Dermatology in the Unhoused

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My background

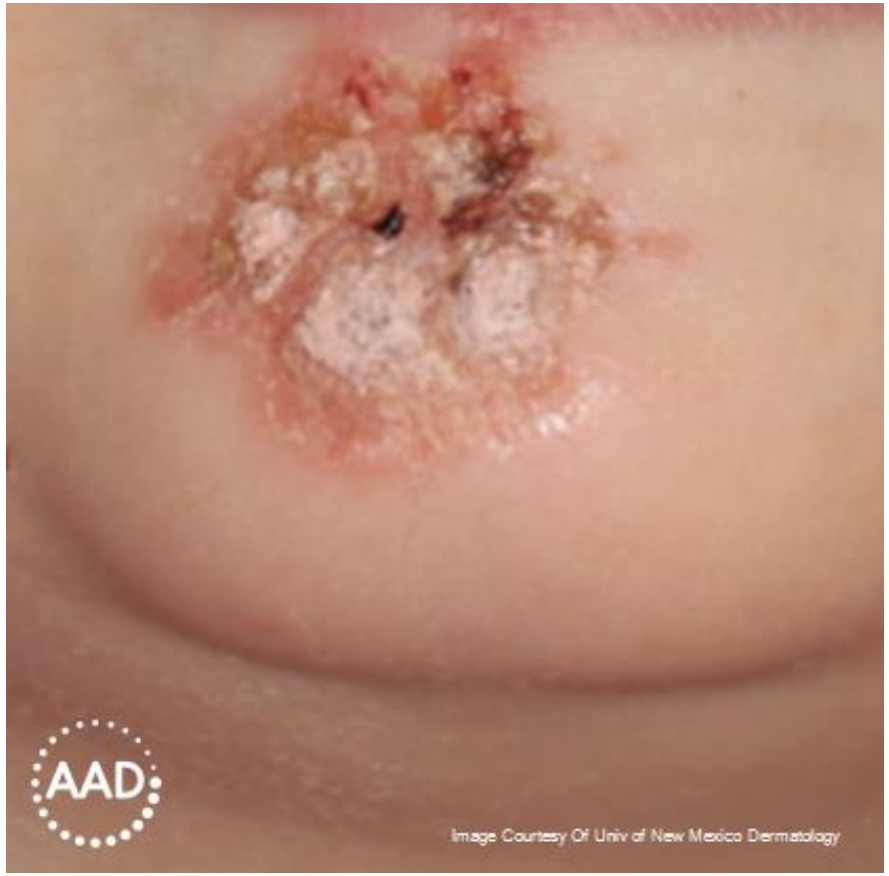
- On-call for ER and inpatient consults at OHSU and the VA 5 days a week 8am-5pm year round since 2015
- Psychodermatology clinic Mon am at OHSU focusing on delusional parasitosis
- VA clinic Fri am at VA focusing on delusional parasitosis, leg ulcers, general derm

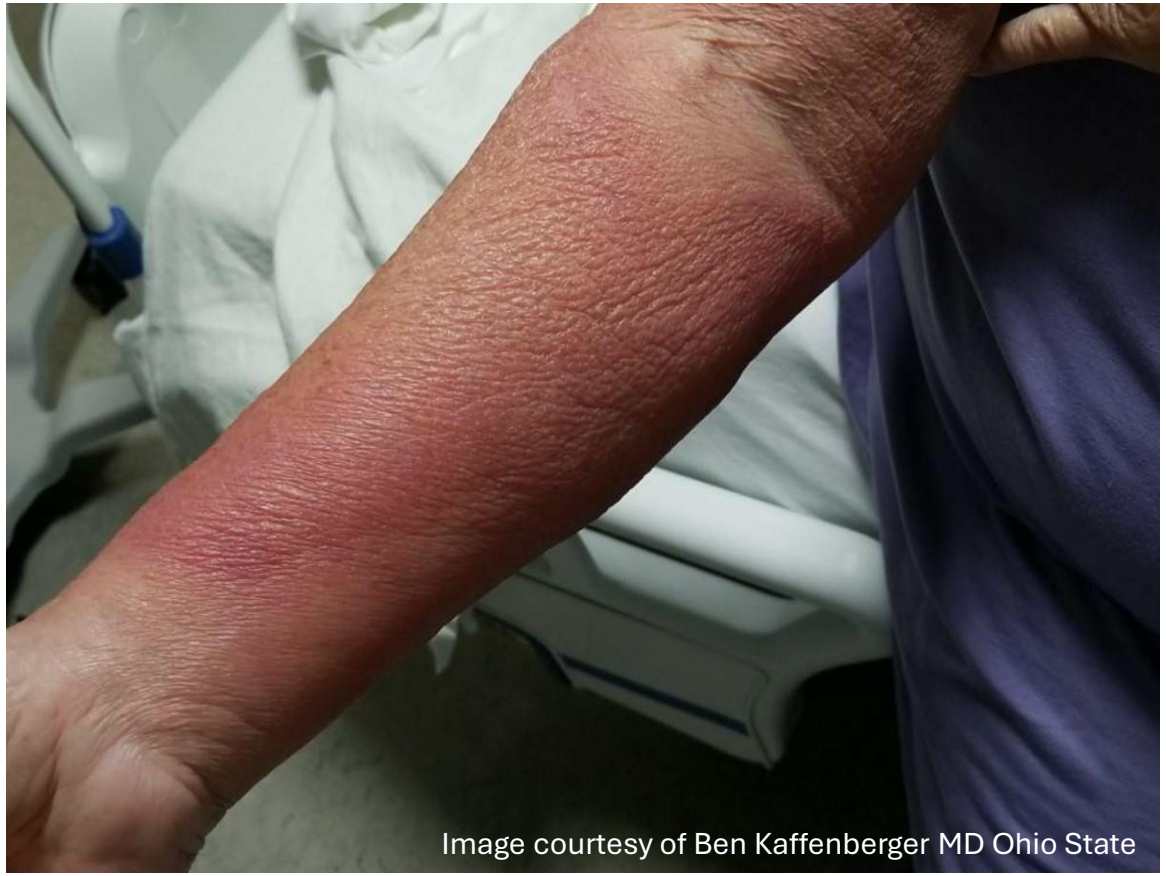
Why should dermatology present differently in an unhoused population?

Why should dermatology present differently in an unhoused population?

