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Insect Bite–Induced Hypersensitivity and the SCRATCH Principles: A New Approach to Papular Urticaria

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ABSTRACT

Insect bites and the associated hypersensitivity reactions known as papular urticaria account for a significant number of all referrals from pediatricians and dermatologists to our pediatric dermatology clinic. Unfortunately, children affected by these eruptions are frequently misdiagnosed and often subject to expensive evaluations including invasive and unnecessary procedures. Here we review the course of 4 children with the typical physical findings and natural history of these reactions. On the basis of our clinical findings and experience with this patient population, we propose a set of principles (termed “SCRATCH”) as clinical features to aid clinicians in making an early and accurate clinical diagnosis. We conclude that a more appropriate term for future study and diagnosis of this entity is insect bite–induced hypersensitivity.

HEALTHY ITCHY CHILDREN provide a diagnostic and therapeutic challenge to parents, pediatricians, and pediatric dermatologists. The complaint of itch may prompt a costly, exhaustive, and invasive evaluation. In our pediatric dermatology practice, insect bite–induced hypersensitivity (IBIH) reactions, known as papular urticaria, account for 5% of these patients. Investigators first used the term “papular urticaria” in the 1820s; however, the exact pathophysiology of the disorder continues to be debated. Papular urticaria is defined by chronic or recurrent eruptions of pruritic papules, vesicles, and wheals resulting from a hypersensitivity reaction to biting or stinging insects.¹ The lesions are most commonly grouped in linear clusters and present on exposed areas with sparing of the genital, perianal, and axillary regions. Intense pruritis accompanies the eruption, resulting in excoriations, secondary infection, scarring, and permanent hyperpigmentation and/or hypopigmentation, particularly in darkly pigmented individuals. Although the prevalence of IBIH peaks in children from 2 to 10 years old, it occurs occasionally in adolescents and adults.²

We found that this common clinical entity is frequently not included in the differential diagnosis for pruritic pediatric rashes, suggesting that this may be an unfamiliar and elusive diagnosis for referring primary care providers and dermatologists alike. Early diagnosis allows for appropriate patient and family counseling re-

sulting in averting use of expensive, unnecessary laboratory studies and avoidance of painful procedures. The following cases and discussion will focus on the characteristic clinical features of IBIH.

CASE REPORTS

CASE 1. An 18-month-old light-pigmented boy with a history of atopic dermatitis was evaluated for pruritic patches on his arms and legs. His eruption initially improved with topical pimecrolimus 1% cream and lubricants. Over a period of 3 months, new red itchy bumps appeared which failed to respond to oral amoxicillin and cefadroxil. Although there was a family history of atopic dermatitis, no other members of the family had active skin disease with the same manifestations of lesions. Examination revealed scattered eczematous patches over his arms and legs. He also had multiple clustered 0.5- to 1.0-cm hyperpigmented macules, many with

Key Words: dermatology, pediatric

Abbreviations: IBIH, insect bite–induced hypersensitivity; DTH, delayed-type hypersensitivity; PSP, pathologic skin picking

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central crusting and hypopigmentation. The face, neck, scalp, chest, abdomen, and diaper areas were not affected (Figs 1 and 2).

CASE 2. A 4-year-old dark-pigmented girl was evaluated for itchy red bumps that healed with brown spots over her arms and legs. The family noted these lesions developed over a period of 6 months. There was no personal or family history of atopic dermatitis. There was also no personal history of other skin disorders in the patient and she was the only family member experiencing the itchy eruption. There were no pets and no history of recent outdoor activity. Examination revealed multiple, grouped, 1-cm hyperpigmented macules. Some of the lesions had central scaling and hypopigmentation. Lesions were organized in groups of 2 to 4, often occurring in a linear fashion. Areas most prominently affected include her arms, upper back, and lower legs. Her face, neck, scalp, chest, abdomen, and underwear area were clear. This patient underwent a skin biopsy, which reported "mild acanthosis with subepidermal edema. A superficial inflammatory infiltrate is appreciated with a predominance of mononuclear cells and eosinophils" (Figs 3 and 4).

