RHEUMATOLOGY
OBJECTIVES

Know and understand:

• How the clinical presentations of rheumatologic diseases can vary

• Components of a thorough physical examination for investigating rheumatoid complaints

• How to differentiate between different rheumatologic diseases

• Evidence-based management of rheumatologic diseases
TOPICS COVERED

- Osteoarthritis
- Rheumatoid Arthritis
- Gout
- Calcium Pyrophosphate Deposition Disease
- Polymyalgia Rheumatica
- Giant Cell Arteritis (Temporal Arteritis)
- Systemic Lupus Erythematosus
- Sjögren Syndrome
- Polymyositis and Dermatomyositis
- Fibromyalgia
OSTEOARTHRITIS (OA): OVERVIEW

- Principal cause of knee, hip, and back pain in older adults, and most common source of chronic pain
- Avoid the reflexive conclusion that all joint pain in older adults is the result of OA
- Can develop in any joint that has suffered injury or other disease
- Hallmark: cartilage degeneration
  - But not purely a degenerative disease; subchondral bone abnormalities and focal synovial inflammation are also seen in pathologic specimens
• Differential diagnosis: inflammatory and crystal arthritides, septic arthritis, bone pain due to malignancy

• Bony enlargement and crepitus suggest OA
  - In the fingers, bony enlargement occurs in the distal interphalangeal joint (Heberden nodes) and in the proximal interphalangeal joints (Bouchard nodes)
  - Osteophytes are the radiographic counterpart of this enlargement, and asymmetric joint space narrowing is common

• Joint tenderness and warmth may appear, but true synovitis suggests an alternative or concomitant diagnosis

• Advanced imaging studies are generally not indicated
• Weight reduction for knee, hip, or spine OA
• Thermal agents for hand, knee, or hip OA
• Physical activity
• Assistive devices
  ➢ Jar or bottle openers
  ➢ Walking aids
  ➢ Braces
  ➢ Insoles
• CDC and Arthritis Foundation recommendations to improve pain and functioning in patients with OA:
  ➢ Minimum of 150 minutes (2.5 hours) of moderate intensity aerobic exercise per week along with 2 days of muscle-strengthening exercise per week
  ➢ These guidelines do not apply specifically to older adults
• A multimodal interdisciplinary approach, including referral to physical and occupational therapy, is key in management of chronic pain related to OA
MEDICATIONS FOR OA

- Traditional approach is “around-the-clock” acetaminophen, but according to recent evidence, this therapy is not as effective as previously thought.

- **Topical therapies** (e.g., analgesic balms, capsaicin, topical NSAIDS) can be helpful in hand or knee OA (SOE=C).

- **Tramadol** can be tried for refractory pain.

- Consider **low-dose opioids** for patients who do not respond to other options; limit to those whose quality of life (QoL) is significantly impacted by pain.

- **Glucosamine and chondroitin sulfate**: conflicting data; for knee OA, no better than placebo (SOE=A).
• **Glucocorticoid injections**
  - Reasonable treatment option for knee OA; provide short-term relief that is superior to placebo
  - Data supporting long-term relief are limited
  - For hip OA, provide only temporary relief compared with placebo

• **Hyaluronic acid and hyaluronan polymers given in a series of weekly injections in the knee**
  - Approved for viscosupplementation therapy
  - Not shown to be more effective than placebo
SURGERY FOR OA

- Consider for patients who have advanced refractory OA, have clearly impaired function and reduced QoL, and are motivated about postsurgical rehabilitation.

- Arthroscopic debridement for knee OA is usually reserved for patients who report mechanical symptoms (e.g., locking, “giveaway” weakness).
  - Effectiveness has not been proved (SOE=C).
  - Initial treatment with physical therapy has similar outcomes.

