Bullous “Cellulitis” With Eosinophilia: Case Report and Review of Wells’ Syndrome in Childhood

Amy E. Gilliam, MD*; Anna L. Bruckner, MD‡; Renée M. Howard, MD*; Brian P. Lee, MD§; Susan Wu, MD§; and Ilona J. Frieden, MD*$

ABSTRACT. A 1-year-old girl presented with acute onset of edematous erythematous plaques associated with bullae on her extremities and accompanied by peripheral eosinophilia. She was afebrile, and the skin lesions were pruritic but not tender. The patient was treated with intravenously administered antibiotics for presumed cellulitis, without improvement. However, the lesions responded rapidly to systemic steroid therapy. On the basis of lesional morphologic features, peripheral eosinophilia, and cutaneous histopathologic features, a diagnosis of Wells’ syndrome was made. Wells’ syndrome is extremely rare in childhood, with 27 pediatric cases reported in the literature. Because it is seen so infrequently, there are no specific guidelines for evaluation and management of Wells’ syndrome among children. The diagnosis should be considered for children with presumed cellulitis and eosinophilia who fail to respond to antibiotics. Evaluation should include a directed history, physical examination, complete blood count, and stool testing for ova and parasites, to identify potential triggers. Treatment is with systemic steroid therapy unless disease is limited, in which case medium/high-potency topical steroids may be indicated. If systemic features are prominent or disease is chronic (lasting >6 months), then a referral to hematology/oncology should be considered. Pediatrics 2005;116:e149–e155. URL: www.pediatrics.org/cgi/doi/10.1542/peds.2004-2273; cellulitis, eosinophilia, eosinophilic cellulitis, Wells’ syndrome.

ABBREVIATIONS. CSS, Churg-Strauss syndrome; HES, hyper-eosinophilic syndrome; IL, interleukin.

Wells’ syndrome, or eosinophilic cellulitis, is a rare, recurrent, inflammatory dermatosis of unknown pathogenesis. In 1971, Wells$ described 4 patients with an acute pruritic dermatitis clinically resembling bacterial cellulitis but with histopathologic findings characterized by dermal eosinophilia, phagocytic histiocytes, and the presence of flame figures. He initially called this syndrome recurrent granulomatous dermatitis with eosinophilia but later simplified the name to eosinophilic cellulitis.$ Wells’ syndrome is seen more commonly among adults but has been observed among children. Some hypothesize that this syndrome may represent a hypersensitivity response to a circulating antigen.\(^2\) Associated precipitants include insect bites, medication reactions, recent immunization, myeloproliferative disorders, malignancies, and infections. We describe a case of a young child with no identifiable triggering factors, and we review the evidence for evaluation and management of these pediatric cases.

CASE REPORT

A previously healthy, 1-year-old girl presented with acute onset of edematous erythematous plaques, with associated bullae, on her lower extremities and left arm (Fig 1). These lesions were pruritic but not painful, and the patient was afebrile. Her parents denied a history of insect bites, ingestion of medications, trauma, or other intercurrent illness. The patient’s most recent immunizations had been received 3 months earlier. The patient did not have a history of asthma, and there was no family history of asthma or atopic disease. Therefore, the patient was admitted with presumed bacterial cellulitis and was treated with intravenously administered oxacillin, without improvement. Her laboratory studies were significant for an elevated white blood cell count of \(30 \times 10^9\) cells per L, with peripheral eosinophilia of 48%. After the patient failed to respond to systemically administered antibiotics, examination of vesicle fluid was performed and revealed numerous eosinophils. Subsequently, the diagnosis of probable Wells’ syndrome was made. Oral steroid therapy was started at 2 mg/kg, and the patient’s cutaneous symptoms improved within 24 hours, leaving residual erythema and hyperpigmentation (Fig 2). Five days after the initiation of oral steroid therapy, a skin biopsy was performed from the left arm of a 1-year-old girl.