- Exposure to cold (thermal injury)
- Reduced access to daily hygiene (infection risk)
- Logistical challenges in communication, transportation (reduced adherence, inability to refrigerate biologic medications, poor follow up)
- More immediate needs including meals, water, safety (neglected chronic skin disease)
- Mental health burden
- Polysubstance use disorder (opioids may mask the pain of a worsening condition; amphetamines may lead to unusual skin sensations)

Skin and soft tissue infections (SSTI)



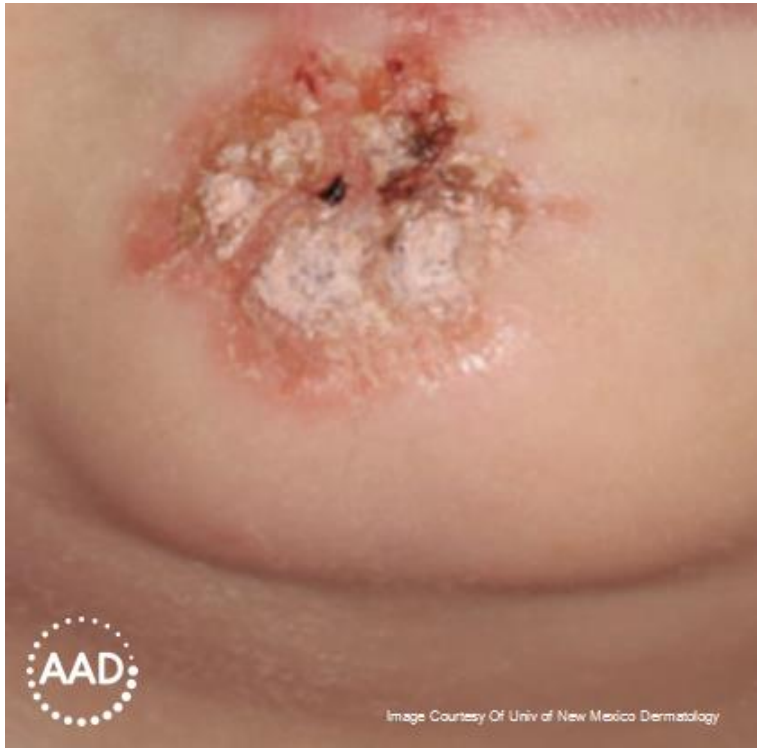


Skin and soft tissue infections

- Presentations may include
  - Cellulitis
  - Impetigo
  - Bullous impetigo
- Common pathogens include MSSA, MRSA, Group A Strep

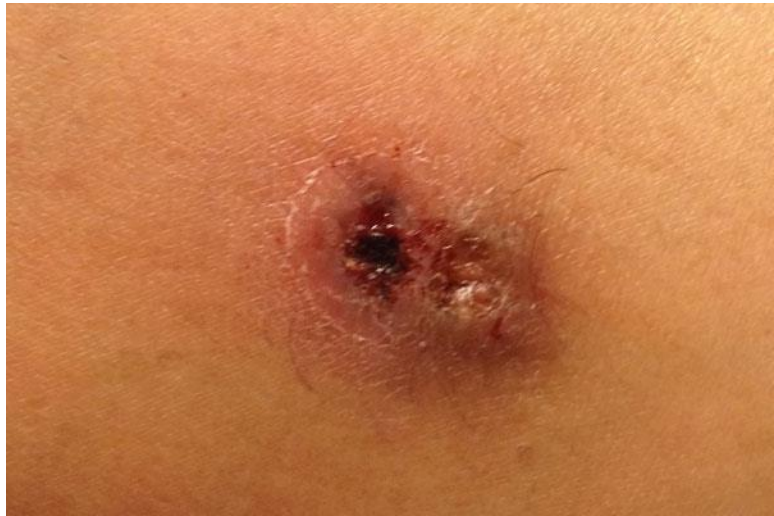
Impetigo

- Crusting is the primary physical exam finding for superficial SSTI
- Can treat with mupirocin or with 3 days of cephalexin or dicloxacillin

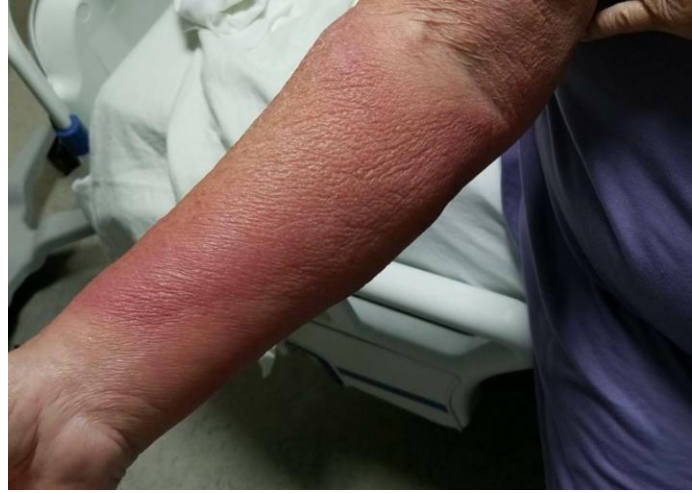


Ecthyma

- SSTI with deeper, more necrotic/ulcerative appearance
- Commonly affects thighs, buttocks
- Seen more often in association with MRSA
- Recommend systemic therapy, ie PO doxycycline for MRSA coverage



