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Sarcoidosis

An APCCMPD Ambulatory Care Curriculum Teaching Script

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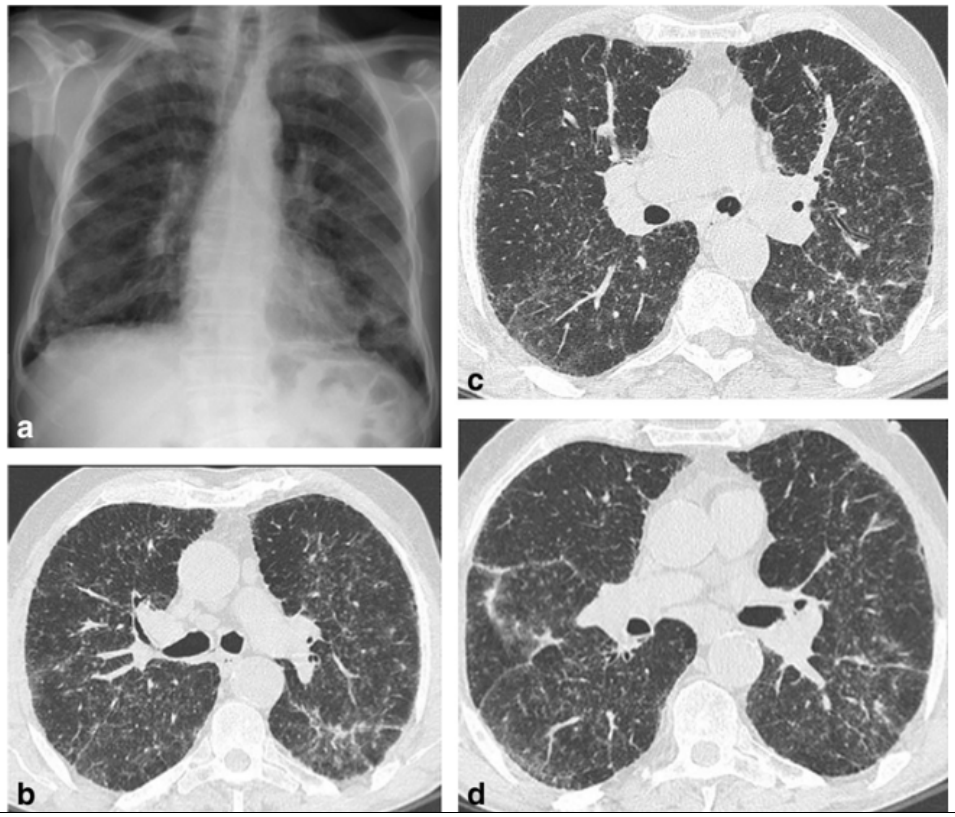
Sarcoidosis

Scenario 1:

Mr. Hernandez is a 60-year-old man with a history of hypertension, recurrent nephrolithiasis for the past two years, and recurrent bronchitis in the 12 months prior to presentation. He continues to have a non-productive cough and mild dyspnea. He has occasional fevers and night sweats but no chills. He has some arthralgias in the knees and hips. He has also developed a red, plaque-like lesion on his chest. He denied any palpitations, chest pain, nausea, vomiting, diarrhea, numbness or weakness. Review of systems is otherwise negative. He is a never smoker and has no significant family history. His physical exam is unremarkable other than the noted skin lesion on the chest. His pulmonary function tests (PFTs) are notable for moderately severe mixed obstruction and restriction (forced expiratory volume in 1 second [FEV1] was 58% predicted). His six-minute walk test was normal without evidence of ambulatory hypoxemia. A chest x-ray (CXR) and a chest CT obtained at presentation are shown (Figure 1).

Figure 1. Imaging on Presentation



CXR (panel a) with symmetric adenopathy and upper lobe predominant parenchymal changes.

Chest CT (panels b-d) demonstrate adenopathy as well as perilymphatic reticulonodular changes.

Images from Silva, et al.[1]

Question 1:
How would you approach the diagnosis in this patient? What are the clinical manifestations of sarcoidosis? What are the stages of sarcoidosis?