- Total joint arthroplasty can be considered in patients with more extensive, disabling knee or hip OA.
RHEUMATOID ARTHRITIS (RA): OVERVIEW

• An inflammatory arthritis linked to increased risk of cardiovascular disease and premature death

• Most older adults with late-onset RA present similarly to younger adults

• Two presentations are unique to older adults:
  - “RS3PE” syndrome — remitting seronegative symmetrical synovitis with pitting edema; accounts for ~10% of late-onset cases
  - Acute-onset, seronegative, inflammatory arthritis of the shoulder and hips — can be difficult to distinguish from polymyalgia rheumatica (PMR); accounts for ~25% of late-onset cases
Compared with younger adults, older adults more frequently present with constitutional symptoms: malaise, fever, fatigue, weight loss, in addition to the characteristic synovitis of RA.

Older adults with RA also more likely to have a higher initial erythrocyte sedimentation rate (ESR).

Check autoantibodies, including rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies.

Radiographic evaluation can be helpful to demonstrate erosions or deformities that appear in more aggressive and long-standing disease.
• **Symptoms** — pain intensity and location, duration of morning stiffness, severity of fatigue, functional status

• **Physical exam** — number of tender and swollen joints; evaluation for extra-articular manifestations, including rheumatoid nodules and other cutaneous manifestations, interstitial lung disease, pleuropericardial disease, vasculitis, ocular disease, neuropathy

• **Lab monitoring** — acute phase reactants, such as ESR and C-reactive protein (CRP); CBC; metabolic panel; liver function tests if the patient is on disease-modifying antirheumatic drugs (DMARDs)

• **Radiographs** periodically to assess for progression of disease
• Start treatment as soon as the diagnosis is made, with the goal of achieving remission or the lowest level of disease activity possible

• Initial approach to treatment should include initiation of one or more oral DMARDs, depending on disease severity and comorbidities

• In general, patients should be optimized on triple therapy with oral DMARDs before moving on to biologic treatments
• **Prednisone** (10–20 mg/d) may be used as the primary treatment for seronegative PMR-like disease and the “RS3PE” syndrome

• In contrast to classic PMR, *late-onset RA may not respond promptly* to low-dose prednisone

• In general, avoid chronic use of prednisone, because its use is associated with increased risk of infectious complications, fluid retention, and *osteoporosis*
GOUT: OVERVIEW

- A crystal-induced arthropathy; more common in older adults than in younger adults
- **Associated with CVD and metabolic syndrome**
- Incidence in postmenopausal women approaches that in men
- **Risk factors:**
  - Obesity
  - Use of diuretics, niacin, or low-dose aspirin
  - Polypharmacy
  - Renal or hepatic impairment
GOUT: CLINICAL PRESENTATIONS

• **Subacute** smoldering oligoarthritis affecting larger joints

• **Acute** monoarticular, incapacitating attack
  
  ➤ Can be precipitated by trauma, acute nonarticular illness requiring hospitalization, or abrupt change in uric acid concentration (for example, due to dehydration, particularly after surgery or admission for CHF with aggressive diuresis)

  ➤ Characterized by abrupt onset of intense inflammation and pain, typically reaching maximum intensity within 12–24 hours, with resolution of symptoms within 7–10 days, even without treatment
GOUT: DIAGNOSIS

• Requires demonstration of monosodium urate crystals from synovial fluid or an aspirate of a tophus

• Radiographs may show juxta-articular erosions of the involved joints
  ➢ An overhanging edge (ie, Martel sign) helps distinguish gout from RA erosions

• Increased serum uric acid + clinical history of episodic mono- or oligoarthritis (often podagra): highly suggestive
  ➢ However, hyperuricemia is not uniformly present at the time of an acute gout attack, and the presence of hyperuricemia alone does not confirm diagnosis of gout
GOUT: MANAGEMENOFACUTEATTACKS

- Topical therapy with ice
- Short-acting NSAID, if tolerated

- **For monoarticular attack** — intra-articular glucocorticoid (assuming septic joint has been excluded)

