week
- Folic acid supplementation 1 mg/day
- Safety monitoring: CBC, Creatinine, AST, ALT

Adenosine pathway is the likely primary down-regulator of RA inflammation.
Methotrexate Toxicity

- One of the mechanisms: folate depletion

- Minor: Nausea, stomatitis, hair loss, headache, fatigue

- Serious but rare:
  - Megaloblastic anemia and pancytopenia
    - Increased risk with folate deficiency or azotemia
  - Liver fibrosis
    - Increased risk in NAFL/NASH patients
  - Hypersensitivity pneumonitis

- Teratogenic effect
RA treatments

- **Treatment goal:** Remission or low disease activity

- **Synthetic DMARDs:** Methotrexate, Sulfasalazine*, Hydroxychloroquine*, Leflunomide

- **Biologic DMARDs:**
  - TNF inhibitors (Infliximab, Adalimumab, Etanercept, Golimumab, Certolizumab*)
  - IL6-receptor antagonists (Tocilizumab, Sarilumab)
  - T-cell co-stimulator blocker (Abatacept)
  - JAK kinase inhibitor (Tofacitinib)
  - Rituximab - antibody against CD20 on B-lymphocytes

* safe in pregnancy and breastfeeding

RA treatment strategies

- Glucocorticoids:
  - bridge-therapy,
  - management of flares,
  - low-moderate doses, limited duration.

Case 2

A 53-year-old woman with a 15-year history of RA is evaluated for intermittent sensory loss in her hands and occasional shock-like sensation from the neck down the back with neck flexion. No muscle weakness.

Medications: Methotrexate, Etanercept, Folic acid

Physical exam: normal vitals signs. No active synovitis. Ulnar deviation of her MCP joints bilaterally. Neck flexion triggers her symptoms. Reflexes, strength and sensation of the upper and lower extremities are normal.
Which of the following is the most appropriate diagnostic test to perform next?

- A) EMG of the upper extremities
- B) Flexion/extension X-rays of the cervical spine
- C) Serum vitamin B12 level
- D) TSH
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- A) EMG of the upper extremities
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- C) Serum vitamin B12 level
- D) TSH
C1-C2 subluxation; cervical myelopathy

Management:

- Immobilization of cervical spine
- MRI/CT with myelography
- High-dose IV steroids
- Neurosurgery consult
Extra-articular manifestations of RA

- Rheumatoid nodules
- Interstitial lung disease (rule out methotrexate toxicity)
- Pleural effusion (Exudate, low glucose, high LDH)
- Pericarditis
- Secondary Sjogren’s
- Rheumatoid vasculitis
- Felty’s syndrome (always sero+)
- Cervical myelopathy
- Amyloidosis

Case 3

- A 32 year old man is evaluated for 10-year history of low back pain. The pain is worse at rest, improves with movement, and can awaken him during the night. He takes Naproxen BID with some relief.

- Family Hx: three paternal uncles with back problems

- Physical exam: normal vital signs. No swollen joints. Tenderness over SI joints bilaterally, reduction in ROM in lumbar spine

- Labs: CBC and inflammatory markers - normal

- Plain A/P X-ray of the pelvis: fusion of SI joints
Which of the following is the most appropriate diagnostic test to perform next?

- A) Anti-neutrophil cytoplasmic antibodies
- B) Anti-CCP antibodies
- C) Antinuclear antibodies
- D) HLA-B27 antigen
- E) No additional testing
Which of the following is the most appropriate diagnostic test to perform next?