Question 2:
What diagnostic approaches should you consider at the time of the bronchoscopy when evaluating for pulmonary sarcoidosis?

Scenario 1 (Continued):

Given concern for sarcoidosis and kidney stones, he should be assessed for hypercalcemia. Lab work including 1,25 vitamin D, parathyroid hormone levels, serum calcium, BUN, creatinine and urinary calcium should be performed.

Question 3:
What studies should be done to work up the history of kidney stones?

Question 4:
What other studies or examinations should be done to rule out other organ involvement in sarcoidosis? What studies should he have performed to monitor his pulmonary disease?

Question 5:
Should you treat Mr. Hernandez? How does treatment vary depending on the extent of pulmonary disease? When should you consider steroid sparing therapies?

Question 6:
What drugs can cause sarcoidosis-like reactions?

Sarcoidosis

Educational Objectives:
At the end of this session, learners will be able to

1. Summarize the criteria for the diagnosis of sarcoidosis
2. Recognize imaging characteristics that can help define activity and organ involvement
3. Describe the initial approach to treatment based on organ involvement

Scenario 1:

Mr. Hernandez is a 60-year-old man with a history of hypertension, recurrent nephrolithiasis for the past two years, and recurrent bronchitis in the 12 months prior to presentation. He continues to have a non-productive cough and mild dyspnea. He has occasional fevers and night sweats but no chills. He has some arthralgias in the knees and hips. He has also developed a red, plaque-like lesion on his chest. He denied any palpitations, chest pain, nausea, vomiting, diarrhea, numbness or weakness. Review of systems is otherwise negative. He is a never smoker and has no significant family history. His physical exam is unremarkable other than the noted skin lesion on the chest. His pulmonary function tests (PFTs) are notable for moderately severe mixed obstruction and restriction (forced expiratory volume in 1 second [FEV1] was 58% predicted). His six-minute walk test was normal without evidence of ambulatory hypoxemia. A chest x-ray (CXR) and a chest CT obtained at presentation are shown (Figure 1).

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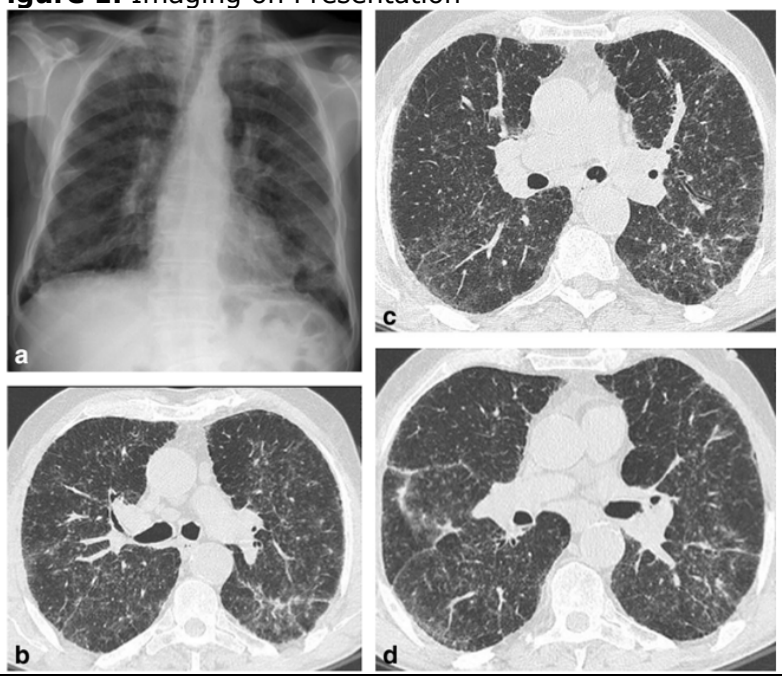

CXR (panel a) with symmetric adenopathy and upper lobe predominant parenchymal changes.