Cellulitis

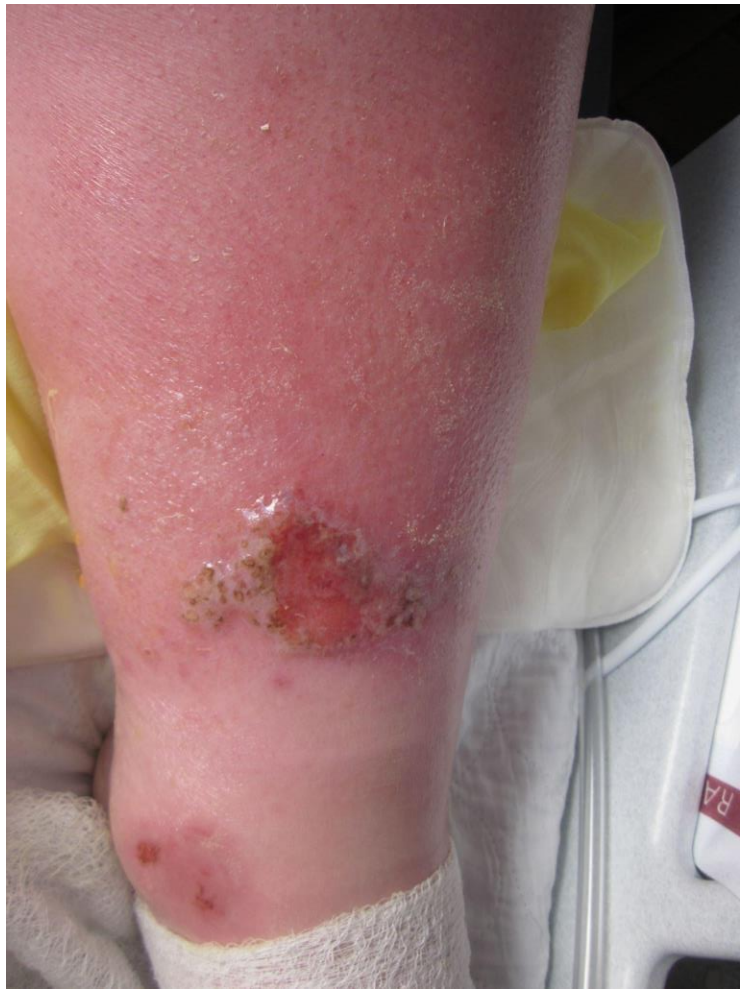


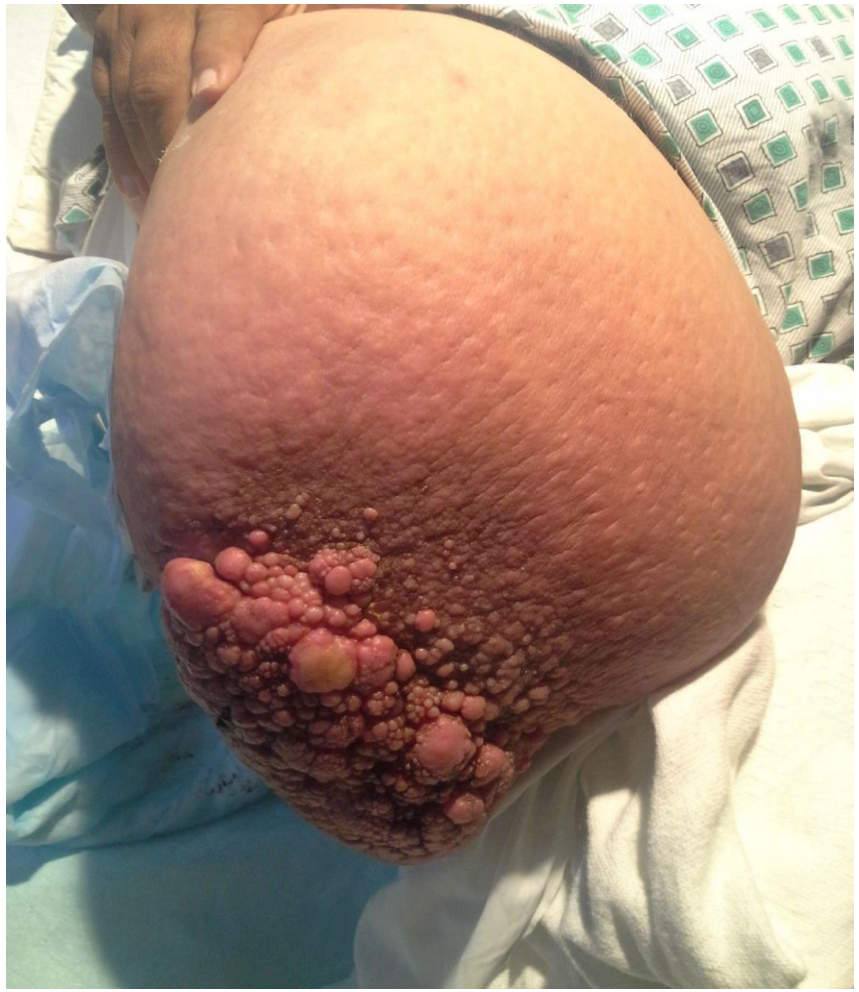
- Deeper process involving subcutis
- Most often Group A Strep, but may be polymicrobial or different pathogens in special groups (DM2, hemochromatosis, cat or dog or human bite)
- Smooth tender red-orange plaques generally affecting one extremity
- May be difficult to distinguish from other mimickers known as pseudocellulitidies (venous stasis, lymphedema, contact dermatitis)
- Scoring tools that may be useful:
  - ALT-70
  - New Havun criteria

Cellulitis

- ALT-70 (61% sens, 71% spec)
  - Asymmetry (3 points), leukocytosis (1), tachycardia (1), age >70 (2 points)
  - 5-7 points = positive
  - 3-4 points = consult derm
  - 0-2 points = negative
- New Havun (100% sens, 95% spec)
  - New onset, erythema, warmth, history of trauma, ache, unilaterality, number of white blood cells)
  - 4-7 points = positive
  - 0-3 = negative

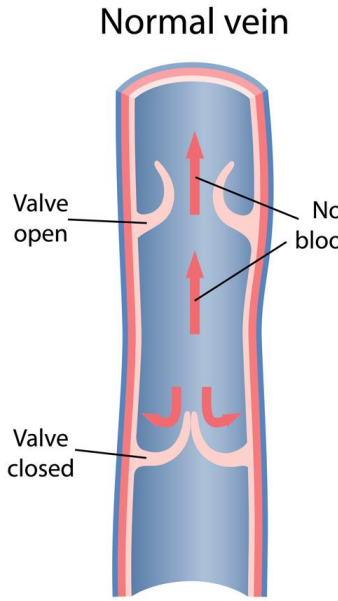
Leg swelling





Leg swelling

- Venous stasis dermatitis is a result of chronic wear and tear of one-way valves in the lower legs that are stressed/stretched with prolonged sitting still or standing still with legs in the dependent position
- Everyone will develop some degree of venous stasis; some develop it decades earlier than others



Preferred sleeping position

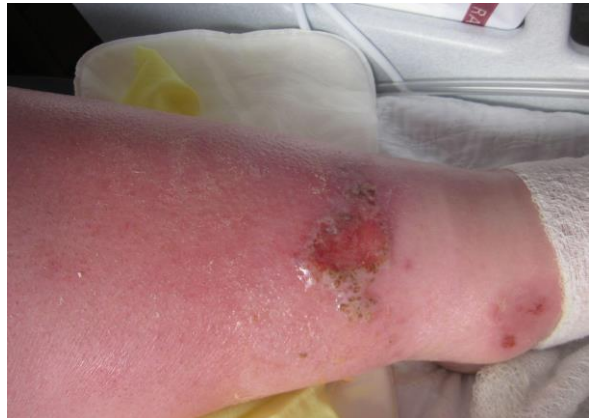
Suboptimal sleeping position

Common Causes of Pseudocellulitis

Vascular

Venous stasis dermatitis

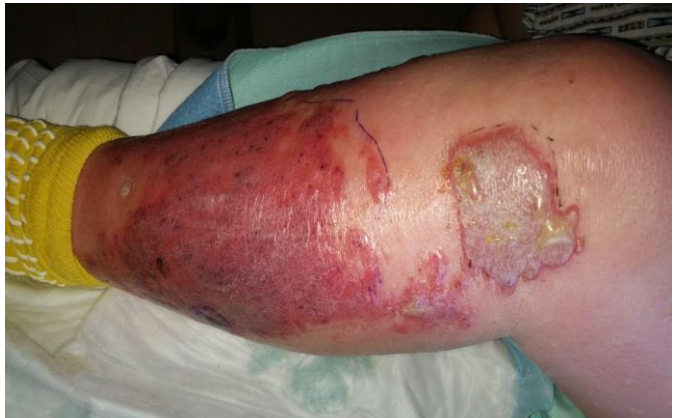
- Inflammation of the skin, typically of the bilateral lower extremities, due to chronic venous insufficiency
- Often associated with varicose veins and dependent chronic edema



Non-infectious Inflammatory

Allergic contact dermatitis

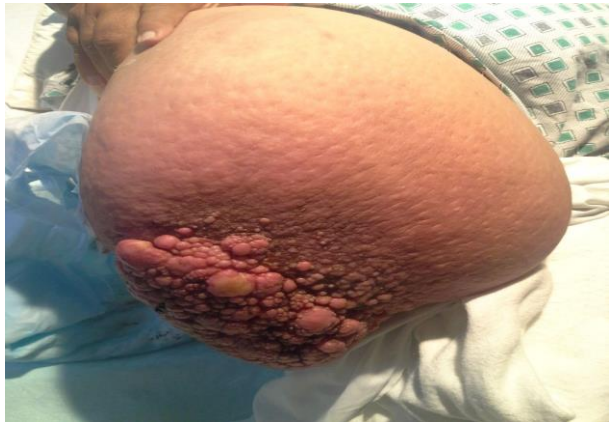
- Delayed hypersensitivity reaction due to contact with exogenous allergens leading to an inflammatory skin reaction



Lymphatics

Lymphedema

- Abnormal accumulation of lymphatic fluid in the soft tissue leading to swelling and skin damage
- Causes include injury, infection, cancer therapies, or congenital abnormalities