• A) Anti-neutrophil cytoplasmic antibodies
• B) Anti-CCP antibodies
• C) Antinuclear antibodies
• D) HLA-B27 antigen
• E) No additional testing
Inflammatory back pain
Hallmark of ankylosing spondylitis

• Age of onset < 40 years
• Insidious onset
• Improvement with exercise or activity
• No improvement with rest
• Pain at night with improvement upon getting up

• If four of the five criteria are present –
  • Sensitivity 80%
  • Specificity 74%

Traditional Classification of the Spondyloarthropathies

- Ankylosing spondylitis
- Psoriatic arthritis
- Reactive arthritis
- Arthritis associated with IBD
Broadening the perspective of spondyloarthropathy classification

Undifferentiated Spondyloarthropathy

• This is the **most common variant** that you will encounter in practice

• Characteristics at disease onset
  • Inflammatory back pain 69%
  • Peripheral arthritis 29%
  • Enthesitis 29%
  • Uveitis 2.5%
  • Dactylitis ("sausage digits") 3.3%
  • Positive FHx 32.1%

Rudwaleit M et al. A&R 2004. 50(suppl):S617
AS/SpA: Diagnostic algorithm (ASAS)

- SpA: spondyloarthritis;
- AS: ankylosing spondylitis;
- X-rays: plain radiographs of the pelvis;
- IBP: inflammatory back pain;
- IBD: inflammatory bowel disease;
- * >3 months, onset < 45.

Association with HLA-B27

- A strong genetic association, controversial mechanisms
- Helpful, but the clinical picture is most useful

<table>
<thead>
<tr>
<th>Disorder</th>
<th>“+” HLA-B27 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankylosing spondylitis</td>
<td>90</td>
</tr>
<tr>
<td>Reactive arthritis</td>
<td>90</td>
</tr>
<tr>
<td>Enteropathic arthritis</td>
<td>35-75</td>
</tr>
<tr>
<td>Psoriatic spondylitis</td>
<td>50</td>
</tr>
<tr>
<td>Undifferentiated SpA</td>
<td>70</td>
</tr>
<tr>
<td>Acute anterior uveitis</td>
<td>50</td>
</tr>
</tbody>
</table>
MRI

Early SI inflammation
Management of spondyloarthritis

• **Treatment goals:** relief of symptoms, maintenance of function, prevention of progression of spinal disease

• Physical activity, smoking cessation

• Except for IBD patients, NSAID trial first

• For axial disease (spondylitis, sacroiliitis):
  • If a patient fails two NSAIDs → TNF inhibitor or anti-IL-17 monoclonal antibody (Secukinumab)

• For peripheral synovitis:
  • Sulfasalazine if not sulfa allergic
  • Methotrexate

Gout
Gout facts

• Most common type of inflammatory arthritis world-wide
  • Genetic component
  • Diet and lifestyle
  • Comorbidities and medications

• Hyperuricemia (> 6.8 mg/dL)

• Clinical manifestations of gout:
  • Recurrent flares of inflammatory arthritis (gout flare)
  • A chronic arthropathy
  • Tophaceous deposits
  • Uric acid nephrolithiasis
  • Chronic nephropathy
Diagnosis

- Physical exam
- Synovial fluid: negatively birefringent needle-shaped crystals using compensated polarizing light microscopy
  
  Mnemonic: “yellow-parallel-allopurinol”

- X-ray: “punched out” lytic lesions
- Dual energy CT

Case 4

- A 54-old man with acute gout flare after recent exacerbation of CHF and optimizing his diuretics dosing. Comorbidities: CHF, CKD, stage 3.
- No improvement with intra-articular steroids and 60 mg/day of IV methylprednisolone for 3 days.

What would you use to treat this acute gout flare?

- A) Colchicine
- B) Ibuprofen
- C) Anakinra
- D) Allopurinol
Management of acute gout

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Adult Regimen</th>
</tr>
</thead>
</table>
| NSAIDs             | Naproxen 3 to 5 days  
Indomethacin 3 to 5 days                                               |
| Colchicine         | 1.2 mg PO at the first sign of a flare → 0.6 mg 1 hour later (MAX 1.8 mg over 1 hour)  
Then 0.6 mg PO 1-2 times a day (MAX 1.2 mg/day) |
| Corticosteroids    | A) Oral prednisone 30 mg/day for 3 to 5 days  
B) IV methylprednisolone 25 mg/day for 3-5 days  
C) Intra-articular injection: methylprednisolone 20 to 80 mg in a single injection |

