CME OUTFITTERS

From Biomarkers to Bedside:

Translating IIM Classification into Targeted Treatment Strategies

Supported through an independent educational grant from argenx



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LEARNING OBJECTIVE 1

Apply biomarker-informed diagnostic strategies to differentiate major IIM subtypes and distinguish inflammatory disease from key mimics

Patient Case: Michael



52-year-old male presenting with four months of progressive difficulty rising from a chair and lifting overhead

No rash. No dysphagia. No dyspnea. No new medications and no family history of muscle disease.

Referred with a working diagnosis of "***polymyositis***."

Examination: symmetric proximal weakness in hip flexors and shoulder abductors; distal strength preserved

CK: 1,840 U/L

Myositis-specific antibody panel: Negative

CK = creatine kinase; U/L = units per liter.

Audience Response



A 52-year-old male has four months of progressive symmetric proximal weakness, CK 1840 U/L, and negative myositis serology. Which interpretation should guide the next diagnostic step?

A. Repeat a broader myositis antibody panel first, because a subtype specific antibody result would determine whether imaging or biopsy is still needed.

B. Proceed directly to muscle biopsy, because seronegative IIM requires tissue confirmation before other neuromuscular testing can refine the differential.

C. Use EMG and muscle MRI to characterize and localize the process, then pursue targeted biopsy if uncertainty remains after multimodal evaluation.

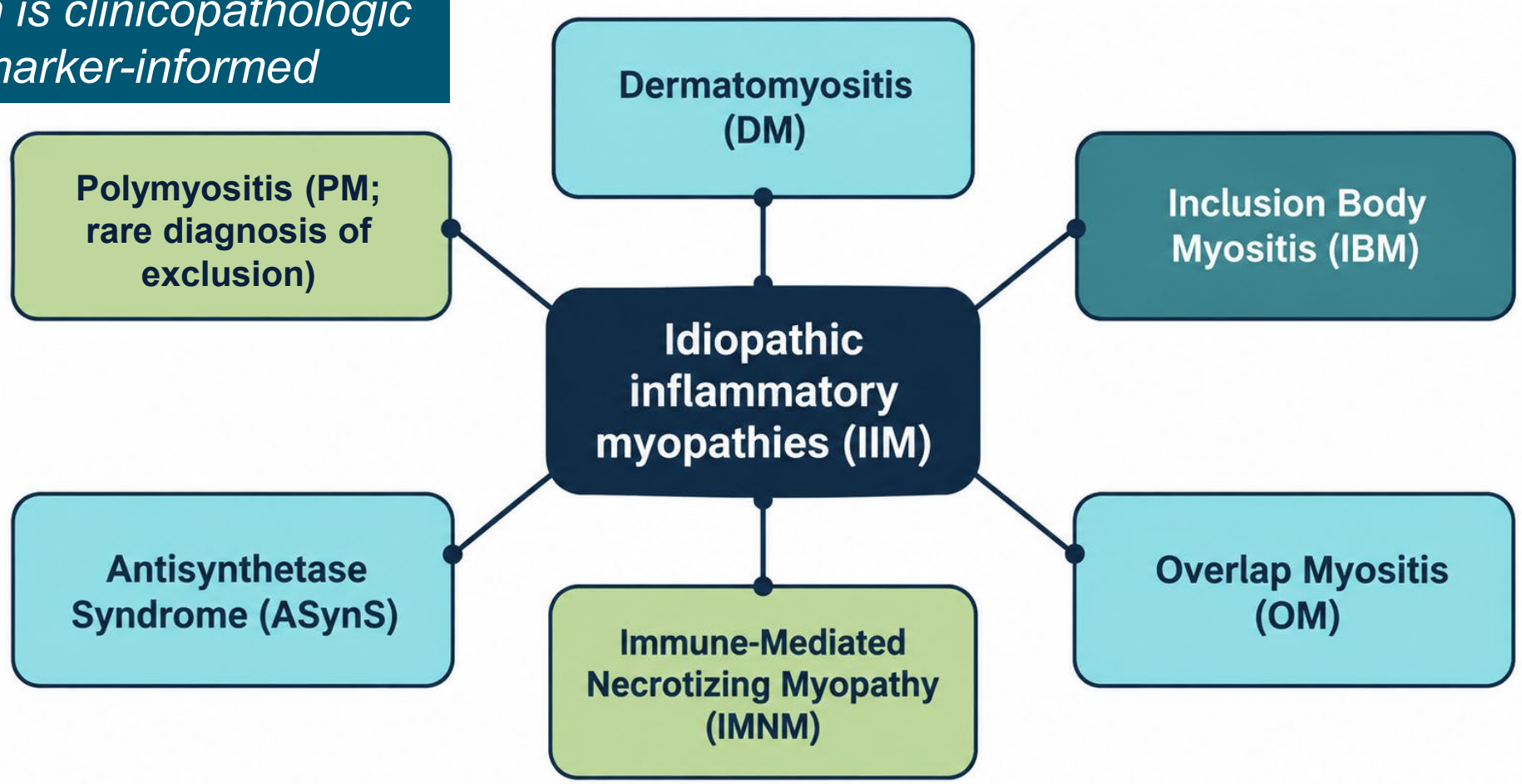
D. Start glucocorticoids and reassess strength and CK, because treatment response can help distinguish inflammatory from noninflammatory myopathy.

EMG = electromyography; IIM = idiopathic inflammatory myopathy; MRI = magnetic resonance imaging.

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Classification of IIM

Classification is clinicopathologic and biomarker-informed



Loarce-Martos J, et al. *Rheumatology (Oxford)*. 2021;60(7):3398-3403. Lundberg IE, et al. *Nat Rev Dis Primers*. 2021;7(1):87.

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Subtypes Separate on Features Seen at Bedside

Feature	PM	DM	IBM	IMNM	ASyS	OM
Weakness Pattern	Symmetric proximal; diagnosis of exclusion	Symmetric proximal; acute/subacute onset	Asymmetric; distal > proximal; finger flexors and quadriceps; slow onset	Symmetric severe proximal; often acute, rapidly progressive	Symmetric mild-to-moderate proximal	Variable; often proximal with overlap features
Cutaneous Presentation	Absent	Heliotrope rash; Gottron's papules/sign; V-sign; Shawl sign	Absent	Absent	Mechanic's hands	Variable, depending on overlap disease
Extramuscular Presentation	Variable systemic overlap	High malignancy risk	Early dysphagia	Minimal systemic involvement	ILD, arthritis; Raynaud's	CTD overlap features, e.g., ILD, arthritis, Raynaud's
Biopsy Pattern	Endomysial inflammatory infiltrates invading non-necrotic fibers	Perifascicular pathology; perifascicular sarcoplasmic MxA upregulation; capillary complement deposition may be present	Endomysial inflammation with invasion of non-necrotic myofibers; protein aggregates; rimmed vacuoles; mitochondrial abnormalities	Extensive necrosis and regeneration; minimal or absent lymphocytic inflammation; diffuse fine granular sarcoplasmic p62 staining may be seen	Inflammatory infiltrates, often perimysial; may show significant perifascicular necrosis/regeneration; MxA usually absent	Variable, depending on overlap disease
Associated autoantibodies	Often: • Anti-Ro • Anti-PM/Scl	• Anti-Mi2 • Anti-TIF1-γ • Anti-NXP2 • Anti-MDA5 • Anti-SAE	• Anti-cN1A	• Anti-SRP • Anti-HMGCR	• Anti-Jo-1 • Anti-PL-7 • Anti-PL-12 • Anti-OJ • Anti-EJ	• Anti-Ro • Anti-PM/Scl

cN1A = cytosolic 5′-nucleotidase 1A; CTD = connective tissue disease; EJ = glycyl-tRNA synthetase; HMGCR = 3-hydroxy-3-methylglutaryl-coenzyme A reductase; ILD = interstitial lung disease; Jo-1 = histidyl-tRNA synthetase; MDA5 = melanoma differentiation-associated protein 5; MHC-1 = major histocompatibility complex class I; NXP2 = nuclear matrix protein 2; OJ = isoleucyl-tRNA synthetase; PL-7 = threonyl-tRNA synthetase; PL-12 = alanyl-tRNA synthetase; PM/Scl = polymyositis/scleroderma; SAE = small ubiquitin-like modifier-activating enzyme; SRP = signal recognition particle; TDP-43 = TAR DNA-binding protein 43; TIF1-γ = transcriptional intermediary factor 1-gamma.

Faghihi-Kashani S, et al. *Arthritis Rheumatol*. 2025;77(4):477-489. Fischer N, et al. *Brain Pathol.* 2020;30(2):261-271. Lundberg IE, et al. *Nat Rev Dis Primers*. 2021;7(1):86. Naddaf E. *Front Neurol.* 2022;13:1020113. Tanboon J, et al. *Neurology.* 2022;98(7):e739-e749. Tanboon J, et al. *Brain Pathol.* 2023;33(4):e13155. Saygin D, et al. *Rheum Dis Clin North Am.* 2025;51(4):563-576.

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Read the Syndrome Before the Serology

"The degree of CK elevation does not distinguish IIMs from other types of myopathies"

Tempo

- Most IIM subtypes present **subacutely**
- IBM and hereditary myopathies are chronic
- **Acute weakness:** infectious myositis, rhabdomyolysis or electrolyte-related myopathy

Distribution

- Muscle predominant IIM often presents with symmetric proximal weakness, commonly involving hip flexors and abductors
- Proximal lower extremity more affected than upper in IMNM and ASyS

Subtype clue

- Proximal predominant weakness, often involving hip flexors and abductors
- Disproportionate finger flexor or knee extensor weakness raises concern for **IBM**

CK in context

- Ranges from normal to markedly raised
- **Normal CK does not exclude DM**; lower CK is particularly seen with anti MDA5 and anti-TIF1γ phenotypes
- **IMNM** typically >**5,000** U/L

Read tempo, distribution, specific muscles and CK together – syndrome-level judgment comes before the serology

De Lorenzo R, et al. *Neurology.* 2018;90(23):e2068-e2076. Harris Love MO, et al. *Rheumatology (Oxford).* 2009;48(2):134-139. Naddaf E. *Front Neurol.* 2022;13:1020113. Roy B, et al. *Curr Rheumatol Rep.* 2026;28(1):12. Tanboon J, et al. *Neurology.* 2022;98(7):e739-e749.