	Cellulitis	Venous Stasis Dermatitis	Allergic Contact Dermatitis	Lymphedema
Onset	**Acute**	Chronic	Acute to chronic	Chronic
Physical Exam	**Poorly demarcated; Classic tetrad: erythema, warmth, edema, pain;** Trauma to the skin allowing for entry of bacteria	Pitting edema, mottled, hyperpigmentation, varicose veins, +/- ulcerations	Well-demarcated rash confined to area of contact to allergen	Swelling, +/- pitting edema, +/- overlying hyperpigmentation, thickening, scale
Symptoms	+/- Systemic symptoms (eg, fever)	Absence of systemic symptoms (eg, no fever)	Pruritus, burning	Feeling of heaviness/discomfort
Location	**Unilateral** > Bilateral Most common: legs or feet	Bilateral, often involving medial malleolus	Area of skin contact to allergen	Commonly arms or legs,may occur in face, neck, trunk, abdomen, or genitals
Diagnostic Tools	CBC, blood culture; histopathology, tissue cultures, Dundee, ALT-70, and New HAvUN scoring assessments	Histopathology, Duplex ultrasound with reflux assessment	Histopathology, Identify trigger(s); patch testing	Histopathology, Lymphoscintigraphy
Histology	Nonspecific inflammatory infiltrate commonly, rarely identification of bacterial or fungal organisms	Neovascularization with dermal fibrosis, perivascular lymphocytic infiltrates, hyperkeratosis	Epidermal edema, acanthosis, +/- hyperkeratosis or parakeratosis	Dilated dermal lymphatics, fibrous tissue deposition in overlying skin, subcutaneous tissue and fascia
Treatment	Antibiotics (depending on severity and area-specific antibiotic nomogram)	Leg elevation, weight loss, exercise, compression therapy, topical	Allergen avoidance, topical corticosteroids or tacrolimus; Severe:	Leg elevation, weight loss, exercise, compression therapy, decongestive

Treatments for Common Causes of Pseudocellulitis

PO: oral
IV: intravenous

Vascular

Venous stasis dermatitis

- Leg elevation
- Weight loss
- Exercise
- Compression therapy
- Topical corticosteroids

Non-infectious Inflammatory

Allergic contact dermatitis

- Allergen avoidance
- Topical corticosteroids
- Topical tacrolimus

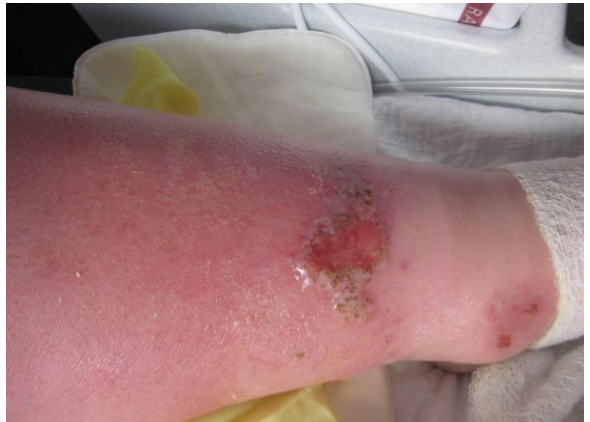
Severe/extensive:

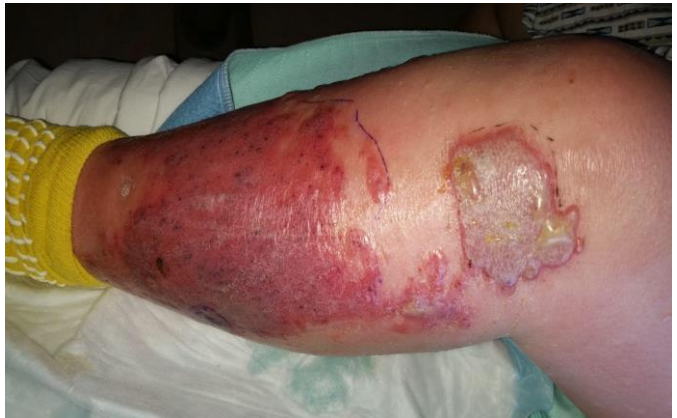
- Oral corticosteroids and/or other immunosuppressants

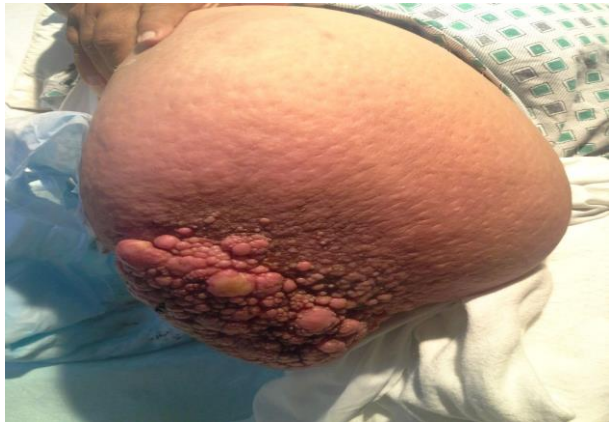
Lymphatics

Lymphedema

- Leg elevation
- Weight loss
- Exercise
- Compression therapy
- Decongestive therapy
- Surgery
- Ablative therapy







Pyoderma gangrenosum

- PG is an autoinflammatory ulcer of the lower legs associated with:
  - Crohn's, UC, RA, Psoriasis
  - Venous stasis/swelling

- Unique factors in the unhoused population include:
  - Lack of consistent and sterile wound care leads to infections
  - Opioids may be used to mask pain while an infection progresses
  - Inadequate leg elevation leads to edema slowing wound healing
  - Inability to travel to follow up appointments, labs, infusion centers for infliximab
  - Inability to refrigerate biologics such as adalimumab

