Precision Management of Chronic Spontaneous Urticaria: Biomarkers, Biologics and Novel Targeted Therapies

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***Abstract*—Chronic spontaneous urticaria (CSU) is a mast-cell-driven inflammatory skin disorder characterized by recurrent wheals, angioedema, or both for six weeks or longer. Although second-generation H1-antihistamines and omalizumab remain central to management, incomplete disease control, variable treatment response and substantial quality-of-life impairment have encouraged development of biomarker-informed and mechanism-based approaches. This review examines the evolution of CSU management toward precision medicine, with emphasis on disease endotypes, biomarkers, biologic therapies and novel targeted agents. Current evidence supports the importance of clinical activity and control measures such as the Urticaria Activity Score over 7 days and Urticaria Control Test, together with selected immunological and inflammatory biomarkers. Omalizumab established the clinical value of IgE-directed treatment, while newer strategies include Bruton tyrosine kinase inhibition with remibrutinib, interleukin-4 receptor alpha targeting with dupilumab, and mast-cell/KIT-directed approaches such as barzolvolimab. The emerging therapeutic landscape may allow treatment selection to move beyond empirical escalation toward mechanism-based decisions. However, validated biomarkers for routine treatment selection remain limited, and long-term comparative effectiveness, safety, cost and sequencing data are still developing. Clinical pharmacists can contribute through medication review, adherence support, adverse-event monitoring, patient education, pharmacovigilance and patient-reported outcome monitoring. Precision management of CSU will increasingly depend on integrating phenotype, endotype, biomarkers, treatment response and patient preferences to achieve sustained disease control and improved quality of life.**

***Index Terms*—Chronic spontaneous urticaria; Precision medicine; Biomarkers; Omalizumab; Remibrutinib; Dupilumab; Bruton tyrosine kinase; KIT; Clinical pharmacy.**

I. INTRODUCTION

Chronic urticaria is a heterogeneous inflammatory disorder with major effects on daily functioning and quality of life. Current international guidance distinguishes chronic spontaneous urticaria (CSU), in which symptoms occur without a consistent external physical trigger, from chronic inducible urticaria, in which reproducible physical or environmental stimuli are involved [1]. CSU is defined by recurrent wheals, angioedema, or both for more than six weeks [1,2].

Historically, treatment has centered on second-generation H1-antihistamines, followed by dose escalation and biologic therapy for patients with inadequate control. Omalizumab, an anti-IgE monoclonal antibody, established the importance of targeted immunotherapy in CSU. Nevertheless, responses are heterogeneous and a proportion of patients remain inadequately controlled [3–5].

The therapeutic landscape is now expanding rapidly. Remibrutinib, a highly selective oral Bruton tyrosine

kinase (BTK) inhibitor, has demonstrated efficacy in phase III CSU trials [6]. Additional strategies include cytokine-pathway modulation and mast-cell-directed therapy, creating an opportunity to develop more individualized treatment pathways [7–10].

This review summarizes current concepts in CSU pathogenesis, disease endotypes, biomarkers, established biologics and emerging targeted therapies, and discusses implications for clinical pharmacy practice.

II. SEARCH STRATEGY

A narrative review approach was used. Literature was considered from PubMed/MEDLINE, guideline publications, randomized controlled trials, systematic reviews, meta-analyses and relevant regulatory or authoritative sources. Priority was given to contemporary evidence from 2021–2026, while seminal studies were retained where necessary to explain established treatment mechanisms. Search concepts included chronic spontaneous urticaria, chronic urticaria, biomarkers, endotypes, omalizumab, remibrutinib, BTK inhibitors, dupilumab, KIT, barzolvolimab, mast cells, precision medicine and quality of life.

III. CLASSIFICATION OF URTICARIA

Table 1. Classification of urticaria

Category	Definition / characteristic	Examples
Acute urticaria	Symptoms lasting less than 6 weeks	Infection-, drug- or food-associated episodes
Chronic spontaneous urticaria	Wheals, angioedema or both for ≥6 weeks without a consistent external trigger	CSU
Chronic inducible urticaria	Symptoms reproducibly triggered by a physical/environmental stimulus	Cold, heat, pressure, cholinergic, solar or dermographism

PATHOPHYSIOLOGY OF CHRONIC SPONTANEOUS URTICARIA

Mast-cell activation is the central effector process in CSU. Activated mast cells release histamine, leukotrienes, prostaglandins, platelet-activating factor and cytokines, producing vasodilation and increased vascular permeability. These changes generate the characteristic wheal-and-flare response and contribute to pruritus and angioedema [2,4].