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EMG and MRI Localize, but not Necessarily Confirm

EMG/nerve conduction

- Delineates the pattern of muscle involvement
- Guides selection of the optimal muscle for biopsy
- Ancillary marker of ongoing disease activity
- **Limit:** fibrillation potentials can be absent in partially treated IIM

Muscle MRI

- **Minimum protocol:** T1-weighted plus STIR or fat-saturated T2 of the thighs
- Edema early in active disease; atrophy and fatty replacement later - both can co-occur
- Can identify the appropriate biopsy site
- **Limit:** Edema is not specific, so MRI alone is insufficient to diagnose myositis

STIR = short tau inversion recovery.

Roy B, et al. *Curr Rheumatol Rep*. 2026;28(1):12.

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Pathology Can Refine the Diagnosis

Michael is seronegative. Biopsy can help identify a characteristic disease pattern and refine subtype assessment.

PATHOLOGY PATTERNS

Classic DM:	Perifascicular atrophy and microangiopathy Capillary C5b-9 deposition on endomysial microvessels Perivascular/perimysial lymphocytic inflammation Perifascicular MHC-I upregulation
IMNM:	Myofiber necrosis with scant lymphocytic inflammation Markedly elevated CK
ASyS:	Perifascicular necrosis/regeneration with perimysial connective tissue inflammation and increased alkaline phosphatase activity

Shared in IIM, not discriminating:

Sarcolemmal MHC-I overexpression and scattered necrotic/regenerating fibers can support inflammatory myopathy but are not subtype specific.



MxA CAN SUPPORT DM DIAGNOSIS

MxA immunohistochemistry

Sarcoplasmic myxovirus resistance protein A separates DM from other IIMs, **including on treatment-modified biopsies**.

46%
Sensitivity
138-specimen series

94%
Specificity
138-specimen series

Across earlier cohorts

57-77% sensitivity

98-100% specificity

Sensitivity varies widely between series; specificity does not. A negative MxA does not exclude DM — read it alongside the morphology, never instead of it.

WHEN CLINICIANS BIOPSY

More likely to biopsy

65.6% undetectable myositis antibodies

58.3% abnormal MRI showing edema

Less likely to biopsy

53.2% positive myositis-specific antibody

70.8% classic dermatomyositis rash

What guides muscle selection

85.2% clinical examination

MRI 51.9% · EMG 45.4%

Non-diagnostic results reported as common or very common

Needle 63.3%

Open surgical 50.5%

Biopsy-practice percentages are the proportion of 116 surveyed expert clinicians reporting that practice or perception; not patient-level rates and not measures of diagnostic accuracy. Non-diagnostic figures are the proportion who rated such results common (1–10%) or very common (>10%).

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C5b-9 = membrane–attack complex; MHC-I = major histocompatibility complex class I; MxA = myxovirus-resistance protein A.

Maxwell S, et al. *Semin Arthritis Rheum*. 2024;68:152519. Milisenda JC, et al. *Rheumatology (Oxford)*. 2023;62(9):3156-3160. Parga A, et al. *Muscles*. 2025;4(4):58. Tanboon J, et al. *Brain Pathol*. 2023;33(4):e13155. Waisayarat J, et al. *J Inflamm Res*. 2023;16:5417-5426.

A Negative Panel Does Not Exclude IIM

Classification criteria and antibody panels inform diagnosis; they do not replace clinical judgment

Criteria are not a diagnosis
- 2017 EULAR/ACR criteria include only anti-Jo-1
- ASyS and IMNM are not classified as distinct subtypes

Panels and assays vary
- Panels may omit relevant antibodies
- Performance varies by antibody and testing method

Labels have shifted
- PM is now a rare diagnosis of exclusion
- Re-evaluation for a better-defined IIM subtype or alternative diagnosis

Interpret MSA results in clinical context; integrate pathology when indicated.

> *"I think the fatigue side seems to be missed quite a bit, that's never sort of talked about too much, it's more just 'what's your strength' and 'are you breathing okay'."*
>
> **- Individual with IIM**

EULAR/ACR = European Alliance of Associations for Rheumatology/American College of Rheumatology; PPV = positive predictive value.

Gono T, et al. *Semin Arthritis Rheum.* 2025;75:152858. Loarce-Martos J, et al. *Rheumatology (Oxford).* 2021;60(7):3398-3403. Lundberg IE, et al. *Ann Rheum Dis.* 2017;76(12):1955-1964. Oldroyd A, et al. *BMC Rheumatol.* 2020;4:47.

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Eight Red Flags That Should Stop Premature Closure

	Red flag	What it suggests
M	Myopathic MUPs without fibrillations	**Consider metabolic, congenital, steroid-induced or non-specific myopathies.** Fibrillation may also be absent in partially treated IIM or some DM presentations.
Y	Young onset or family history	**Hereditary myopathies**: neonatal to childhood onset favors hereditary disease. **Inherited myopathies**: family history supports an inherited cause.
O	Onset atypical for IIMs	**Chronic course**: hereditary myopathies or IBM. **Acute course**: infectious myositis, rhabdomyolysis, electrolyte-related myopathy. Most IIM subtypes present sub acutely.
S	Seronegative or discordant serology	**Low-titer or weakly positive MSAs are not by themselves diagnostic.** Interpret antibody results alongside phenotype and, when indicated, muscle pathology.
I	Iatrogenic exposure	**Consider toxic myopathy and exposure to myotoxic drugs.** Weakness may begin weeks to months after exposure.
T	Treatment refractoriness	**IBM and genetic myopathies may explain lack of the expected response.** Reconsider the diagnosis rather than assuming treatment-resistant IIM.
I	Irregular weakness distributions	**Distal, asymmetric, scapuloperoneal, ocular, bulbar or early respiratory patterns suggest mimics.** Finger-flexor and quadriceps weakness is characteristic of IBM.
S	Systemic features	**Rash, arthritis or interstitial lung disease may support IIM.** The absence of these features does not exclude IIM.

GVHD = graft-versus-host disease; MSAs = myositis-specific autoantibodies; MUP = motor unit potential.

Parga A, et al. *Muscles.* 2025;4(4):58. Roy B, et al. *Curr Rheumatol Rep*. 2026;28(1):12.

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Five Reasons to Refer



The diagnosis remains unclear



Phenotype and serology remain discordant



The clinical course is unexpected



Response to treatment does not fit the presumed diagnosis



The patient has received prolonged treatment without expected improvement

Roy B, et al. *Curr Rheumatol Rep*. 2026;28(1):12.

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Audience Response Reask

A 52-year-old male has four months of progressive symmetric proximal weakness, CK 1840 U/L, and negative myositis serology. Now which interpretation should guide the next diagnostic step?

A. Repeat a broader myositis antibody panel first, because a subtype specific antibody result would determine whether imaging or biopsy is still needed

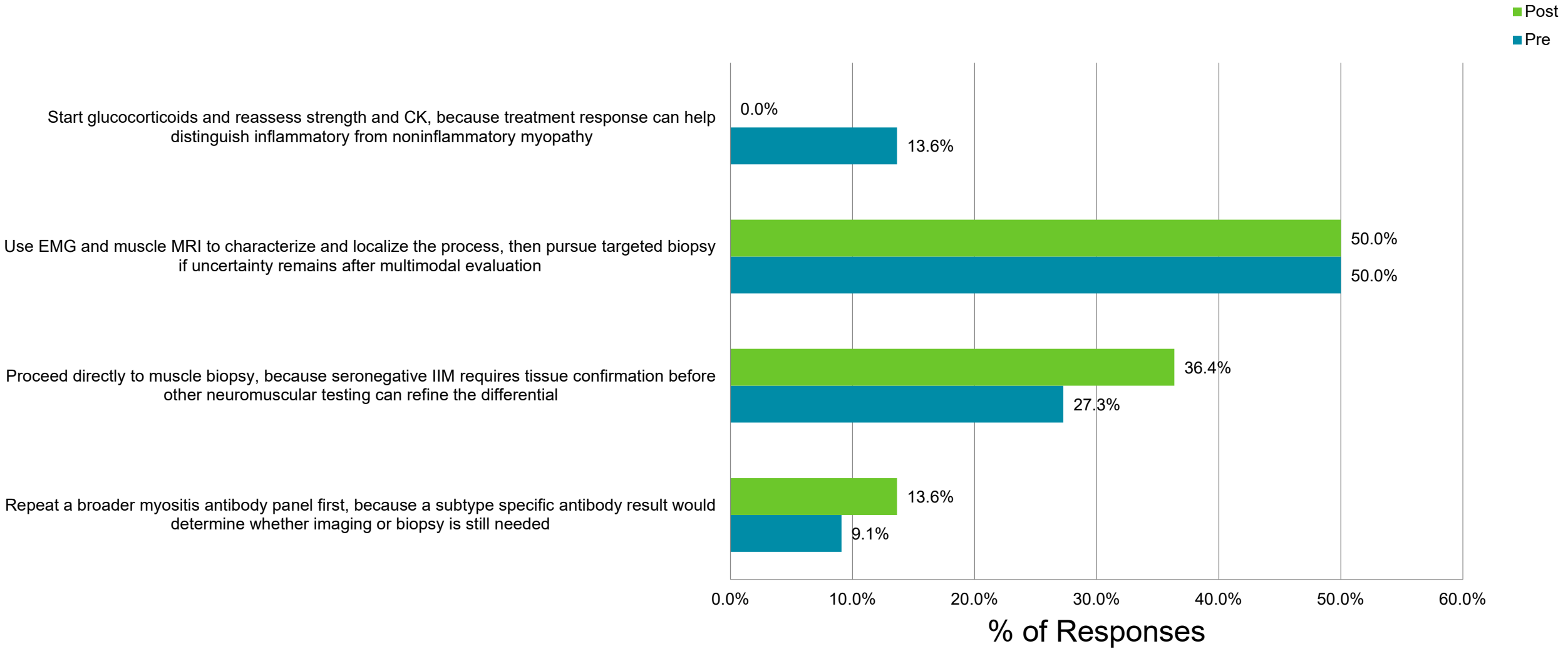
B. Proceed directly to muscle biopsy, because seronegative IIM requires tissue confirmation before other neuromuscular testing can refine the differential

C. Use EMG and muscle MRI to characterize and localize the process, then pursue targeted biopsy if uncertainty remains after multimodal evaluation

D. Start glucocorticoids and reassess strength and CK, because treatment response can help distinguish inflammatory from noninflammatory myopathy

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A 52-year-old male has four months of progressive symmetric proximal weakness, CK 1840 U/L, and negative myositis serology. Now which interpretation should guide the next diagnostic step?