CASE 3. A 4-year-old dark-pigmented girl complained of itchy bumps on her arms, legs, and back for 3 weeks. She failed to improve with topical triamcinolone cream, oral diphenhydramine, and a course of cephalexin. There was no personal or family history of atopic dermatitis or other dermatoses, and no other family members were affected. Examination revealed multiple, clustered, edematous, mildly erythematous, hyperpigmented, 1-cm papules that were most dense on the



FIGURE 1
Urticarial papules have a target-like appearance.



FIGURE 2
Clustering of papules on one extremity.

forearms. Her face, neck, scalp, chest, abdomen, and underwear area were spared (Fig 5).

CASE 4. A 2-year-old dark-pigmented boy presented with itchy bumps on his arms, legs, and face. Although he developed lesions continually for >1 year, they tended to increase in severity during the spring and summer. Treatments included oral antibiotics and topical moisturizers without improvement. There was no family history of skin disease, and no other members of the family were affected. There was a pet dog in the home, and pets in the homes of many relatives. Examination showed multiple grouped 2- to 5-mm red hyperpigmented papules with central scale and crusting scattered over his forehead, temples, and exposed areas of the arms and legs. His trunk and diaper area were spared. A skin biopsy found "mild spongiosis, superficial and deep mixed cel-



FIGURE 3
This 4-year-old has uniform 2- to 4-mm hyperpigmented macules and papules with hypopigmented centers grouped in a linear pattern. Note the collarette of scale and central crust on some lesions.



FIGURE 4
Many lesions demonstrate a hypopigmented center with a target-like appearance. Also note the pleiomorphic nature of the lesions in varying stages of healing.

lular infiltrate with eosinophils consistent with a dermatitis" (Fig 6).

DISCUSSION

Although there are few reports citing the incidence of papular urticaria, we noted that 5% of office visits to the Johns Hopkins Pediatric Dermatology Clinic over a 4-week period (December 12, 2003 to January 11, 2004) were attributed to papular urticaria or insect bite reactions. Many of these patients had undergone extensive laboratory testing and skin biopsies as demonstrated in our case reports. A majority of these patients had been referred to our clinic by their primary pediatrician or dermatologist for evaluation of a persistent pruritic rash.

The prevalence of papular urticaria has been speculated to increase in the spring and summer months when insect populations are peaking in the United States.² We observed, however, that the eruption invariably recurs during the fall and winter months (for reasons discussed later in this section), making prompt di-



FIGURE 5
This patient demonstrates the symmetric nature of papular urticaria with both arms being affected equally. There are varying degrees of scarring and hyperpigmentation.



FIGURE 6
An itchy healthy 2-year-old girl developed uniform, red, itchy papules measuring 2 to 5 mm that were disseminated over exposed areas of his skin.

agnosis difficult unless the clinician is aware of the characteristic skin lesions, possible exposures, and natural history of the disease.

IBIH has been primarily associated with cat and dog fleas in addition to mosquitoes.^{3,4} Interestingly, the common bedbug (*Cimex lectularius*) has long been known to produce the identical reaction but is less commonly considered an offending agent in IBIH.⁵ This small blood-sucking insect was largely eradicated in developing countries around the 1950s secondary to the widespread use of DDT. Within the past 3 years, however, studies have shown an astounding 500% increase in the number of cases of bedbug infestations in the United States and Europe.^{6,7} Many factors have been proposed as culprits for this epidemic. In fact, the frequency and ease of international travel in today's modern world may be a primary cause of bedbug resurgence. It is easy to imagine how an "international exchange" of bedbug colonies between infested and noninfested countries has reintroduced these pesky insects into the United States.

Moreover, bedbugs, unlike mosquitoes, are not dependent on the surrounding environment for reproduction. These hearty insects can be transported to any climate and thrive in many geographic regions.⁸ In addition, the life cycle of the common bedbug allows for it

to survive up to 1 year without a blood meal, making them a difficult vermin to exterminate even after appropriate use of insecticides. A study by Hwang et al⁶ showed that urban cities in particular provide ideal environments in which bedbugs can thrive. These heavily populated locations allow for the frequent transfer and proliferation of bedbugs in places such as shelters, hotels, and apartment units. Consequently, pediatricians should be aware of not only the association between IBIH and bedbugs but also of the fact that these insects are rising in incidence and in their distribution throughout the United States.

