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Review Article

Wells Syndrome (Eosinophilic Cellulitis): Current Clinical, Histopathological, and Therapeutic Aspects

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Abstract

Wells syndrome, also known as *eosinophilic cellulitis*, is a rare, relapsing inflammatory dermatosis characterized by erythematous and edematous plaques, accompanied by a distinctive histopathological feature known as flame figures. Since its initial description by G. Wells in 1971, fewer than one hundred well-documented cases have been reported in both children and adults. This review summarizes the clinical, histopathological, and therapeutic aspects of Wells syndrome, with particular emphasis on diagnostic challenges and emerging treatment options. A systematic analysis of classical and contemporary literature, including case series and clinical observations, was conducted. The discussion encompasses the clinical presentation, histological characteristics, presumed etiopathogenesis, and therapeutic outcomes. Clinically, the disease manifests as acute-onset erythematous, urticarial, or annular plaques that often resemble infectious cellulitis but fail to respond to antibiotic therapy. Histopathological examination typically reveals a dense eosinophilic infiltrate with flame figures and granulomatous elements. Although the disease course is generally benign, it is prone to recurrence. Systemic corticosteroids remain the mainstay of treatment; however, recurrent or refractory cases may require agents such as dapsone, cyclosporine, or methotrexate. Recent advances include the use of biologic therapies targeting the IL-5 and IL-4/IL-13 pathways—such as omalizumab, mepolizumab and dupilumab—as well as JAK inhibitors, which have shown promising results in improving disease control and reducing relapse frequency. Further research is needed to establish standardized treatment protocols and optimize long-term management strategies.

Keywords

Wells Syndrome, Eosinophilic Cellulitis, Flame Figures, Eosinophilia, Corticosteroids, Biologic Therapy

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1. Introduction and Historical Background

Eosinophilic cellulitis, or Wells syndrome, is a rare, relapsing dermatosis characterized by distinct clinical and histological features, although its etiopathogenesis remains incompletely understood.

The condition was first described in 1971 by G. Wells, who reported four cases under the term *recurrent granulomatous dermatitis with eosinophilia* [1]. Subsequently, in 1979, G. Wells and N. Smith [2] published eight additional cases, providing a detailed account of the clinical and histopathological characteristics and introducing the term *eosinophilic cellulitis.*

Over time, the eponym "Wells syndrome" became widely adopted in recognition of the author who first described and systematized its defining features.

2. Epidemiology

Wells syndrome is an exceptionally rare disorder, with only a few dozen well-documented cases reported in the literature to date, including the series by R. Caputo *et al.* [3] and the reviews by G. Guhl and A. Garc á-D éz [4]. The condition occurs in both children and adults, with no consistent evidence of sex predilection, although some authors have reported variable findings [5-7].

The reported age range is broad, extending from early childhood (approximately 1–2 years) to over 70 years of age. While Wells syndrome was initially believed to affect both sexes equally, subsequent studies have suggested a slight female predominance [8, 9].

Overall, current evidence indicates that Wells syndrome can develop at any age and in either sex, with no clearly defined demographic predisposition [10, 11].

3. Clinical Presentation

The onset of Wells syndrome is typically acute. In some cases, a low-grade fever (up to 38 ℃) may occur, occasionally accompanied by nonspecific systemic symptoms such as arthralgia, diarrhea, or asthma attacks. These systemic manifestations are rare and inconsistent, with cutaneous lesions representing the predominant feature.

Within two to three days, erythematous–edematous plaques appear, often with a violaceous peripheral rim and variable size. Lesions may be solitary but are more commonly multiple, distributed across various body regions with a predilection for the trunk and extremities, while the face and scalp are usually spared. In some cases, lesions may involve an entire limb or large areas of the trunk.

The morphology of lesions is diverse: urticarial, annular, and indurated plaques have been described, and in certain cases, vesiculobullous variants may occur [12, 13]. Bullae are typically tense and filled with clear fluid, although hemorrhagic content has also been reported. As the process evolves, edema gradually subsides; the central portion of the plaque fades and softens, while the periphery remains indurated and violaceous with a sharply demarcated border. The lesions progress through edematous, indurated, and resolving stages, leading to complete recovery (*restitutio ad integrum*) within two to six weeks.

During a single episode, multiple plaques often develop simultaneously or sequentially, each following its own course of evolution. The initial presentation frequently mimics acute bacterial cellulitis; however, the lack of response to antibiotic therapy and the presence of marked tissue eosinophilia support the designation *eosinophilic cellulitis.* In later stages, the indurated nature of the lesions combined with granulomatous inflammation justifies the alternative term *granulomatous dermatitis with eosinophilia*.

Cutaneous manifestations are commonly associated with pruritus or a burning sensation and, less frequently, pain. General symptoms such as fever and arthralgia may occur but are inconsistent [7, 14].