• Anakinra – IL-1 inhibitor - off label use: SubQ: 100 mg once daily x 3 days

## Management of chronic gout

<table>
<thead>
<tr>
<th>Medication</th>
<th>Action</th>
<th>Adult Regimen</th>
<th>Relative Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allopurinol*</td>
<td>Reduced uric acid production</td>
<td>Starting dose 100 mg/day with up-titration to achieve a goal uric acid of &lt; 6 mg/dL (&lt; 5 mg/dL if tophaceous gout).</td>
<td>• interactions with azathioprine, mercaptopurine, didanosine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Renal insufficiency</td>
</tr>
<tr>
<td>Febuxostat</td>
<td>Reduced uric acid production</td>
<td>40 mg PO daily, can be increased to 80 mg PO daily goal uric acid:&lt; 6 mg/dL (&lt; 5 mg/dL if tophaceous gout).</td>
<td>• CV thromboembolic events</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Hepatic dysfunction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• interactions with azathioprine, mercaptopurine, didanosine</td>
</tr>
<tr>
<td>Probenecid (in underexcretors of uric acid)</td>
<td>Increased uric acid excretion</td>
<td>250 mg PO BID for 1 week; if tolerated, increase to 500 mg PO BID.</td>
<td>• Renal impairment: creatinine clearance &lt;50 mL/min</td>
</tr>
</tbody>
</table>

- * Screen for HLA-B*5801 in Thai, Han Chinese or Korean patients: high risk for allopurinol hypersensitivity (DRESS syndrome)
- Pegloticase – porcine-derived uricase in patients with high tophaceous burden. IV administration

Calcium pyrophosphate deposition disease (CPPD) and pseudogout

- Mono-/ oligo- or polyarthritis
- Chondrocalcinosis on X-rays
- Assess for: hyperparathyroidism, hypothyroidism, hypophosphatasia, hypomagnesemia, hemochromatosis
- Rhombus-shaped calcium pyrophosphate crystal: positively birefrigent on polarized light microscopy
- Intra-articular steroid injections, NSAIDs,
- +/- colchicine
Acute monoarthritis

• Aspirate
• Synovial fluid WBC counts:
  <200/μL – normal
  200-2000/μL – non-inflammatory conditions
  >2000/μL – inflammatory states
  >50,000/μL (>75% neutrophils) – septic arthritis, until proven otherwise, regardless of presence of crystals
  “+” Gram stain, “+” culture

Management: Joint debridement, antibiotics
Case 5

• A 21-year-old woman is evaluated for an 8-week history of fatigue, low-grade fever, facial rash, oral sores. Medications: multivitamin.

• Physical exam: normal vital signs. Tenderness of multiple MCPs and PIPs bilaterally. T 37.3°C (99.1°F). No other findings.

• CBC: normocytic anemia
Which of the following is the most likely diagnosis for the rash?

- A) Acute cutaneous lupus erythematosus
- B) Erysipelas
- C) Rosacea
- D) Seborrheic dermatitis
- E) Subacute cutaneous lupus erythematosis
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Systemic Lupus Erythematosus

- Rare disease with genetic predisposition
- Strong female predominance

<table>
<thead>
<tr>
<th>2012 SLICC CLASSIFICATION CRITERIA FOR SYSTEMIC LUPUS ERYTHEMATOSUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biopsy proven LUPUS NEPHRITIS and ANA or anti-DNA</td>
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</table>

**CLINICAL**
- Acute cutaneous LE
- Chronic cutaneous LE
- Oral ulcer
- Alopecia
- Synovitis
- Serositis
- Renal
- Neurologic
- Hemolytic anemia
- Leucopenia/lymphopenia
- Thrombocytopenia

**IMMUNOLOGIC**
- ANA
- Anti-dsDNA
- Anti-Sm
- aPL antibodies
- Low complement
- Direct Coomb’s test

Flare

AT LEAST 4 CRITERIA (1 Needs to be IMMUNOLOGIC)

SLE management

• Mild lupus: hydroxychloroquine, NSAIDs, low dose prednisone

• Moderate lupus: Azathioprine, mycophenolate mofetyl (CellCept), methotrexate, corticosteroids
  • Belimumab: blocks binding of B lymphocyte stimulator protein (BLyS) to receptors on B lymphocytes. IV or SQ