- **For polyarticular attack** — oral glucocorticoid tapered over 5–10 days
  - Glucocorticoids may be preferable to NSAIDs for treating gout in older adults with multiple comorbidities

- Low-dose colchicine is also an option, but its use is often limited by presence of renal impairment
GOUT: LIFESTYLE MODIFICATIONS

- Weight loss
- Avoidance of purine-rich foods such as shellfish, mollusks, organ meats, and full-fat dairy products
- Avoidance of alcohol, particularly beer, which is especially purine-rich
- Reinforce the importance of lifestyle modifications at each follow-up visit

➤ These changes can be difficult to implement and sustain but can be invaluable in prevention of future attacks
Consider uric acid–lowering therapy, such as allopurinol, febuxostat, or probenecid, for patients with more than 2 attacks in a year, evidence of tophi, or erosive disease:

- **Goal:** lower serum uric acid to 5–6 mg/dL
- **Educate** patient that this may precipitate a flare

  - As the uric acid–lowering therapy is titrated up, it is reasonable to use a prophylactic agent such as colchicine at dosages as low at 0.6 mg/d, renal function permitting, or low-dose prednisone for several months

- If the patient develops an acute gout attack while on a uric acid–lowering agent, he or she should continue the medication daily while adding an abortive treatment.
Calcium pyrophosphate deposition disease (CPDD), like gout, is a crystal-induced arthropathy that is more common in older adults than younger adults.

Can mimic RA, inflammatory OA, gout, or septic arthritis.

Associated with:
- Disorders of calcium metabolism (for example, hypomagnesemia, hypophosphatemia, and hyperparathyroidism)
- Hypothyroidism
- Hemochromatosis
CPDD: CLINICAL PRESENTATIONS

- Can result in an acute, intermittently inflammatory arthritis of the knee, hip, wrist, and metacarpophalangeal joints
  - Elbow, shoulder, or ankle involvement is less common
- Can mimic an acute gout attack
  - Sudden onset of pain and swelling that coincide with or immediately follow an acute illness or traumatic event such as surgery
• **Arthrocentesis** with crystal analysis is diagnostic and useful to distinguish CPPD from gout and infection
  - When fever is present, distinguishing CPPD from septic arthritis is imperative, and diagnostic arthrocentesis is indicated

• CPPD is commonly suspected by the **radiographic finding of chondrocalcinosis** on plain films
  - Appears as a stippled or linear calcification of the articular cartilage of the knee, wrist, hip, shoulder, and symphysis pubis
CHONDROCALCINOSIS IN CPPD
- **Intra-articular steroid injection**
  - Can result in significant relief of painful symptoms
  - May be the preferred option if only one joint involved

- **Short-acting NSAIDs and oral steroidal agents** are useful in patients who can tolerate them (SOE=C)

- **Evidence for use of colchicine in CPPD is limited**
  - Oral colchicine for acute flares is typically dosed 0.6 mg q8–24 hours, similar to acute gout treatment
  - Colchicine can also be useful to prevent future acute episodes of CPPD, typically dosed 0.6 mg q12–24 hours
POLYMYALGIA RHEUMATICA (PMR): OVERVIEW

- Inflammatory rheumatic disease that occurs almost exclusively in patients ≥50 years old
- Persistent pain or stiffness of bilateral upper arms, shoulders, hips, or thighs — accompanied by significant morning stiffness and constitutional symptoms (fatigue, low-grade fever, anorexia, weight loss)
- Approximately 10%–20% of patients with PMR also have giant cell arteritis (GCA), and up to 50%–60% of patients with GCA have symptoms of PMR

➤ Diagnosis of one condition should prompt evaluation for the other
- **Criteria:** age $\geq 50$ years, bilateral shoulder pain or aching not explained by another etiology, and increased ESR and/or CRP

- If all 3 required criteria are present, the diagnosis of PMR is suggested based on a scoring system that includes morning stiffness that lasts $>45$ minutes, negative RF and/or anti-CCP antibodies, pain or limited range of motion of the hips, or absence of other joint involvement