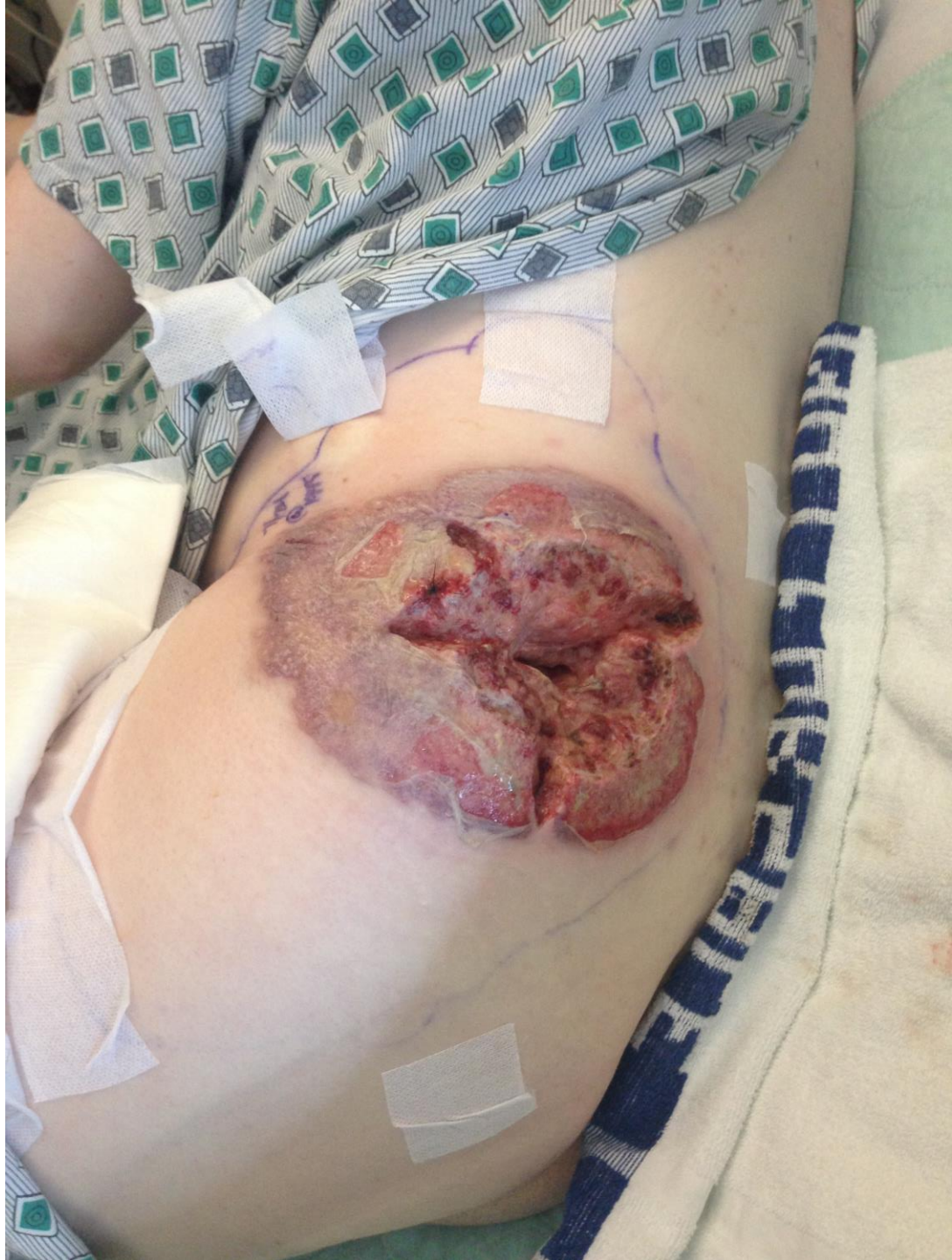
Pyoderma gangrenosum workup

- Consider skin biopsy to rule out alternate etiologies (BCC, medium vessel vasculitis, vasculopathy)
- Consider Hep B, Hep C, ANCA, G6PD

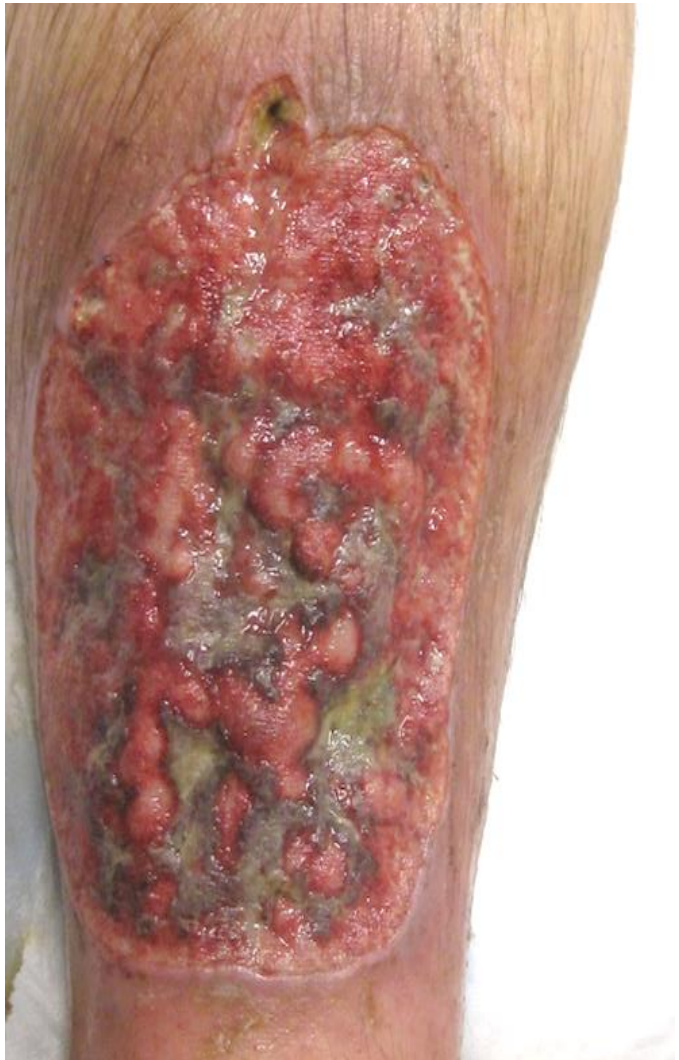
- Inflammatory phase: Prednisone or cyclosporine are first-line. TNF inhibitors or dapsone may also provide some benefit

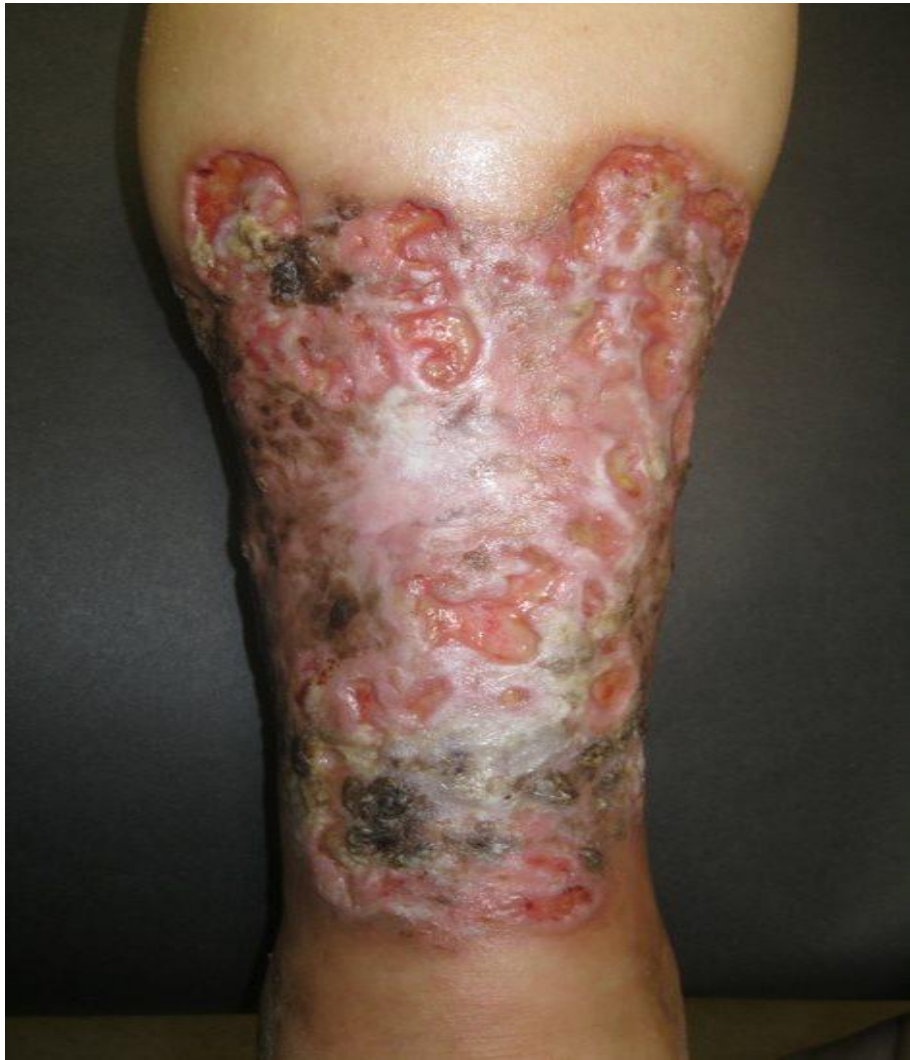
- "Burnt-out" phase: Ulcer is established but the borders are not actively inflamed. Needs gentle conservative wound care, leg elevation, and compression as tolerated