Previously reported histopathologic findings in papular urticaria have been shown to vary with the particular arthropod, age of the lesion, and sensitivity of the patient.⁹ The characteristic urticarial lesions will demonstrate prominent papillary dermal edema and perivascular lymphocytes, eosinophils, and mast cells. Early lesions may even demonstrate some exudate and neutrophils. Vesicular lesions show intraepidermal and subepidermal edema and, occasionally, some hemorrhage. In addition, there may be superficial and deep perivascular and interstitial infiltrate with variable density of lymphocytes and eosinophils present. Chronic lesions may demonstrate pseudoepitheliomatous hyperplasia and atypical dermal infiltrates. On the basis of these findings, the histologic differential diagnosis would include conditions such as atopic dermatitis, contact allergic dermatitis, drug-hypersensitivity reaction, and, in rare cases, lymphoma. If found in a newborn, one would need to consider incontinentia pigmenti and erythema toxicum neonatorum. However, because of the required period of sensitization, papular urticaria is not seen in the newborn period. Other clinical entities to consider in the differential diagnosis are discussed in Table 1.

Early experiments on papular urticaria by Mellanby in the 1940s demonstrated an acute and predictable allergic skin response in humans who had intradermal injection of mosquito antigens (Table 2).⁴

Identifying this sequence of events provided the basis for understanding the immune mechanisms involved in insect bite reactions. After a period of sensitization during early infancy, when bites occur with a minimal reaction (stage 1), patients develop a delayed-type hypersensitivity (DTH) IV reaction, histologically characterized by a diffuse mononuclear infiltration of the mid- and deep dermis (stage 2). In stage 3, sensitized individuals first develop an immediate hypersensitivity type I urticarial papule followed by a delayed reaction. This is considered to be the individual's peak of sensitization. The immediate reactions in stages 3 and 4 are distinguished by the presence of large numbers of eosinophils in the skin biopsy.¹⁰

Recent immunohistochemical studies have supported these historical findings. Histopathologic examination of lesions in 30 patients with papular urticaria revealed that

eosinophils were present in 86% of the samples.¹¹ This study, therefore, speculated that papular urticaria was primarily mediated through a type I hypersensitivity reaction. However, in 2004, Garcia et al¹² conducted studies to evaluate the characteristic cell types in lesions of patients diagnosed with papular urticaria after cutaneous injection of flea antigen. They noted a predominance of CD4⁺ T cells in all lesions including vesicles, wheals, and papules. It is noteworthy that in addition to elucidating a delayed type mechanism in papular urticaria, this study also supported previous studies by showing an abundant number of eosinophils throughout the dermis in all lesions. The presence of these 2 types of cell populations lends support to the theory that both immediate and delayed mechanisms are involved in mediating IBIH.

DTH, however, is the basis for both the clinical chronicity and variable severity of IBIH in pediatric patients. The elapsed time between an insect bite and the formation of a firm, intensely itching papule begins to lengthen as children gain exposure to these allergens. Consequently, it can become difficult for a patient and/or parent to accurately report "bite events." Continued and repeated exposure to the inciting antigen results in not only immediate skin reactions but also a cycle of DTH-mediated lesions. Parents subsequently go on to find more new skin findings as the child persists at itching and scratching from previous sites. This sequence is perpetuated until the offending agent is identified or the individual becomes desensitized, which can take weeks, months, and sometimes years.⁴

Furthermore, a phenomenon known as reactivation can result in variable severity of pruritis and skin lesions even when the number of new lesions seems to be waning. This was first described in the 1960s, when clinicians noted that some individuals reactivated previous lesions after appearance of new bites.¹⁰ This effect is now thought to be secondary to circulating insect antigen stimulating cutaneous T cells in previously sensitized sites.¹ Clinical evidence of this is seen in children with generalized eruptions and pruritis after only limited exposure to biting insects at a focal site. The idea of reactivation again complicates clear diagnosis of IBIH in that it may be difficult, if not impossible, to distinguish between new-onset lesions and a reactivation event. One can expect, however, that given the DTH mechanism of the cutaneous T-cell response, a majority of reactivation events will resolve within 4 to 6 weeks.

