

CASE REPORT

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# Relapsing polychondritis associated with pustular psoriasis successfully treated with secukinumab: a case-based review

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## Abstract

**Rationale** Relapsing polychondritis (RP) is an immune-mediated systemic inflammatory disease involving cartilage and proteoglycan-rich tissues. Pustular psoriasis (PP) is a psoriasis subtype characterized by skin erythema and sterile pustules. In previous studies, there were few reports on patients with RP combined with psoriasis, and treatment strategies are not standardized.

**Patient concerns** An 80-year-old Chinese woman with a 7-year history of atrial fibrillation, a 1-year history of coronary atherosclerotic heart disease, and no familial history, had a 2-month history of skin rash, erythema, swelling and pain in both hands, swollen bilateral auricles, and fingertip gangrene.

**Diagnosis** Based on the diagnostic criteria for generalized pustular psoriasis proposed by Fujita et al. in 2018 and RP proposed by McAdam et al. in 1975, we diagnosed RP with PP as the predominant manifestation.

**Interventions** We started therapy with subcutaneous secukinumab 150 mg weekly for the first month, then 150 mg monthly thereafter.

**Outcomes** After 2 weeks of secukinumab administration, the patient showed significant remission of pustular skin lesions, with almost no joint pain, swollen bilateral auricles, and no adverse reaction.

**Conclusions** Pustular lesions secondary to RP-associated gangrene and swollen auricles were observed, demonstrating a potential immune correlation between RP and psoriasis in some patients. Although data related to the use of secukinumab for RP and PP is very limited due to the rarity of the two conditions presented together, secukinumab provides a novel therapeutic option. Further prospective studies are needed to support our findings.

**Keywords** Relapsing polychondritis, Pustular psoriasis, Secukinumab, Gangrene, Dermatopathology

## Introduction

Relapsing polychondritis (RP) is an immune-mediated systemic inflammatory disease involving cartilage and proteoglycan-rich tissues, such as ears, nose,

laryngo-tracheo-bronchial tree, joints, skin, and blood vessels [1]. The clinical manifestations vary depending on the affected areas, including distal necrosis, sterile pustules, erythema elevatum diutinum, and Raynaud's phenomenon [2, 3]. The etiology and pathophysiology of RP remains uncertain. However, RP probably results from a combination of genetic susceptibility, triggering factors, and a subsequent abnormal autoimmune reaction [4–6]. A prior study demonstrated that one-third of the patients with RP may display associated autoimmune diseases, such as antineutrophil cytoplasmic antibody-associated vasculitides, and hematological disorders [7]. Similarly,

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patients with psoriasis have been shown to develop autoimmune diseases, such as rheumatoid arthritis, systemic lupus erythematosus, and Sjögren's syndrome [8]. The association between RP and psoriasis has only been reported in a few cases over many decades of study. Here, we report a case of RP with pustular psoriasis (PP) who experienced significant improvement in joint and skin symptoms after receiving two doses of secukinumab. The reporting of this study conforms to CARE guidelines [9].

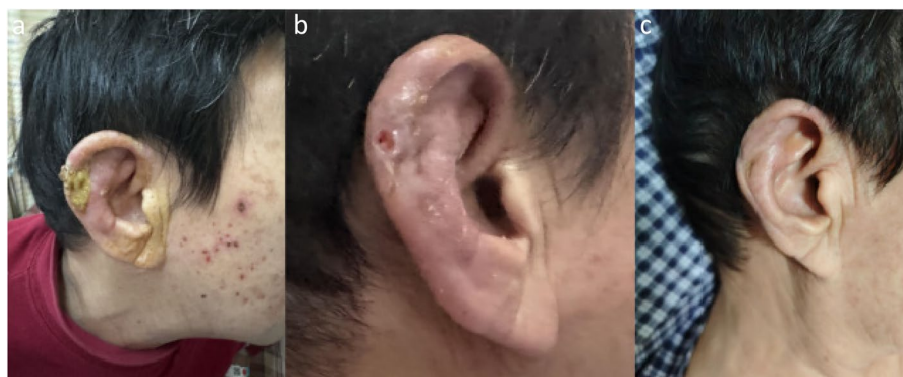
### Patient information and clinical findings