![Fig 1. Acute onset of edematous erythematous plaques and bullae on the left arm of a 1-year-old girl.](http://example.com/image1.jpg)
are several reports of associated elevated IgE lev-
tation rate is elevated for some patients, and there
active phase of disease.5,6 The erythrocyte sedimen-
tation, resembling morphea.2 There is usually no
weeks, leaving residual skin atrophy and hyperpig-
therapy, showing residual erythema, induration, and hyperpig-
sei.7–14 Fever, lymphadenopathy, arthralgias, and

discussion
Wells’ syndrome is extremely rare in childhood, with
on 27 pediatric cases reported (Table 1). It is
characterized by a combination of distinct clinical
and histopathologic findings. Classically, patients
with pruritic erythematous plaques, sometimes with associated bullae, that evolve rapidly over 2 to 3 days. These resolve spontaneously over 2 to 8 weeks, leaving residual skin atrophy and hyperpig-
mentation. There is usually no improvement with antimicrobial therapy; instead, a rapid response to oral corticosteroid treatment is ob-
served, as in our case. It is not uncommon for pa-
ents to have recurrent disease, with exacerbations and remissions occurring over several years.

The histopathologic findings are quite specific and
are characterized by flame figures, which are com-
plicated by eosinophilic granules surrounded by
collagen bundles.9 With resolution, there is a granu-
omatous phase of histiocytes palisading around the
flame figures. Vasculitis is absent, and direct immu-

fig. 2. Appearance of skin lesions after 5 days of oral steroid therapy, showing residual erythema, induration, and hyperpig-
mentation.

els.7–14 Fever, lymphadenopathy, arthralgias, and
other systemic symptoms (such as pulmonary in-
volvement) have been described for Wells’ syn-
drome, and these findings may be indicative of a
more severe or progressive course.7

The differential diagnosis of Wells’ syndrome in-
cludes bacterial cellulitis, Churg-Strauss syndrome
(CSS), eosinophilic fasciitis, and hypereosinophilic
syndrome (HES) (Table 2). The skin lesions of Wells’
syndrome are distinguished from those of bacterial
cellulitis by the absence of tenderness and the pres-
ence of pruritus, which is often the primary symp-
tom of Wells’ syndrome. Lack of warmth, failure to
respond to antibiotic therapy, and characteristic his-
tologic findings are the other features that differen-
tiate Wells’ syndrome from bacterial cellulitis.

CSS should be considered for patients with persis-
tent peripheral eosinophilia and skin lesions. Al-
though more commonly seen among adults, CSS can
present in childhood.15–17 This syndrome is charac-
terized by asthma, peripheral eosinophilia, and vas-
culitis and is associated with autoantibodies to pe-
rinuclear antineutrophil cytoplasmic antibody, as
well as cutaneous and systemic granulomas. Palpa-
ble purpura, tender subcutaneous nodules, and cu-
taneous infarctions are more often the associated
skin findings in CSS, whereas patients present with
bullae and vesicular lesions in Wells’ syndrome. In
both conditions, flame figures can be identified his-
topathologically, as can peripheral blood and tissue
eosinophilia. However, the presence of vasculitis
with extensive fibrinoid necrosis of collagen is more
suggestive of CSS.2–9

Eosinophilic fasciitis is another condition that can
resemble Wells’ syndrome. Also seen more fre-
cently among adults but reported in children, it
presents with acute onset of skin inflammation and
resolves with hyperpigmentation and scleroderma-
like skin changes. Unlike Wells’ syndrome, eosino-
philic fasciitis is characterized by arthritis as a prom-
inent symptom, and it follows a more chronic course,
with individual lesions requiring months or years to
resolve.12 It is distinguishable from Wells’ syndrome
by the depth of inflammation, with eosinophilic in-
vasion into deeper fascial tissues.

Finally, idiopathic HES also should be considered
when the diagnosis of Wells’ syndrome is being en-
tertained. This condition is extremely rare in the
pediatric age group but has been reported in child-
hood.20 HES is a lymphoproliferative disorder char-
acterized by overproduction of eosinophils with a
predilection to damage specific organs, especially the
cardiovascular system. It is defined by sustained eo-
sinophilia (>1.5 × 10^9 cells per L, lasting for >6
months), with evidence of multiple-organ system in-
volve ment, in the absence of parasitic disease, aller-
gic diatheses, or other conditions known to cause
eosinophilia. The heart, lungs, central and peripheral
nervous systems, kidneys, and gastrointestinal tract
can be affected, and the cutaneous findings are simi-
lar to those of Wells’ syndrome, including erythem-
ato us pruritic papules and nodules, urticaria, and
angioedema.9,21–23 Histopathologically, the skin le-
sions of HES are nonspecific, and the flame figures
and granulomatous infiltrate seen in Wells’ syndrome are absent.21