- **Ultrasound finding** of unilateral or bilateral subdeltoid bursitis, biceps tenosynovitis, glenohumeral synovitis, hip synovitis, or trochanteric bursitis increases the specificity of the criteria
PMR: DIAGNOSIS

• Unlike in RA, usually no evidence of inflammatory arthritis of the small hand joints

• Unlike in the inflammatory myopathies, normal muscle bulk and strength

• Typical lab findings:
  - Increased ESR and/or CRP
  - Negative RF, anti-CCP antibodies, and anti-nuclear antibodies (ANA)
  - Normal levels of muscle enzymes

• Plain radiographs of involved joints should not reveal abnormalities, and the arthritis of PMR is not erosive
PMR: MANAGEMENT

- Symptoms should respond dramatically and quickly, within 7 days, to prednisone about 10–20 mg/d
  - Consider RA or concomitant GCA in patients whose response is incomplete or not sustained
- The duration of treatment required varies from 3 months to several years
- Dosage reduction of steroids should be gradual, with periodic monitoring for symptom recurrence and lab studies (CRP, ESR) suggesting recurrence
- Consider methotrexate as a steroid-sparing agent for patients at high risk of complications and those unable to taper the dose effectively
GIANT CELL ARTERITIS (GCA): OVERVIEW

- Also called temporal arteritis
- A granulomatous vasculitis that involves large and medium-sized arteries
- Like PMR, a disease unique to older adults, occurring almost exclusively in patients ≥50 years old and increasing in incidence with age
- The overlap between PMR and GCA is notable
  - Patients with PMR who have any symptoms above the neck should have a temporal artery biopsy to evaluate for GCA
• Best known manifestation: cranial arteritis that may result in loss of vision

• But may present initially with extracranial arteritis, systemic inflammatory symptoms, PMR, or any combination

• Head and neck manifestations: headache; scalp tenderness; jaw or tongue claudication; diplopia; and prominent tender, erythematous, or nodular temporal arteries, which are typically pulseless

• Constitutional symptoms, including fatigue, malaise, weight loss, and fever, are common and may be the only manifestations
• Optic nerve pallor or swelling portends ischemia with impending blindness that warrants immediate glucocorticoid therapy

• GCA can present as sudden blindness with no prior systemic illness

• GCA may present with claudication in the arms, respiratory symptoms, TIA, stroke, peripheral neuropathies, syncope, ischemic necrosis of tongue or scalp, or, rarely, MI

• Aortic aneurysm, predominantly thoracic, is a late manifestation of GCA even when previously appropriately treated
GCA: DIAGNOSIS

• Lab findings:
  - Increased ESR and/or CRP
  - Possibly anemia of chronic inflammation, thrombocytosis, leukocytosis, hypoalbuminemia, increased alkaline phosphatase
  - Serologic tests are generally not helpful

• Temporal artery biopsy is the only test able to definitively diagnose GCA
  - Because of the focal and segmental nature of GCA, inflammation may be missed initially
  - A section of the asymptomatic side can be obtained if the initial specimens are negative
• With a positive biopsy, or a convincing history and symptoms, start oral prednisone 40–60 mg/d to reduce the risk of sudden blindness
  ➢ IV glucocorticoid for patients with transient or permanent vision loss, diplopia, TIA, or stroke
• Maintain dose for at least 1 month
• Treatment typically continues for 1–3 years
• Concomitant low-dose aspirin decreases the risk of vision loss and cranial ischemic complications
• Relapse is common — After steroid is withdrawn, monitor for return of symptoms of GCA or PMR, increase of inflammatory markers, and aortic aneurysms
• Autoimmune multisystem disease that most commonly affects women of childbearing age

• ~20% of cases have late onset (after 50 years of age)