Inflammatory phase



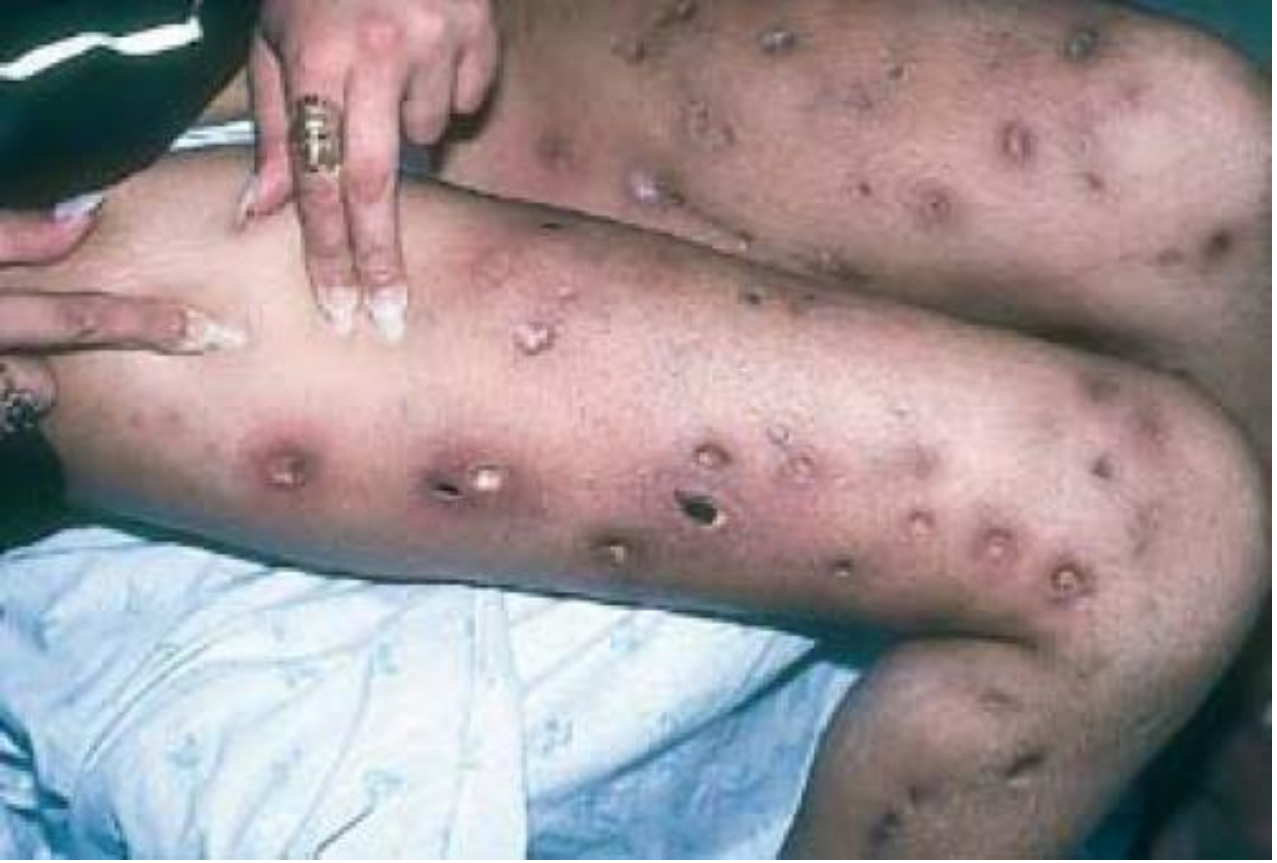
Burnt out phase





Amphetamine associations

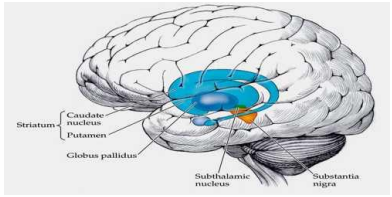
- Methamphetamine is a cheap, widely, and easily accessible drug that activates the dopamine reward/pleasure centers in the brain
- Common side effects include skin picking, skin crawling, and obsessive-compulsive behavioral changes



Reviewing the chart: “Why this patient, and why now?”

	Past medical history	Medications	Substance use	Psychiatric history
Risk factors for DI/MD	Neurologic lesions (CVA,vascular dementia, MS)	Amphetamines	Amphetamines	PTSD, ADHD, Depression, Anxiety
	Chronic pain syndromes (spinal stenosis, fibromyalgia)	Opioids	Opioids	
	Iron deficiency	Ropinirole Pramipexole		
	Postmenopausal female			

Dopamine: A delicate balance



low dopamine

↓

Parkinson's
Restless leg syndrome

↓

Treat with: dopamine agonist

high dopamine

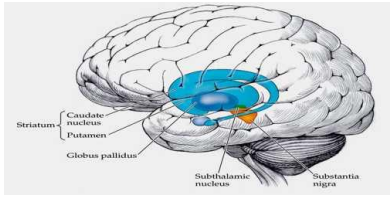
↓

Morgellons/Delusional parasitosis

↓

Treat with: dopamine antagonist (antipsychotic)

Dopamine: A delicate balance



low dopamine

↓

Parkinson's
Restless leg syndrome

↓

Treat with: dopamine agonist

high dopamine

↓

Morgellon's/Delusional parasitosis

↓

Treat with: dopamine antagonist (antipsychotic)

First step in management?

- Discontinue contributing medications

- Adderall (replace w/)
  - methylphenidate
  - modafinil
  - Vyvanse
- pramipexole, ropinirole (replace w/)
  - pregabalin
  - gabapentin
- methamphetamine
  - If patient spontaneously offers that they take this, encourage reduction/cessation

Second step in management

- Assess for secondary dermatologic or systemic abnormalities

- Evidence of atopy/primary skin rash/prurigo nodules
- Peripheral eosinophil level
- Iron status

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RESEARCH LETTER

Iron deficiency in Morgellons disease: unveiling the link

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Introduction

Iron deficiency (ID) is the most common nutritional deficiency with prevalence averaging 8–15% [1]. ID can be associated with pruritus, dopamine regulation, and psychotic spectrum disorders [2]. One case report links ID to a case of delusional infestation (DI) that resolved with iron supplementation [3]. Morgellons disease (MD), presumably the same condition as DI, is a psychocutaneous disorder characterized by cutaneous paresthesias and visualization of dermal fibers. Removal of foreign material from the skin is associated with excoriations. This study aims to characterize the prevalence of ID in a population with MD.