The pathogenesis of Wells’ syndrome is not well defined. One hypothesis is that it represents a hypersensitivity mechanism triggered by factors such as infections, drugs, or internal disease. However, in approximately one half of reported cases among children, there is no identifiable precipitating factor.24 Reported precipitants have included bites or stings from ticks, bees, and spiders8,9,12,25–28 and infections with mumps, molluscum contagiosum, varicella, and herpes simplex virus.1,24,29,30 There are also several reports of Wells’ syndrome associated with bacterial, parasitic, and fungal infections.5,9,14,31–33 Numerous medications have been implicated as triggers for Wells’ syndrome.1–3,5,9,34–38 Also, several cases of Wells’ syndrome occurred after vaccinations,39,40 and it was proposed that the preservative thimerosol was the causative agent in those cases.39 Several cases of Wells’ syndrome among adults have been associated with hematologic disorders,9,41 lymphoproliferative malignancies,1,34,42 and carcinoma.34,43–45 Zachary et al45 reported a case of Wells’ syndrome in a 17-year-old girl with nasopharyngeal carcinoma, which is the only pediatric case of Wells’ syndrome associated with malignancy reported in the literature.

One of the key events in disease expression of Wells’ syndrome appears to be aberrant and inadequate eosinophil skin homing. Increased interleukin (IL)-5 levels have been observed in Wells’ syndrome, and IL-5 not only mobilizes eosinophils from the bone marrow but also promotes homing of eosinophils by altering expression of adhesion molecules. In addition, increased levels of IL-5 appear to induce expression of CD25, the α chain of the IL-2 receptor, which enhances eosinophil degranulation and subsequent tissue destruction.46–48

Treatment for Wells’ syndrome is sometimes unnecessary, because cases often resolve spontaneously. If an infection or other treatable precipitating factor can be identified, then there is often improvement with treatment of the underlying condition. However, when no treatable underlying factor can be identified, systemic corticosteroid therapy is used frequently for both adults and children. Most cases resolve after a single course of systemic corticosteroid therapy; when recurrences occur, however, alternative treatments should be considered, to avoid the side effects of chronic systemic steroid therapy.36 Topical steroid treatment has also been reported as successful therapy, both alone,2,29,39 and in combination with systemic steroid therapy.5 Specifically, topical steroid therapy alone resolved skin lesions for 2 children, which suggests that topical steroid therapy may be a safe alternative to systemic corticosteroid therapy in the pediatric age group.28,39 Other therapies reported to be successful include various antimicrobial agents,2,5,6,9,10,13,49 colchicine,13 antimalarial drugs, cyclosporine,50 azathioprine,5 interferon-α,51 psoralen with ultraviolet A,52 and antihistamines.10,43,53

The small numbers of cases and the fact that most reports are anecdotal make it difficult to draw conclusions regarding whether these therapies are truly effective or these cases resolved spontaneously. However, we think that first-line treatment for children should be systemic corticosteroid therapy, with the addition of topical steroid treatment depending on the extent of disease. A dose of orally administered prednisolone or prednisone of 2 mg/kg per day for 5 to 7 days, with a taper over 2 to 3 weeks, is appropriate. Topical steroid treatment may be used in combination. In cases in which there is limited body surface area involved (15–30%) and an absence of systemic symptoms, it may be prudent to consider medium-potency topical steroid therapy alone, with close follow-up monitoring. This would also be appropriate for recurrent cases of Wells’ syndrome identified early, when disease may be limited.

Evaluation should be directed at ruling out other conditions that mimic Wells’ syndrome, as well as evaluating possible triggering factors. We recommend a complete history and review of systems, with specific attention to recent medications, vaccinations, insect bites, infections, or illnesses and associated