• Compared with SLE in younger adults, late-onset SLE:
  - Shows less of a female predominance
  - Is less commonly characterized by malar rash, photosensitivity, arthritis, and nephritis, and other familiar features of SLE
  - Typically fulfills fewer ACR criteria for the classification of SLE
  - Is more commonly characterized by pulmonary involvement and serositis
• Use the 1997 ACR criteria for the classification of SLE

• Evaluate for rash, oral or nasal ulcers, non-erosive arthritis, evidence of serositis, cognitive or neurologic disorders, history of sicca symptoms that are not medication-induced, and history of Raynaud phenomenon

• **Initial lab investigation:** CBC to evaluate for cytopenias, metabolic panel, and urinalysis

• Serologic testing for ANA, anti-double-stranded DNA, and anti-Smith antibodies
  
  ➢ In late-onset lupus, anti-ribonucleoprotein or anti-Smith antibodies are less likely, but patients are more frequently positive for RF, ANA, anti-Ro/Sjögren syndrome antibodies (anti-Ro/SSA), and anti-La/SSB
• Distinguish late-onset lupus from drug-induced lupus erythematosus
  ➢ This is often clinically difficult: both have a positive ANA test, although drug-induced lupus erythematosus is associated with a speckled pattern with antihistone antibodies

• Renal biopsy for patients who have proteinuria or active urine sediment, to determine histopathology before starting treatment

• Serologic evaluation for antiphospholipid syndrome for patients with evidence of thromboembolic disease
• No clinical trials have involved older adults
• **Short-acting NSAID, if tolerated, to treat arthritis and serositis**
• **Hydroxychloroquine (200–400 mg/d) is effective in managing skin and joint manifestations**
  - Macular degeneration is a contraindication
  - **Monitor patient for visual field deficits**
  - Consider low-dose corticosteroid for patients with symptoms refractory to hydroxychloroquine
    • But significant side effects with long-term steroid use may outweigh potential benefits
• Depending on the organ system involved and disease manifestation, methotrexate, azathioprine, mycophenolate mofetil, or cyclosporine (all off-label) can be of benefit as corticosteroid-sparing agents.
  - The safety of these drugs has not been studied specifically in older adults.

• Patients with evidence of antiphospholipid syndrome should receive preventive anticoagulant as well as immunosuppressive therapy.

• High-dose steroids and cyclophosphamide are reserved for severe neuropsychiatric and renal manifestations of SLE.
SJÖGREN SYNDROME: OVERVIEW

- Systemic, multiorgan chronic disease characterized by lymphocytic infiltration of exocrine glands
- May develop alone or in conjunction with other rheumatologic diseases, including RA, lupus erythematosus, scleroderma, or inflammatory myopathy
- Presence of palpable purpura and C4 hypocomplementemia are potential predictors for development of lymphoma
 SJÖGREN SYNDROME: CLINICAL PRESENTATIONS

- **Best known manifestations:** xerophthalmia and xerostomia

- **Not limited to lacrimal and salivary glands;** may present with dysphagia, weight loss, vaginal dryness, sexual dysfunction

- **May cause extraglandular disease,** including renal, bladder, liver, biliary, thyroid, nervous system, and skin manifestations
  - Consider Sjögren syndrome in patients with interstitial lung disease, malabsorption, CNS disease that mimics multiple sclerosis, rash, and unexplained renal, liver, or thyroid disease
**Sjögren Syndrome: Diagnosis**

- **Exclude:**
  - Drug-induced sicca symptoms, particularly due to anticholinergic adverse effects
  - Age-related exocrine gland fibrosis or fatty infiltration
  - Other connective tissue syndromes

- **Schirmer test and slit-lamp examination** to assess for corneal damage and confirm the presence of keratoconjunctivitis

- **Lab evaluation:** ANA, anti-Ro/SSA, anti-La/SSB, and RF (usually positive)
SJÖGREN SYNDROME: MANAGEMENT

• For xerostomia:
  ➢ Sugar-free candies, artificial saliva
  ➢ Counsel patients on maintaining good dental health to prevent caries, gum disease, and dental erosions
  ➢ Cholinergic adverse effects limit the use of pilocarpine and cevimeline in older adults

• For xerophthalmia:
  ➢ Lubricating ointments, artificial tears
  ➢ Consider punctal plugs to retain tears

• For inflammatory eye disease: ophthalmic cyclosporine or tacrolimus
Inflammatory muscle diseases, including polymyositis and dermatomyositis, form a heterogeneous and uncommon group of skeletal muscle diseases.