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Images from Silva, et al.[1]

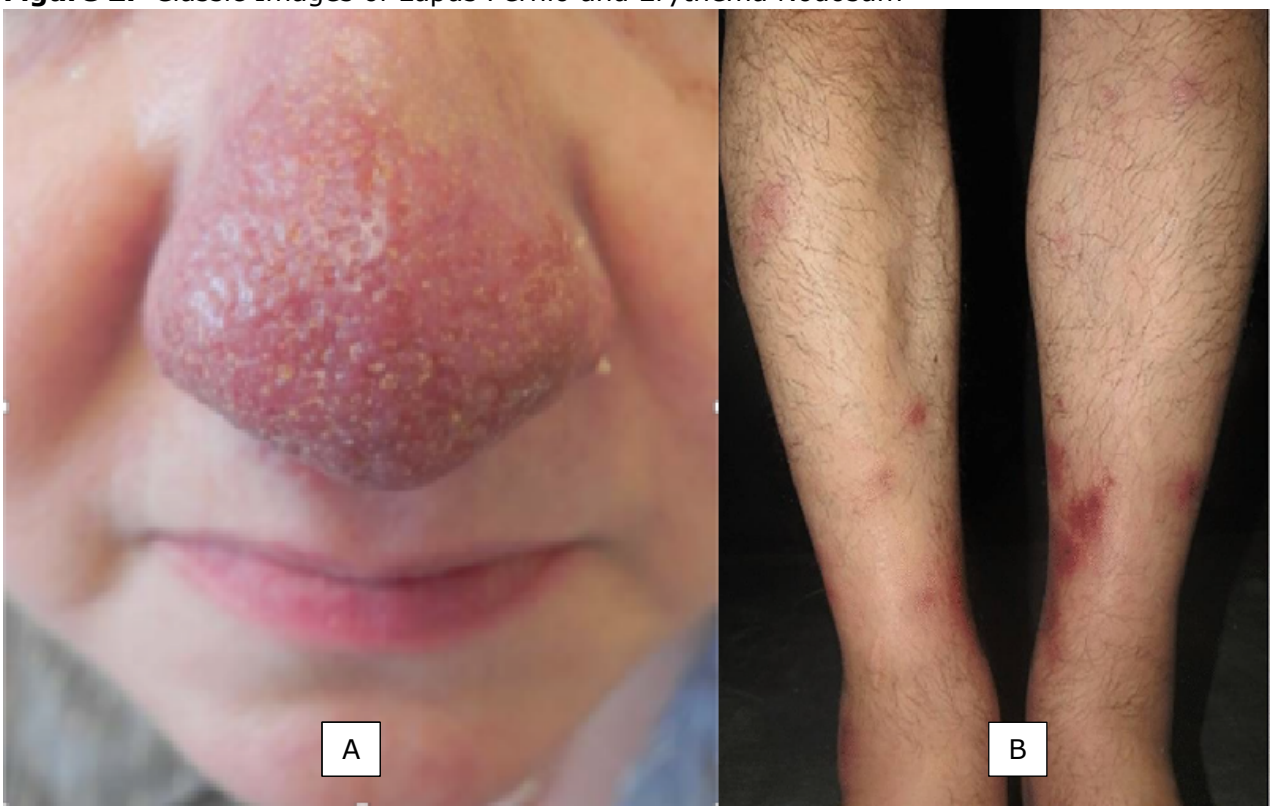
Question 1: How would you approach the diagnosis in this patient? What are the clinical manifestations of sarcoidosis? What are the stages of sarcoidosis?

Mr. Hernandez's presentation is consistent with a diagnosis of sarcoidosis. Clinical symptoms of pulmonary sarcoidosis typically include cough (usually non-productive), dyspnea, and chest discomfort. Systemic symptoms including fevers, night sweats, weight loss, depression, fatigue, and myalgias may also be present. Although sarcoidosis can be familial, most patients report no family history. PFT findings are widely varied with most patients having normal results.[2]

Sarcoidosis is a diagnosis of exclusion that requires a clinical presentation or pattern on imaging consistent with the diagnosis granulomatous inflammation on histopathology (Table 1), and exclusion of sarcoidosis mimics. Histopathology showing noncaseating granulomas is necessary to make a diagnosis in all cases apart from the following presentations, which do not require a biopsy:

- Lupus Pernio: Chronic raised and indurated lesion of the skin over nose, often purplish in color (2A)
- Lofgren's syndrome: Characterized by bilateral hilar lymphadenopathy, erythema nodosum (Figure 2B), migratory polyarthralgias and fever
- Heerfordt's syndrome: Facial nerve palsy, parotid gland enlargement, anterior uveitis, and low-grade fever

Figure 2. Classic Images of Lupus Pernio and Erythema Nodosum



Lupus pernio (left) and erythema nodosum (right).
Images taken from Caplan, et al and Perez-Garza et al.[3,4]

Table 1. Clinical, Radiographic, and Pathologic Findings of Sarcoidosis

	Highly Probable	Probable
History	Löfgren's syndrome*	Seventh cranial nerve paralysis Treatment-responsive renal failure Treatment-responsive CM or AVNB Spontaneous/inducible VT with no risk factors
Physical	Lupus pernio Uveitis Optic neuritis Erythema nodosum	Maculopapular, erythematous, or violaceous skin lesions Subcutaneous nodules Scleritis Retinitis Lacrimal gland swelling Granulomatous lesions on direct laryngoscopy Symmetrical parotid enlargement Hepato-/splenomegaly
Imaging	Bilateral hilar adenopathy (CXR, CT, and PET) Perilymphatic nodules (chest CT) Gadolinium enhancement on MRI (CNS) Osteolysis, cysts/punched-out lesion, trabecular pattern bone (X-ray, CT, and MRI) Parotid uptake (gallium and PET)	Upper lobe or diffuse infiltrates (CXR, CT, and PET) Peribronchial thickening (CT) Two or more enlarged extra thoracic nodes (CT, MRI, and PET) Increased inflammatory activity in heart (MRI, PET, and gallium) Imaging showing enlargement or nodules in liver or spleen (CT, PET, and MRI) Inflammatory lesions in bone (gallium, PET, and MRI)
Other testing	Hypercalcemia or hypercalciuria with abnormal vitamin D metabolism†	Reduced LVEF with no risk factors (echo and MRI) Elevated ACE level test‡ Nephrolithiasis with calcium stone, no vitamin D testing BAL lymphocytosis or elevated CD4:CD8 ratio Alkaline phosphatase greater than three times the upper limit of normal New-onset, third-degree AV block in young or middle-aged adults

Definition of abbreviations: ACE = angiotensin-converting enzyme; AV = atrioventricular; AVNB = atrioventricular node block; CM = cardiomyopathy; CNS = central nervous system; CT = computed tomography; CXR = chest X-ray; LVEF = left ventricular ejection fraction; MRI = magnetic resonance imaging; PET = positron emission tomography; VT = ventricular tachycardia.
*Löfgren's syndrome is defined as bilateral hilar adenopathy with erythema nodosum and/or periarticular arthritis.
†Abnormal vitamin D metabolism is defined as normal to low parathyroid hormone, normal to elevated 1,25-dihydroxyvitamin D, and normal to low 25-hydroxyvitamin D.
‡ACE elevated above 50% of the upper limit of normal was considered abnormal.

Features favoring the diagnosis of sarcoidosis.
Taken from Crouser et al.[5]

When histopathologic confirmation is required, the least invasive biopsy feasible should be performed. For Mr. Hernandez, a skin biopsy is likely the best first step to document a granulomatous process. Other non-pulmonary sites to consider for biopsy include peripheral lymph nodes, lacrimal glands, and conjunctiva. If the skin biopsy is negative and there is evidence of disease in a visceral organ, the lungs in this case, then invasive pulmonarybiopsies could be performed to document granulomatous inflammation.

For patients presenting with asymptomatic bilateral hilar lymphadenopathy, the diagnosis is almost always sarcoidosis, though lymphoma or infection remain on the differential diagnosis. The American Thoracic Society (ATS) guidelines recommend the decision to biopsy be made with the patient, considering things such as patient preference, risk for malignancy, and likelihood of achieving follow-up.[5] If lymph node sampling is not obtained, close clinical follow-up is a reasonable alternative approach.[5] For patients without apparent lung involvement, PET-CT can be useful to identify potential targets for biopsy (e.g., liver, spleen). FDG-PET and MRI with gadolinium can detect cardiac and neurologic involvement. Of note, cardiac, hepatic, and neurosarcoidosis can occur in isolation without apparent disease activity elsewhere in the body. However, clinical sarcoidosis most commonly

manifests as intrathoracic lymph node enlargement, pulmonary parenchymal abnormalities, cutaneous, or ocular changes in the vast majority of patients.

