Pruritus: Diagnosis and Management

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Pruritus is the sensation of itching; it can be caused by dermatologic and systemic conditions. An exposure history may reveal symptom triggers. A thorough skin examination, including visualization of the finger webs, anogenital region, nails, and scalp, is essential. Primary skin lesions indicate diseased skin, and secondary lesions are reactive and result from skin manipulation, such as scratching. An initial evaluation for systemic causes may include a complete blood count with differential, creatinine and blood urea nitrogen levels, liver function tests, iron studies, fasting glucose or A1C level, and a thyroid-stimulating hormone test. Additional testing, including erythrocyte sedimentation rate, HIV screening, hepatitis serologies, and chest radiography, may also be appropriate based on the history and physical examination. In the absence of primary skin lesions, physicians should consider evaluation for malignancy in older patients with chronic generalized pruritus. General management includes trigger avoidance, liberal emollient use, limiting water exposure, and administration of oral antihistamines and topical corticosteroids. If the evaluation for multiple etiologies of pruritus is ambiguous, clinicians may consider psychogenic etiologies and consultation with a specialist. (*Am Fam Physician*. 2022;105(1):55-64. Copyright © 2022 American Academy of Family Physicians.)

Pruritus is the sensation of itching. Although large-scale epidemiologic data on prevalence are limited, pruritus is a common symptom encountered in primary care.[1,2] The etiology of pruritus is complex and can include histamine, serotonin, and neuropeptide release, and neuronal itch signal transmission.[1] Risk factors include older age, known or new dermatologic disease, and systemic conditions, such as renal and hepatic disease.[1] When inadequately treated, pruritus can adversely affect a patient's quality of life by altering mood, stress levels, and sleep.[3]

Differential Diagnosis

The differential diagnosis of pruritus is broad and includes acute and chronic (i.e., at least six weeks of symptoms) presentations.[1,2] Primary and secondary skin lesions suggest dermatologic etiologies of pruritus *(Table 1[4])*. When distinct exposures result in symptoms, the underlying etiology is often discovered. However, the absence of obvious triggers or examination findings coupled with chronic symptoms makes identifying the underlying etiology of pruritus more challenging.

The International Forum for the Study of Itch has proposed a formal classification system for chronic pruritus with three different clinical classes: pruritus on diseased skin (Group I), pruritus on nondiseased skin (Group II), and chronic reactive lesions acquired from skin manipulation, such as rubbing, picking, or scratching (Group III).[5] Group I presentations suggest a dermatologic etiology. Group II presentations suggest systemic, neurogenic, or psychogenic etiologies. Group III presentations may result from any one of the previously mentioned etiologies or a mixed presentation. The classification also includes chronic pruritus of unknown origin, for which there are no known effective interventions.[6] Pathognomonic skin findings and the extent of bodily involvement can also suggest certain diagnoses *(Figure 1[7])*.

CME This clinical content conforms to AAFP criteria for CME. See CME Quiz on page 15.

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Patient information: A handout on this topic, written by the authors of this article, is available at https://www.aafp.org/afp/2022/0100/p55-s1.html.

Common Dermatologic Etiologies of Pruritus

ATOPIC DERMATITIS

Atopic dermatitis is an inflammatory skin condition that is often associated with secondary lesions that result from scratching or other skin manipulation findings, such as excoriations, lichenification, and hyperpigmented or erythematous papules or plaques *(Figure 2)*. Patients scratch