Results recorded on October 14, 2026.

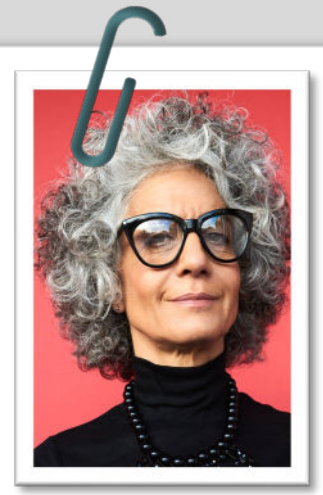
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LEARNING OBJECTIVE 2

Utilize IIM subtype to inform comorbidity assessment, multidisciplinary management, and QoL improvement

Patient Case: Diana



68-year-old female with **anti-Jo-1 positive antisynthetase syndrome**, diagnosed three years ago

Mild proximal weakness, stable on methotrexate; Mechanic's hands; Raynaud phenomenon

Three months of progressive exertional dyspnea and a dry cough

Referred back to rheumatology as a “myositis flare”

Muscle examination essentially unchanged; CK mildly elevated

FVC: 68% predicted

DLCO: 52% predicted

Which comorbidity should be prioritized for immediate evaluation, and what is the next step in management?

DLCO = diffusing capacity of the lung for carbon monoxide; FVC = forced vital capacity.

Audience Response

A 68-year-old female with anti-Jo1 positive ASyS has progressive dyspnea and dry cough, unchanged muscle findings, FVC 68% predicted, and DLCO 52% predicted. Which next step should be prioritized?

A. Repeat full PFTs with respiratory pressures before imaging, because reduced FVC may reflect respiratory muscle weakness rather than interstitial lung disease.

B. Obtain HRCT and pulmonary evaluation now while maintaining rheumatology and neurology involvement in the broader antisynthetase syndrome care plan.

C. Increase immunosuppression for presumed myositis activity and repeat PFTs soon, using pulmonary response to determine whether separate lung evaluation is necessary.

D. Complete malignancy and swallowing assessment first, because subtype linked screening obligations should be addressed before escalating organ specific evaluation.

HRCT = high-resolution computed tomography; PFTs = pulmonary function tests.

The Subtype Tells You What to Screen For

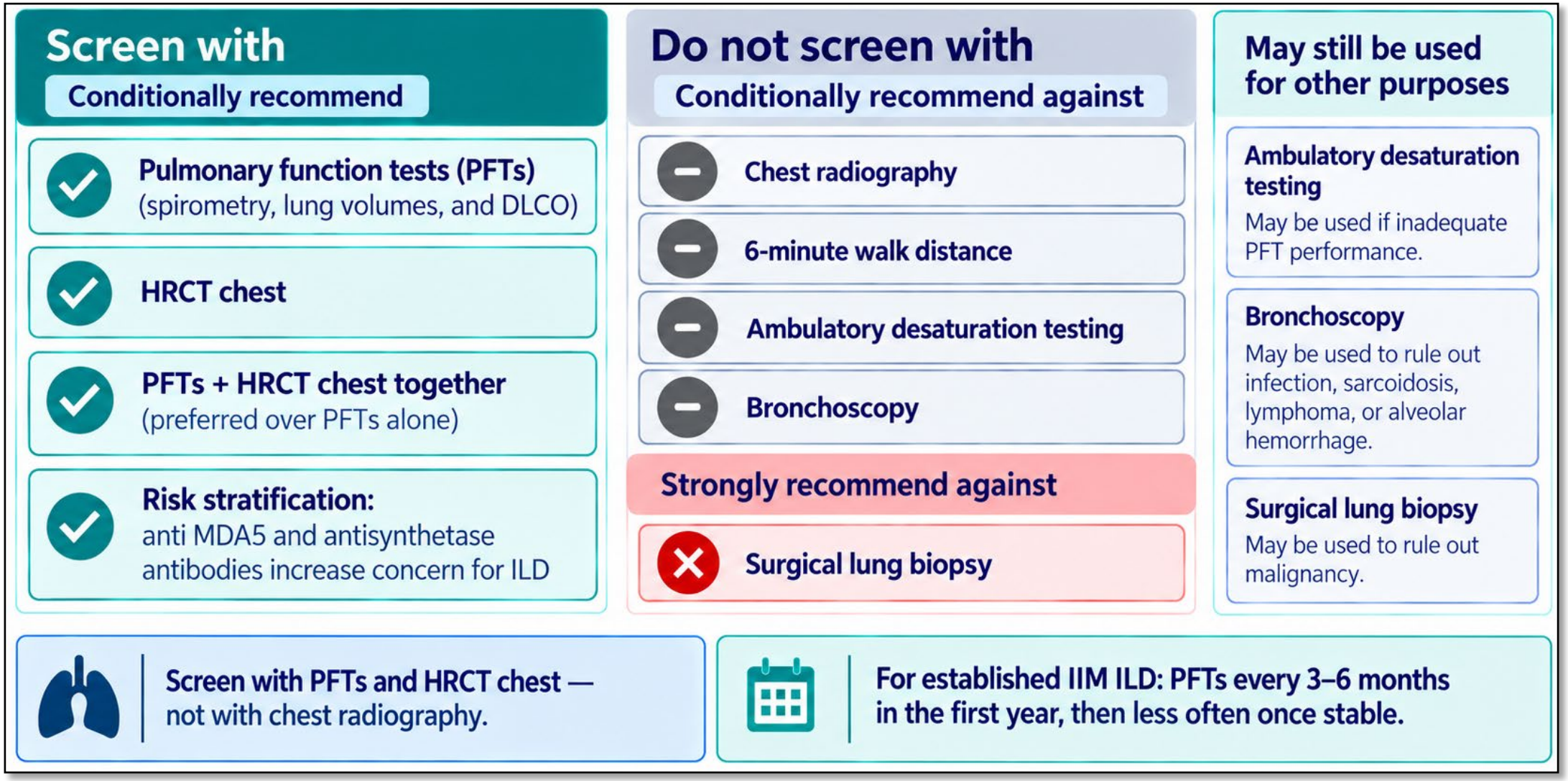
Screening Assessment	DM	ASyS	IMNM	IBM
Lung	• High ILD risk; prevalence varies by cohort and antibody profile	• High ILD risk; prevalence varies by cohort and antibody profile • Chronic ILD is the most common presentation	• ILD less common overall • More frequent with anti-SRP than anti-HMGCR	• Rare
Malignancy	• HIGH risk factor • anti-TIF1γ and anti-NXP2 positivity carry higher risk	• LOW risk factor • Anti-Jo-1 and non-Anti-Jo-1 anti-synthetase positivity also low	• INTERMEDIATE risk factor • Anti-HMGCR positivity: intermediate	• Screening not routinely required in verified IBM

Johnson SR, et al. *Arthritis Care Res (Hoboken)*. 2024;76(8):1070-1082. Oldroyd AGS, et al. *Nat Rev Rheumatol*. 2023;19(12):805-817. Sehgal S, et al. *Lancet Respir Med*. 2025;13(3):272-288.

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IIM Requires Risk Guided ILD Assessment

Patients with IIM should be assessed for ILD risk, with PFTs and HRCT used when subtype, antibody profile, or symptoms indicate increased risk



Johnson SR, et al. *Arthritis Care Res (Hoboken)*. 2024;76(8):1070-1082.

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Use FVC and DLCO Together to Guide the Next Clinical Step

Pulmonary function abnormalities can reflect respiratory muscle involvement as well as ILD

PFT pattern	What it suggests	Next question
Low FVC and low DLCO (restrictive, low TLC)	The most common finding in IIM-related lung disease	Characterise the interstitial disease - HRCT chest
Normal FVC with isolated mild reduction in DLCO	May reflect mild disease	Do not dismiss - HRCT chest
DLCO reduced out of proportion to FVC	May reflect concomitant emphysema or pulmonary hypertension	Look beyond ILD alone
Low FVC, Normal DLCO, >20% fall in FVC when supine, reduced maximal inspiratory and expiratory pressures	Pattern usual in respiratory muscle weakness	Assess respiratory muscle involvement rather than assuming ILD

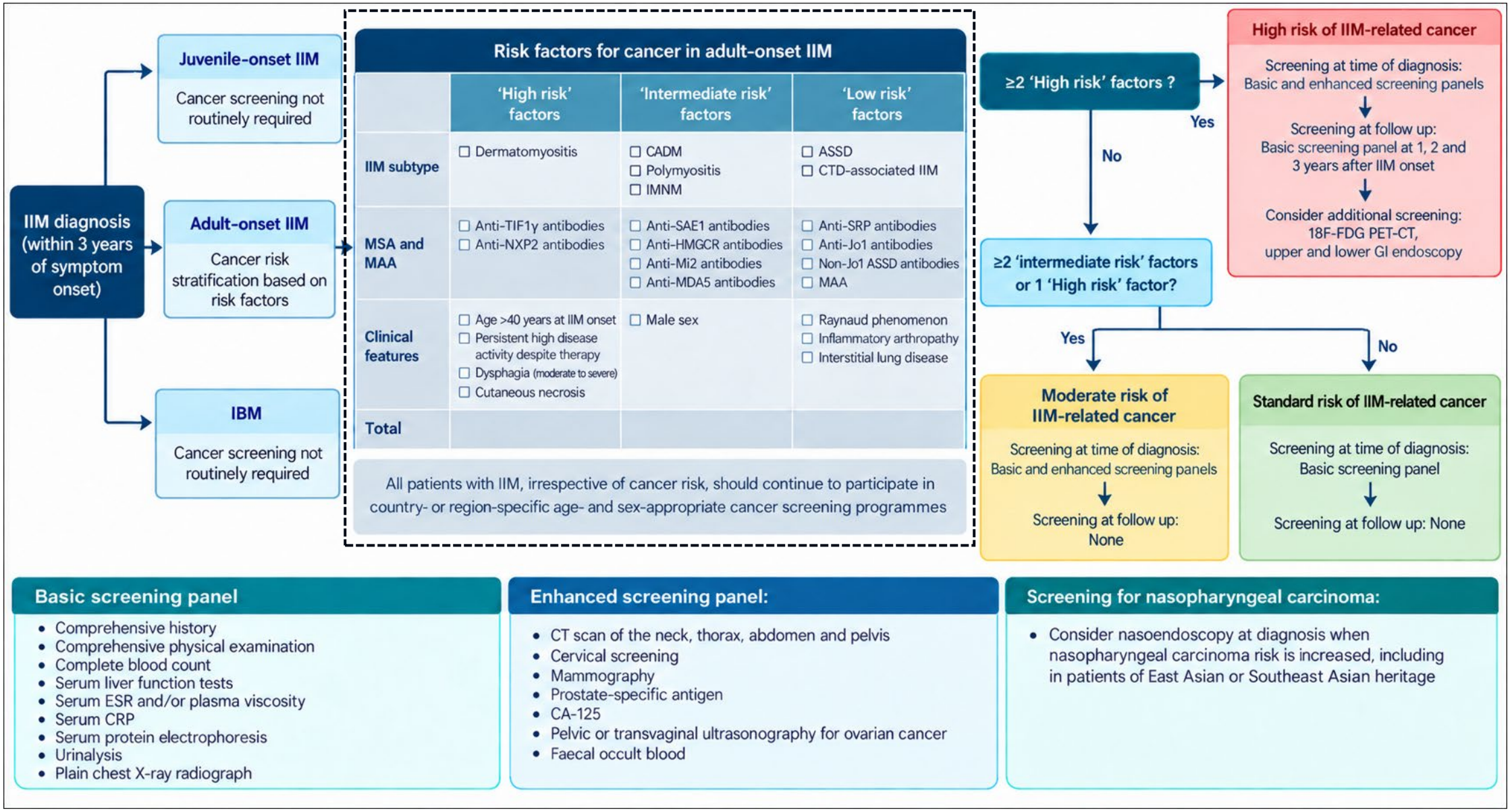
HRCT Patterns: NSIP is the most common in chronic ILD; an organizing pneumonia pattern is also seen, and both are often observed together in IIM-associated ILD