Clinical Features

We suggest that the clinical diagnosis may be aided by using the mnemonic "SCRATCH," which is described below and in Table 3.

TABLE 1 Differential Diagnosis for an Itchy Rash

Dermatologic Process	Clinical Features	Clinical Pearls
Scabies	Severe pruritis J- or S-shaped burrows 5-10 mm in length, seen on superficial dermis Most commonly seen on web spaces of fingers and toes, flexural aspect of wrists, and antecubital fossa Head and face commonly affected in young children	Multiple family members generally affected Nocturnal pruritis is a characteristic complaint Diagnosis can be confirmed with skin scraping of burrow
Atopic dermatitis	Chronic pruritis Distribution (infancy) includes widespread, generalized lesions with sparing of diaper area Early childhood: lesions involve flexural and extensor surfaces of distal extremities; head and neck may also be involved	Two thirds of patients have a family history of asthma, allergic rhinitis, or atopic dermatitis 60% of patients have initial outbreak by first birthday; another third of patients develop disease between 1 and 5 y of age Look for hyperlinear palms, facial pallor, and infraorbital darkening
Contact dermatitis	Acute onset of severe pruritis, erythema, and edema Distribution of rash typically restricted to areas of exposure	Common allergens include poison ivy, nickel, rubber, glues, and dyes Continued exposure to allergen may result in scaling and lichenification
Id reaction	Recent and/or current history of fungal infection Defined as acute onset of an extremely pruritic, erythematous, maculopapular, or papulovesicular eruption; occurs 1-2 weeks after primary infection or dermatitis Secondary lesions are distant from site of primary infection	Common inciting pathogens include dermatophytes, mycobacteria, viral/bacterial infections, or parasites (ie, pediculosis) Treatment of primary pathogen is essential for cure; however, patient may benefit from topical corticosteroids
Pityriasis lichenoides	Rash is often asymptomatic; however, pruritis may be intense Rash consists of round 2-mm to 1-cm reddish-brown papules with shiny, brown central scale Crops of papules heal over several weeks, leaving postinflammatory pigment changes	Look for a "frosted-glass" appearance of lesions on torso and back Typically seen in adolescent population, with slight predominance in males
Linear IgA bullous dermatosis	Lesions are distinctive, 1- to 2-cm tense bullae with erythematous bases as well as urticarial plaques Lesions commonly appear on lower trunk and perineum; however, may also be seen on face and trunk Mucous membrane involvement is rare; however, may occur with disseminated disease	Look for "string-of-pearls" phenomenon: annular vesicles occurring around a healing or inflamed base Direct immunofluorescence shows diagnostic linear IgA deposition along dermal-epidermal junction Many children are responsive to treatment with dapsone
HIV-associated dermatosis	Variable morphology including papules, vesicles, and hyperpigmentation Disseminated distribution affecting face, extremities, and trunk	Systemic symptoms such as failure to thrive or lymphadenopathy should arouse concern Consider with fulminant presentation of common rashes such as molluscum contagiosum or scabies
Lymphatoid papulosis	Chronic, benign, self-limiting disorder Lesions similar in appearance to acute form of pityriasis lichenoides Exception is that rash is often nonpruritic and widespread	Histopathology is characterized by atypical lymphohistiocytic infiltrate suggestive of lymphoma; however, eruptions are rarely a presenting sign for systemic lymphoma
Urticaria pigmentosa	Often first observed in infancy as solitary brown mastocytoma or multiple brown macules and papules around the trunk Lesions range from 1cm in diameter to large infiltrated confluent plaques that cover entire chest and back	Darier's sign: rubbing of one of the brown lesions causes erythema, swelling, and itching, confirming the presence of mastocytosis

Id reaction indicates autoeczematization or a secondary dermatitis, derived from the Greek for father-son relationship.

