## Managing PG

### There are no FDA-approved therapies for PG[12]

- Since PG is a rapidly progressing disease, prompt treatment is key[1]
- Treatment strategies depend on PG disease severity[13]

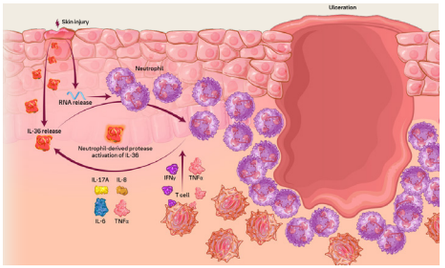
**Mild PG**
One ulceration, dermis and/or ≤4 $cm^2$ area

**Moderate to severe PG** (Multiple) ulceration(s), subcutaneous/ muscle/ tendon/bone and/or >4 $cm^2$ area

- Topical corticosteroids and/or calcineurin inhibitor
- Wound and pain management, compression therapy
- Systemic corticosteroids and/or cyclosporine

**Response to current therapies is unpredictable and can be slow[14,15]**

## Pathophysiology of PG




### The IL-36 pathway may play a key role in PG pathophysiology[16-20]

IL-36 is shown to be significantly increased in lesional skin of PG and may contribute to inflammatory response and progression of disease[16-20]

FDA, US Food and Drug Administration; IBD, inflammatory bowel disease; IFN, interferon; IL, interleukin; PG, pyoderma gangrenosum; TNF, tumor necrosis factor.

1. Maverakis E, et al. *Nat Rev Dis Primers.* 2020;6:81. 2. Xu A, et al. *J Am Acad Dermatol.* 2020;83:425–429. 3.Narla S, Silverberg JI. *Arch Dermatol Res.* 2021;313(4):245–253. 4. Li SJ, et al. *Dermatology*. 2024;240(1):26–31. 5. Yamamoto T, et al. *J Dermatol*. 2023;50:e253–e275. 6. Langan SM, et al. *J Invest Dermatol.* 2012;132(9):2166–2170. 7. Marzano AV, et al. *Clin Rev Allergy Immunol.* 2018;54(1):114–130. 8. Romagnuolo M, et al. *Front Med (Lausanne).* 2023;10:1197273. 9. Ye MJ, Ye JM. *Dermatol Res Pract.* 2014;2014:461467. 10. George C, et al. *Clin Med (Lond).* 2019;19:224–228. 11. Ighani A, et al. *Br J Dermatol.* 2019;180:672–673. 12. Rick J, et al. *Arch Dermatol Res.* 2023;315(4):983–988. 13. Dissemond J, et al. *Drugs*. 2023;83:1255–1267. 14. Argüelles-Arias F, et al. *Dig Dis Sci.* 2013;58(10):2949–2954. 15. Miller J, et al. *J Am Acad Dermatol.* 2010;62(4):646–654. 16. Mercurio L, et al. *Cell Death Dis.* 2018;9(11):1104. 17. Wang EA, et al. *Front Immunol.* 2018;8:1980. 18. Łyko M, et al. *Int J Mol Sci.* 2024;25(4):2440. 19. Yuan ZC, et al. *Front Immunol.* 2019;10:2532. 20. Carrier Y, et al. *J Invest Dermatol.* 2011;131(12):2428–2437.

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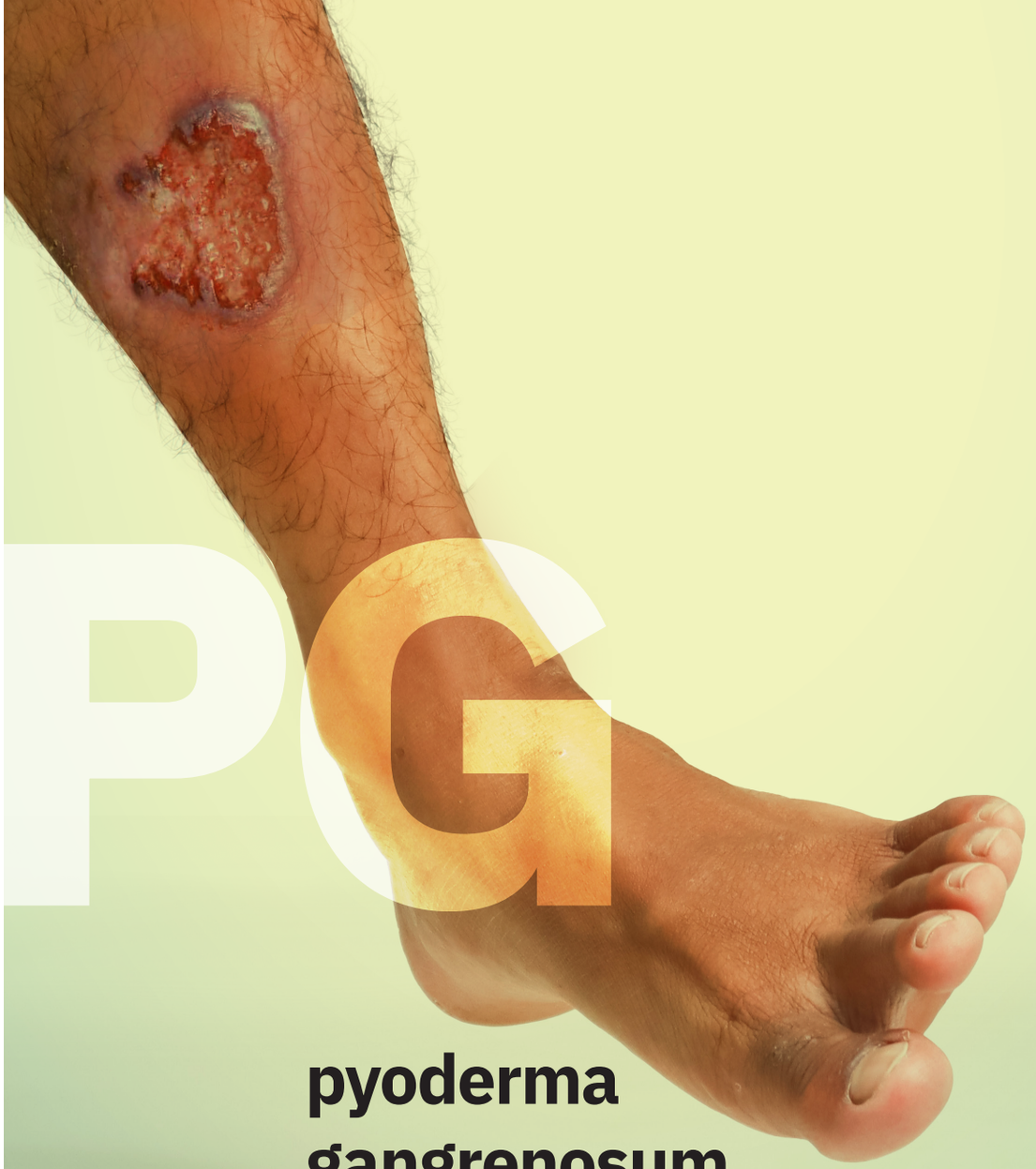
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MAT-94535 April 2026