An 80-year-old Chinese female presented at our hospital with a 2-month history of skin rash, erythema, swelling, and pain in both hands and fingertip gangrene. Two months prior, the patient developed the scattered appearance of a bright red, spot-like rash and erythema with skin desquamation and itching over the entire body without any obvious cause. Subsequently, some of the rash subsided, ulcerated, and scabbed, while other ulcer surfaces and the surrounding scabs developed secondary pustules. Simultaneously, the patient's left fourth fingertip turned dark and gradually became necrotic. Subsequently, a 10-mm black dry gangrenous lesion appeared that was accompanied by pain. The edge of the lesion ruptured, showing secondary pustules and a bright red wound. The patient's bilateral auricles were swollen with no involvement of the earlobe. The right ear had secondary ruptured pustules. She had paroxysmal hoarseness without any other accompanying symptoms. The patient was diagnosed with psoriasis 20 years prior and had been taking prednisone and methotrexate regularly for about 1 year. After the psoriatic lesions resolved, she discontinued the medication without relapse. Additionally, she had a history of atrial fibrillation lasting more than 7 years and regularly takes rivaroxaban (15 mg qd po). The

patient has a history of coronary atherosclerotic heart disease and regularly takes aspirin (100 mg qd po) and atorvastatin (20 mg qn po). However, the patient has no history of food and drug allergies, and other thrombotic diseases. At the time of admission, the physical examination results of the patient were as follows: temperature, 36.2 °C; pulse rate, 79 beats/min; respiratory rate, 18 breaths/min; blood pressure, 122/83 mmHg; and weight, 46 kg. Raynaud's phenomenon was observed in both hands, and a black carbonized area of gangrene approximately 10 mm in length was visible at the tip of the left fourth finger. Pustules, ulceration, and yellowish exudate were observed on the edge of the gangrenous area. Multiple distal nail detachments, subungual hyperkeratosis, and nail wrinkles were observed on both hands. The auricles of both ears were red and swollen with pustules and ulcerations. Erythema covered by white scales at the edge, thickened skin, rash, and pustules were distributed over the entire body. Bilateral interphalangeal joints and wrist joints were tender (Figs. 1, 2, and 3). Relevant laboratory examinations are shown in Table 1. Histopathology of the gangrene margin at the digital end showed Kogoj spongiform pustules and acanthosis elongation of the stratum spinosum (Fig. 4). Cardiac ultrasound indicated right atrial enlargement and mild tricuspid valve regurgitation. Chest CT showed thickening of the tracheal wall. Finally, wrist radiography revealed a small amount of effusion in wrist joints, with no significant bone destruction (Fig. 5). Based on the diagnostic criteria proposed by Fujita et al. in 2018 (extensive flush accompanied by multiple sterile pustules and Kogoj's spongiform pustules) [10] and RP proposed by McAdam et al. in 1975 (bilateral ear chondritis, respiratory tract chondritis, and non-erosive seronegative polyarthritis) [11], we diagnosed RP with PP as the predominant manifestation.



**Fig. 1** **a** Scattered appearance of a bright-red spot-like rash, erythema with skin desquamation, and necrotic left fourth fingertip. **b** The edge of the gangrenous area is ruptured, showing secondary pustules, nail wrinkles, distal nail detachment, and subungual hyperkeratosis. **c** The edge of the gangrenous area is ruptured, showing secondary pustules and a bright red wound



**Fig. 2** **a, b** The patient's auricles are red and swollen, with no involvement of the earlobe. The right ear shows ruptured pustules. **c** The auricles are normal after treatment



**Fig. 3** **a** Rash, skin ulcers, and desquamation. **b** Erythroderma-like lesion of psoriasis and secondary pustules. **c** After treatment, the skin erythema and pustules are resolved. The scab surface is dry

### Timeline

The patient was admitted on December 27, 2023. Her symptoms were rated using a series of scales: severity criteria for generalized pustular psoriasis [10], a score of 5; and RPDAI [12], a score of 27. We started therapy with subcutaneous secukinumab 150 mg weekly for the first month and monthly thereafter. Two weeks later, symptoms such as skin pustules, erythema, ulcers, erythema and swelling of auricles, and joint pain improved compared to that experienced prior to treatment (Severity criteria for GPP, 2; RPDAI, 14) (Figs. 2 and 3). Subsequently, the patient's necrotic fingertips were continuously treated with debridement and dressing changes, with no recurrence during the follow-up period (Table 2).