TABLE 1

Dermatologic Etiologies of Pruritus

Etiology	Features
Allergic or irritant contact dermatitis	Bullae, vesicles, erythema, and edema localized to area in contact with exposure Reaction within days of exposure
Atopic dermatitis	Erythematous papules, patches, or plaques; pruritic area where rash appears when scratched in patients with atopic conditions (e.g., allergic rhinitis, asthma) Involvement of crease areas (axillae, wrists, ankles, popliteal and antecubital fossae) Chronic, worsens with itch-scratch cycle
Bullous pemphigoid	Initially pruritic urticarial lesions, often in intertriginous areas Formation of tense blisters
Cutaneous T-cell lymphoma (mycosis fungoides)	Oval eczematous patch on skin with no sun exposure (e.g., buttocks) Possible presentation of new eczematous dermatitis in older adults Possible presentation of erythroderma
Dermatitis herpetiformis	Rare vesicular dermatitis affecting the lumbosacral spine, elbows, or knees
Dermatophyte infection	Can occur on several sites, including the feet, trunk, groin, scalp, and nails Localized pruritus and rash characterized by peripheral scaling and central clearing Patchy alopecia on scalp Dystrophic or discolored nails
Folliculitis	Pruritus out of proportion to appearance of dermatitis Papules and pustules at follicular sites on chest, back, or thigh
Lichen planus	Lesions often located on the flexor wrists Characterized by the six P's (pruritus, polygonal, planar, purple, papules, plaques)
Lichen simplex chronicus	Localized, intense pruritus Initial erythematous, well-defined plaques with excoriations lead to thickened, lichenified, violaceous patches if scratching continues
Pediculosis (lice infestation)	Adult organisms and nits on hair shafts Occiput in school-aged children; genitalia in adults (sexually transmitted)
Psoriasis	Plaques on extensor extremities, low back, palms, soles, and scalp
Scabies	Burrows, vesicles, and papules in finger web spaces and axillae and on wrists, ankles, genitals, and extensor surfaces Pruritus worse at night Can persist after mite eradication
Sunburn	Possible photosensitizing cause (e.g., with use of nonsteroidal anti-inflammatory drugs or cosmetics)
Urticaria (hives)	Intensely pruritic, well-circumscribed, erythematous, and elevated wheals Lesions are transient, may coalesce, and wax and wane over several hours
Xerosis	Intense pruritus, often during winter months in northern climates Involvement of back, flank, abdomen, waist, and lower extremities More common in older people

Adapted with permission from Reamy BV, Bunt CW, Fletcher S. A diagnostic approach to pruritus. Am Fam Physician. *2011;84(2):198.*

pruritic areas and subsequently develop secondary lesions, worsening dermatitis and associated pruritus; this process is often called the itch-scratch cycle. Flexural areas are commonly affected, including ankles, regions behind the ears, and the antecubital and popliteal fossae. Patients with this condition often have a personal or family history of asthma or allergic rhinitis, and symptoms commonly begin during childhood. Treatment includes limiting exposure to water, liberal emollient use, and topical corticosteroid application.[8,9]

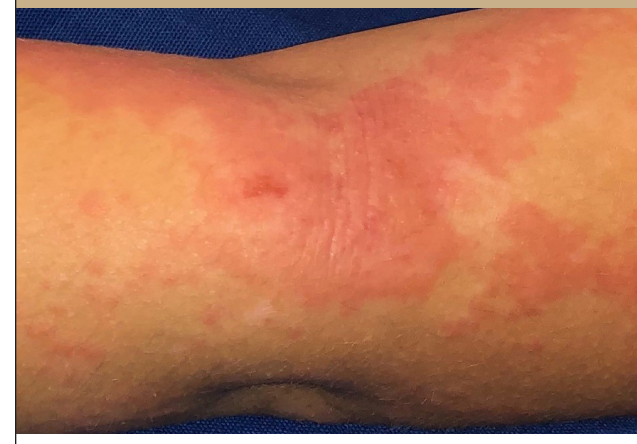
CONTACT DERMATITIS

Contact dermatitis is an inflammatory reaction that typically erupts within days of direct skin contact with an environmental trigger. Associated primary skin findings include bullae, vesicles, erythema, and edema, localized to areas that directly contacted the trigger. Irritant and allergic mechanisms are common. Irritant contact dermatitis is not immunologically mediated and progressively compromises the physical and chemical composition of the epidermis. In contrast, allergic contact dermatitis is dependent on a delayed hypersensitivity reaction and is more common in people with atopy. Contact dermatitis treatment includes avoidance of triggers, such as rough textiles, detergents, perfumes, chemicals, and dyes,[1,4,10] and topical corticosteroid application.[11]