A hallmark of Wells syndrome is its strong tendency to recur, with relapses appearing after variable intervals and following a similar clinical course. The disease typically exhibits a chronic–relapsing pattern without visceral involvement and consistently resolves completely. Rare extension to the underlying muscles and fascia has been reported in a single case by M. Zloczower *et al.* [15].

4. Biological Parameters

A typical laboratory finding in Wells syndrome is peripheral eosinophilia, which is common but not constant. It occurs in approximately half of the patients and usually coincides with the active phase of the disease, subsiding or disappearing during remission [7]. In addition to peripheral eosinophilia, bone marrow eosinophilia has also been reported, as confirmed by sternal puncture findings [13, 14, 16].

Elevated levels of mediators and biomarkers associated with eosinophil activation — such as eosinophilic cationic protein (ECP), interleukin-5 (IL-5), and immunoglobulin E (IgE) — have been documented in several cases [14, 17]. These alterations correlate with disease activity and support the immune-mediated nature of the inflammatory process.

Additional laboratory abnormalities may include a mildly elevated erythrocyte sedimentation rate (ESR), the presence of circulating immune complexes, and increased serum concentrations of IgG and IgE [14, 18]. In rare instances, positive antinuclear antibodies with a nonspecific staining pattern have also been observed [19].

Overall, eosinophilia — whether peripheral or bone mar-

row — represents the most characteristic biological feature of Wells syndrome, and its fluctuations typically parallel the clinical course of the disease.

5. Histopathology

The histopathological findings in Wells syndrome are characteristic and diagnostically suggestive, although not entirely specific. Lesions typically show a dense eosinophilic infiltrate involving the dermis and, in some cases, extending into the hypodermis [4, 20]. A granulomatous inflammatory component is frequently observed, particularly in the subacute and chronic stages, often featuring palisading multinucleated giant cells [21-23].

A distinctive morphological hallmark is the presence of *flame figures*—aggregates of eosinophilic granules deposited on collagen bundles and surrounded by histiocytes and foreign-body–type giant cells. Although considered pathognomonic for Wells syndrome, flame figures are not disease-specific; similar structures may occur in bullous pemphigoid, insect bite reactions, parasitic infestations, certain eczematous disorders, and drug-induced eruptions [23, 24].

Histopathological changes follow a staged pattern that closely correlates with the clinical evolution of the lesions [4, 16, 21]:

1) *Acute stage (first few days):* Dense perivascular and interstitial eosinophilic infiltrates, sometimes extending into vessel walls and fat lobules, accompanied by extracellular eosinophilic granules and marked dermal edema. In exudative forms, bullae are typically subepidermal, with minimal epidermal alteration.
2) *Subacute stage (around the second week):* The eosinophilic infiltrate becomes less dense, and flame figures begin to form within collagen bundles, surrounded by histiocytic and multinucleated giant cell reactions that create palisading microgranulomas.
3) *Involutive stage (around the fourth week):* Flame figures and the associated histiocytic response persist, while eosinophilic infiltrates largely disappear.

In conclusion, the histological picture of Wells syndrome is highly characteristic and of significant diagnostic value, though not entirely specific. Similar morphological alterations may also be encountered in other eosinophilic or granulomatous dermatoses. Flame figures, while a distinctive and frequently observed feature, are not pathognomonic and may likewise occur in other eosinophil-mediated conditions such as bullous pemphigoid, eosinophilic vasculitis, insect bite reactions, and drug eruptions.

6. Histopathogenesis of Flame Figures

Flame figures, regarded as a characteristic and almost constant feature of Wells syndrome, raise important questions about their histopathogenesis, which remains only partially understood. Two principal hypotheses have been proposed to explain their formation [20, 25]:

1) *Secondary formation* – resulting from the aggregation of eosinophilic granules and disintegrated eosinophils within the dermal connective tissue.
2) *Necrobiotic process* – representing foci of collagen fiber necrobiosis onto which eosinophilic products are subsequently deposited.

Electron microscopy and immunofluorescence studies have demonstrated the accumulation of eosinophilic granules and nuclear fragments adherent to morphologically intact collagen fibers, supporting the hypothesis that flame figures are of secondary origin [26]. The eosinophilic granules contain major basic protein (MBP) and eosinophilic cationic protein (ECP), both of which exhibit cytotoxic activity and can damage collagen fibers, thereby facilitating the deposition of granular material and the subsequent formation of flame figures.