• Severe lupus: Cyclophosphamide, CellCept, pulse-steroids

Hahn B. et al. Arthritis Care & Res 2012; 64: 797–808
Neonatal lupus

• In children from mothers with positive SS-A/SS-B
• Risk of complete heart block
• Follow with OB/GYN: OB ultrasound
• Hydroxychloroquine
• Steroids
Systemic sclerosis, classification

Limited cutaneous scl
- Raynaud phenomenon for years
- Skin thickening: hands, face, feet, and forearms (acral distribution)
- Nail-fold capillary changes
- 10-15% late incidence of pulmonary hypertension
- Renal disease rare
- Anti-centromere antibody

Diffuse cutaneous scl
- Raynaud phenomenon followed, within 1 year, by skin thickening
- Truncal and acral skin involvement; tendon friction rubs
- Nail-fold capillary changes
- Early and significant incidence of renal, interstitial lung, diffuse gastrointestinal, and myocardial disease
- Anti-Scl-70 and anti-RNA polymerase-I, II, or III antibodies

Allanore Y. Nat Rev Dis Primers. 2015;1:15002
Cutaneous manifestations of limited scleroderma

- Abnormal nail-fold capillaroscopy
- Raynaud’s phenomenon
- Telangiectasia
- Digital pitting and ulcerations
- Calcinosi
Case 6

• A 42 year old woman developed Raynaud’s phenomenon last summer. Gradually, her fingers got tight and her grip weakened.
• This morning she awoke with a severe generalized headache. Her husband brought her to the ER: BP 232/118 mmHg
• Her peripheral smear:
What is the next most appropriate action?

• A) Hematology consult
• B) Prednisone 30 mg daily
• C) ACE inhibitor
• D) Neurology consult
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Scleroderma renal crisis

- 5-20% of patients with systemic sclerosis
- AKI, severe hypertension, mild proteinuria, MAHA, thrombocytopenia
- Histology (kidney): intimal proliferation and thickening, with concentric "onion-skin" hypertrophy
- Emergent inpatient care
  - Normalize BP within 72 hours with captopril
  - Increase captopril dose Q 6 to 8 hours
  - Add other agents if necessary
- Dialysis

Penn H, et al. CP SOQJM. 2007;100:485
Case 7

- A 47-year-old woman presents with low-grade fever for 3 weeks; skin peeling on the sides of the second digits and palms, pain and swelling of 2\textsuperscript{nd} and 4\textsuperscript{th} PIP joints.

- Physical exam: $t=37.6^\circ C$ (99.7F), pulse 95/min, respirations 20/min. Blood pressure and O2 sat normal.

- Erythema of her forehead and chin. Crackles at the lung bases bilaterally. No weakness.

- Labs: CK 115 U/L (normal); ANA 1:1280; anti-DS-DNA negative, Anti-Jo1-antibodies – positive, anti-Smith antibodies – negative
Which of the following is the most likely diagnosis?

- A) Anti-synthetase syndrome
- B) Sjogren syndrome
- C) SLE
- D) Systemic sclerosis
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- A) Anti-synthetase syndrome
- B) Sjogren syndrome
- C) SLE
- D) Systemic sclerosis
Anti-synthetase syndrome

• Acute onset

• Interstitial lung disease, risk of pulmonary hypertension

• Fever, inflammatory arthritis, Raynaud’s phenomenon, inflammatory myopathy, “mechanic’s hands.”