Sarcoidosis is a granulomatous disease of uncertain etiology that can involve multiple organs. Classic CT findings include lymphadenopathy, and nodules in a perilymphatic and central distribution.[6] Less common findings include large consolidation, perihilar opacities, and pulmonary fibrosis, pleural effusions, and cavitation.[6] The radiographic findings are typically organized into stages which reflect an anatomic guide of lung involvement rather than disease activity (Table 2).

Table 2. Scadding Stage of Pulmonary Sarcoidosis

	Radiographic Findings	**Frequency at Presentation**	**Spontaneous Resolution**
Stage 0	Normal chest radiograph	8-10%	
Stage I	Hilar or mediastinal nodal enlargement only	40-51%	49-82%
Stage II	Nodal enlargement and parenchymal disease (often upper lung zone predominant reticular opacities)	29-40%	31-68%
Stage III	Parenchymal disease only	12-20%	10-38%
Stage IV	Pulmonary fibrosis;reticular opacities with evidence of volume loss; +/- traction bronchiectasis; +/- calcification, cavitation or cyst formation	2-5%	0%

Description of Scadding Stages with frequency of occurrence and of resolution.[6]
Data derived from: Belperio JA, Shaikh F, Abtin FG, et al. Diagnosis and Treatment of Pulmonary Sarcoidosis: A Review. *JAMA*. 2022;327(9):856–867. doi:10.1001/jama.2022.1570

A biopsy of the skin lesion on Mr. Hernandez's chest was negative for granulomatous inflammation, so a bronchoscopy was scheduled.

Question 2: What diagnostic approaches should you consider at the time of the bronchoscopy when evaluating for pulmonary sarcoidosis?

When evaluating a patient for pulmonary sarcoidosis, the following diagnostic studies should be considered (Table 3):

Table 3. Commonly Used Bronchoscopic Techniques in the Diagnosis of Pulmonary Sarcoidosis

Technique	**Diagnostic Yield**
Bronchoalveolar lavage	
Endobronchial biopsy	20-61%
Endobronchial ultrasound guided transbronchial needle aspiration (EBUS-TBNA)	80-94%
Transbronchial bronchoscopic biopsy (TBBx)	37-90%
Transbronchial lung cryobiopsy	67-92.6%

Common diagnostic bronchoscopic techniques and their yield.[7]
Adapted from J. Clin. Med. 2019, 8, 1327; doi:10.3390/jcm8091327

Bronchoalveolar lavage is minimally invasive and can both provide information to support the diagnosis of sarcoidosis and to rule out sarcoidosis mimics. An analysis of the T- cell subtypes can be valuable as a CD4+/CD8+ ratio $\geq$3.5 is suggestive of sarcoidosis. Typically, this is utilized along with EBUS-TBNA to maximize yield. Ideally, 3-5 TBNAs are performed with rapid onsite pathology available to determine the need for additional tissue sampling.

The yield of endobronchial biopsies increases with patients who experience airway related symptoms such as wheezing or cough, or those with cobblestone mucosa of the airways. A minimum of 3-4 different samples should be obtained. Endobronchial biopsy would not be used as a standalone technique and should be used in concert with others, such as EBUS-TBNA and TBBx, to maximize yield.[7] Transbronchial lung biopsies are most helpful when there is visible parenchymal involvement on imaging. At least 4 samples should be obtained as higher samples numbers increase the yield. Finally, in experienced centers, transbronchial cryobiopsies can provide even greater yields due to the larger pieces of lung tissue obtained with less crush artifact.[7]

Once pathology confirming the presence of granulomas is obtained, it is important to consider whether the patient's presentation still fits the diagnosis of sarcoidosis or if other mimics should be considered (Table 4).