7. Diagnosis

The diagnosis of Wells syndrome is established through a comprehensive evaluation integrating clinical, laboratory, and histopathological findings. Three major diagnostic criteria are generally recognized:

1) *Clinical syndrome* — acute onset of erythematous and edematous plaques resembling cellulitis, which evolve into indurated or granulomatous lesions. The eruptions typically resolve spontaneously within two to six weeks but tend to recur.
2) *Laboratory syndrome* — presence of peripheral eosinophilia, frequently observed during the active phase of the disease, although it is neither constant nor essential for diagnosis.
3) *Histological syndrome* — dense dermal eosinophilic infiltrate with formation of characteristic flame figures, the prominence of which varies according to the stage of disease evolution.

The combination of a characteristic clinical course, transient eosinophilia, and distinctive histopathological features provides a reliable basis for differentiating Wells syndrome from other eosinophilic dermatoses and inflammatory disorders presenting with edema and induration.

8. Differential Diagnosis

Despite its well-defined clinical, biological, and histopathological characteristics, Wells syndrome requires a broad differential diagnostic approach [23, 24, 27].

Clinically, it should be differentiated from:

1) *Bacterial cellulitis and erysipelas*, due to their similar acute erythematous and edematous presentation; however, Wells syndrome is characteristically unresponsive to antibiotic therapy.

2) *Urticaria and urticarial vasculitis*, which lack lesion induration and the typical relapsing–remitting course.
3) *Angioedema and insect bite reactions*, which may present with comparable local edema and erythema.
4) *Bullous dermatoses* (e.g., bullous pemphigoid), *erythema multiforme*, and *Sweet's syndrome*, particularly in exudative variants featuring vesiculobullous lesions.

In the subacute phase, the formation of annular lesions may mimic *erythema chronicum migrans* (early Lyme disease), *localized scleroderma*, *granuloma annulare*, *erythema elevatum diutinum*, and *dermatophytosis*.

Histopathologically, Wells syndrome must be distinguished from disorders exhibiting similar eosinophilic–granulomatous infiltrates, such as *toxocariasis*, *sporotrichosis*, *post-scabetic nodules*, and other eosinophil-mediated dermatoses. In most cases, differentiation is feasible because the histopathological picture of Wells syndrome does not fully correspond to the clinical features of these entities.

Diagnostic challenges may arise in *cutaneous larva migrans*, where parasitologic identification of larvae is decisive. In *eosinophilic granulomatosis with polyangiitis* (Churg–Strauss syndrome, EGPA), overlap is greater; however, the presence of vasculitic changes and systemic involvement distinguishes it from Wells syndrome.

From a *biological perspective*, *hypereosinophilic syndrome (HES)* must also be considered. HES is defined by persistent eosinophilia (>1500/μL for over six months) with systemic involvement—most commonly hepatic, pulmonary, cardiac, neurologic, and cutaneous. The skin manifestations of HES (erythematous papules, nodules, or urticarial lesions) and its poor prognosis clearly differentiate it from Wells syndrome.

9. Etiopathogenesis

The etiopathogenesis of Wells syndrome remains incompletely understood. The condition is generally considered a hypersensitivity reaction, likely immune complex–mediated, triggered by a variety of exogenous and endogenous stimuli. Reported potential triggers include insect bites, medications, viral, bacterial, fungal, and parasitic infections, vaccinations, surgical interventions, radiotherapy, and hormonal factors such as pregnancy and menstruation [12, 24, 28].

Immunofluorescence studies have demonstrated the presence of circulating immune complexes and the deposition of complement (C3), IgM, and fibrin within dermal vessels and along the basement membrane, supporting an immune complex–mediated mechanism [26]. This reaction likely leads to extensive eosinophil degranulation, with deposition of eosinophil granule proteins on intact collagen bundles, resulting in the formation of the characteristic flame figures. Subsequently, a secondary foreign-body–type granulomatous reaction develops, characterized by palisading histiocytes and multinucleated giant cells.

Recent studies have emphasized the central role of Th2 immune pathways and IL-5–mediated eosinophil activation. Accumulation of eosinophil granule proteins within skin lesions, as well as elevated levels of IL-5 and peripheral eosinophilia during the active phase of the disease, have been demonstrated [5, 17, 29]. IL-5 is thought to contribute to the persistence of inflammation by promoting eosinophil recruitment, activation, and degranulation.

An autoimmune component is supported by positive immunofluorescent findings and by the favorable therapeutic response to systemic corticosteroids. Nevertheless, in a substantial proportion of patients, no specific triggering factor can be identified.

In summary, Wells syndrome can be regarded as a localized, Th2-driven eosinophilic dermatosis of multifactorial origin. Its pathogenesis involves a complex interplay between immune complex deposition, eosinophil activation, and cytokine-mediated inflammation, ultimately producing the distinctive histopathologic pattern with flame figures. The disease thus represents a model of immune dysregulation characterized by pronounced eosinophil-mediated tissue injury confined to the skin.