DERMATOPHYTOSIS

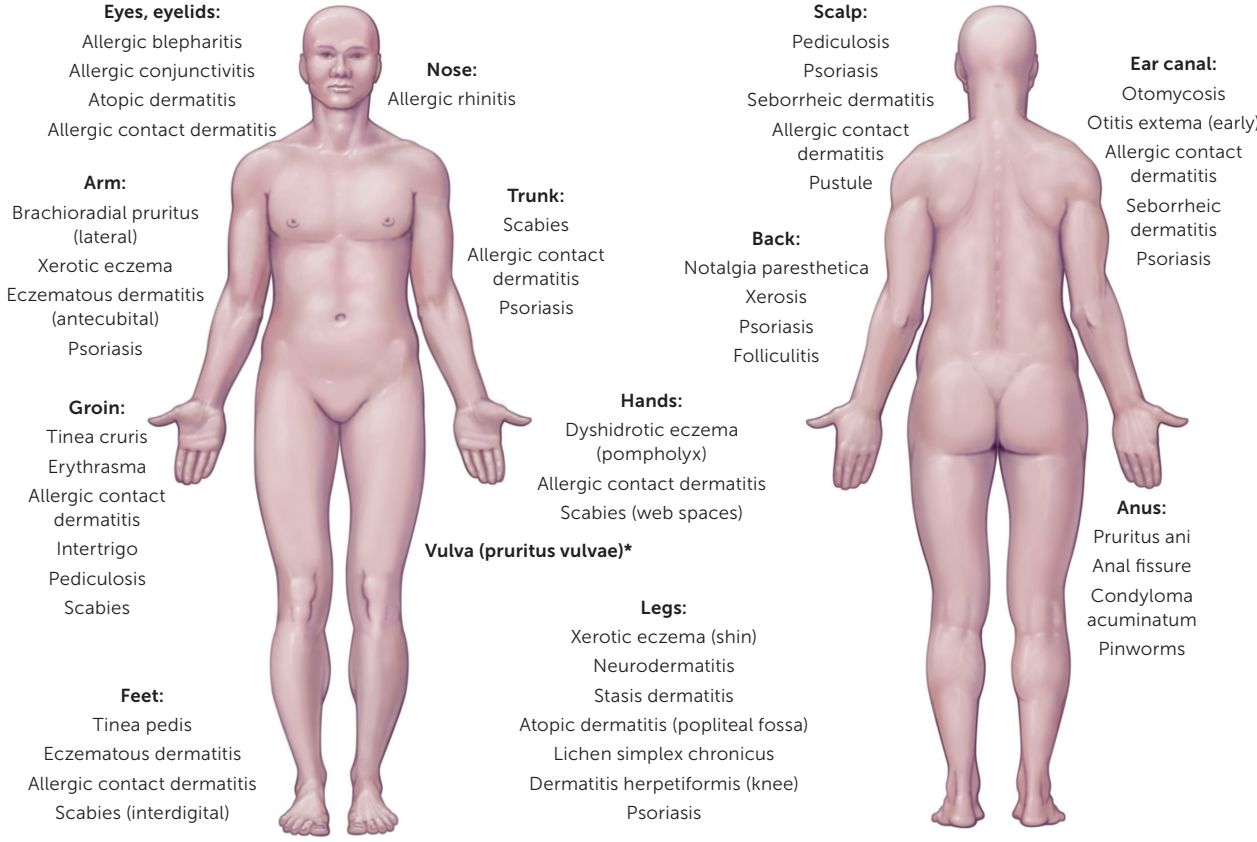
Tinea infections are caused by fungi that can survive only on dead keratin, including the most superficial layer of the epidermis (i.e., stratum corneum), hair, and nails. The characteristic ringworm rash appears as a well-demarcated region with a red, scaly, elevated border containing the highest concentration of fungal hyphae. Tinea pedis more often affects toe webs and soles of the feet, and the skin can become dry, scaly, fissured, or soft and macerated. Tinea capitis may present with patchy alopecia and scaling of the scalp. Onychomycosis is associated with dystrophic, discolored nails. Direct visualization of fungal hyphae on scale scrapings, hair shafts, and nail clippings prepared with

FIGURE 2



Urticaria superimposed on lichenification from atopic dermatitis.

FIGURE 1



*—Causes of pruritus vulvae: in patients who are prepubertal – poor hygiene, streptococcal infection, *Escherichia coli* infection, pinworms, scabies, allergic contact dermatitis; in patients who are postmenarchal – vaginitis, allergic contact dermatitis, hidradenitis suppurativa, lichen simplex chronicus; in patients who are postmenopausal – atrophic vaginitis, lichen sclerosus, vulvar cancer, Paget disease of bone; in patients with diabetes mellitus – candidiasis, other dermatophyte infections.

Drawings showing causes of pruritus by location.

Illustration by Scott Bodell

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potassium hydroxide solution aids in the diagnosis. Treatment includes topical and oral antifungals.[12]

INFESTATIONS AND INSECT BITES

Common infestations that cause pruritus include scabies and lice. Low socioeconomic status, poor hygiene, and overcrowding can be risk factors.[13,14] The patient's history may reveal other contacts with similar signs and symptoms.