Two major autoimmune concepts are frequently discussed. In type I autoimmunity, also described as autoallergic CSU, IgE antibodies recognize self-antigens and activate mast cells. In type IIb autoimmune CSU, IgG autoantibodies directed against IgE or its high-affinity receptor FcεRI may contribute to mast-cell and basophil activation [4,11]. These mechanisms may overlap and are better viewed as biologic endotypes rather than rigid clinical categories.

IV. DISEASE ENDOTYPES AND PRECISION MEDICINE

Precision medicine aims to match an individual's biological disease mechanism and clinical phenotype with the most appropriate treatment. In CSU, this approach remains an evolving concept because no single biomarker currently provides a universally validated treatment-selection algorithm [4,11].

Clinical phenotyping should incorporate disease activity, disease control, angioedema burden, quality of life, comorbidities, previous treatment response and patient preferences. Biomarker information may then help characterize the underlying immune phenotype and guide future therapeutic research.

V. BIOMARKERS IN CSU

Table 2. Potential biomarkers and clinical relevance

Biomarker	Potential relevance	Current limitation
Total IgE	May help characterize IgE-associated immune phenotype and treatment response patterns	Not sufficiently specific for routine treatment selection
Specific IgE	May identify auto allergic sensitization in selected patients	Clinical utility varies by antigen and assay
Basophil activation tests	May support identification of autoimmune/type IIb phenotype	Limited availability and standardization

CRP / ESR	May reflect systemic inflammatory activity	Non-specific
Thyroid autoantibodies	May identify associated autoimmune thyroid disease	Association does not establish causality
IgG anti-FcεRI / anti-IgE	Supports autoimmune mechanisms	Testing is not standardized for routine care
Eosinophils / basophils	May provide phenotypic information	Predictive value remains inconsistent

VI. CLINICAL ASSESSMENT AND PATIENT-REPORTED OUTCOMES

Disease activity and treatment response should be assessed systematically. Urticaria Activity Score over 7 days (UAS7) quantifies wheal and itch burden. The Urticaria Control Test (UCT) assesses disease control, while the Chronic Urticaria Quality of Life Questionnaire (CU-Q2oL) evaluates the broader effect of disease on patients [1,12].

Table 3. Major outcome measures in CSU

Instrument	Main domain	Clinical use
UAS7	Wheals and itch over 7 days	Disease activity and response
UCT	Disease control	Routine follow-up
CU-Q2oL	Quality of life	Patient-centered outcome
AAS	Angioedema activity	Patients with recurrent angioedema

VII. CURRENT PHARMACOLOGICAL MANAGEMENT

Contemporary guidelines recommend a stepwise approach beginning with a standard-dose second-generation H1-antihistamine. In patients with inadequate control, dose escalation can be considered according to guideline recommendations, followed by biologic therapy, particularly omalizumab, for persistent disease [1]. Treatment should be accompanied by regular assessment of disease activity, control, quality of life and adherence.

Table 4. Current pharmacological approaches

Therapy	Target / role	Major considerations
Second-generation H1-antihistamines	H1 receptor blockade	First-line symptomatic treatment; generally favorable safety profile
Up-dosed second-generation H1-antihistamines	Enhanced H1 blockade	Used when standard dosing does not adequately control symptoms
Omalizumab	Circulating IgE	Established biologic option for antihistamine-refractory CSU
Systemic corticosteroids	Broad anti-inflammatory effect	Not suitable for long-term routine CSU control; short rescue use only in selected situations
Emerging targeted agents	Specific immune/mast-cell signaling pathways	Increasing role in refractory disease and future precision treatment

VIII. OMALIZUMAB: ESTABLISHED BIOLOGIC THERAPY

Omalizumab is a recombinant humanized monoclonal antibody that binds free IgE and reduces IgE-dependent signaling. Its clinical development changed the management of antihistamine-refractory CSU and established anti-IgE therapy as a key component of targeted treatment [3,5].

Response to omalizumab varies among individuals. Clinical factors and biomarkers such as baseline IgE have been investigated as potential predictors, but no biomarker has sufficient consistency to replace clinical assessment [4,11]. Treatment decisions therefore continue to rely on disease control, response, tolerability, access and patient preference.