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Fig 3. High-power view of a biopsy specimen from a resolving lesion, showing a waning flame figure, represented by collections of eosinophilic granules surrounded by a palisade of histiocytes. (Hematoxylin-eosin; original magnification: ×100.)
<table>
<thead>
<tr>
<th>Reference</th>
<th>Age</th>
<th>Gender</th>
<th>Triggering Event(s)</th>
<th>Associated Findings</th>
<th>Treatment and Course of Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wells and Smith,2 1979</td>
<td>11 y</td>
<td>M</td>
<td>Mumps, penicillin</td>
<td>Elevated WBC count (16 × 10⁹ cells per L), fever</td>
<td>Prednisolone and sulfapyridine, recurrences over 3 y</td>
</tr>
<tr>
<td>Wells and Smith,2 1979</td>
<td>12 y</td>
<td>M</td>
<td>Erysipelas, penicillin</td>
<td>Elevated WBC count (26 × 10⁹ cells per L), eosinophilia (44%)</td>
<td>Prednisolone, recurrences over 2-3 y</td>
</tr>
<tr>
<td>Nielsen et al,25 1981</td>
<td>11 y</td>
<td>M</td>
<td>Unknown</td>
<td>Elevated WBC count (42 × 10⁹ cells per L), eosinophilia (13%), fever, ANA-positive,</td>
<td>Prednisone, 1 recurrence</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>arthralgia, alopecia</td>
<td></td>
</tr>
<tr>
<td>Saulsbury et al,22 1983</td>
<td>7 y</td>
<td>M</td>
<td>Possible insect bites</td>
<td>Elevated WBC count (17 × 10⁹ cells per L), eosinophilia (48%), fever, elevated IgE</td>
<td>Ampicillin and antihistamines, spontaneous resolution in 1 wk, with recurrences</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>level, bone marrow eosinophilia, oculomotor nerve palsy</td>
<td></td>
</tr>
<tr>
<td>Bonvalet et al,4 1983</td>
<td>2 y</td>
<td>M</td>
<td>Unknown</td>
<td>Eosinophilia</td>
<td>No treatment, spontaneous resolution in 2 wk with recurrences</td>
</tr>
<tr>
<td>Schorr et al,28 1984</td>
<td>3.5 y</td>
<td>M</td>
<td>Flea bite</td>
<td>Fever</td>
<td>No record of treatment, multiple recurrences</td>
</tr>
<tr>
<td>Brehmer-Andersson et al,34</td>
<td>14 y</td>
<td>M</td>
<td>Unknown</td>
<td>None</td>
<td>No treatment, spontaneous resolution in 1 wk, multiple</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>recurrences over 8 y</td>
</tr>
<tr>
<td>Wood, et al,26 1986</td>
<td>18 mo</td>
<td>M</td>
<td>Unknown</td>
<td>None</td>
<td>Systemic steroids, recurrences responsive to steroids</td>
</tr>
<tr>
<td>Wood et al,26 1986</td>
<td>10 y</td>
<td>M</td>
<td>Insect bites</td>
<td>Eosinophilia</td>
<td></td>
</tr>
<tr>
<td>Kamani and Lipsitz,7 1987</td>
<td>7 wk</td>
<td>M</td>
<td>Genetic (?), mother and brother</td>
<td>Elevated WBC count (22 × 10⁹ cells per L), eosinophilia (16%), fever, elevated IgE</td>
<td>Systemic steroids, recurrences responsive to steroids</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with Wells’ syndrome</td>
<td>level, pericarditis, pulmonary infiltrates with eosinophilic exudates/effusions</td>
<td></td>
</tr>
<tr>
<td>Kamani and Lipsitz,7 1987</td>
<td>3 wk</td>
<td>M</td>
<td>Genetic (?), mother and brother</td>
<td>Elevated WBC count (29 × 10⁹ cells per L), eosinophilia (32%), fever, generalized</td>
<td>Systemic steroids, recurrences responsive to steroids</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with Wells’ syndrome</td>
<td>seizure, meningitis, pulmonary infiltrates with eosinophilic cerebrospinal fluid</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>pleocytosis and pleural effusions</td>
<td></td>
</tr>
<tr>
<td>Correia and Garcia e Silva,54 1988</td>
<td>12 y</td>
<td>F</td>
<td>Possibly diabetes mellitus</td>
<td>Muscular weakness</td>
<td>No record of treatment in English abstract</td>
</tr>
<tr>
<td>Lindskov et al,11 1988</td>
<td>4 y</td>
<td>F</td>
<td>Unknown</td>
<td>Elevated WBC count (13 × 10⁹ cells per L), eosinophilia (17%), fourfold increase in</td>
<td>Oral antibiotics, acyclovir, and prednisone, gradual</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>varicella titer</td>
<td>resolution over 2 mo, recurrence treated with steroids</td>
</tr>
<tr>
<td>Lindskov et al,11 1988</td>
<td>5 y</td>
<td>F</td>
<td>Unknown</td>
<td>Elevated WBC count (47 × 10⁹ cells per L), eosinophilia (55%), fever, ESR of 62</td>