### Discussion

This represents the first case report of RP associated with PP that was successfully treated with secukinumab. The presence of Raynaud's phenomenon is not uncommon in some patients with RP. A clinical study of 66 patients with RP showed that 17% of patients had Raynaud's

phenomenon, and there was a numerically higher proportion of patients carrying Discoidin, CUB, and LCCL

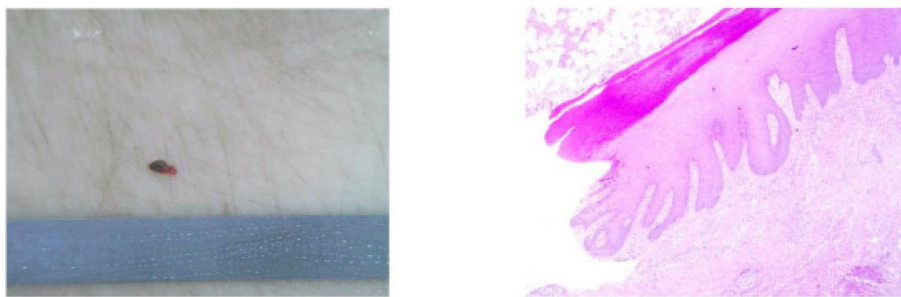
Domain Containing 2 (DCBLD2) ultra-rare damaging qualifying variants with cardiovascular manifestations, including venous thromboembolism, Raynaud's phenomenon, and aortitis [3]. Unlike patients with primary Raynaud's phenomenon, persistent digital ischemia, ulceration and gangrene can occur in patients with secondary Raynaud's phenomenon [13]. Immune-associated endothelial dysfunction and overproduction of vasoconstrictors have been suggested to be important in the pathogenesis of connective tissue disease-associated RP [14]. Based on the patient's diagnosis, the formation of gangrene may involve multiple factors: (1) a large number of vasoactive molecules associated with psoriasis are up-regulated, including vascular endothelial growth factor which enhances vascular permeability and intercellular/vascular cell adhesion molecules involved in trafficking of lymphocytes to inflammatory lesions, and modification of the cutaneous microvasculature maintains the clinical lesional state and promotes inflammatory cell migration [15]. These factors trigger immune-associated endothelial dysfunction. (2) RP causes vasculitis (including leukocytoclastic vasculitis and lymphocytic vasculitis) and thrombosis [6, 14]. (3) Age-related thickening, hardening, and loss of elasticity of arteries.