REMIBRUTINIB AND BTK INHIBITION

Bruton tyrosine kinase is an intracellular signaling component involved in pathways relevant to B-cell,

basophil and mast-cell activation. Remibrutinib is a selective oral BTK inhibitor designed to suppress these signaling processes. Phase III REMIX-1 and REMIX-2 trials demonstrated clinically meaningful reductions in CSU disease activity compared with placebo [6]. The importance of remibrutinib extends beyond another drug option. Its oral route and mechanism-based action represent a shift from predominantly injectable biologics toward targeted oral therapy. Questions remain regarding comparative effectiveness versus biologics, long-term safety, treatment sequencing, cost and patient selection [6,7].

DUPILUMAB AND IL-4Rα TARGETING

Dupilumab targets the interleukin-4 receptor alpha subunit and inhibits signaling mediated by IL-4 and IL-13. Because type 2 inflammatory pathways may contribute to a subset of CSU phenotypes, IL-4Rα blockade represents an alternative mechanism-based strategy [7,8].

The clinical position of dupilumab should be interpreted in the context of evolving evidence and regulatory indications. Further studies are needed to define which CSU patients derive the greatest benefit and how IL-4Rα-directed treatment should be sequenced with anti-IgE and BTK-directed approaches.

ANTI-KIT THERAPY AND MAST-CELL TARGETING

KIT is a receptor tyrosine kinase activated by stem cell factor and is essential for mast-cell survival, development and function. Directly targeting KIT therefore represents a mechanistically attractive strategy for diseases driven by mast-cell activation.

Barzolvolimab, an anti-KIT monoclonal antibody, has generated considerable interest as an emerging CSU therapy.

Its development illustrates a shift toward targeting the mast cell itself rather than downstream mediators such as histamine. Further studies are required to determine long-term safety, optimal dosing, durability and treatment positioning [7–10].

OTHER EMERGING TARGETED APPROACHES

Table 5. Emerging therapeutic targets in CSU

Target / pathway	Therapeutic concept	Development status / research question
BTK	Block mast-cell/basophil signaling	Remibrutinib has advanced clinical evidence
KIT	Reduce mast-cell survival/function	Anti-KIT therapies are under clinical investigation
IL-4/IL-13 / IL-4Rα	Modulate type 2 inflammation	Clinical development and positioning continue
JAK/TYK2	Interrupt cytokine signaling	Investigational; efficacy and safety need clarification
MRGPRX2	Reduce non-IgE mast-cell activation	Early-stage mechanistic target
Other mast-cell pathways	Direct modulation of mast-cell activation	Research stage

IX. COMPARISON OF BIOLOGICS AND NOVEL TARGETED THERAPIES

Table 6. Comparison of selected targeted therapies

Therapy	Target	Route	Potential advantage	Important evidence gap
Omalizumab	IgE	Subcutaneous	Established efficacy and clinical experience	Predicting responders and optimal duration
Dupilumab	IL-4Rα	Subcutaneous	Targets type 2 inflammatory signaling	Optimal CSU positioning and patient selection
Remibrutinib	BTK	Oral	Mechanism-based oral targeted therapy	Long-term and comparative evidence
Barzolvolimab	KIT	Subcutaneous	Direct mast-cell targeting	Long-term safety, sequencing and durability
Other investigational agents	Multiple pathways	Varies	Potential precision/endotype-directed therapy	Efficacy, safety and clinical positioning

TREATMENT-RESISTANT CSU

A subset of patients remains inadequately controlled despite guideline-directed treatment. Apparent treatment resistance may reflect severe disease biology, autoimmune endotypes, adherence problems, incorrect diagnosis, uncontrolled inducible triggers, comorbid disease or inadequate monitoring. Before labeling a patient as refractory, clinicians should reassess diagnosis, adherence, treatment exposure and disease activity.

Novel targeted therapies may provide alternatives for patients with persistent symptoms, but treatment sequencing should be individualized. Comparative trials and real-world studies will be important in determining whether specific endotypes benefit preferentially from particular mechanisms.

QUALITY OF LIFE AND PATIENT-CENTERED CARE

CSU can interfere with sleep, work, social functioning and psychological well-being. Angioedema may add substantial functional and emotional burden. Consequently, disease control should not be judged solely by visible wheals. Patient-reported outcomes should be integrated into treatment decisions and longitudinal follow-up [1,12].