Lice infestations often present on the scalp, pubic area, and body. Head lice commonly present in children, and pubic lice are sexually transmitted. Body lice live in the seams and folds of clothing. Physical examination findings associated with lice include direct visualization of adult organisms and their nits on hair shafts.[13]

Patients with scabies infestations typically report pruritus that worsens at night and can persist despite mite eradication. Skin findings common to scabies include burrows, vesicles, and papules in finger web spaces and axillae and on wrists, ankles, genitals, and extensor surfaces. Secondary skin lesions may be caused by scratching. Microscopic examination of skin scrapings may reveal scabies, mites, eggs, and feces.[13]

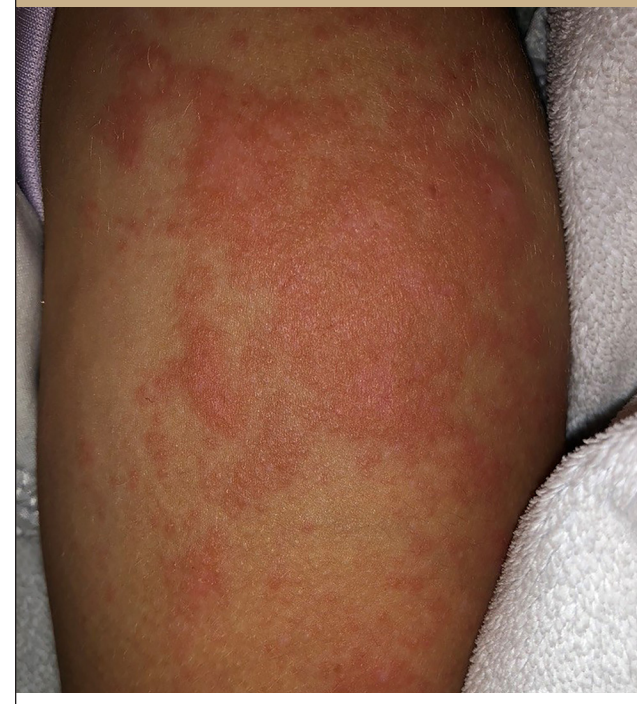
Insects commonly known to cause pruritic bites include mosquitoes, fire ants (Solenopsis), bed bugs (Cimex lectularius), gnats (Sciaridae), fleas (Siphonaptera), and chiggers (trombiculids). These insects injure the skin through stabbing mechanisms that expose the affected skin to the insect's saliva. An individual's allergic response to the injury and insect saliva determines the severity of the localized reaction. Patients with sensitive skin can quickly develop localized hypersensitivity reactions, including pruritic, erythematous urticarial papules and plaques.[13]

Treatment for infestations and insect bites is dependent on the causative species. For scabies and lice, permethrin is a first-line antiparasitic. *N,N*-diethyl-*m*-toluamide, also known as DEET, is the most effective insect repellent against biting insects. Treatment for insect bites includes symptom management with cool compresses, oral antihistamines, and topical corticosteroids.[13]

PSORIASIS

Psoriasis is a common chronic inflammatory skin disease that occurs because of a complex immune-mediated process that causes hyperproliferation of the skin, leading to scale and plaque formation. Psoriasis often develops at sites of skin trauma (Koebner phenomenon) and classically demonstrates pinpoint bleeding after removal of the scale overlying the plaque (Auspitz sign). Topical corticosteroids are the primary therapy for localized plaques; however, psoriatic pruritus can extend beyond the plaques. Liberal use of topical emollients can maintain skin moisture and suppleness, minimizing pruritus and subsequent trauma from scratching, ideally preventing the formation of new plaques.[15]

FIGURE 3



Urticaria following ingestion of a cashew.

URTICARIA

Urticarial lesions (i.e., hives) are typically pruritic plaques with pale, centrally edematous wheals surrounded by erythematous flare regions *(Figure 3)*. The lesions evolve or wax and wane over hours to days, are transient, change size and shape, and persist for less than 24 hours. Immunologic, autoimmune, and mechanical etiologies are possible. Examples include serum sickness and dermographism. Treatment of urticaria includes oral antihistamines.[16]

XEROSIS

Xerosis is dry, scaly skin that is more likely to occur in older adults because of excessive washing of the skin or during winter when homes are heated with relatively low humidity. Treatment of the associated pruritus should include using mild skin cleansers, avoiding excessive washing and abrasive scrubbing of the skin, applying skin moisturizers daily, and using a humidifier in the home.[17]

Common Systemic Etiologies of Pruritus

Systemic etiologies of pruritus should be considered in the absence of symptoms or findings suggestive of dermatologic disease *(Table 2)*.[4] Malignancy, including cervical, prostate, and colon cancers, can cause chronic generalized pruritus.[1] Chronic conditions, including renal and

TABLE 2

Systemic Etiologies of Pruritus

Autoimmune
- Dermatitis herpetiformis
- Dermatomyositis
- Linear immunoglobulin A disease
- Sjögren syndrome