S
Most patients present with a symmetric eruption. Commonly affected areas include exposed surfaces such as the face, neck, arms, and legs, whereas the diaper area, palms, and soles are invariably spared. The trunk is commonly spared; however, careful inspection may reveal bites along the sockline and waistline or other sites where clothing may cause pressure sites. This contrasts with the distribution found in scabies, in which burrows are commonly found on the palms, soles, and interdigital

TABLE 2 Mellanby's Stages of Bite Reactions^{4,10}

Stage 1	A period of induction of hypersensitivity; no observable skin reaction
Stage 2	Delayed skin reactions
Stage 3	Immediate skin reactions followed by delayed reactions
Stage 4	Immediate reactions only (not delayed)
Stage 5	No reactivity

spaces. IBIH also commonly involves the scalp, particularly in young children who are close to the ground. This is another helpful finding that distinguishes IBIH from scabies, in which burrows are rarely seen on the scalp and face.

C

Lesions usually appear in a “meal cluster,” sometimes described as “breakfast, lunch, and dinner.” These linear or triangular groupings of lesions are characteristic of bedbug bites; however, they may also be seen in flea bites. We noted this type of clustering very regularly in children with IBIH (demonstrated in Figs 3–5). Lesions in sensitized patients usually begin as itchy, red, edematous, indurated, urticarial papules, which develop a central vesicle within 1 to 3 days. Excoriation leads to central crusting, erosions, and occasionally secondary infection. The eruption heals over 4 to 6 weeks, leaving residual hyperpigmented macules with central hypopigmentation in dark-pigmented individuals and a persistent violaceous discoloration in some light-pigmented patients.

R

Many clinicians have a problem with the diagnosis of papular urticaria when there is no history of a pet in the patient's home. Cat flea bites are, in fact, the most common cause of IBIH; however, several older studies established that only a remote history of pet exposure (eg, from a relative's house, visited only once to twice weekly) was necessary to produce the itchy rash.¹³ Therefore, lack of “Rover” in the immediate home should not eliminate IBIH from the pediatrician's differential diagnosis. As mentioned earlier, exposures to mosquitoes (eg, outdoor play, camping) and bedbugs (ie, inhabiting a shelter, apartment complex, or a history of

a recent hotel visit) are other important components to consider in the patient's history.

A

Age is a very important factor to consider, because few patients develop the eruption before their first birthday and the prevalence peaks after age 2. Fully blossomed papular urticaria requires a long period of sensitization with repeated exposures to blood-sucking insects. Tolerance develops in most children by 10 years of age.

T

Target lesions are characteristic for IBIH, particularly in dark-pigmented patients (Figs 1–3). This “T” also stands for “time” to emphasize the chronic nature of the eruption and the need for patience and watchful waiting. Prevention of secondary infection with appropriate use of nail and hand hygiene and use of moisturizers is perhaps the biggest intervention.

C

Including the “confused pediatrician and parent” component in our mnemonic is our way of introducing a very real reaction that we encountered in a large percentage of our patients who went on to have the diagnosis of IBIH. After having been seen by numerous physicians, the last thing the parent wanted to hear was that this ongoing and concerning rash was caused by insect bites. The expression of disbelief and confusion was uniform throughout our panel of patients. We came to realize that the sheer implication that there might be bedbugs or fleas in the home was quite an insult (not to mention a disappointing diagnosis after being told that there are few remedies besides observation). This diagnosis was also difficult to explain to the well-meaning general pediatrician who referred the family to an allergist and a dermatologist for help with a diagnostic dilemma. In summary, we decided that if a family did not meet us with disbelief and confusion, we most likely had the wrong diagnosis.