NSIP = nonspecific interstitial pneumonia; TLC = total lung capacity.

Sehgal S, et al. *Lancet Respir Med. 2025*;13(3):272-288.

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Count the Risk Factors, Then Screen*



*Adults within 3 years of IIM symptom onset. Not routinely required for juvenile-onset IIM or verified IBM.

Oldroyd A.G.S., et al. *Nat Rev Rheumatol*. 2023;19(12):805-817.

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Ask Directly, Do Not Wait for Patients to Volunteer Symptoms



Any difficulty swallowing?



Any breathing problems?



Any symptoms of respiratory muscle weakness?



Any decline in what you can do day to day?



What matters most to you right now about your symptoms and treatment?

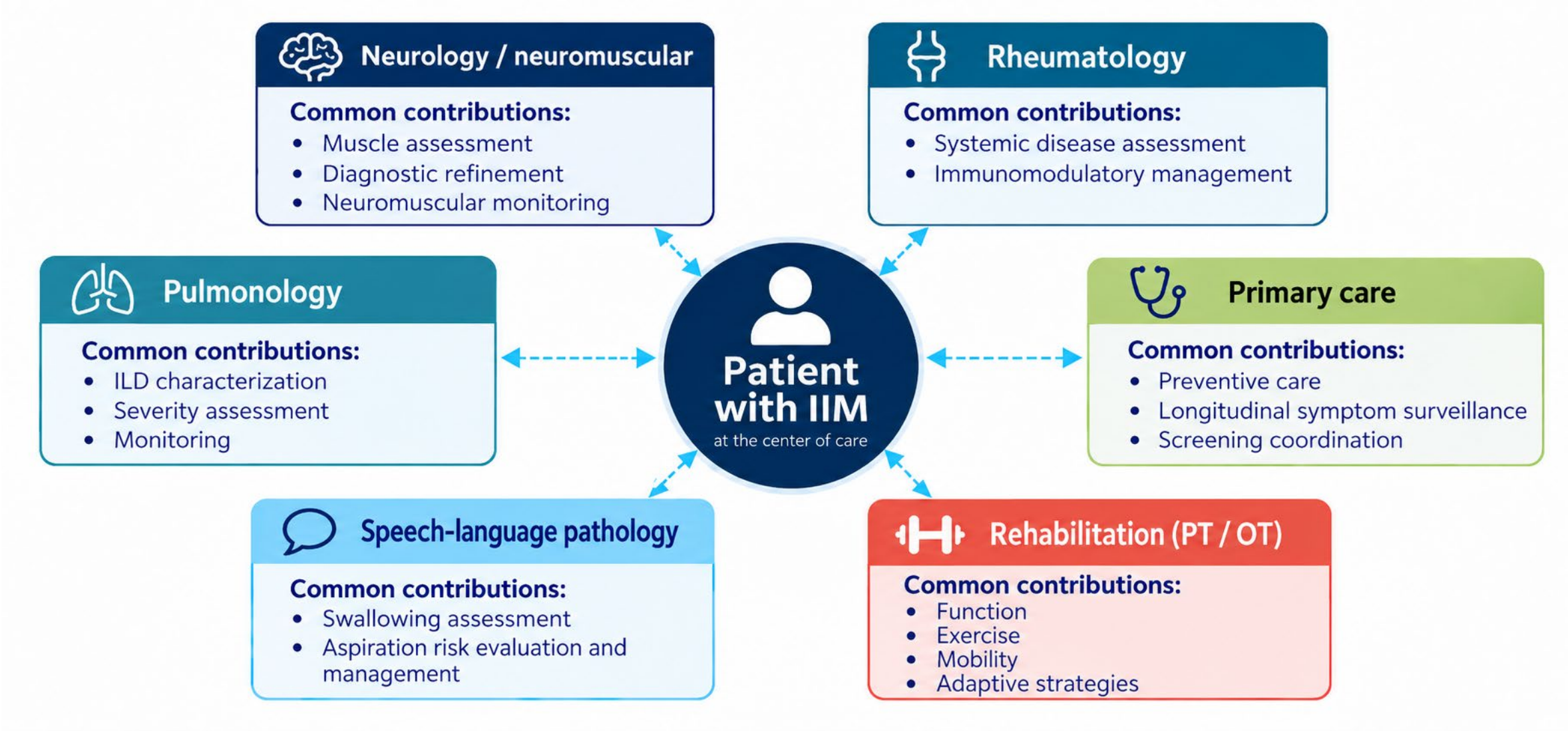
> *"I will be fine one day, the next day I can feel absolutely terrible."*
> **- Individual with IIM**

Labeit B, et al. *J Clin Med.* 2020;9(7):2150. Oldroyd A, et al. *BMC Rheumatol*. 2020;4:47. Sehgal S, et al. *Lancet Respir Med*. 2025;13(3):272-288.

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Team-Based Approach: Coordinate Roles and Close the Loop

Examples of team contributions; roles vary by setting and patient need.



Ambrocio KR, et al. *Rheumatology (Oxford)*. 2023;62(10):3227-3244. Johnson SR, et al. *Arthritis Care Res*. 2024;76(8):1070-1082. Oldroyd AGS, et al. *Nat Rev Rheumatol*. 2023;19(12):805-817. Raaphorst J, et al. *Lancet Neurol.* 2025;24(9):776-788.

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Audience Response Reask

A 68-year-old with anti-Jo1 positive ASyS has progressive dyspnea and dry cough, unchanged muscle findings, FVC 68% predicted, and DLCO 52% predicted. Now, which next step should be prioritized?

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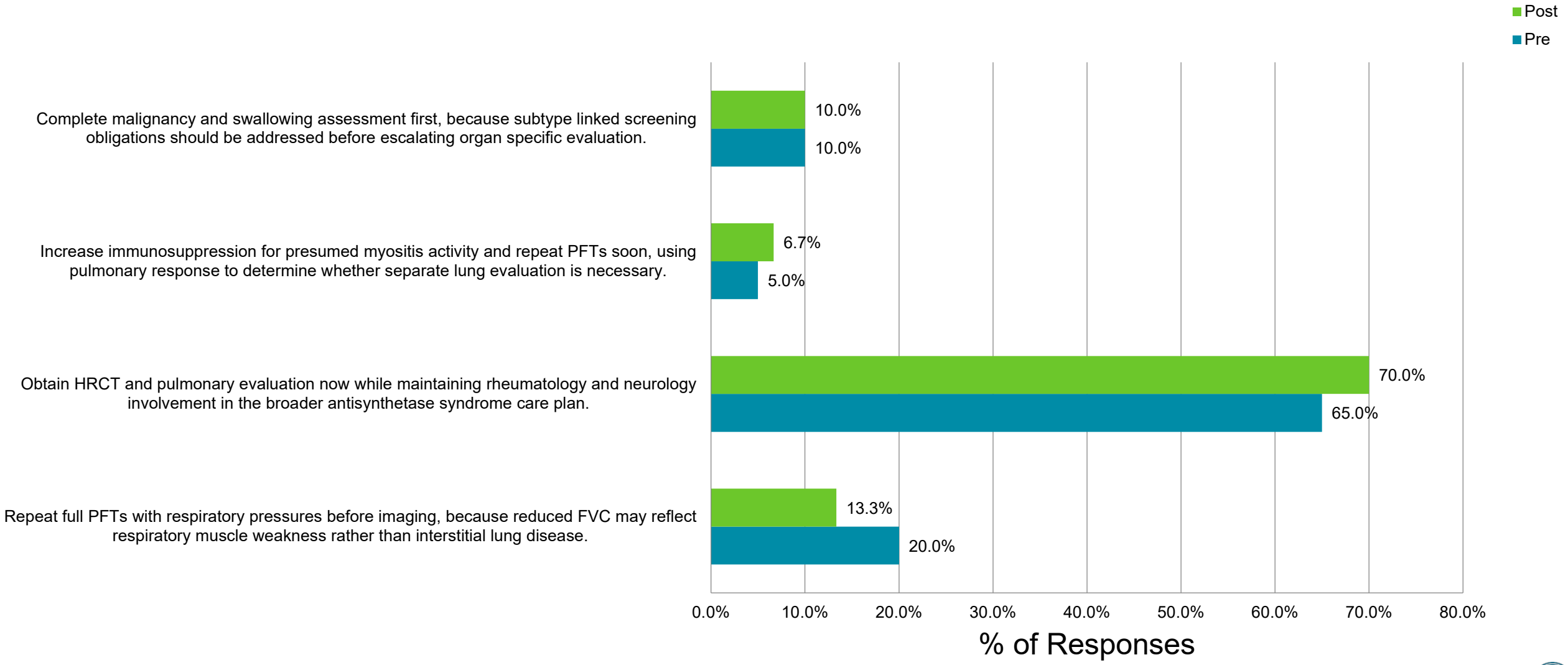
B. Obtain HRCT and pulmonary evaluation now while maintaining rheumatology and neurology involvement in the broader ASyS care plan

C. Increase immunosuppression for presumed myositis activity and repeat PFTs soon, using pulmonary response to determine whether separate lung evaluation is necessary

D. Complete malignancy and swallowing assessment first, because subtype linked screening obligations should be addressed before escalating organ specific evaluation

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A 68-year-old female with anti-Jo1 positive ASyS has progressive dyspnea and dry cough, unchanged muscle findings, FVC 68% predicted, and DLCO 52% predicted. Which next step should be prioritized?