Hematologic
- Hemochromatosis
- Iron deficiency anemia
- Mastocytosis
- Plasma cell dyscrasias
- Polycythemia vera

Hepatobiliary
- Biliary cirrhosis
- Cholestasis
- Chronic pancreatitis with obstruction of biliary tracts
- Hepatitis (particularly hepatitis C)
- Sclerosing cholangitis

Infectious disease
- Herpes zoster
- HIV/AIDS
- Human parvovirus B19
- Parasitic disease (ascariasis, giardiasis, onchocerciasis, schistosomiasis)
- Prion disease

Malignancy
- Cerebral tumor
- Leukemia
- Lymphoma
- Multiple myeloma
- Solid tumors with paraneoplastic syndrome

Metabolic and endocrine
- Carcinoid syndrome
- Chronic renal disease
- Diabetes mellitus
- Hyperparathyroidism
- Thyroid disorders

Neurologic
- Cerebral abscess
- Multiple sclerosis
- Nostalgia paresthetica
- Peripheral neuropathy
- Stroke

Other
- Drug ingestion or eruption
- Eating disorders with rapid weight loss
- Neuropsychiatric disorders
- Pemphigoid gestationis
- Pruritic urticarial papules and plaques of pregnancy

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hepatic failure, thyroid disease, diabetes mellitus, pregnancy, and multiple sclerosis, can also precipitate diffuse pruritus without concurrent skin disease. Psychogenic etiologies are diagnoses of exclusion; however, a history of emotional stress or chronic or transient psychiatric conditions may increase suspicion for pruritus.[10,18]

Clinical Evaluation

A detailed history helps build the differential diagnosis for pruritus *(Table 3)*.[4] An acute episode is less than six weeks, and a chronic presentation is six weeks or longer in duration.[5] Physicians should inquire about the extent of bodily involvement; the frequency, quality, intensity, duration, and triggers of itching; and alleviating factors. Interview questions should also focus on topical, oral, and airborne exposures, such as detergents, hygiene products, occupational materials, illicit drug use, and medications *(Table 4)*.[19] Exposure duration and frequency may demonstrate other associations from hobbies, travel history, and sick contacts. Personal history or family history of skin disorders may suggest a predisposition to certain skin diseases. Systemic conditions should be suspected when pruritus is not accompanied by any reported or noticeable skin changes. A history of emotional stress and chronic psychiatric conditions increases consideration of a psychogenic etiology. Systemic symptoms, such as fever, fatigue, unintentional weight loss, and night sweats, are

TABLE 3

Historical Findings That Suggest Etiologies of Pruritus

Historical finding	Possible etiologies
Hobby or occupational exposure to solvents, adhesives, cleaners	Irritant contact dermatitis, xerosis, atopic dermatitis, urticaria
Malaise, nausea, decreased urine output	Renal failure with generalized pruritus
New animal exposures	Flea infestation, allergic contact dermatitis, urticaria, dermatophytosis
New medications, supplements, or illicit drugs	Urticaria, drug eruptions
New skin or hair products (e.g., cosmetics, creams, soaps, detergents)	Allergic contact dermatitis, urticaria, photodermatitis
Personal or family history of atopic dermatitis, allergic rhinitis, or asthma; childhood onset; or prolonged water exposure	Atopic dermatitis
Recent travel	Pediculosis, scabies infestation, photodermatitis, urticaria
Sick contacts, especially people with febrile diseases and rashes	Rubeola, mumps, varicella, scarlet fever, cellulitis, fifth disease, folliculitis
Unexplained weight changes, menstrual irregularity, heat or cold intolerance	Thyroid disease with or without secondary urticaria or xerosis cutis
Unexplained weight loss, night sweats, unexplained fevers, fatigue	Malignancy, to include lymphoma with secondary generalized pruritus