**Table 1** Changes in patient-related laboratory examination indicators

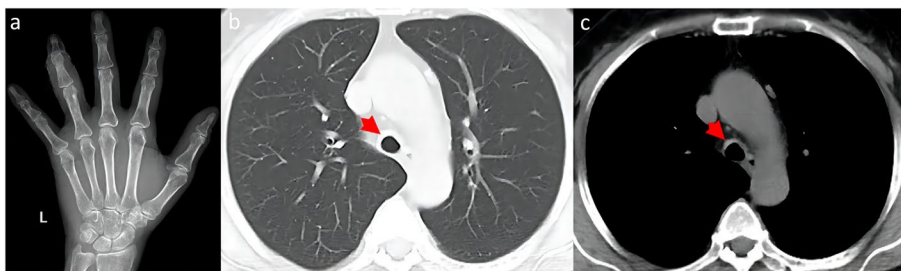
Laboratory examination indicators	At admission	After 2 weeks of receiving secukinumab treatment	Reference value
White blood cell	$8.51 \times 10^9/L$	$9.03 \times 10^9/L$	$(3.5-9.5) \times 10^9/L$
Neutrophil count	$6.14 \times 10^9/L$	$6.29 \times 10^9/L$	$(1.8-6.3) \times 10^9/L$
Red blood cell	$4 \times 10^{12}/L$	$4.04 \times 10^{12}/L$	$(3.8-5.1) \times 10^{12}/L$
Hemoglobin	132 g/L	134 g/L	115–150 g/L
Platelet count	$298 \times 10^9/L$	$227 \times 10^9/L$	$(125-350) \times 10^9/L$
Alanine aminotransferase	33.9 $\mu/L$		< 40 $\mu/L$
Aspartate aminotransferase	23.9 $\mu/L$		< 35 $\mu/L$
Serum albumin	31.1 g/L	31.7 g/l	40–55 g/l
Creatinine	40 $\mu\text{mol}/L$		45–84 $\mu\text{mol}/L$
Uric acid	159 $\mu\text{mol}/L$		142.8–339.2 $\mu\text{mol}/L$
Urine protein	Negative		Negative
Cardiac troponin I	0.012 ng/ml		0–0.034 ng/ml
Creatine kinase	34 $\mu/L$		26–140 $\mu/L$
Creatine kinase isoenzyme	15.48 $\mu/L$		7–25 $\mu/L$
B-type natriuretic peptide	235.46 pg/ml	260.72 pg/ml	0–100 pg/ml
ANA	1:320 the nuclear Dense granular pattern		Negative
Anti-ENA antibody profile	Negative		Negative
Anti-Proteinase 3 antibody	0 RU/ml		0–20 RU/ml
Myeloperoxidase antibody	1.01 RU/ml		0–20 RU/ml
Anticardiolipin antibody	4 RU/ml		0–12 RU/ml
Anti-ntilodyiolipin tibodytideu	32 RU/ml		0–20 RU/ml
Lupus anticoagulant	Negative		Negative
Procalcitonin	0.04 pg/ml		0–0.05 pg/ml
C-reactive protein	8.92 mg/L	3.22 mg/L	0–3.48 mg/L
Erythrocyte sedimentation rate	64 mm/h	19 mm/h	0–20 mm/h
Prothrombin time	11.7 s		9.8–12.1 s
International normalized ratio	1		0.84–1.16
Activated partial thromboplastin time	24.3 s		22.7–31.8 s
Thrombin time	17.5 s		14–21 s
Fibrinogen	2.91 g/L		1.8–3.5 g/L
Fibrinogen degradation products	2.5 mg/L		0–5 mg/L
D-Dimer	0.23 mg/L		0–0.55 mg/L
Protein C	77.7%		70–140%
Protein S	75.9%		60–130%

A systematic search of articles published in PubMed/MEDLINE, Web of Science, ChinaInfo System, LILACS, and SciELO, registered from 1960 to February 2024 was performed. In addition to our case report, five studies consisting of eight patients with RP and psoriasis were retrieved. Table 1 summarizes this report and all cases of RP associated with psoriasis published in the literature [16–21]. Sex predilection (5 females, and 4 males) was not observed, and age varied from 15 to 80 years. Psoriasis preceded the onset of RP in all cases by 1–20 years. Among the patients with concurrent RP, four patients had psoriasis vulgaris (PsV), two had PsV complicated

by psoriatic arthritis (PsA), one had generalized pustular psoriasis (GPP) complicated by PsA, and one had GPP complicated by ankylosing spondylitis. The treatment regimen comprised glucocorticoids, non-steroidal anti-inflammatory and analgesic agents, and biologics (adalimumab, etanercept, and secukinumab). Except for two cases without documented outcomes, the remaining patients all exhibited a favorable prognosis. The skin lesions were predominantly characterized by erythema and pustules. The incidence of RP-related damage (77.78% in ears, 44.44% in the nose) was similar to the probabilities reported in the literature (85% in ears,



**Fig. 4** Histopathology of the gangrene margin at the digital end. The pathological results of the skin tissue at the edge of gangrene and the other skin lesions show hyperkeratosis and parakeratosis of the epidermis and subcutaneous tissue, formation of local ulcers, Kogoj spongiform pustules, acanthosis elongation of the stratum spinosum, severe acute and chronic inflammation of the dermis and subcutaneous tissue, formation of multiple abscesses, and fibrogranular tissue hyperplasia



**Fig. 5** a Degenerative changes in the left hand, slightly narrowed joint space in the fourth distal interphalangeal joint, and no significant abnormalities in the remaining joints. b, c Thickening of the wall of the main bronchial tube

54% in the nose) [6]. No significant tendency towards the development of erosive or non-erosive arthritis was observed.