Results recorded on October 14, 2026.

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LEARNING OBJECTIVE 3

Describe current and emerging steroid-sparing and targeted treatment strategies in IIM, including the clinical rationale for FcRn inhibition within a balanced therapeutic landscape

Patient Case: Amber



58-year-old female with **anti-Mi2 positive dermatomyositis**

Fourteen months on **prednisone**, currently **15 mg daily**
Methotrexate 20 mg weekly for eleven months
New **hyperglycemia** and **8 kg weight gain**
Asks whether anything would let her come off steroids

Persistent proximal weakness and disabling fatigue
Gottron papules still active

What factors should guide the decision to escalate to alternative or emerging therapies?

Audience Response

A 58-year-old with anti-Mi2 positive DM has persistent proximal weakness, disabling fatigue, and active Gottron papules despite methotrexate. After 14 months of prednisone, she has developed hyperglycemia and substantial weight gain. Which approach should guide the next treatment decision?

A. Choose treatment according to the most active disease domain, then revisit route, monitoring burden, and patient preferences after clinical response is established

B. Escalate along a conventional treatment sequence after methotrexate failure, reserving targeted or infusion therapy until another conventional agent has been tried

C. Prefer an FDA approved adult DM therapy with steroid sparing potential, using regulatory status and route as the main basis for treatment selection

D. Compare reasonable options using disease domains, steroid toxicity, comorbidities, safety, monitoring, route, access, and patient priorities before selecting therapy

CME OUTFITTERS

Treatment Is Not Interchangeable Across IIM

Dermatomyositis	Antisynthetase syndrome	Immune-Mediated Necrotizing Myopathy
• **Foundation:** glucocorticoids with methotrexate, azathioprine, mycophenolate mofetil or calcineurin inhibitors • **Skin:** Topical glucocorticoids, topical calcineurin inhibitors, sun protection • **Persistent/refractory DM:** consider IVIG, brepocitinib in adults, or selected off label approaches based on active disease domain and patient factors	• **Initial:** calcineurin inhibitors or mycophenolate mofetil, with close pulmonary monitoring • **Progressive or severe ILD:** escalate according to ILD severity, pace, phenotype, comorbidities, and prior response	• **Often requires combination immunotherapy:** IVIG is particularly relevant in anti HMGCR disease, while rituximab is often considered earlier in anti SRP disease • Plasma exchange in extreme cases • Treatment can vary by antibody status: Rituximab for anti-SRP, IVIG for anti-HMGCR

Inclusion body myositis sits apart

- Refractory to both immunosuppressive and non-immunosuppressive therapies; corticosteroids and immunosuppressants give only transient benefit
- No dedicated pharmacological treatment is available for IBM
- The 2026 Phase 2/3 MUSCLE trial of ulviprubart (n = 272) did not meet its primary endpoint
- Management is supportive

IVIG = intravenous immunoglobulin.

Johnson SR, et al. *Arthritis Care Res (Hoboken)*. 2024;76(8):1051-1069. Medical Professionals Reference (MPR). MPR Website. 2026. https://www.empr.com/news/ulviprubart-fails-to-meet-endpoints-in-inclusion-body-myositis-study/. Paik JJ, et al. *Rheumatology (Oxford)*. 2025;64(6):3288-3302. Selva-O'Callaghan A, et al. *Curr Treatm Opt Rheumatol*. 2025;11:21. Suh J, et al. *Muscle Nerve*. 2024;70(2):166-172.

CME OUTFITTERS

Off-Label Steroid-Sparing Therapy*

Agent	IIM Subtype Utilized	Treatment Role	What drives the choice
MUSCLE-PREDOMINANT DISEASE: conventional first steroid-sparing agents			
Azathioprine	• DM, OM, PM	• Alternative early agent when MTX unsuitable	• First-line option for SARD-ILD
Methotrexate (MTX)	• DM, OM, PM	• Early conventional agent, often started with glucocorticoids	• Muscle disease; • ***Caution with established ILD***
ORGAN-DRIVEN DISEASE: When ILD or refractory skin disease directs the choice			
Cyclophosphamide	• ASyS, DM	• Reserved for severe or rapidly progressive disease	• Rapidly progressive IIM-ILD; • *Toxicity limits routine use*
Mycophenolate mofetil (MMF)	• ASyS, DM	• Common early ILD-directed and refractory cutaneous option	• IIM-ILD • Refractory cutaneous DM
Tacrolimus / cyclosporine	• ASyS, DM	• ILD-directed escalation	• Antisynthetase-associated ILD • acute or subacute DM-ILD
REFRACTORY OR ORGAN-THREATENING DISEASE			
Rituximab	• ASyS, IMNM, Refractory DM/PM	• Refractory muscle, skin, or lung disease • First-line ILD option	• Refractory DM/PM • Antisynthetase syndrome • IIM-ILD

*These agents are not FDA approved, but guideline-recommended.
CTD = connective tissue disease; IMACS = International Myositis Assessment and Clinical Studies Group; MTX = methotrexate; SARD = systemic autoimmune rheumatic disease.
Johnson SR, et al. *Arthritis Care Res (Hoboken)*. 2024 Aug;76(8):1051-1069. Kocyigit M, et al. *J Neuromuscul Dis.* 2026;13(4):553-572. Moghadam-Kia S, et al. *Expert Rev Clin Immunol*. 2015;11(11):1265-1275. Oddis CV, et al. *Arthritis Rheum*. 2013;65(2):314-324. Selva-O'Callaghan A, et al. *Curr Treatm Opt Rheumatol*. 2025;11:21. Wang R, et al. *J Cachexia Sarcopenia Muscle*. 2025;16(6):e70143.

CME OUTFITTERS

Approved Therapeutic Agents for IIM: Specifically Adult DM*

Immune Globulin Intravenous (Human), 10%

PIVOTAL PHASE 3

- **ProDERM** (NCT02728752)
- n = 95; 47 IVIG vs 48 placebo
- Adults 18–80 with active DM; MMT-8 <142

PRIMARY ENDPOINT

- TIS ≥20 at week 16 without confirmed deterioration:
  - **79% (37/47) vs 44% (21/48)**
  - adjusted difference **35 percentage points**; 95% CI, 16.7 to 53.2; **P<0.001**

OPEN-LABEL EXTENSION

- **Concomitant glucocorticoid reduction:**
  - **15/84 patients (18%)** reduced dose or discontinued

SAFETY IN PIVOTAL TRIAL

- Treatment-related AEs: **58% vs 23% placebo**
- Serious related AEs: **3/52 (6%)**
- Deaths: **none**; hemolytic reactions: **none**

Brepocitinib

PIVOTAL PHASE 3

- **VALOR** (NCT05437263)
- n = 241; 81 / 81 / 79 to 30 mg, 15 mg, placebo
- Adults 18–75 with DM; inadequate response to ≥1 prior therapy

PRIMARY ENDPOINT

- Mean TIS at week 52 (continuous measure):
  - **46.5 (30 mg) vs 37.5 (15 mg) vs 31.2 (placebo)**
  - 30 mg vs placebo: difference **15.3**; 95% CI, 6.7 to 24.0; **P<0.001**
  - 15 mg vs placebo: 6.3; 95% CI, −2.4 to 14.9 — **did not meet the threshold**

KEY SECONDARY ENDPOINT

- **Glucocorticoid-sparing composite at week 52:**
  - TIS ≥40 + prednisone-equivalent dose ≤2.5 mg/day at weeks
  - TIS ≥40 + prednisone-equivalent dose ≤2.5 mg/day at weeks 48 and 52: **54% (30 mg) vs 27% placebo**
  - Difference: **26 percentage points** (95% CI, 11 to 40); **P<0.001**

SAFETY IN PIVOTAL TRIAL

- Any AE: **90% / 86% vs 91% placebo**
- Serious infection: **10% / 2% vs 1% placebo**
- Deaths: **none**

*Trial populations, endpoints, treatment exposure, dosing regimens, and safety assessment periods differ. Results are shown within each trial and should not be interpreted as comparative efficacy or safety across agents.

**One IVIG agent is FDA approved for adult DM.

AEs = adverse events; CI = confidence interval; MMT-8 = Manual Muscle Test 8; TIS = Total Improvement Score.

Aggarwal R, et al. *N Engl J Med*. 2022;387:1264-1278. Brepocitinib [package insert]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/220106Orig1s000lbl.pdf. Immune globulin intravenous (human) [package insert]. https://www.fda.gov/media/70911/download. Vleugels RA, et al. *N Engl J Med*. 2026;394(19):1883-1893.

CME OUTFITTERS

Safety Profiles: FDA-Approved Therapies in Adult DM*

Intravenous Immunoglobulin 10%

ADVERSE REACTIONS (≥5% patients)

Headache	Vomiting	MSK pain	Infusion site reaction
Fever	Increased BP	Increased HR	
Nausea	Chills	Dyspnea	

WARNINGS AND PRECAUTIONS

- Hypersensitivity
- Renal dysfunction/failure
- Blood glucose monitoring
- Hyperproteinemia
- Hemolysis
- Aseptic meningitis
- TRALI

CONTRAINDICATIONS

- History of anaphylactic or severe systemic reactions to human immunoglobulin
- IgA-deficient patients with anti-IgA antibodies
- History of hypersensitivity reactions

Brepocitinib

ADVERSE REACTIONS (≥5% patients)

URTI	UTI	Arthralgia	Fall
Headache	Nausea	Diarrhea	Influenza
Fatigue	Bronchitis	Back pain	Acne

WARNINGS AND PRECAUTIONS

- Serious Infections
- Hypersensitivity Reactions
- GI Perforations
- Hypoglycemia in Patients with Diabetes
- Laboratory Abnormalities
- Immunizations
- Embryofetal Toxicity

CONTRAINDICATIONS

- History of hypersensitivity reactions to brepocitinib or any of the excipients

***Trial populations, treatment exposure, dosing regimens and safety assessment periods differ. Adverse reactions are shown within each product's own labeling and should not be interpreted as comparative safety across agents.**

BP = blood pressure; GI = gastrointestinal; HR = heart rate; IgA = immunoglobulin A; MSK = musculoskeletal; TRALI = transfusion-related acute lung injury; URTI = upper respiratory tract infection; UTI = urinary tract infection.

