Mechanistic Narrative Report

Analysis period — 11 Nov 2025 — 09 Apr 2026

Unified psoriatic-spectrum interpretation versus separate local dermatosis

WHAT HAPPENED

Upadacitinib interruption inflammatory arthropathy worsened.

Sulfur soap use during the skin episode skin improved locally.

WHAT CONNECTIONS STOOD OUT

MRI documented synovitis, mild tenosynovitis, and erosive change as the initial objective signal.

A seronegative, lab-silent pattern and reported heel symptoms reinforced the same inflammatory picture.

WHAT IT MAY MEAN

A unified psoriatic-spectrum inflammatory diathesis is the leading model.

The pattern holds because off-drug worsening and interruption-window skin timing align with the leading model.

QUICK READING

What this report currently suggests

Main signal

The current best-supported interpretation is a single seronegative inflammatory process that links the established arthritis to a possible interruption-associated psoriasiform skin eruption. This view is favored because one framework accounts for MRI-proven inflammatory joint disease, heel involvement, normal inflammatory markers, worsening during upadacitinib interruption, and skin change appearing in the off-drug window. The strongest competing explanation keeps the arthritis as real and JAK-sensitive but treats the skin event as a separate superficial dermatosis that improved because sulfur soap acted directly on a local skin process. The central unresolved issue is not whether sulfur helped, but whether the skin behavior is reproducibly tied to the same stop-gap-restart pattern as the joint disease. Uncertainty remains because the eruption was not dermatologist-confirmed and only one clearly described interruption-window episode is available.

CHRONOLOGY

What happened over time

- Established inflammatory arthropathy documented on MRI
- Maintained on upadacitinib with suppression period
- Two-month drug interruption occurred
- Joint worsening and skin eruption appeared off-drug
- Sulfur soap was used and skin improved

PATTERN FORMATION

How this pattern became visible

Initial signal

MRI documented synovitis, mild tenosynovitis, and erosive change as the initial objective signal.

What repeated

A seronegative, lab-silent pattern and reported heel symptoms reinforced the same inflammatory picture.

What was ruled out

There is no dermatologist confirmation, biopsy, nail description, or repeated prospective stop-restart skin record.

Why this connection stands

The pattern holds because off-drug worsening and interruption-window skin timing align with the leading model.

OBSERVED PATTERN

What the data most clearly shows

- MRI showed synovitis, mild tenosynovitis, and erosive change.
- Inflammatory markers were normal in a seronegative pattern.
- Heel symptoms and a skin eruption during a two-month off-drug interval were reported.

CASE FRAMING

Question under examination

מאובחן בדלקת מפרקים, מטופל ברינבוק, היתה הפסקה של חודשיים, בזמני ההפסקה מופיע בעור פסוריאזיס שטופל בהצלחה בעזרת סבון גפרית.
זה יכול להעיד על מסלול סיסטמי שאפשר לנצל?

INTERPRETATION

Best current interpretation

- A unified psoriatic-spectrum inflammatory diathesis is the leading model.
- It links synovitis, tendon-sheath involvement, heel symptoms, and off-drug skin eruption within one framework.
- This fit is supported by inflammatory arthropathy, interruption-related worsening, and skin appearance during washout.

ALTERNATIVE VIEW

Alternative explanation

- The alternative separates the skin event from the JAK-sensitive seronegative synovitis.
- In this model, the eruption is a superficial barrier or follicular dermatosis better matching sulfur responsiveness.
- Its limitation is weaker explanation for interruption timing and loss of a single linked framework.

BIOLOGICAL MEANING

Underlying biological interpretation

MAIN INTERPRETATION

The leading model is a unified psoriatic-spectrum inflammatory diathesis affecting more than one tissue compartment. In this account, a seronegative TNF and IL-23/Th17-skewed program, supported by JAK-dependent cytokine signaling, drives synovitis, tendon-sheath involvement, likely entheseal symptoms, and can also permit a psoriasiform eruption when upadacitinib suppression is removed. The core clues are the established inflammatory arthropathy, off-drug worsening, and the appearance of skin disease during the interruption window. Secondary clues strengthen but do not prove the model: heel involvement fits an enthesis-linked phenotype, normal ESR and CRP do not exclude it, and sulfur soap improving the skin does not contradict a persistent upstream systemic axis.

ALTERNATIVE EXPLANATION

The strongest alternative keeps the arthritis as a genuine JAK-sensitive seronegative synovitis but separates the skin event from that systemic process. In this model, the eruption is a superficial barrier or follicular dermatosis, such as a seborrheic or eczematous mimicker, whose main drivers are local epidermal turnover and surface amplification rather than the same inflammatory axis affecting the joints. This explanation handles sulfur responsiveness more directly, because sulfur could be acting on the dominant skin mechanism itself. Its main limitation is that it does not naturally explain why the eruption appeared specifically during the upadacitinib interruption, and it gives up the advantage of a single framework linking heel symptoms, lab silence, and skin timing.

TECHNICAL APPENDIX

Why this interpretation currently leads

The leading model currently wins because it explains more of the observed pattern with fewer separate assumptions. A unified psoriatic-spectrum process accommodates MRI-proven inflammatory arthropathy, heel involvement, normal inflammatory markers, and a skin event that arose during withdrawal of a drug already known to suppress the joint disease. The competitor remains viable because the skin lesion was never formally classified and sulfur responsiveness is compatible with a surface-dominant dermatosis. The key discriminator is reproducible coupling: whether skin activity follows the same upadacitinib-sensitive stop-gap-restart curve as the arthritis. Morphology can clarify what the lesion is, but timing is what most directly tests whether both compartments belong to one disease axis.

NEXT INVESTIGATION

What may be worth testing next

- Create a prospective stop-restart timeline with exact upadacitinib dates, daily symptoms, photos, and sulfur exposure.
- If the leading model is true, off-drug skin and musculoskeletal activity should improve after restart.
- If the competitor is true, skin behavior will be nonreproducible or track sulfur use more than exposure.

FURTHER EXPLORATION

Where this can be explored further

Questions worth testing next

Was skin onset repeatedly tied to exact stop and restart dates? Did joint, heel, and skin changes move together across the same timeline?

Relevant directions to explore

Track exact last and first dose dates alongside daily morning stiffness, wrist, hand, heel, and skin changes. Record sulfur soap start and stop dates with standardized photos.

Related topics or mechanisms

Exposure-response timing around upadacitinib interruption and restart is the key topic. Skin site of onset and symptom chronology should be captured prospectively.

Useful sources or search angles

Use the prospective stop-restart timeline, symptom diary, and standardized skin photos. Include exact upadacitinib and sulfur soap dates.