Interestingly, the clinical course and severity of psoriasis did not correlate with the occurrence or clinical course of RP in these previous cases. However, we observed the occurrence of psoriatic pustules at the skin lesion margin of RP (digital gangrene and auricular swelling) in our case, and the nature of the skin lesions was confirmed by pathology. In genetically susceptible individuals with RP, molecular mimicry may occur between cartilage micro-injuries, infections, or stimuli and cartilage structure, which may trigger a series of inflammatory responses similar to other autoimmune diseases [22].

Relapsing polycondritis usually exhibits immunological abnormalities. A large infiltration of CD4+ T lymphocytes, B cells, or plasma cells occurs in the lesion [23]. After stimulation with mitogens and collagen antigens, increased levels of various cytokines, including TNF- $\alpha$ , IL-1, IL-6, and IL-17, as well as autologous chondroitin sulfate proteoglycans are expressed by T cells. These cytokines can lead to the degradation of chondroitin sulfate proteoglycans or direct inhibition of the synthesis of glucosaminoglycans by chondrocytes [1]. Additionally, inflammation increases protease activity and

causes cartilage degradation [7]. Psoriasis is considered a disease primarily mediated by T helper cell subsets and the cytokines that they secrete. The cells infiltrating the dermis are a mixture of CD4+ and CD8+ T cells, which are predominantly memory T cells expressing cutaneous homing receptors, namely, cutaneous leucocyte-associated antigen (CLA) receptor and chemokine receptor 4 (CCR4). These cells migrate into the epidermis by binding to basement membrane type IV collagen through the expression of  $\alpha 1\beta 1$  integrin and participate in the formation of psoriatic lesions [24]. Innate immune cytokines such as IL-1, IL-6, IL-17, and TNF- $\alpha$  are upregulated in psoriatic lesions [24]. The shared pathogenic mechanisms may ultimately lead to secondary PP in our patient due to RP-related skin lesions. Therefore, we acknowledge the persistent systemic inflammation and immune damage caused by RP, which expose the skin lesions to immune attacks and make them susceptible to immune aggression.

In psoriasis, expression of IL-17 mRNA is higher in skin with lesions than in skin without lesions [25]. Additionally, IL-17A levels are significantly correlated to disease severity [26, 27]. The current paradigm for RP pathogenesis involves cytokine-mediated immunologic activity via IL-17A and TNF- $\alpha$ , leading to the expression

**Table 2** Cases of relapsing polychondritis associated with psoriasis described in the literature

Author, year	Age, years	Sex	Complication (disease duration)	RP locations and symptoms	Psoriasis symptoms	Other rheumatic disease symptoms	Treatment	Outcome
Song et al. 2024 (present article)	80	F	PsV (20 years)	Bilateral ear swelling and erythema, NON-erosive polyarthritis, tracheal chondritis, fingertip gangrene	Nail wrinkles, distal nail detachment, subungual hyperkeratosis, pustular lesions	No	Secukinumab	Recovered
Matsuo et al. 2017 [16]	38	M	PsV (13 years)	Bilateral ear swelling and erythema, nasal chondritis	Erythema with skin desquamation, Nail psoriasis	No	Adalimumab	Recovered
Murao et al. 2011 [17]	42	F	PsV (10 years) AA (1 year)	Bilateral ear swelling and erythema, non-erosive polyarthritis	Erythema with skin desquamation, nail psoriasis	No	PSL 30 mg/day	Recovered
Lee et al. 2011 [18]	43	M	PsV (11 years) PsA (11 years)	Bilateral ear swelling and erythema	Erythema with skin desquamation, peripheral arthritis and sacroiliac arthritis	No	Etanercept 50 mg twice weekly	Recovered
Lee et al. 2011 [18]	25	M	PsV (1 year) PsA (2 years)	Bilateral ear swelling and erythema	Red plaques covered by scales, psoriatic arthritis	No	Etanercept 50 mg twice weekly	Recovered
Borbujo et al. 1989 [19]	62	F	PsV (20 years)	Nasal and laryngeal chondritis, conjunctivitis, episcleritis, and non-erosive polyarthritis	Red plaques covered by scales, psoriatic arthritis	No	NSAIDs	Unknown
Borbujo et al. 1989 [19]	15	F	PsV (2 years)	Nasal and laryngeal chondritis, ocular inflammation, fever	Red plaques covered by scales	No	Antibiotics, NSAIDs	Unknown
Hager et al. 1987 [20]	45	F	GPP (19 years) PsA (9 years)	Bilateral ear swelling and erythema, nasal chondritis	Cutaneous pustules, psoriatic arthritis	No	Spontaneous remission	Recovered
Pearson et al. 1960 [21]	47	M	GPP (8 years) AS (16 years)	Bilateral ear swelling and erythema	Cutaneous pustules, nail psoriasis	Progressive arthritis of the spine and several peripheral joints caused by AS	PSL30 mg/day	Recovered