Brepocitinib [package insert]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/220106Orig1s000lbl.pdf. Immune globulin intravenous (human) [package insert]. https://www.fda.gov/media/70911/download.

CME OUTFITTERS

Safety Profiles: Boxed Warnings

IVIG 10%

WARNING
THROMBOSIS, RENAL DYSFUNCTION AND ACUTE RENAL FAILURE

1. Thrombosis may occur with immune globulin intravenous (IGIV) products, including Octagam 10%. Risk factors may include: advanced age, prolonged immobilization, hypercoagulable conditions, history of venous or arterial thrombosis, use of estrogens, indwelling vascular catheters, hyperviscosity, and cardiovascular risk factors.
2. Renal dysfunction, acute renal failure, osmotic nephropathy, and death may occur with the administration of Immune Globulin Intravenous (Human) (IGIV) products in predisposed patients. Renal dysfunction and acute renal failure occur more commonly in patients receiving IGIV products containing sucrose. Octagam 10% does not contain sucrose.
3. For patients at risk of thrombosis, renal dysfunction or renal failure, administer Octagam 10% at the minimum infusion rate practicable. Ensure adequate hydration in patients before administration. Monitor for signs and symptoms of thrombosis and assess blood viscosity in patients at risk for hyperviscosity.

Brepocitinib

WARNING
SERIOUS INFECTIONS, MORTALITY, MALIGNANCY, MAJOR ADVERSE CARDIOVASCULAR EVENTS, and THROMBOSIS

1. Increased risk of serious bacterial, fungal, viral, and opportunistic infections, including tuberculosis (TB), leading to hospitalization or death. Test for latent and active TB prior to brepoctinib treatment; treat if positive. Monitor for active TB, even with initial negative latent TB test. Interrupt brepocitinib if a serious infection occurs until the infection resolves or is adequately treated.
2. Higher rate of all-cause mortality, including sudden cardiovascular (CV) death, occurred with another Janus kinase (JAK) inhibitor vs. tumor necrosis factor (TNF) blockers in rheumatoid arthritis (RA) patients. Brepoctinib is not approved for RA.
3. Malignancies have occurred in patients treated with brepoctinib. Higher rate of malignancies occurred with another JAK inhibitor vs. TNF blockers in RA patients.
4. Major adverse cardiovascular events (MACE) (CV death, myocardial infarction, and stroke) occurred in patients treated with brepoctinib. Higher rate of MACE occurred with another JAK inhibitor vs. TNF blockers in RA patients.
5. Thromboses have occurred in patients treated with brepoctinib. Higher rate of thromboses occurred with another JAK inhibitor vs. TNF blockers in RA patients.

Brepocitinib [package insert]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/220106Orig1s000lbl.pdf.
Immune globulin intravenous (human) [package insert]. https://www.fda.gov/media/70911/download.

CME OUTFITTERS

Audience Response Reask



A 58-year-old with anti-Mi2 positive DM has persistent proximal weakness, disabling fatigue, and active Gottron papules despite methotrexate. After 14 months of prednisone, she has developed hyperglycemia and substantial weight gain. Which approach should guide the next treatment decision?

A. Choose treatment according to the most active disease domain, then revisit route, monitoring burden, and patient preferences after clinical response is established

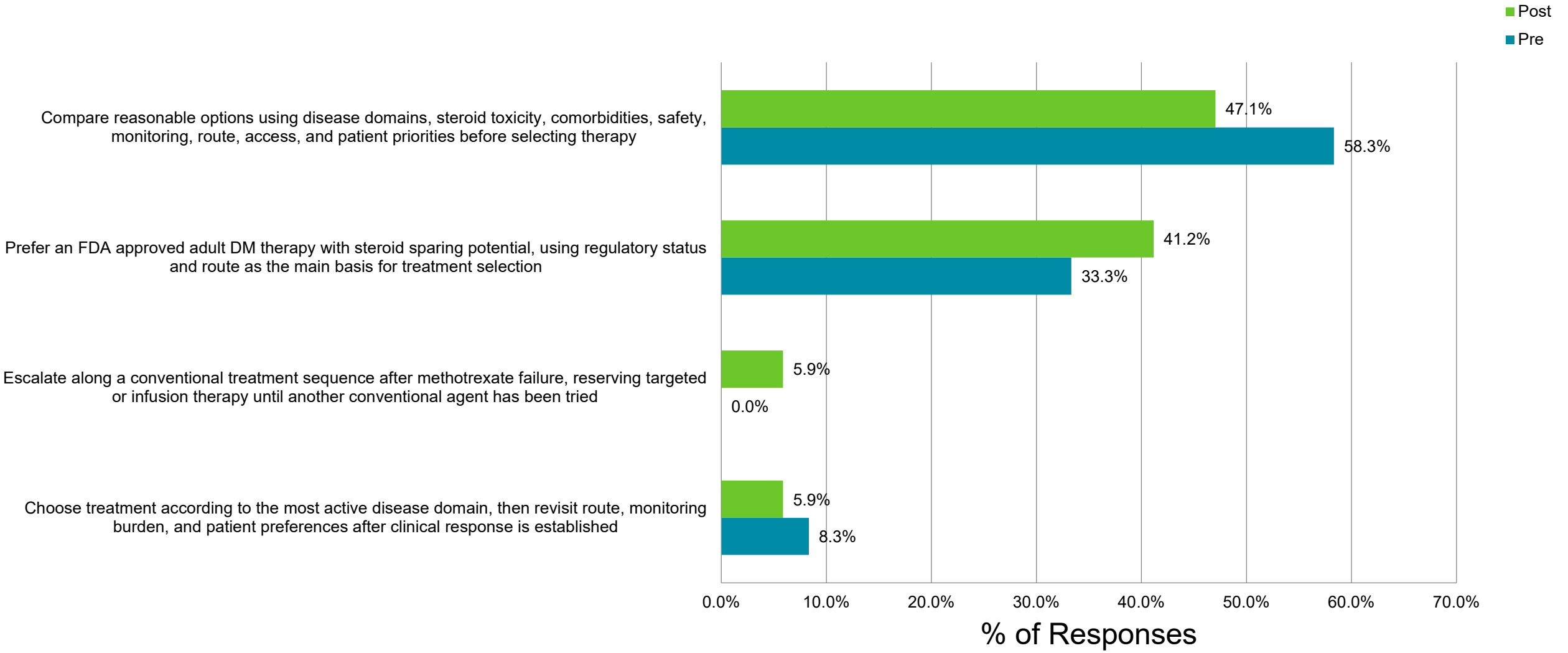
B. Escalate along a conventional treatment sequence after methotrexate failure, reserving targeted or infusion therapy until another conventional agent has been tried

C. Prefer an FDA approved adult DM therapy with steroid sparing potential, using regulatory status and route as the main basis for treatment selection

D. Compare reasonable options using disease domains, steroid toxicity, comorbidities, safety, monitoring, route, access, and patient priorities before selecting therapy

CME OUTFITTERS

A 58-year-old with anti-Mi2 positive DM has persistent proximal weakness, disabling fatigue, and active Gottron papules despite methotrexate. After 14 months of prednisone, she has developed hyperglycemia and substantial weight gain. Which approach should guide the next treatment decision?



Results recorded on October 14, 2026.

Selected Emerging Agents in IIM*

Class	Agent	Mechanism of Action	Trial	Current Status	Target IIM subtype(s)
B-cell-directed	**Resecabtagene Autoleucel**	• Autologous CD19-directed CAR T-cell therapy	**RESET-Myositis** Phase 1/2	**Recruiting**	ASyS, DM, IMNM, Juvenile IIM
	Ublituximab	• Glycoengineered anti-CD20 monoclonal antibody; B-cell depletion	**SUMMIT** Phase 2	**Recruiting**	Early autoantibody-positive IMNM
FcRn antagonist	**Efgartigimod SC**	• Neonatal Fc receptor antagonist; reduces circulating pathogenic IgG	**ALKIVIA** Phase 2/3	**Completed**	ASyS, DM/JDM, IMNM, PM
			ALKIVIA OLE Phase 3	**Enrolling by Invitation**	
	Nipocalimab		**SPIREA** Phase 2	**Active, not recruiting**	ASyS, DM, IMNM
Type I interferon pathway	**Anifrolumab**	• Type I interferon receptor subunit 1 antagonist	**JASMINE** Phase 3	**Recruiting**	DM, PM
	Baricitinib	• Oral JAK1/JAK2 inhibitor; suppresses type I interferon signaling	**BIRD** Phase 3	**Recruiting**	Relapsing or Treatment-naïve DM
			MYOJAK Phase 2	**Completed**	DM, PM
	Dazukibart	• IFNβ ligand-specific monoclonal antibody	**PF-06823859** (Phase 3); **OLE** (Phase 3)	**Recruiting**	DM and PM

*Selected examples; not a complete pipeline review.
JDM = juvenile dermatomyositis; OLE = open-label extension.
Aggarwal R, et al. *Clin Cosmet Investig Dermatol*. 2026;19:581733. ClinicalTrials.gov Identifiers: NCT05523167, NCT05979441, NCT05379634, NCT07103746, NCT06154252, NCT06455449, NCT04972760, NCT04208464, NCT05895786, and NCT06698796.