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TABLE 4

Medications That May Cause Pruritus

Group of drugs	Examples	Possible mechanism	Frequency
Antiarrhythmic drugs	Amiodarone	Cholestatic liver injury	Case reports
Antibiotics and chemotherapeutics	Antimalarials	Unknown, but release of histamine or activation of μ-receptors was postulated	Case reports
	Carbapenems	Cholestatic liver injury	Rare
	Cephalosporins	Unknown or secondary to skin lesions	< 2%
	Lincosamides	Secondary to skin lesions or cholestatic liver injury	Rare
	Macrolides	Secondary to skin lesions or cholestatic liver injury	< 0.3%
	Metronidazole (Flagyl)	Unknown or secondary to skin lesions	< 5%
	Monobactams	Secondary to skin lesions	Rare
	Penicillins	Secondary to skin lesions or cholestatic liver injury	2% to 20%
	Quinolones	Unknown or secondary to skin lesions	1% to 4%
	Rifampin	Unknown	Case report
	Streptogramins	Secondary to skin lesions	2.5%
	Tetracyclines	Unknown or cholestatic liver injury	1% to 2%
	Trimethoprim/sulfamethoxazole	Secondary to skin lesions	2% to 10%
		Cholestatic liver injury	Rare
Anticoagulants	Fractionated heparins	Urticarial reaction	Case reports
	Ticlopidine	Cholestatic liver injury	Case reports
Antidiabetic drugs	Biguanides	Cholestatic liver injury	Case reports
	Sulfonylurea derivatives	Unknown	< 5%
Antiepileptics	Carbamazepine (Tegretol), fosphenytoin (Cerebyx), oxcarbazepine (Trileptal), phenytoin (Dilantin), topiramate (Topamax)	Secondary to skin lesions, allergic reaction	Rare
Antihypertensive drugs	Angiotensin-converting enzyme inhibitors	Increase of bradykinin level or cholestatic liver injury or secondary to skin lesions	1% to 15%
	Angiotensin-II inhibitors (sartans)	Cholestatic liver injury	Case reports
	Beta-adrenergic blockers	Secondary to skin lesions	Frequent, if administered transdermally
		Cholestatic liver injury	Rare
	Calcium channel blockers	Secondary to skin lesions or unknown	< 2%
		Cholestatic liver injury	Case reports
	Methyldopa	Unknown or secondary to skin lesions	< 2%
	Sildenafil (Viagra)	Cholestatic liver injury	Case report
Cytokines, growth factors, and monoclonal antibodies	Granulocyte-macrophage colony-stimulating factor	Unknown	Common
	Interleukin-2	Direct pruritogenic effect of interleukin-2	Very common
	Lapatinib (Tykerb)	Unknown or urticarial reaction	3%
	Matuzumab (Leukeran)	Unknown	< 10%

continues

concerning for malignancy, particularly in older patients with chronic generalized pruritus without an obvious exposure association.[4]

The physical examination should include a complete dermatologic assessment, particularly for diffuse and chronic pruritus. Inspection of finger webs, the anogenital region, nails, and the scalp is suggested.[1] During the assessment of skin lesions, primary lesions (i.e., present before the onset of pruritus) should be differentiated from reactive lesions (i.e., secondary to skin manipulation, including scratching,

TABLE 4 *(continued)*

Medications That May Cause Pruritus

Group of drugs	Examples	Possible mechanism	Frequency
Cytostatics or chemotherapeutics	Chlorambucil	Secondary to skin lesions	Case reports
	Paclitaxel (Taxol)	Unknown or secondary to skin lesions	10% to 14%
	Tamoxifen	Sebostasis or xerosis	3% to 5%
Hypolipidemic drugs	Statins	Unknown or secondary to skin lesions	16%
Plasma volume expanders	Hydroxyethyl starch	Deposition of hydroxyethyl starch in small peripheral nerves or in Schwann cells of cutaneous nerves	12.6% to 54%
Psychotropic drugs	Neuroleptics	Cholestatic liver injury	Rare
	Selective serotonin reuptake inhibitors	Activation of peripheral serotonin receptors or secondary to skin lesions	Rare
	Tricyclic antidepressants	Cholestatic liver injury	Rare
Others	Antithyroid agents	Cholestatic liver injury	Rare
	Corticosteroids	Cholestatic liver injury	Very rare
	Inhibitors of xanthine oxidase	Secondary to skin lesions	0.8% to 2.1%
	Nonsteroidal anti-inflammatory drugs	Increased synthesis of leukotrienes	1% to 7%
		Cholestatic liver injury	Rare
	Opioids	Centrally mediated process via μ-opioid receptor	2% to 100%
	Sex hormones	Cholestatic liver injury	Rare

Adapted with permission from Reich A, Ständer S, Szepietowski JC. Drug-induced pruritus: a review. Acta Derm Venereol. *2009;89(3):238.*

rubbing, or picking).[5] If systemic disease is suspected, examination for lymphadenopathy and hepatosplenomegaly and additional diagnostic testing should be performed.