10. Associated Diseases

Wells syndrome is frequently associated with other disorders, most notably hematologic abnormalities, which may also have etiopathogenetic significance. Reported associations include myeloproliferative disorders such as chronic lymphocytic and non-lymphocytic leukemia, polycythemia vera, myelofibrosis, and thrombocytosis [11]. Although a direct causal relationship has not been definitively established, W. Aberer *et al.* [30] recommend targeted hematologic evaluation in all patients with suspected eosinophilic cellulitis.

In addition to hematologic abnormalities, the syndrome may coexist with autoimmune and rheumatologic conditions, including rheumatoid arthritis, alopecia areata or pseudopelade, and Raynaud's phenomenon [9-11]. These associations remain controversial and likely reflect a shared immunologic predisposition.

An increased prevalence of allergic disorders such as bronchial asthma, chronic urticaria, angioedema, fixed drug eruption, and migraine has also been reported [13, 31]. Isolated cases have been described in association with Ofuji's disease, hypereosinophilic syndrome, HIV infection, lymphomas, solid neoplasms, bullous pemphigoid, and herpes gestationis [32–34].

Practical relevance:

Recognition of potential associated disorders is of clinical importance, as targeted screening for underlying systemic, hematologic, or autoimmune conditions is recommended in patients diagnosed with Wells syndrome.

11. Course and Prognosis

The disease follows a chronic, relapsing course with marked individual variability, ranging from single, self-limited episodes lasting several weeks to recurrent relapses persisting over many years [3, 5, 13]. Cases with a disease duration exceeding 18 years have been reported in the literature [16].

Wells syndrome generally has a benign course without visceral involvement and typically resolves completely. In some instances, residual post-inflammatory hyperpigmentation or mild atrophic skin changes may persist following lesion regression [14, 15, 35].

Overall, the prognosis is favorable. Relapses occur unpredictably in both frequency and intensity but do not result in permanent functional impairment or systemic complications.

12. Treatment

First-line therapy:

Systemic corticosteroids remain the most commonly used and most effective treatment for acute, extensive, or symptomatic episodes, providing a rapid clinical response [3, 5, 12]. In cases with limited or localized lesions, topical corticosteroids may be appropriate [11].

Steroid-sparing and alternative agents:

In recurrent forms, or in patients with steroid intolerance or resistance to conventional therapy, favorable outcomes have been reported with dapsone, cyclosporine, methotrexate, antihistamines, and minocycline [4, 21]. These agents may serve as effective alternatives or adjuncts in maintaining remission and reducing corticosteroid dependence.

Novel therapeutic approaches:

Recent studies have demonstrated promising results with biologic agents such as omalizumab [35], mepolizumab [36], and dupilumab [37-39], as well as with the topical JAK inhibitor ruxolitinib [40].

Therapeutic strategies targeting the underlying triggering factor—such as infection, medication, or insect bite—are also essential for achieving and maintaining remission [12, 24, 28, 41, 42].

13. Practical Recommendations

1. Diagnostic approach:

In patients presenting with cellulitis-like lesions unresponsive to antibiotic therapy, an early skin biopsy with histopathological examination is essential to confirm or exclude a diagnosis of eosinophilic cellulitis (Wells syndrome).

2. Therapeutic choice:

Once the diagnosis is established, treatment should be individualized according to the extent and severity of the disease. Topical corticosteroids are suitable for limited lesions, whereas systemic corticosteroids are indicated for extensive or symptomatic forms. In cases associated with marked eosinophilia or suspected systemic involvement, periodic hematologic monitoring is recommended.

3. Steroid-sparing and novel therapies:

When a steroid-sparing strategy is necessary or in refractory cases, biologic agents may be considered as alternative or adjunctive options.

14. Conclusion

Wells syndrome is a rare but clinically and histologically well-defined eosinophilic dermatosis, characterized by a typical relapsing course and the distinctive histopathological presence of flame figures. Early recognition, timely skin biopsy, and appropriate selection of immunomodulatory therapy are essential for achieving favorable clinical outcomes.

Accurate differentiation from bacterial cellulitis and other inflammatory or bullous dermatoses is critical to prevent unnecessary antibiotic use and to ensure correct initiation of immunosuppressive or immunomodulatory treatment.

Emerging therapeutic modalities, including biologic agents and JAK inhibitors, have demonstrated encouraging results in refractory or recurrent cases. However, current evidence is limited to isolated case reports and small series, underscoring the need for larger, controlled studies to better define their efficacy, safety, and long-term outcomes.

Abbreviations

C	Complement
CRP	C-reactive Protein
ECP	Eosinophilic Cationic Protein
ESR	Erythrocyte Sedimentation Rate
Ig	Immunoglobulin
IL	Interleukin
JAK inhibitors	Janus Kinase Inhibitors
MBP	Major Basic Protein

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Conflicts of Interest

The authors declare no conflict of interest.

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