<td>Oral antibiotics and acyclovir, gradual resolution over 2 mo,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>mm/h, anemia, arthralgia, lymphadenopathy, bone marrow eosinophilia, and fourfold</td>
<td>no recurrences</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>increase in varicella titer</td>
<td></td>
</tr>
<tr>
<td>Lindskov et al,11 1988</td>
<td>20 mo</td>
<td>M</td>
<td>Unknown</td>
<td>Elevated WBC count (20 × 10⁹ cells per L), eosinophilia (13%), anemia, elevated IgE</td>
<td>Oral and topical antibiotics, gradual resolution over 2 mo,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>level</td>
<td>recurrences over 2 y</td>
</tr>
<tr>
<td>Authors</td>
<td>Age</td>
<td>Gender</td>
<td>Diagnosis</td>
<td>Laboratory Findings</td>
<td>Treatment</td>
</tr>
<tr>
<td>------------------</td>
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</tr>
<tr>
<td>Lindskov et al, 1988</td>
<td>9 y</td>
<td>M</td>
<td>Unknown</td>
<td>Elevated WBC (19 × 10⁹ cells per L), eosinophilia (13%), fever</td>
<td>Topical antibiotics, spontaneous resolution over 2 wk with recurrences</td>
</tr>
<tr>
<td>Reichel et al, 1991</td>
<td>6 y</td>
<td>M</td>
<td>Varicella</td>
<td>Eosinophilia (3%), varicella zoster virus titer of 1:32</td>
<td>Spontaneous improvement over 1 mo, topical steroids cleared residua</td>
</tr>
<tr>
<td>Paquet et al, 1992</td>
<td>10 y</td>
<td>M</td>
<td>Unknown</td>
<td>Eosinophilia (13.4%), elevated IgE level</td>
<td>Systemic antibiotics and oral steroids, no recurrences after 9 mo of follow-up monitoring</td>
</tr>
<tr>
<td>Anderson et al, 1995</td>
<td>4 y</td>
<td>F</td>
<td>Bee sting</td>
<td>Elevated WBC (45 × 10⁹ cells per L), eosinophilia, elevated IgE and septicemia</td>
<td>Systemic antibiotics and oral steroids, course complicated by systemic infection, improvement over 4 wk with recurrences</td>
</tr>
<tr>
<td>Garty et al, 1997</td>
<td>&lt;1 mo</td>
<td>F</td>
<td>Possibly danazol, ingested by mother during pregnancy</td>
<td>Elevated WBC count (15 × 10⁹ cells per L), eosinophilia (25%), ESR of 30 mm/h, mild hepatosplenomegaly, lymphadenopathy</td>
<td>Topical steroids, topical antibiotics, and antifungal medications ineffective, spontaneous resolution over months but with recurrences</td>
</tr>
<tr>
<td>Davis et al, 1998</td>
<td>7 y</td>
<td>M</td>
<td>Unknown</td>
<td>Eosinophilia of blood, tissue, and bone marrow, familial case associated with short stature, mental retardation, distinctive habitus</td>
<td>Systemic and topical steroids, frequent recurrences without resolution</td>
</tr>
<tr>
<td>Davis et al, 1998</td>
<td>5 y</td>
<td>F</td>
<td>Unknown</td>
<td>Eosinophilia of peripheral blood and bone marrow and elevated IgA, IgM, and IgG levels, familial case associated with short stature, mental retardation, and distinctive habitus</td>
<td>No treatment, recurrences over years</td>
</tr>
<tr>
<td>Aroni et al, 1999</td>
<td>12 y</td>
<td>F</td>
<td>Unknown</td>
<td>Eosinophilia</td>
<td>Cetirizine, resolved after 4 wk of treatment, recurrence after 18 mo</td>
</tr>
<tr>
<td>Kuwahara et al, 2001</td>
<td>Birth</td>
<td>F</td>
<td>Possibly penicillin or danazol ingested by mother during pregnancy</td>
<td>None</td>
<td>None, resolved by the 2-y follow-up visit</td>
</tr>
<tr>
<td>Moossavi and Mehregan, 2003</td>
<td>21 mo</td>
<td>F</td>
<td>Unknown</td>
<td>None</td>
<td>Prednisone and topical steroids, resolved without recurrence</td>
</tr>
<tr>
<td>Koh et al, 2003</td>
<td>3.5 y</td>
<td>M</td>
<td>Hepatitis B and DPT vaccination</td>
<td>Slight peripheral eosinophilia</td>
<td>Topical steroids (rapid improvement), recurrences treated with topical steroids</td>
</tr>
<tr>
<td>Stavropoulos et al, 2003</td>
<td>9 y</td>
<td>F</td>
<td>Molluscum contagiosum treated with cryosurgery</td>
<td>Eosinophilia (12%), ESR of 26 mm/h</td>
<td>Oral prednisolone, no recurrences</td>
</tr>
<tr>
<td>Gilliam et al, 2005</td>
<td>1 y</td>
<td>F</td>
<td>Unknown</td>
<td>Elevated WBC count (30 × 10⁹ cells per L), eosinophilia (48%)</td>
<td>Oral prednisolone and topical steroids, no recurrences in 1 y of follow-up monitoring</td>
</tr>
</tbody>
</table>