L E O



# pyoderma gangrenosum

DISEASE BURDEN AND UNMET NEEDS

*Not an actual patient—for illustrative purposes only.*

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L E O

# What is Pyoderma Gangrenosum (PG)?

- Rapidly progressing, ulcerative neutrophilic dermatosis[1]
- Rare disease, with an estimated prevalence of 5.8 per 100,000 adults in the US[1,2]
- Associated with systemic disease, including inflammatory bowel disease (IBD), rheumatoid arthritis, and hematologic malignancies[1,3]

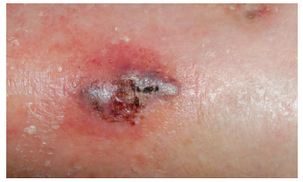
# Diagnosing PG

## PG is a diagnosis of exclusion with nonspecific histological features[4]

When evaluating a patient for PG, it is necessary to exclude other potential causes of ulceration, including infections, circulatory disorders, antiphospholipid antibody syndrome, livedo vasculopathy, malignant neoplasms, and vasculitis[5]

## PG disease course

BEGINS AS



- Sterile small pustules, papules, and blisters[1]
- Mostly occurs on extensor surface of the legs[6,7]
- Can be triggered by minor skin trauma or certain drugs, including anti–TNF-α agents[7,8]

ULCERATION



**Rapidly evolves** into painful ulcers with purulent and necrotic floors and elevated violaceous borders[1,7]

In the **healing stage**, the wound edge has projections of epithelium extending into the ulcer, known as Gulliver's sign. After healing, scars persist[7]



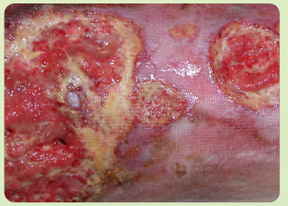
There is a high **recurrence rate** of lesions[9]

HEALING

Image: Sterile small pustule. Reproduced with permission from © DermNet www.dermnetnz.org 2024. Image: Ulcers with purulent and necrotic floors. Reproduced with permission from © DermNet www.dermnetnz.org 2024. Image: Healing scars "Gulliver's sign." Reprinted from Marzano AV, et al. *Clin Rev Allergy Immunol*. 2018;54(1):114-130. Copyright © 2017, Springer Science Business Media, LLC. Image: Lesions. Reproduced with permission from © Te Whatu Ora www.dermnetnz.org 2024.

## PG subtypes[10]

### Classical (ulcerative)



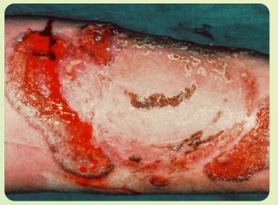
Most common form (≈85% of cases); presents with painful, **rapidly progressing ulcers**.

### Pustular



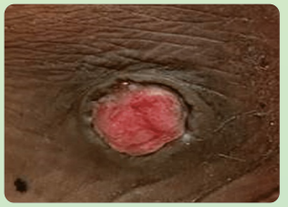
Typically occurs in the context of flaring IBD; presents with painful **pustules on an erythema background**

### Bullous



Presents with rapidly evolving, painful, **superficial vesicles and bullae** arising in waves

### Granulomatous superficial



Presents with **verrucous** and **ulcerative lesions**; usually progresses more slowly

### Peristomal



Mostly seen in the context of stomas in patients with **IBD**

### Malignant



Rare variant; presents with **destructive ulcerations** typically affecting the upper torso, head, and neck

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# Burden of PG

PG is associated with a substantial burden on quality of life[11]



- Excessive pain[1]
- Unsightly ulcers[1]
- Persistent cribriform scars[1,7]



- Psychosocial and emotional burden[11]
- Depression[11]
- Fear of recurrence[1]