Table 4. Sarcoidosis Mimics

Infectious	**Non-Infectious**	
• Tuberculosis • Non-tuberculous mycobacteria • Cryptococcus • Endemic fungi	• Hypersensitivity pneumonitis • Chronic beryllium disease • Occupational exposures • Chronic aspiration • Talc • Inflammatory bowel disease Granulomatous-lymphocytic interstitial lung disease	• Granulomatosis with polyangiitis • Eosinophilic granulomatosis with polyangiitis • Rheumatoid nodule • Sarcoid like reaction to malignancy • Medications (i.e., checkpoint inhibitors)

List of other conditions causing granulomatous inflammation on biopsy.[8]

Data derived from: Harper LJ, Farver CF, Yadav R, Culver DA. A framework for exclusion of alternative diagnoses in sarcoidosis. *Journal of autoimmunity*. 2024;149:103288. doi:https://dx.doi.org/10.1016/j.jaut.2024.103288

Question 3: What studies should be done to work up the history of kidney stones?

Scenario 1 (Continued):

Given concern for sarcoidosis and kidney stones, he should be assessed for hypercalcemia. Lab work including 1,25 vitamin D, parathyroid hormone levels, serum calcium, BUN, creatinine and urinary calcium should be performed.

Question 4: What other studies or examinations should be done to rule out other organ involvement in sarcoidosis? What studies should he have performed to monitor his pulmonary disease?

Evaluation of other organ involvement in all patients with newly diagnosed sarcoidosis should consist of the following[5]:

- Detailed history and physical
- Baseline eye exam
- Baseline labs: complete blood count, creatinine, alkaline phosphatase, calcium, 25-OH vitamin D, 1,25-OH vitamin D
- Baseline electrocardiogram

Other evaluations should be ordered as needed based on the history and physical and initial testing.

On follow-up, the calcium, creatinine, and alkaline phosphatase should be re-evaluated annually. Special attention should be paid on follow-up to review of systems and physical exam to guide additional investigation of other organ involvement. Specifically, the following should trigger additional evaluation:

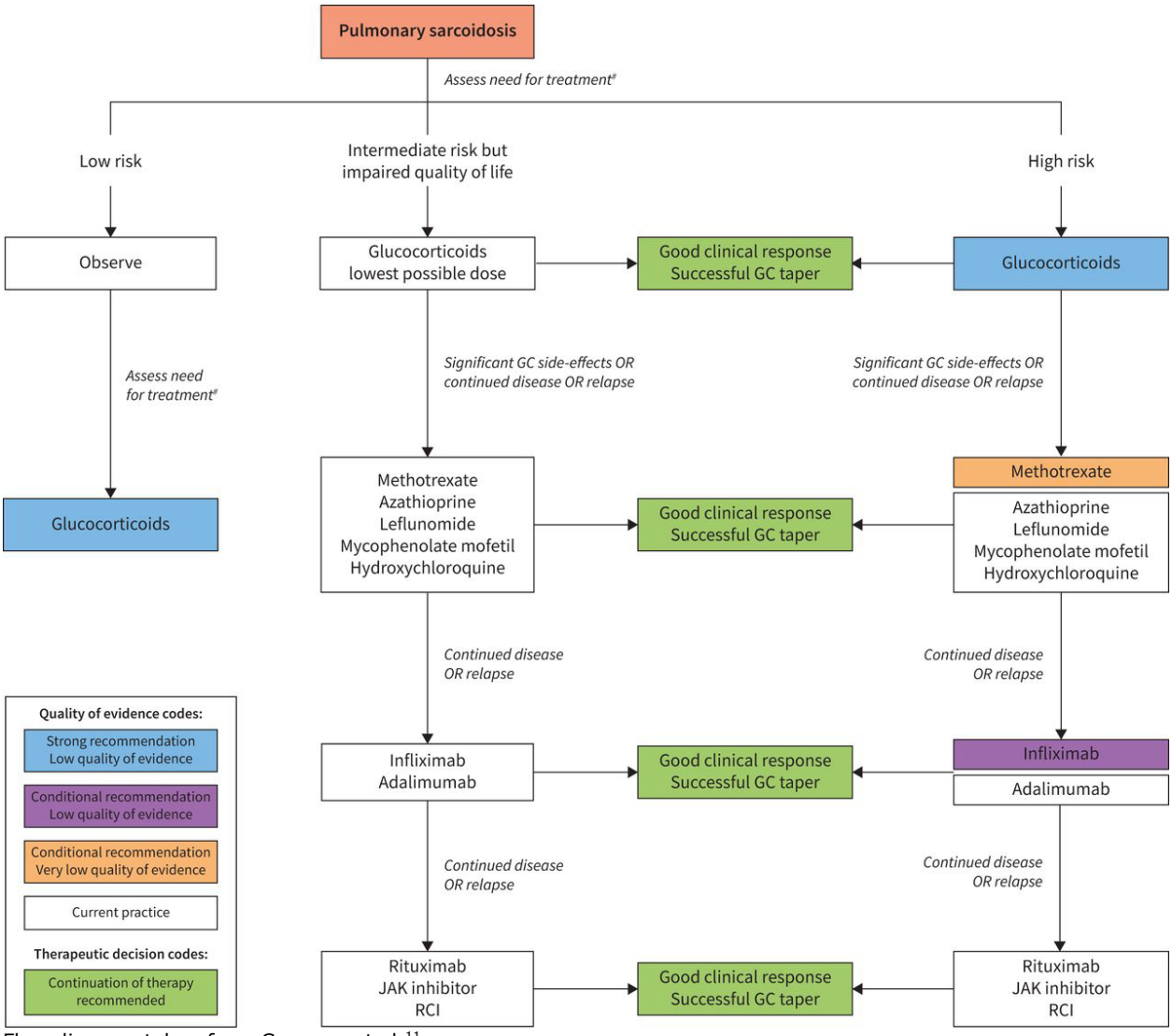
- Pulmonary: new symptoms such as dry cough or dyspnea
- Cardiac: palpitations, presyncope, dyspnea out of proportion to lung involvement
- Ophthalmologic: dry eyes, redness
- Neurologic: strokes, seizures, memory issues