Methods

Results

Seventy-three MD patients were included in the study (55 female, 18 male) with a mean age of 53.8 years. Thirty-two (43%) patients had a history of amphetamine use, 29 (39%) had a history of opioid use, and 28 (38%) had a diagnosis of ADHD (Table 1). Of 52 MD patients tested for ferritin within the last year, 20 (38%) had iron deficiency and 9 (17%) had iron insufficiency (IS), indicated by serum ferritin levels below 20 ng/mL or 50 ng/mL, respectively. Substance use appeared to be overrepresented in younger MD patients, and ID appeared to be overrepresented in female MD patients, though these findings were not statistically significant. In a multivariable model including age, gender, amphetamine use, opioid use, and ADHD, only age was inversely associated with ID/IS ($p=0.003$), Table 2).

SKIN

BRIEF ARTICLE

Successful Treatment of Delusional Infestation with Dupilumab: Case Report

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ABSTRACT

Delusional infestation (DI) is a psychodermatologic disorder marked by fixed, false belief of parasitic infestation, often accompanied by significant distress. The pathogenesis remains incompletely understood. Currently, the standard of care is antipsychotic medication. We report the case of a 71-year-old man with Parkinson's disease and a four-year history of treatment-resistant DI, who experienced complete symptom resolution following the initiation of dupilumab. The patient presented with sensations of worms, bugs, and fibers invading his body, severely impacting his quality of life. Skin findings were absent, and blood work revealed persistent peripheral eosinophilia. Multiple treatments had been trialed, including antiparasitics, antihistamines, topical steroids, antifungals, and immunosuppressants, which were unsuccessful or discontinued due to side effects. Following the initiation of dupilumab, the patient reported near-complete relief from DI symptoms. This case demonstrates dupilumab's potential to act as a novel treatment for DI. Dupixent blocks interleukins 4 and 13, key contributors for Th2 inflammatory pathways, which may indirectly reduce delusional ideation by resolving symptoms of formication and itch. To our knowledge, this is among the first reported cases to describe complete symptom resolution of DI with dupilumab. These findings suggest that immunomodulatory therapy may be an effective treatment in managing select cases of DI, especially in the presence of peripheral eosinophilia. Further research is warranted to explore the neuroimmunologic underpinnings of DI and expand therapeutic options for this challenging condition.

Therapeutic ladder

- gabapentin 300-600mg tid – easiest to prescribe
- pregabalin 75-150mg bid – more potent than gabapentin
- duloxetine 60-90mg daily
- naltrexone 50-100mg qhs – nonsedating

- pimozide 1-3mg qhs – most acceptable to patients, as not FDA approved for psychosis and therefore not as stigmatizing
- risperidone 2-6mg qhs

Cardiac arrhythmias? Sudden death?

- Pimozide's black box warning regarding QT prolongation, ventricular arrythmias, and sudden death can be intimidating

- The background for this black box warning is that pimozide was once studied for schizophrenia and 2 sudden cardiac deaths occurred in patients who were rapidly titrated up to 70 to 80mg daily in less than 2 weeks

Cardiac arrhythmias? Sudden death?

- Controversy exists as to the need for ECG monitoring with dosages of <10 mg/day.

- Shatzberg et al. recommends that if dosages above the FDA's upper limit of 10 mg/day are to be used, an ECG should be obtained after every dosage increase, implying that dosages <10 mg/day may not require ECG checks.

- QTc interval >500 milliseconds would be cause for concern.

Stage 1

`I feel crawling, biting and stinging sensations on my skin but it's probably not a parasite because I haven't seen any on my skin'	• Requires general pruritus work up • Treat for pruritus, if appropriate • Patients still may respond to pimozide despite having no ideation regarding parasites or foreign bodies • Can talk to these patients normally

Stage 2

'I know this sounds crazy and sorry for mentioning this but I worry that my crawling and biting sensations are due to parasites'	• Can openly discuss several diagnostic possibilities • Can even warn the patient about the possibility of becoming delusional • Can introduce oral medication even on the first visit

Stage 3

'I am very worried about parasites but ultimately I really don't care if I have parasites or not, as long as we can get rid of this problem'	• Requires good rapport before risking asking the critical question 'how important is it for you that this problem is caused by something alive?' (helps to better categorize the patient) • If the patient proves not fully delusional, then the rest of the discussion regarding treatment can be a normal conversation

Stage 4

'I am infested with parasites and I am so miserable so I am willing to try this medication to try to get out of the present misery even though you tell me that nobody really knows how this medication works for my condition'	• Requires strong rapport to treat patient • Solely for practicality, recommend using eponym 'Morgellons disease', not 'delusions of parasitosis' so as not to offend the patient • Offer oral medication as 'trial and error approach' • Emphasize their misery and de-emphasize etiology to help encourage oral therapy • If the patient is still fixated on etiology and not ready to try oral medications, encourage the patients to see other specialists such as parasitologists, entomologists, infectious disease, etc. but still offer future assistance

Stage 5 (Terminal)

• The only thing these patients want is validation about their parasitic beliefs • As soon as they sense that the providers are not going to agree with them, the patient may storm out of the room • Very difficult to treat • Have seen multiple providers already	'The only thing I care about is that you confirm for me that my symptoms are caused by parasites'	• Requires short, frequent follow up visits to show support (short visits also help the provider to avoid burnout) • Ok to offer patient referral to parasitologist, entomologist etc. • May not be possible to introduce oral medication but a supportive/positive rapport may still be beneficial • Consider interdisciplinary care with a psychiatrist but this may be very difficult

Our goals differ from the patient

Goals	Physician	Patient
	Antipsychotic = cure	“It’s not in my head”
		“I’ll prove it. Look at these samples.”
		Validation
		Extensive testing
		Antimicrobials; “please kill it”

- How to reconcile these goals?
  - Compassion/supportive listening
  - Avoiding direct confrontation
  - Accepting samples and photos “show and tell”
  - Labs (ferritin, CBC with diff, consider additional at your discretion. Look for prior urine drug screen)
    - I generally try to talk patients out of biopsies as they are not helpful
  - Antimicrobials
    - “Ivermectin is a very powerful medication and should cure you after one dose if you have a parasite”
    - Tetracyclines
  - Tactfully offer antipsychotic when the time is right

Antipsychotic timing and framing

- If patient presents as mild, ie no agitation, no trust issues, you may be able to offer pimozide on the first visit.
- If patient comes in with a lot of agitation, mistrust, opposition to stigma around neuro/psych meds, it will take longer to earn their trust, if ever

- “Pimozide is a medication that in the US is only FDA approved for a neurologic condition called Tourette’s syndrome. It has also been used in other countries for schizophrenia. You do not have these conditions but we use this medication in much lower doses in dermatology for skin conditions.”
- Many medications have several uses
  - Minoxidil – hypertension → hair growth
  - Spironolactone – heart failure → acne
  - Pimozide – schizophrenia → cancer, antimicrobial

Antipsychotic timing and framing

- “Please don’t waste my time. Does this medication kill parasites or not?”

Can pimozide kill parasites? Surprisingly, the most honest answer is 'yes'

Georgia Marquez-Grap [1], Allison Kranyak [1], Nicholas Brownstone [2], John Koo [1]

Affiliations + expand

PMID: 39988333 DOI: 10.1080/09546634.2025.2466635

Free article

Abstract

Purpose: One of the most well-known medications for treating delusional infestation (DI) is pimozide. Many patients may be reluctant to initiate treatment unless a medication has anti-pathogenic properties, as they feel otherwise it does not address their concerns regarding infestation. In this article, we explore the evidence that pimozide has a range of antipathogenic effects and how this fact can aid in patient care.