Diagnostic Approach

When primary or secondary skin lesions are identified, but the etiology is not apparent, diagnostic testing of lesions should be considered *(Figure 4[4])*. Skin biopsy, scrapings, and cultures may provide additional diagnostic information.[1,2,10]

If an occult exposure or underlying etiology is not apparent on initial evaluation, physicians should consider other systemic causes *(Table 2[4])* by obtaining laboratory and imaging studies. Serologic testing should include a complete blood count with differential and iron studies because iron deficiency anemia is the most common cause of generalized pruritus in patients with underlying systemic disease.[10] These studies can help identify other hematologic or malignant causes of pruritus, including polycythemia vera, hemochromatosis, and Hodgkin lymphoma. Serum creatinine and blood urea nitrogen tests can assess for chronic kidney disease, and liver function tests can evaluate for hepatic and biliary etiologies. Fasting glucose, A1C, and thyroid-stimulating hormone tests can evaluate for diabetes and thyroid disorders.[1,2,4,10]

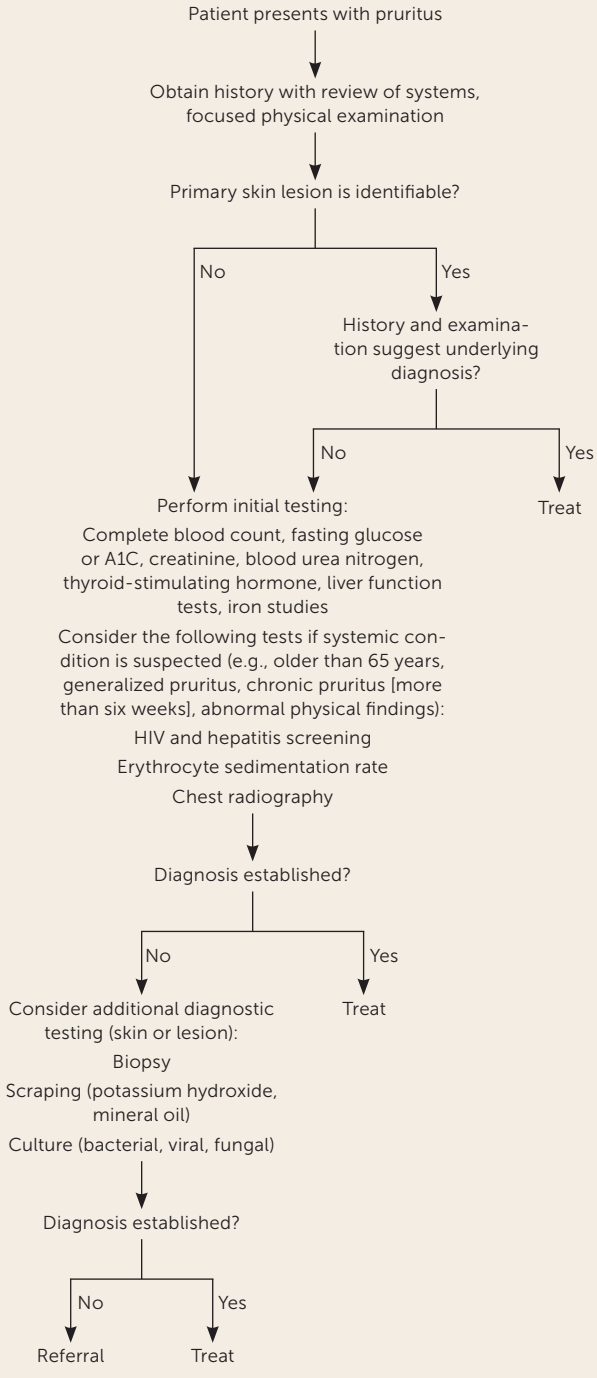
An erythrocyte sedimentation rate and chest radiography are appropriate if a systemic inflammatory condition or malignancy in an older patient is suspected. HIV screening is appropriate for individuals with increased risk based on history and physical examination.[10]

General Management

The determination of underlying etiologies affects treatment selection. Diagnosis and treatment of an underlying condition often resolves the associated pruritus. Although general management principles are provided in European guidelines, randomized controlled trials supporting these measures are lacking, and the recommendations are consensus based *(Table 5[1,20-46])*. Dry skin and prolonged water exposure aggravate pruritus; liberal use of emollients is helpful, particularly following showering and bathing.[1,2,4,47,48] Water exposure from bathing should not exceed a maximum of 20 minutes with lukewarm water, and the use of mild, perfume-free soaps and other hygiene products is recommended.[1] Maintaining cool room temperatures at night can increase patient comfort while sleeping.[1] Cooling wraps are an as-needed application option.[1] Patient education promotes awareness of contact irritants and triggers[1,4,10] *(Table 1[4])*.