Incidence peaks in adults in their 50s, but these diseases can occur at any age, and up to 20% of cases are in adults ≥65 years old.

Muscle weakness is the central feature of polymyositis and dermatomyositis, most prominently in the proximal muscle groups. Muscle tenderness is unusual; should raise suspicion of other conditions.
Arthritis, when present, is inflammatory and occasionally erosive, suggesting overlap with RA.

Esophageal dysmotility can cause dysphagia, hoarseness, and aspiration.

Arrhythmia, symptoms of congestive heart disease, dyspnea on exertion, or persistent cough suggest cardiac muscle involvement or coexistent interstitial lung disease.

Pulmonary involvement is a poor prognostic sign.

Raynaud phenomenon or Sjögren syndrome can be present.

Dermatomyositis is characterized by a rash (example: heliotrope or gottron papules).
• Differential diagnosis: endocrine, metabolic, musculoskeletal, and medication-related disorders, including thyroid disorders, diabetes, vitamin D deficiency, electrolyte abnormalities, PMR, and adverse effects from steroids or statins

• Initial evaluation: exclude more common causes of muscle weakness (example, statin use)

• Serum levels of muscle enzymes (creatine kinase, aldolase, and sometimes transaminases) are usually increased in myositis; normal levels suggest an alternative diagnosis
Electromyography is used to exclude neuropathy and identify an irritable myopathy.

**MRI** using fat-suppression sequences helps to confirm myositis and can help select which muscle to biopsy.

**Muscle biopsy** remains the gold standard to confirm the diagnosis and distinguish among subtypes of myositis.

Diagnosis of polymyositis warrants evaluation for cardiac and pulmonary disease.

Diagnosis of dermatomyositis, and less so with polymyositis, warrants heightened suspicion of an underlying malignancy — recommend age-appropriate cancer screening.
• Typical initial therapy: **oral prednisone 1 mg/kg/d**
  - Try to taper the total dose by 10%–20% per month
  - For severe disease, methylprednisolone 1,000 mg IV over 3 consecutive days
• **Methotrexate** (off-label), given orally or parenterally in a **weekly regimen**, can be combined with corticosteroids and can also have a steroid-sparing effect in long-term therapy
• **Weekly oral methotrexate** or **mycophenolate mofetil** (also off-label) may be effective in managing refractory skin manifestations of dermatomyositis
• **Azathioprine** (off-label) may be a favorable alternative in patients with interstitial lung disease or underlying liver disease

• **Intravenous immunoglobulin** can be effective in refractory dermatomyositis and is generally considered relatively safe, although older adults are at increased risk of renal failure and thrombotic events

• **Supervised exercise** has proved beneficial in polymyositis (SOE=B)
FIBROMYALGIA: OVERVIEW

• A pain syndrome that has physical and psychological components
• Hallmark: Generalized body pain
• Can be associated with other complaints, such as:
  ➢ Mood disturbance
  ➢ Fatigue
  ➢ Cognitive disorders
  ➢ Somatic symptoms
• May coincide with chronic pain from a variety of sources, including OA, neuropathy, and degenerative spinal disease
FIBROMYALGIA: DIAGNOSIS

• Diagnosis is made using Widespread Pain Index and Symptom Severity scores, with symptoms present for at least 3 months and no other explanation for symptoms.

• Diagnosis no longer includes tender point examination.