ANA indicates antinuclear antibody; WBC, white blood cell; ESR, erythrocyte sedimentation rate; DPT, diphtheria-pertussis-tetanus.
## Differential Diagnosis of Wells' Syndrome

**TABLE 2.**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Associated Findings</th>
<th>Histopathologic Findings</th>
<th>Associated Findings</th>
<th>Standard Treatment*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wells' syndrome</td>
<td>Eosinophilic and granulomatous dermatitis with eosinophilia.</td>
<td>Eosinophilic and granulomatous infiltrate with flame figures, no vasculitis and lymphocytic infiltrate with dermal lymphedema</td>
<td>Eosinophilic fasciitis</td>
<td>Oral steroids with or without cyclophosphamide, with addition of steroid-sparing agents for maintenance</td>
</tr>
<tr>
<td>Bacterial cellulitis</td>
<td>Recurrent, erythematous, pruritic plaques</td>
<td>Eosinophils, neutrophils, and mononuclear cells, absence of vasculitis, flame figures, or granulomas</td>
<td>Palpable purpura, tender subcutaneous plaques</td>
<td>Oral or intravenous chemotherapy</td>
</tr>
<tr>
<td>CSS</td>
<td>Palpable purpura, tender subcutaneous plaques</td>
<td>Palpable purpura, tender subcutaneous plaques</td>
<td>Palpable purpura, tender subcutaneous plaques</td>
<td></td>
</tr>
<tr>
<td>Eosinophilic fasciitis</td>
<td>Acute onset of symmetric induration of extremities, resolves with sclerodermatous skin changes</td>
<td>Acute onset of symmetric induration of extremities, resolves with sclerodermatous skin changes</td>
<td>Acute onset of symmetric induration of extremities, resolves with sclerodermatous skin changes</td>
<td></td>
</tr>
<tr>
<td>HES</td>
<td>Purpuric papules and plaques or urticaria and angioedema</td>
<td>Purpuric papules and plaques or urticaria and angioedema</td>
<td>Purpuric papules and plaques or urticaria and angioedema</td>
<td></td>
</tr>
</tbody>
</table>

*WBC indicates white blood cell.*

Medical problems such as asthma. A thorough physical examination should be performed, with attention to the liver, spleen, and lymph nodes. Stool samples should be sent for ova and parasite testing, and a complete blood count with differential evaluation should be performed. If there is uncertainty regarding the diagnosis, then a skin biopsy should be performed to distinguish between Wells' syndrome and other conditions that mimic it. We do not recommend a complete hematologic evaluation in all cases, because it is rare to see cases of either HES or Wells' syndrome triggered by hematologic or oncologic disorders in childhood. However, if a child presents with either systemic features, such as fevers, arthralgias, or other organ system involvement, or a chronic course, defined as >6 months of peripheral eosinophilia or recurrences of clinical disease, then a referral to hematology/oncology should be considered.

### REFERENCES


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