CME OUTFITTERS

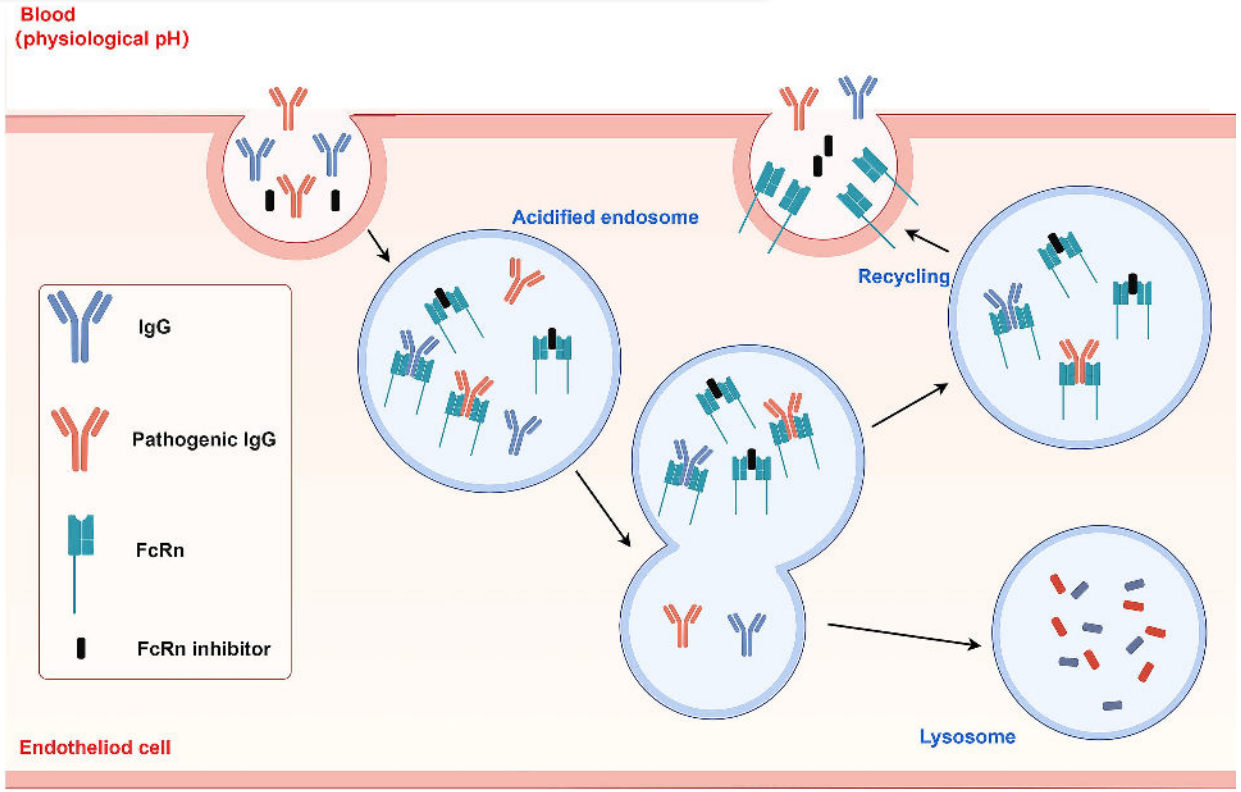
The Rationale for FcRn Inhibition in IIM

1 FcRn recycles IgG

FcRn binds IgG and returns it to the circulation, extending IgG half-life, including that of IgG autoantibodies.

2 Blockade stops recycling

Efgartigimod, a human IgG1 antibody fragment, binds FcRn and competitively inhibits IgG–FcRn binding.



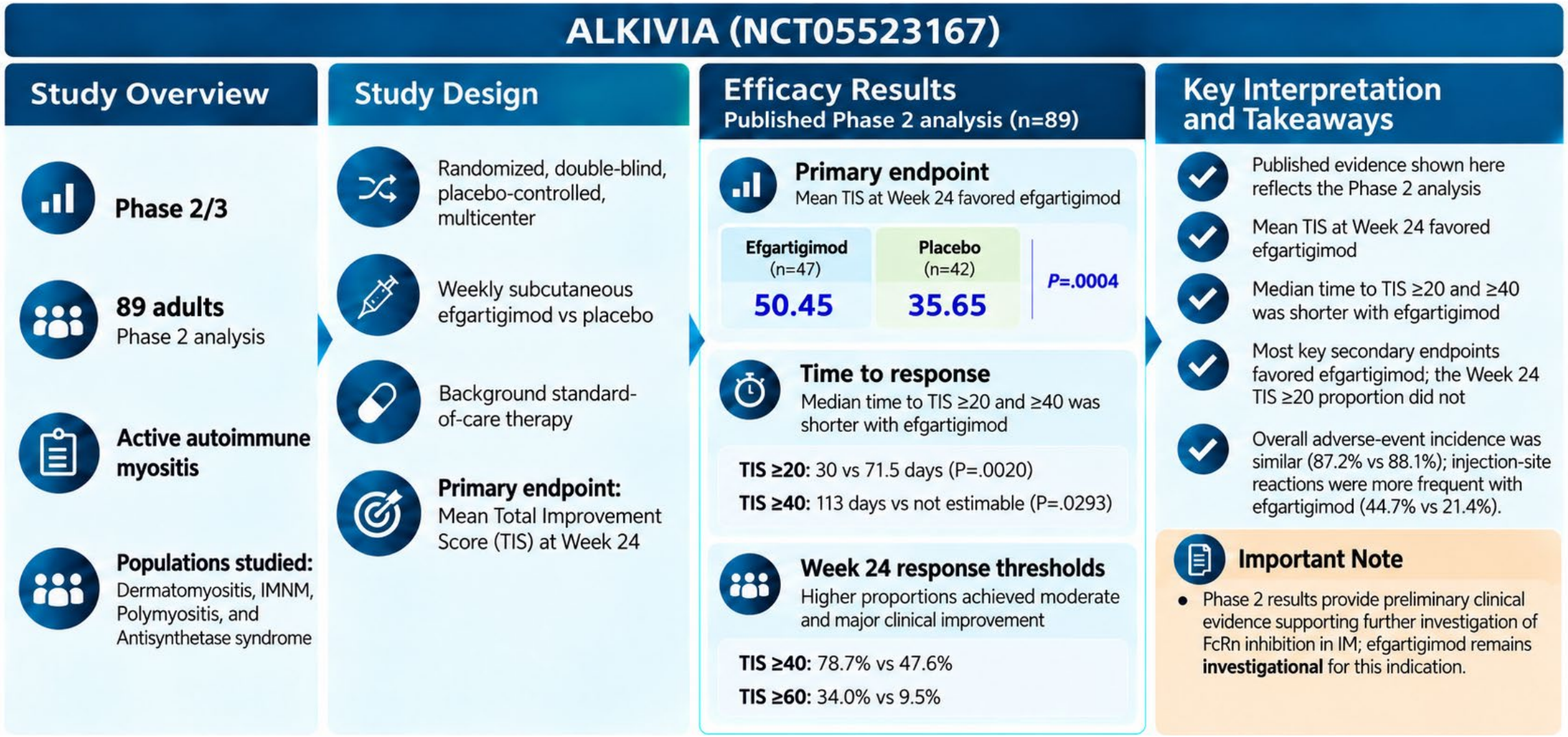
3 IgG is cleared

Unrecycled IgG is degraded, lowering circulating total IgG, including IgG autoantibodies.

Sandhu M, et al. *Antibodies (Basel)*. 2025; 14(3):65. Wang R, et al. *J Cachexia Sarcopenia Muscle*. 2025;16(6):e70143.

CME OUTFITTERS

FcRn Antagonist Application in IIM



**Activity was recorded on October 14, 2026 date. New Phase 3 abstract data on ALKIVIA was released on October 20, 2026 and is reflected above*

ClinicalTrials.gov. Identifier: NCT05523167; Aggarwal R, et al. ACR Convergence 2026; 2026. Abstract No. LB24. https://acrabstracts.org/abstract/efgartigimod-ph20-sc-in-adults-with-idiopathic-inflammatory-myopathy-results-from-the-phase-3-randomized-placebo-controlled-alkivia-trial/

CME OUTFITTERS

FcRn Antagonist Application in IIM

ALKIVIA Phase 2 Cohort

Study Overview

Phase 2 Cohort

89
adults
Phase 2 analysis

Active autoimmune myositis

Populations studied:

- Dermatomyositis
- IMNM
- Polymyositis
- Antisynthetase syndrome

Study Design

Randomized, double-blind, placebo-controlled, multicenter

Weekly subcutaneous **efgartigimod PH20**
vs
placebo

Primary endpoint:
Mean Total Improvement Score (TIS) at Week 24

Efficacy Results

Analysis

Primary endpoint
Mean TIS at Week 24

Efgartigimod (n = 47)	Placebo (n = 42)
50.45	35.65

p = .0004

Time to response
Median time to TIS ≥20 and ≥40 was **shorter** with efgartigimod
TIS ≥20: 30 vs 71.5 days (p = .0020)
TIS ≥40: 113 days vs not estimable (p = .0293)

Week 24 response thresholds
Higher proportions achieved moderate and major clinical improvement
TIS ≥40: 78.7% vs 47.6%
TIS ≥60: 34.0% vs 9.5%

Key Interpretation and Takeaways

- Mean TIS at Week 24 favored efgartigimod.
- Median time to TIS ≥20 and ≥40 was shorter with efgartigimod.
- Most key secondary endpoints favored efgartigimod; the Week 24 TIS ≥20 proportion did not.
- Overall adverse-event incidence was similar (87.2% vs 88.1%); injection-site reactions were more frequent with efgartigimod (44.7% vs 21.4%).

ClinicalTrials.gov. Identifier: NCT05523167; Chinoy H, et al. *Ann Rheum Dis*. 2025;84(Suppl 1):2-3

CME OUTFITTERS

FcRn Antagonist Application in IIM

ALKIVIA Phase 3 Cohort

Study Overview

175
randomized adults

IMNM	**n = 92**
DM	**n = 54**
PM including ASyS	**n = 29**

Primary efficacy population:
Combined IMNM + DM

Study Design

Randomized, double-blind, placebo-controlled, multicenter.

Weekly subcutaneous
efgartigimod PH20
vs
placebo

Primary endpoint:
Mean Total Improvement Score (TIS) at Week 52

Efficacy Results

Adjusted between-group mean TIS difference

Combined IMNM + DM
+15.4 points
p = 0.0011

IMNM **+14.8**
p = 0.0048

DM **+14.5**
p = 0.1093
Not statistically significant

Key Interpretation and Takeaways

- The primary endpoint was met in the combined IMNM + DM population.
- The IMNM subgroup showed a statistically significant difference; the DM subgroup did not.
- Several secondary endpoints favored efgartigimod.
- No new safety signals were identified

Efgartigimod remains investigational for IIM.