Materials and methods: A scoping literature review was performed using The National Library of Medicine (PubMed). The search terms used were pimozide AND anti-microbial OR anti-bacterial OR anti-infective. All relevant articles were reviewed up to September 2024.

Results: Our findings show that pimozide has antibacterial and antiparasitic effects through several unique mechanisms. Additionally, several older first-generation antipsychotics also have demonstrated anti-pathogenic properties. While the studies identified are entirely *in vitro*, the potential antipathogenic effects of pimozide may be pivotal to patients with DI as they make the critical decision to accept or reject treatment.

Conclusion: With adequate disclaimers that pimozide's therapeutic efficacy may not have to do with its anti-pathogen profile, the evidence that pimozide has anti-pathogenic properties may enable dermatology providers to strengthen their therapeutic approach and alliance with patients with DI and make life-changing therapy more acceptable to the patient.

› mSphere. 2016 Mar 2;1(2):e00042-15. doi: 10.1128/mSphere.00042-15. eCollection 2016 Mar-Apr.

Drug Repurposing Screening Identifies Novel Compounds That Effectively Inhibit Toxoplasma gondii Growth

Ashley J Dittmar [1], Allison A Drozda [1], Ira J Blader [1]

Affiliations + expand

PMID: 27303726 PMCID: PMC4894684 DOI: 10.1128/mSphere.00042-15

Abstract

The urgent need to develop new antimicrobial therapies has spawned the development of repurposing screens in which well-studied drugs and other types of compounds are tested for potential off-label uses. As a proof-of-principle screen to identify compounds effective against Toxoplasma gondii, we screened a collection of 1,120 compounds for the ability to significantly reduce Toxoplasma replication. A total of 94 compounds blocked parasite replication with 50% inhibitory concentrations of <5 μM. A significant number of these compounds are established inhibitors of dopamine or estrogen signaling. Follow-up experiments with the dopamine receptor inhibitor pimozide revealed that the drug impacted both parasite invasion and replication but did so independently of inhibition of dopamine or other neurotransmitter receptor signaling. Tamoxifen, which is an established inhibitor of the estrogen receptor, also reduced parasite invasion and replication. Even though Toxoplasma can activate the estrogen receptor, tamoxifen inhibits parasite growth independently of this transcription factor. Tamoxifen is also a potent inducer of autophagy.

Dopaminergic inhibitors inhibit *Toxoplasma* growth. A significant number of compounds that target dopaminergic signaling were identified in our screen, which was intriguing since *Toxoplasma* has been proposed to alter host dopaminergic signaling for growth (15, 16). Therefore, we examined two different classes of these inhibitors, 3-CPMT, a dopamine reuptake inhibitor (IC_{50} = 2.1 μM), and pimozide (IC_{50} = 1.8 μM), a dopamine D2 receptor antagonist. *Toxoplasma* has been proposed to use dopamine for growth (17), and we hypothesized that 3-CPMT affects parasite growth by reducing host cell dopamine levels by blocking its reuptake from the extracellular milieu. Thus, we tested whether addition of 1 μM dopamine (>30× the K_i of 3-CPMT for the dopamine transporter [18]) enhanced parasite growth and found that the neurotransmitter neither increased the IC_{50} of 3-CPMT nor significantly increased parasite growth on its own (Fig. 2A). We next tested whether exogenous dopamine could reverse pimozide inhibition of parasite growth due to the drug's inhibition of dopamine receptor signaling. Similar to its effect on 3-CPMT, 1 μM dopamine (~500 times its IC_{50} for dopamine receptor signaling [19]) did not affect the ability of pimozide to impact parasite replication (Fig. 2B). Pimozide is also reported to inhibit serotonergic, histaminergic, and noradrenergic signaling (20, 21). Addition of serotonin, histamine, norepinephrine, or epinephrine did not affect the sensitivity of *Toxoplasma* to pimozide (Fig. 2C). Together, these data indicate that off-target effects of 3-CPMT and pimozide are the basis of their inhibition of *Toxoplasma*. Since pimozide has a lower IC_{50} than 3-CPMT, the remaining experiments were performed only with pimozide.

Pimozide inhibits *Toxoplasma* invasion and replication. *Toxoplasma* replicates via a lytic cycle composed of repeated rounds of invasion, replication, and egress (22). *Toxoplasma* invasion is a highly coordinated process in which invasion is started by parasites attaching to the host cell through a low-affinity interaction between unidentified parasite and host factors. An unknown trigger then induces the calcium-dependent release of micronemal proteins that act as adhesins that form an intimate attachment between the parasite and the host cell. Finally, the parasite traverses the surface of the host cell until it begins to penetrate the host cell while simultaneously forming the nascent parasitophorous vacuole (PV) (23). To test whether pimozide affected parasite invasion, host cells were pretreated with pimozide or the vehicle control for 60 min and then RH–β-Gal–green fluorescent protein (GFP) parasites were added in the presence of pimozide or the vehicle control, respectively. After 60 min, the cells were fixed but not permeabilized and then stained with anti-SAG1 antiserum to discriminate between intracellular (GFP^+ $SAG1^-$) and extracellular (GFP^+ $SAG1^+$) parasites. We found that pimozide significantly reduced the number of intracellular parasites by ~50% (Fig. 3A). Pimozide had no apparent effect on the ability of ethanol to induce calcium-dependent secretion of the MIC2 micronemal protein (24) or on the ratio of intracellular to extracellular parasites (Fig. 3B and C), indicating that the drug did not affect the steps involved in intimate attachment or host cell penetration. In contrast, pimozide reduced the total number of parasites associated with the host cell by ~50% (Fig. 3D), indicating that the drug affected the initial step in parasite invasion, which is the loose association of the parasite with the host plasma membrane.

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Dopamine signaling drives skin invasion by human-infective nematodes

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Abstract

Skin-penetrating nematodes are one of the most prevalent causes of disease worldwide. The World Health Organization has targeted these parasites for elimination by 2030, but the lack of preventative measures is a major obstacle to this goal. Infective larvae enter hosts through skin and blocking skin penetration could prevent infection. However, in order to prevent worm ingress via the skin, an understanding of the behavioral and neural mechanisms that drive skin penetration is required. Here, we describe the skin-penetration behavior of the human-infective threadworm *Strongyloides stercoralis*. We show that *S. stercoralis* engages in repeated cycles of pushing, puncturing, and crawling on the skin surface before penetrating. Pharmacological inhibition of dopamine signaling inhibits these behaviors in *S. stercoralis* and the human hookworm *Ancylostoma ceylanicum*, suggesting a critical role for dopamine signaling in driving skin penetration across distantly related nematodes. CRISPR-mediated disruption of dopamine biosynthesis and chemogenetic silencing of dopaminergic neurons also inhibit skin penetration. Finally, inactivation of the TRPN channel TRP-4, which is expressed in the dopaminergic neurons, blocks skin penetration. Our results suggest that drugs targeting TRP-4 and other nematode-specific components of the dopaminergic pathway could be developed into topical prophylactics that block skin penetration, thereby preventing infections.

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