• Anti-synthetase antibodies - target tRNA synthetase enzymes

• Fair response to therapy (steroids)

Dermatomyositis

Shawl sign

Heliotrope rash

Gottron’s papules

T2 weighted image of thighs. Inflammation can be seen

Perivascular infiltrate around fascicle

Treatment: glucocorticoids, Azathioprine, Methotrexate, Hydroxychloroquine, IVIG; Rituximab, Cyclophosphamide, CellCept

Amato AA, Barohn RJ SOJ Neurol Neurosurg Psychiatry. 2009;80:1060
Inclusion body myositis

- Age >50 years
- Muscle weakness in proximal and distal muscles of UE and LE with involvement of either finger flexors, wrist flexors, or quadriceps.
- Dysphagia
- Muscle biopsy with intracellular amyloid deposits or filamentous inclusions, rimmed vacuoles – focal destruction of muscle fibers
- Resistant to treatment
Case 8

- A 79-year-old man presents with aching in bilateral shoulders and hips, stiffness for 2 hours in the morning, aching in his jaw when chewing. Reports left-sided headaches.

- Physical exam: vital signs are normal. Tenderness and slight swelling over the left temple. No vision changes. Painful and limited ROM in hips and shoulders. T 37.3C (99.1F).

- ESR = 85 mm/hr
Which of the following is the most appropriate initial management?

• A) CT of the head
• B) Low-dose aspirin
• C) Methotrexate
• D) Prednisone
• E) Temporal artery biopsy
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- A) CT of the head
- B) Low-dose aspirin
- C) Methotrexate
- D) Prednisone
- E) Temporal artery biopsy
Vasculitis by vessel size

Giant cell arteritis

- Consider GCA in a patient >50 years of age if:
  - New headaches
  - Monocular vision loss
  - Jaw claudication
  - Fever, anemia
  - PMR-like symptoms
  - High ESR, CRP

→ TA biopsy: panarteritis, CD4+ lymphocytes and macrophages
→ Vascular contrast imaging

- Treatment:
  Glucocorticoids
  - Vision changes – emergency: IV methylprednisone 1000 mg x 3 days
  - No vision changes – Prednisone 1 mg/kg/day PO with slow taper (60 mg/day)
  - Methotrexate
  - Tocilizumab

Polyarteritis nodosa

- Associated with Hep B infection
- Fever, malaise, weight loss
- Mononeuritis multiplex
- Skin purpura, necrotic ulcers
- Renal artery vasculitis with renal infarction, aneurysms (do not obtain kidney biopsy!)
- Orchitis
- Mesenteric vasculitis (GI bleed/ perforation)
- No serological markers
  → Angiography
  → Hep B serology, HIV serology, r/out ANCA
  → Nerve, muscle or deep skin biopsy
Small Vessel Vasculitis

A Practical Classification

• Primary small vessel vasculitis
  • Anti-neutrophil cytoplasmic antibody (ANCA) vasculitis:
    • GPA = granulomatosis with polyangiitis (c-ANCA/ PR3)
    • MPA = microscopic polyangiitis (p-ANCA/ MPO)
    • EGPA = eosinophilic granulomatosis with polyangiitis
  • IgA-Associated Vasculitis (“Henoch-Schönlein purpura”)

• Secondary small vessel vasculitis
  • Cutaneous only – think of drugs 1st!
  • Multisystem disorders: Cryoglobulinemia, SLE, RA, HIV, IBD, Paraneoplastic syndromes

Evaluation of Small Vessel Vasculitis

Histology Confirmation
- Skin; Nerve; Kidney; Lung

Laboratory Testing
- Creatinine and UA
- ANCA panel
- ANA panel
- RF/CCP
- Monoclonal protein study
- C3, C4
- Hepatitis B and C serology
- Chest imaging

Indirect immunofluorescence assay
Initial immunosuppressive treatment

- Glucocorticoids; Methotrexate
- Cyclophosphamide OR Rituximab

Take home points

- Arthritis: Inflammatory or degenerative?
  - Monoarthritis – aspirate!
  - Extra-articular/ systemic features?
- CTD: assess for organ damage (kidney, lung, heart)
- Vasculitis: vessel size, work-up (serologies, biopsy, imaging)
- Recognize rheumatologic emergencies
Helpful resources

- https://www.acponline.org/featured-products/mksap-18
- https://www.rheumatology.org/Practice-Quality/Clinical-Support/Clinical-Practice-Guidelines
- https://www.rheumatology.org/Learning-Center/Medication-Guides
- https://www.uptodate.com/contents/search
Questions & Discussion