**Activity was recorded on October 14, 2026 date. New Phase 3 abstract data on ALKIVIA was released on October 20, 2026 and is reflected above*

ClinicalTrials.gov. Identifier: NCT05523167; Aggarwal R, et al. ACR Convergence 2026; 2026. Abstract No. LB24. https://acrabstracts.org/abstract/efgartigimod-ph20-sc-in-adults-with-idiopathic-inflammatory-myopathy-results-from-the-phase-3-randomized-placebo-controlled-alkivia-trial/

CME OUTFITTERS

Efficacy Narrows the Options; Everything Else Decides Between Them

Disease

- Expected efficacy for the relevant subtype
- Organ involvement

Monitoring

- **Monitoring burden:** Lymphocytes, neutrophils, hemoglobin, liver enzymes including GGT, and lipids
- **Pre-treatment thresholds:**
  - Avoid with:
    - **ALC <500/mm³**
    - **ANC <1,000/mm³**, or
    - **Hemoglobin <8 g/dL**
- **Route:** IV/SubQ/Oral

Patient safety

- Relevant comorbidities - including diabetes
- Thrombotic or clotting risk
- Steroid burden
- Infection risk

Patient Factors

- Functional status
- Treatment burden
- Ability to self-administer
- Distance from an infusion center

> *"There's definitely a finite resource with the fatigue effect and I know that's very difficult to quantify."*
>
> ***- Individual with IIM***

ALC = absolute lymphocyte count; ANC = absolute neutrophil count; GGT = gamma-glutamyl transferase; SubQ = subcutaneous.

Aggarwal R, et al. *N Engl J Med*. 2022;387(14):1264-1278. Brepocitinib [package insert]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/220106Orig1s000lbl.pdf. Oldroyd A, et al. *BMC Rheumatol*. 2020;4:47. Vleugels RA, et al. *N Engl J Med*. 2026;394(19):1883-1893.

CME OUTFITTERS

Key Takeaways

1. **IIM is a group of distinct diseases, not one:** Phenotype directs the initial workup; subtype then informs what organs you screen, who you involve, and how you frame treatment. Classification is the first management decision, not a labelling exercise.

2. **A negative myositis panel does not exclude IIM:** Seronegative disease is common. When proximal weakness or unexplained CK elevation persists, extend the evaluation with MRI, EMG, or biopsy rather than defaulting to an empiric steroid trial.

3. **The organ that threatens the patient is often the quiet one:** ILD, dysphagia, and respiratory muscle weakness can be missed unless clinicians ask directly and use risk guided screening. Baseline PFTs with HRCT and direct questioning about swallowing and breathing at every visit are what turn subtype knowledge into earlier intervention.

4. **Steroid burden is a treatable target, not an inevitability:** Reassess cumulative exposure at every visit. Choose among approved, off-label, and investigational options by subtype, organ involvement, and patient-specific risk, and name which is which.

CME OUTFITTERS



Put information into action!

Takeaways from this program can be implemented into your practice to improve patient care.

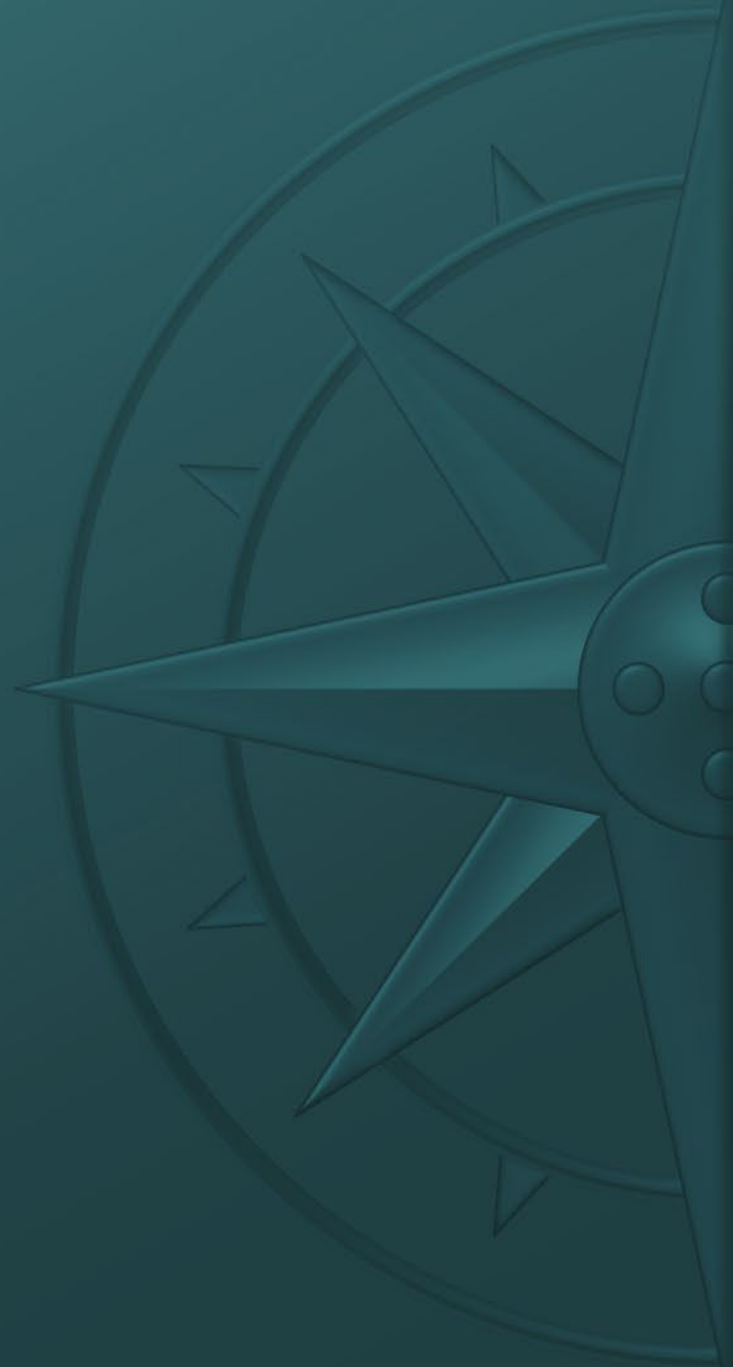
Within the next 6 months…

- In patients evaluated for suspected IIM, apply a phenotype and biomarker-informed diagnostic approach to establish a working subtype and pursue additional evaluation when findings are discordant or a mimic remains plausible.
- In the next 3 patients with confirmed or suspected IIM seen in practice, apply subtype-guided comorbidity, symptom assessment, and patient reported concerns to guide indicated screening for appropriate referral planning.
- In the next 3 patients with IIM receiving systemic glucocorticoids, document the rationale to continue, taper, or modify therapy, incorporating subtype, disease activity, toxicity, safety, treatment burden and patient priorities.

CME OUTFITTERS

Questions and Answers

Ask the Experts!



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2. Complete the post-test and evaluation at the conclusion of the webcast
3. Enter your **ABIM ID number** and **DOB** (MM/DD) on the evaluation, so credit can be submitted to ABIM

ABIM CME MOC ACCREDITED

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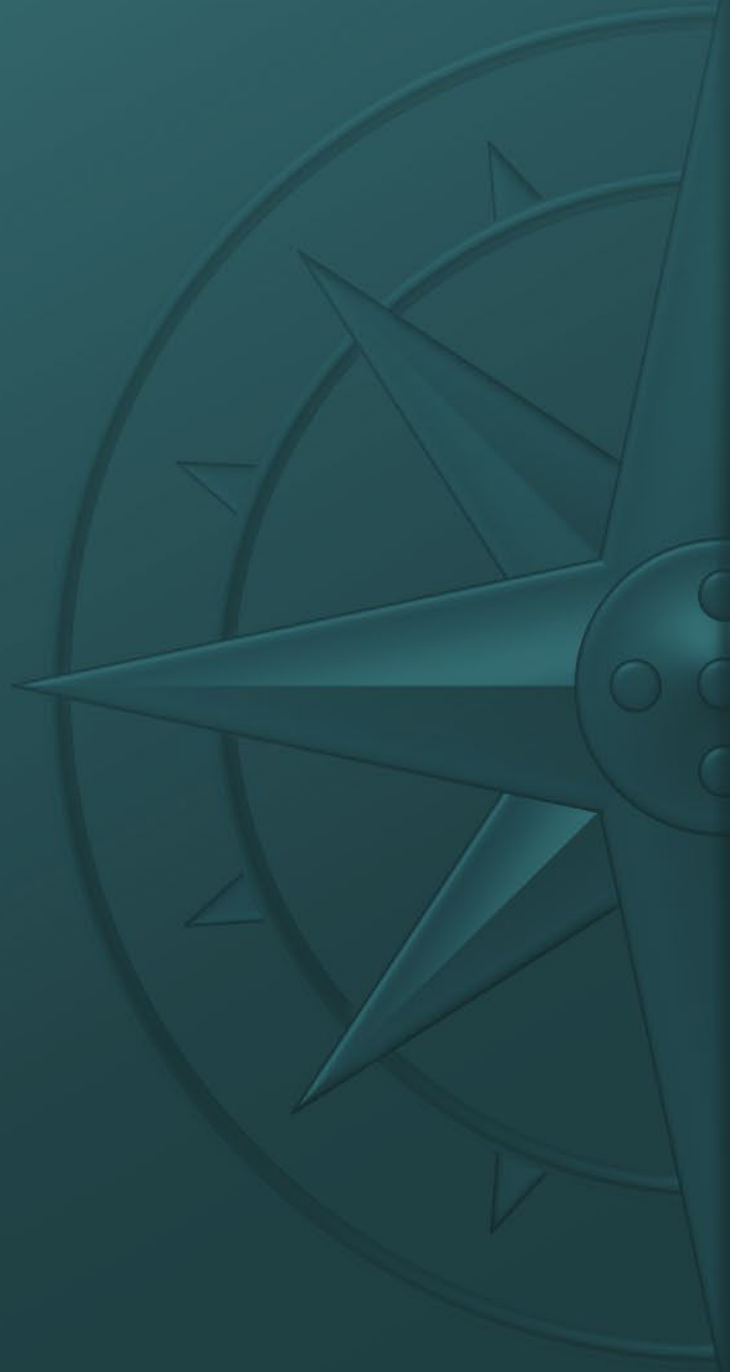
- Actively participate today by responding to ARS questions and/or asking the faculty questions
- Complete the post-test and activity evaluation at the link provided
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- In approximately 3 months, complete the follow-up survey from CME Outfitters

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