H

Finally, unlike scabies and atopic dermatitis, which invariably have some component of family history in their development, IBIH very often only affects a single family member. Mothers often stated to us: “He's the only one! I don't understand!” Common sense tells us that if there were fleas or mosquitoes, other family members would have the same rash, right? Because this condition is caused by hypersensitivity, some individuals, by definition, will react while others will not. It is rare, in fact, for multiple family members to have similar symptoms in IBIH because of the fact that each individual has different thresholds for being “sensitized”; some children become sensitized after a single bite, whereas others require more frequent and repeated inoculation with the

TABLE 3 The SCRATCH Principles

S	Symmetric distribution (scalp, neck, face, torso, extremities)
C	Crops/clusters of different coloration (erythema, hypo-/hyperpigmentation)
R	Rover not required: pets are not necessary criteria for diagnosis
A	Age specific (usually occurring between 2 and 10 y of age)
T	Target lesions and time (may take weeks to years to resolve)
C	Confused pediatrician/parent: “We don't have fleas!”
H	Household with single family member affected

same antigen before synthesizing the required antibodies to induce a skin reaction. In this case, a negative family history is very helpful in making the diagnosis.

Differential Diagnosis

As we have observed, IBIH is a difficult diagnosis to make because of the fact that so many other conditions share the observed features and presenting symptoms. We have listed what we consider common dermatologic conditions similar to IBIH and their defining clinical features in Table 1.

Another less common clinical entity to consider in the differential diagnosis is neurotic excoriations or pathologic skin picking (PSP). Patients affected by this disorder will typically have lesions similar in appearance and distribution to IBIH; however, skin findings and pruritis are uniformly a physical manifestation of an emotional problem. If a previous history of anxiety, obsessive compulsive disorder, or depression is elicited, PSP should be considered; however, there are few case reports of this disorder in the otherwise healthy pediatric population. Therefore, PSP should be a diagnosis of exclusion after the listed conditions have been considered.

Management

Treatment of IBIH is usually disappointing to families and practitioners alike. Fortunately, however, this condition is rarely serious and always improves with time. Below we emphasize the 3 "P's" of management.

Prevention

This includes the wearing of protective clothing for outdoor play with judicious use of insect repellents. Families with pets should participate in aggressive flea-control measures for the home, including flea collars, flea medication, frequent bathing of the dog/cat, and frequent washing of personal bedding in hot water. If bedbugs are thought to exist in the home, measures such as laundering bedding and mattress pads every 2 to 4 weeks in combination with applying double-sided tape to the legs of the bed have been shown to prevent bedbugs from becoming long-term residents in their common dwelling places.⁶ Although flea and bedbug infestations often respond to these conservative measures, families should consider professional application of pesticide treatments to assure removal of allergens.

Pruritis Control

High-potency topical steroids may help with individual lesions. However, the inflammatory process may extend deep into the dermis and fat, rendering topical agents ineffective. Some symptomatic relief has been reported with use of antihistamines such as cetirizine and Benadryl if findings are acute in nature, suggesting a type 1 mechanism. There is little evidence to support widespread use of these medications in children with

recurrent and chronic lesions because of the predominance of T-cell-mediated response and a lack of histamine-mediated lesions. In older children and adults with localized irritation, intralesional steroids may suppress pruritis, but this is often not a feasible option for young patients with widespread lesions.

Patience

Parents should be advised of the frustrating, persistent, recurrent nature of IBIH and reassured by the eventual development of tolerance and resolve of symptoms. Their understanding of the natural history of IBIH is necessary to prevent multiple consultations and invasive, unnecessary investigative studies.

CONCLUSIONS

We found that identification of IBIH in children is difficult because of the variability of symptoms. We have proposed the SCRATCH principles as a means to help the practitioner with the diagnosis. The terminology used to describe insect bite reactions is confusing, and after consideration of both historical and current histologic data, we suggest the term IBIH to better describe the findings in this group of patients.

Finally, the 3 "P's" of therapy, with the emphasis on patience and understanding of the pathophysiology of IBIH, should be discussed with patients and parents to prevent unnecessary laboratory studies and multiple consultations. Areas of future investigation might include flea-antigen patch testing to help facilitate the diagnosis of IBIH.

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