Shared decision-making is increasingly relevant as treatment options expand. Route of administration, speed of response, expected duration, adverse effects, affordability and patient preference can influence treatment selection.

X. ROLE OF THE CLINICAL PHARMACIST

Clinical pharmacists can support precision management through comprehensive medication review, identification of possible drug triggers, adherence assessment, patient education, monitoring for adverse drug reactions and interactions, and pharmacovigilance.

Pharmacists can also reinforce standardized use of UAS7 and UCT and help patients understand the purpose and expected benefits of targeted therapy.

- Medication reconciliation and assessment of suspected drug-related triggers.
- Counseling on correct use and adherence to antihistamines.
- Education regarding biologic and targeted therapies.
- Monitoring adverse drug reactions and clinically relevant interactions.
- Support for UAS7, UCT and quality-of-life monitoring.
- Patient counseling on treatment expectations and follow-up.
- Contribution to pharmacovigilance and real-world evidence generation.
- Participation in multidisciplinary and shared-decision-making models.

PHARMACOVIGILANCE AND SAFETY

As the number of targeted therapies increases, safety surveillance becomes increasingly important. Monitoring should consider agent-specific adverse effects, infections where relevant, laboratory abnormalities, hypersensitivity, injection-related reactions and clinically significant drug interactions. Long-term real-world pharmacovigilance is particularly important for newer mechanisms because clinical trials may not detect uncommon or delayed adverse events.

PHARMACOECONOMIC CONSIDERATIONS

Biologics and novel targeted therapies may impose substantial costs compared with conventional antihistamines. However, the economic burden of uncontrolled CSU includes repeated healthcare visits, loss of productivity, sleep impairment and reduced quality of life. Future pharmacoeconomic studies should compare total healthcare costs and patient-centered outcomes across treatment sequences, particularly in resource-constrained healthcare systems.

CURRENT CHALLENGES

- Absence of a universally validated biomarker-guided treatment algorithm.
- Variable response to existing biologic therapy.
- Limited ability to predict individual treatment response.
- High cost and unequal access to advanced therapies.
- Limited long-term real-world safety data for newer agents.
- Uncertainty regarding optimal treatment sequencing.

- Need for head-to-head comparative effectiveness studies.
- Need to integrate patient preferences into precision treatment decisions.

XI. FUTURE PERSPECTIVES

The future of CSU management is likely to involve increasingly individualized treatment based on clinical phenotype, immune endotype, biomarkers and treatment-response patterns. Oral BTK inhibition, mast-cell/KIT targeting and cytokine-directed therapy demonstrate how advances in disease biology can generate new therapeutic strategies. Artificial intelligence and digital health may eventually support symptom tracking, prediction of treatment response and remote monitoring, although these applications require validation before routine clinical use.

Research priorities include identification of robust predictive biomarkers, comparative trials between targeted mechanisms, long-term safety studies, pharmacoeconomic evaluations, treatment de-escalation strategies and real-world evidence from diverse populations.

XII. DISCUSSION

The evolution of CSU therapy demonstrates a broader transition in clinical pharmacotherapy from symptom-based treatment toward mechanism-based intervention. Antihistamines remain essential, while omalizumab provides an established targeted option for inadequately controlled disease. Remibrutinib introduces a clinically important oral BTK-directed strategy, and anti-KIT therapies offer the possibility of directly modifying mast-cell biology [6–10].

However, precision medicine should not be equated with simply selecting a new molecular target. True precision management requires integration of disease activity, patient-reported outcomes, endotype, safety, treatment access, adherence and patient preference. At present, biomarker-guided prescribing remains an emerging rather than fully established practice in CSU.

XIII. CONCLUSION

Chronic spontaneous urticaria is evolving from a predominantly symptom-managed condition into a biologically characterized disease with an expanding targeted therapeutic landscape. Omalizumab established anti-IgE therapy, while remibrutinib has demonstrated the potential of oral BTK inhibition. IL-4Rα-directed treatment and KIT-directed mast-cell strategies further broaden therapeutic possibilities. Nevertheless, validated biomarkers for routine treatment selection remain limited. Future precision management should combine clinical phenotype, disease endotype, biomarkers, patient-reported outcomes and treatment-response data. Clinical pharmacists have an important role in medication optimization, patient education, adherence, safety monitoring and pharmacovigilance. Continued translational and comparative research will be essential to transform emerging biological knowledge into practical individualized CSU care.

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