• Aside from generalized soft-tissue sensitivity, physical exam should be normal for age.
  - Importantly, patients will not have evidence of inflammation or synovitis on physical exam.
  - Lab abnormalities such as an increased ESR or CRP should prompt investigation for an alternative diagnosis.
FIBROMYALGIA: NONPHARMACOLOGIC TREATMENT

- Physical activity, patient education, and cognitive behavioral therapy are key to symptom relief

- When encouraging physical activity:
  - Reassure patient that increased pain on activity is probably temporary and not necessarily harmful
  - Increase physical activity slowly and in small increments that are easily sustainable

- Address chronic pain, sleep disturbances, and depression and anxiety individually

- Critical to form a therapeutic alliance between physician and patient
FIBROMYALGIA: PHARMACOLOGIC TREATMENT

- 3 FDA-approved medications: pregabalin, duloxetine, and milnacipran
- Acetaminophen, anticonvulsants, antidepressants, and weak opioids such as tramadol have not been studied specifically for older adults with fibromyalgia
- Most medications used to treat fibromyalgia provide only modest benefits despite considerable risk of adverse effects
- Individualize pharmacologic therapy based on specific symptoms, and reassess frequently for effectiveness and adverse effects
SUMMARY

- Osteoarthritis is the most common cause of chronic pain among older adults.
- History and physical examination remain the most important tools for diagnosing and distinguishing among the rheumatologic diseases.
- When developing treatment plans for older adults with rheumatologic disease, keep in mind each patient’s comorbid medical conditions, functional status, and potential for drug-drug and drug-disease interactions.
- Interventions ideally use a multimodal approach, including pharmacologic and nonpharmacologic treatments as well as rehabilitation modalities.
Recommendation for Rheumatology, based on the American Board of Internal Medicine Foundation’s Choosing Wisely® Campaign:

Use biologic DMARDs only after failure of nonbiologic DMARDs.
• A 70-year-old man with a 12-year history of RA asks about vaccinations for patients receiving biologic agents.

• RA had been controlled with methotrexate and prednisone until recent acute flare.
  - RA improved rapidly in hospital with high-dose prednisone and a single infusion of infliximab.

• He was discharged 1 week ago.
  - Per discharge plan, he will receive next dose of infliximab in 1 week.
• Vaccination history

- Receives intradermal influenza vaccine yearly
- Received 23-valent pneumococcal polysaccharide vaccine (PPSV23) at age 63
- Most recent tetanus vaccine was at age 59
Which one of the following should the patient receive?

A. Tetanus and reduced diphtheria toxoid vaccine (Td)
B. 13-Valent pneumococcal conjugate vaccine
C. Hepatitis B vaccine
D. Varicella zoster vaccine
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A 75-year-old man comes to the office to establish care. He feels well overall, and does not smoke.

History: type 2 diabetes, benign prostatic hyperplasia, hypertension, temporal arteritis

Current medications: metformin, hydrochlorothiazide, tamsulosin, multivitamin

- He stopped statins because of intolerable myalgia.
- He stopped prednisone treatment for temporal arteritis 5 years ago.

He asks whether he should have any evaluation related to history of temporal arteritis.
Physical examination:
- BP is 123/82 mmHg, heart rate is 72 bpm.
- BMI is 24 kg/m²
- No temporal artery tenderness
- Cardiac, pulmonary, and abdominal findings are normal.
- Distal pulses are normal.

Lab findings:
- Hemoglobin -- 13.0 g/dL
- ESR -- 50 mm/h
- Hemoglobin A1c -- 7.3%
- Blood urea nitrogen -- 14 mg/dL
- Creatinine -- 0.7 mg/dL
- Urine analysis is positive for trace albumin
Which one of the following is the most appropriate response to the patient’s concerns about temporal arteritis?

A. Reassurance that no additional follow-up is needed
B. Referral to rheumatology
C. Stress test to exclude subclinical coronary artery disease
D. Screening for aortic aneurysm
E. Temporal artery biopsy
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