Given that Mr. Hernandez has known pulmonary sarcoidosis, routine pulmonary function testing and annual chest x-rays should be considered, with the use of high-resolution chest CT scans reserved for further investigation of these.[9]

Question 5: Should you treat Mr. Hernandez? How does treatment vary depending on the extent of pulmonary disease? When should you consider steroid sparing therapies?

Most patients with sarcoidosis do not require treatment and treatment should only be considered to avoid danger (end-organ damage) or to improve quality of life.[10] Generally speaking, patients should be treated if there is evidence of active inflammation, bothersome symptoms related to organ involvement, impaired organ function or the patient is at risk for progressing to fibrosis. Imaging findings suggesting sarcoidosis activity include nodules, recurrent consolidation, and occasionally ground glass opacities. The suggested algorithm for the treatment of pulmonary sarcoidosis is outlined in Figure 3.

Figure 3. Suggested Approach to Treatment of Pulmonary Sarcoidosis



Flow diagram taken from Crouser, et al.[11]

Given that Ms. Hernandez has moderate symptoms and moderately severe impairment in her PFTs, starting systemic steroids should be considered, usually in a range of 20-40 mg daily at the start. A recent open-label RCT compared prednisolone 40 mg vs. 20 mg in patients with pulmonary sarcoidosis, which found that high-dose prednisolone did not lead to different rates of relapse, treatment failure, adverse events or quality of life scores.[12] For him, we might opt for 20 mg with a planned taper over the next several months given the modest degree of impairment, but again, there will be variability by provider preference.