Initial specific pharmacotherapy may include oral antihistamines for histamine-associated pruritic disorders or topical corticosteroids for pruritus associated with inflammatory dermatoses.[1,2,37,38,40,41] Lifestyle modifications can include cognitive behavior therapy to address coping mechanisms.[1,49] Frequent follow-up is recommended to assess responses to treatment modalities.

FIGURE 4



Diagnostic algorithm for pruritus.

Adapted with permission from Reamy BV, Bunt CW, Fletcher S. A diagnostic approach to pruritus. Am Fam Physician. *2011;84(2):196.*

TABLE 5

Treatment Options for Pruritus

General	
Keep skin cooler for comfort	Cool room temperatures at night[1] Cool wraps, menthol, camphor, ice[1]
Reduce skin drying effects	Limit bathing to 20 minutes with lukewarm water[1] Limit soap use to oily and intertriginous areas[1]
Medications	
Cholestatic hepatic disease	Cholestyramine (Questran)[20] Selective serotonin reuptake inhibitors[21]
Intrahepatic cholestasis of pregnancy	Ursodeoxycholic acid[22]
Lichen simplex chronicus	Salicylic acid or dichloromethane solution[23]
Liver dysfunction related	Naltrexone (Revia)[24]
Neuropathic pruritus	Gabapentin (Neurontin)[25,26] Pregabalin (Lyrica)[25,27]
Paraneoplastic syndrome	Selective serotonin reuptake inhibitors[28]
Renal dysfunction related	Capsaicin[29] Gabapentin, pregabalin[30-33] Montelukast (Singulair)[34] Selective serotonin reuptake inhibitors[35] Topical anesthetics (1% pramoxine hydrochloride)[36]
Urticaria, allergic or atopic dermatitis	First- or second-generation antihistamines[37,38] Topical naltrexone (1% cream)[39] Topical steroids[40,41]
Nonspecific	
Anti-inflammatory therapies*	Topical calcineurin inhibitors[42] Topical corticosteroids[1,43]
Nerve modulation	Systemic doxepin[44] Topical doxepin†[45]
Phototherapy	Narrowband ultraviolet B and ultraviolet A-1[46]

*—When used nonspecifically for inflammatory dermatoses.
†—Specifically for atopic dermatitis.

Information from references 1 and 20-46.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	Comments
Differentiate lesions as primary to pruritus or secondary (e.g., excoriations, scarring). Primary skin lesions indicate skin disease.[1,5]	C	Expert opinion
Counsel patients with a history of irritant and allergic contact dermatitis to avoid contact irritants and other triggers (e.g., rough textiles, detergents, perfumes, chemicals, dyes).[1,4,10]	C	Professional society guideline based on expert opinion
The physical examination for pruritus should include a complete dermatologic assessment.[1]	C	Professional society guideline based on expert opinion
Consider additional skin testing (e.g., biopsy, scraping, culture) for persistent, unexplained pruritus.[1,2,10]	C	Professional society guideline based on expert opinion
Consider the following serologic studies when pruritus is undifferentiated after initial evaluation: complete blood count, iron studies, renal and hepatic function tests, thyroid-stimulating hormone, and fasting glucose or A1C.[1,2,4,10]	C	Professional society guidelines based on case-control studies and expert opinion
Encourage liberal use of emollients and limiting water exposure to reduce dry skin.[1,47,48]	B	Professional society guideline and multiple RCTs for treatment of atopic dermatitis
Use oral antihistamines and topical corticosteroids for initial symptomatic therapy in patients with pruritus.[1,37,38,40,41]	B	Professional society guideline and several small RCTs
Lifestyle modifications and cognitive behavior therapy can be considered for resistant symptoms of pruritus.[1,49]	B	Professional society guideline based on a Cochrane review of 10 RCTs

RCT = randomized controlled trial.

A = consistent, good-quality patient-oriented evidence; **B** = inconsistent or limited-quality patient-oriented evidence; **C** = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to https://www.aafp.org/afpsort.

Specialist consultation is reasonable if a cause is uncertain after the initial evaluation and additional diagnostic testing or if a patient is symptomatic following appropriate first-line treatment modalities for an underlying condition.

This article updates previous articles on this topic by Reamy, et al.,[4] and Moses.[7]

Data Sources: PubMed and Cochrane databases were searched using key terms including pruritus and itch. The search included randomized controlled trials, meta-analyses, clinical trials, and clinical reviews. An Essential Evidence Plus summary report on this topic was reviewed and utilized to assist in literature review. Search dates: September 2020, October 2020, and August 2021.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the U.S. Air Force or the Department of Defense.

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