AA alopecia areata, AS ankylosing spondylitis, F female, GPP generalized pustular psoriasis, M male, NSAIDs non-steroidal anti-inflammatory drugs, PsA psoriatic arthritis, PSL prednisolone, PsV psoriasis vulgaris, RP relapsing polychondritis

of matrix-degrading proteinases in chondrocytes [28]. Secukinumab is a recombinant human IgG1 kappa monoclonal antibody that selectively targets IL-17A and blocks its interaction with the IL-17 receptor. Inhibition of the downstream effects of this proinflammatory cytokine thereby interferes with key psoriatic disease pathways while promoting normalization of immune function and skin histology [29].

In prior research, secukinumab has been successfully employed in the therapeutic management of psoriasis (a dose of 300 mg or 150 mg), exhibiting a commendable efficacy and safety profile [30, 31]. Based on the body mass (46 kg) of the patient, a weekly dosage of 150 mg was recommended. Ultimately, the patient exhibited a favorable response to treatment, with amelioration of symptoms and a reduction in inflammatory indices (CRP decreased from 8.92 mg/L to 3.22 mg/L, ESR from 64 mm/h to 19 mm/h). However, due to the very limited data related to the use of secukinumab for RP and PP, whether low-dose secukinumab is applicable in a broader spectrum of patients with RP complicated by psoriasis and has the potential to reduce the risk of relapse requires further investigation.

## Conclusions

This is the first case report of RP associated with PP that was successfully treated with secukinumab, adding to new evidence emphasizing a potential immune correlation between RP and psoriasis in some patients. The clinical course of psoriasis correlates with the clinical course of RP in our case, and partially shared pathogenic mechanisms may ultimately lead to secondary PP in our patient due to RP-related skin lesions.

## Abbreviations

AA	Alopecia areata
AS	Ankylosing spondylitis
BNP	B-type natriuretic peptide
BSA	Body surface area
CCR4	Chemokine receptor 4
CLA	Cutaneous leucocyte-associated antigen
CRP	C-reactive protein
DCBLD2	Discoidin, CUB, and LCCL Domain Containing 2
ESR	Erythrocyte sedimentation rate
F	Female
FIB	Fibrinogen
GPP	Generalized pustular psoriasis
IgE	Immunoglobulin E
M	Male
NSAIDs	Non-steroidal anti-inflammatory drugs
PASI	Psoriasis Area and Severity Index
PP	Pustular psoriasis
PsA	Psoriatic arthritis
PSL	Prednisolone
PsV	Psoriasis vulgaris
RP	Relapsing polychondritis
RPDAI	Relapsing Polychondritis Disease Activity Index

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## Authors' contributions

Conceptualization: Zhankui Wang. Writing—original draft: Qingcheng Song. Writing—review and editing: Qingcheng Song, Zhankui Wang. All authors read and approved the final manuscript.

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## Availability of data and materials

Data availability is not applicable to this article as no new data were created or analyzed in this study.

## Declarations

### Ethics approval and consent to participate

Formal written consent for publication was obtained from the patient and is available on request. This study is a clinical retrospective case report. No ethics approval.

### Consent for publication

The manuscript is approved by all authors for publication.

### Competing interests

The authors declare that they have no competing interests.

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