Based on the results of a recent study, initiation of methotrexate as first line monotherapy should also be considered for Mr. Hernandez. In the PREDMETH study, patients with pulmonary sarcoidosis were randomized to prednisone or methotrexate as first line therapy with a primary end point of mean change in FVC after 24 weeks. Methotrexate was non-inferior to prednisone and adverse events were similar between the two groups. The decision of which medication to start rests in patient preference and tolerance of the side

effect profiles of the two medications.[13] A separate study demonstrated that over longer periods of time, patients tolerated methotrexate better than prednisone, reporting a better quality of life with 78% of patients reporting side effects with prednisone use compared with 48% of those on methotrexate.[14]

Steroid-sparing therapies should be considered for all patients who have symptomatic pulmonary sarcoidosis and are at higher risk of permanent sequelae from their disease despite treatment with steroids (e.g., fibrotic lung disease), or those who cannot tolerate steroids. Evidence also supports the use of infliximab, particularly in those with extrapulmonary sarcoid.[15] Many of the therapies however also have a number of side effects (Table 5). Treatment recommendations vary with the presence of extrapulmonary sarcoidosis depending on the specific organ involved and the severity of disease. Additional treatment algorithms for extrapulmonary sarcoidosis can be found through the European Respiratory Society.[11]

Table 5. Commonly Used Steroid Sparing Therapies in Sarcoidosis

Drug	Usual dosage	Major toxicities	Recommended monitoring	Comments
Prednisone or prednisolone	Initial 20 mg once a day; follow-up 5–10 mg once a day to once every other day	Diabetes; hypertension; weight gain; osteoporosis; cataracts; glaucoma; moodiness	Bone density; blood pressure and serum glucose	Cumulative toxicity
Methotrexate	10–15 mg once a week	Nausea; leukopenia; hepatotoxicity; pulmonary	CBC, hepatic, renal serum testing	Cleared by kidney, avoid in significant renal failure
Leflunomide	10–20 mg once a day	Nausea; leukopenia; hepatotoxicity; pulmonary	CBC, hepatic, renal serum testing	Cleared by kidney, avoid in significant renal failure
Azathioprine	50–250 mg once a day	Nausea; leukopenia; infections; malignancy	CBC	
Mycophenolate mofetil	500–1500 mg twice a day	Diarrhea; leukopenia; infections; malignancy	CBC	Less experience in sarcoidosis than other agents
Infliximab or biosimilars	3–5 $mg \cdot kg^{-1}$ initially, 2 weeks later, then once every 4–6 weeks	Infections; allergic reaction	Screen for prior TB; monitor for allergic reactions; contraindicated in severe CHF, prior	Allergic reactions can be life threatening

Drug	Usual dosage	Major toxicities	Recommended monitoring	Comments
			malignancy, demyelinating neurologic disease, active TB, deep fungal infections	
Adalimumab	40 mg every 1–2 weeks	Infections	Screen for prior TB; monitor for allergic reactions; contraindicated in severe CHF, prior malignancy, demyelinating neurologic disease, active TB, deep fungal infections	Less toxic than infliximab
Rituximab	500–1000 mg every 1–6 months	Infections	Screen for viral hepatitis; check IgG level with chronic therapy	High risk for viral reactivation; can lead to IgG deficiency
Hydroxychloroquine	200–400 mg once a day	Loss of vision	Ocular exams periodically depending on age and renal function	Minimal impact on cardiac and neurologic disease

Common medications used in the treatment of sarcoidosis along with dosing, recommended lab monitoring and potential toxicities.
Taken from: Baughman RP, Valeyre D, Korsten P, et al. ERS clinical practice guidelines on treatment of sarcoidosis. European Respiratory Journal. 2021;58(6)

Question 6: What drugs can cause sarcoidosis-like reactions?

Some medications can induce a multisystem granulomatous inflammatory response that mimics sarcoidosis. It is important to recognize this as stopping the causative medication will typically result in complete resolution of symptoms. The diagnosis of drug-induced sarcoidosis should be considered in patients who have no history of sarcoidosis and whose symptoms begin during or after exposure to a medication known to cause this type of reaction. Medications known to cause drug-induced sarcoidosis include:

- Immune checkpoint inhibitors
- Anti-retroviral therapy
- Interferons
- Tumor necrosis factor-alpha antagonists.[16]

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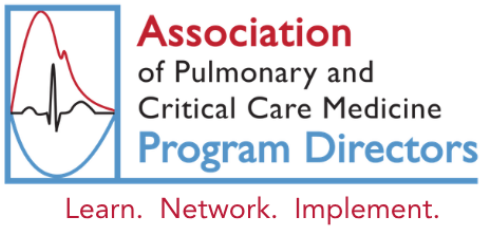

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