Original Article

Sex-Related Differences in Laboratory and Autoreactivity Biomarkers in Chronic Spontaneous Urticaria

Muhammed Burak Yücel[1], Esranur Ünal[2]
[1]Clinic of Dermatology, Memorial Kayseri Hospital, Kayseri, Türkiye
[2]Department of Dermatology, Ufuk University Faculty of Medicine, Ankara, Türkiye

Abstract

Aim: Chronic spontaneous urticaria (CSU) affects female more frequently than men, but whether this epidemiological difference is accompanied by a reproducible sex-related laboratory phenotype remains unclear. To evaluate and quantitatively synthesize sex-related differences in laboratory, inflammatory, autoimmune, and autoreactivity biomarkers in CSU.

Materials and Methods: MEDLINE/PubMed, Embase.com, Scopus, and Web of Science Core Collection were searched from inception through 16 June 2026. Eligible studies either directly compared female and male patients with CSU or reported sex-stratified data sufficient for effect estimation. Two investigators independently screened the records and assessed the risk of bias. One investigator extracted the data, and a second investigator independently verified all entries against the source reports. Random-effects pooling was performed using restricted maximum likelihood estimation with Hartung-Knapp inference.

Results: Of 4,083 unique records screened, 25 studies were included. Ten adult or mixed-population studies contributed to the primary quantitative synthesis of autologous serum skin test (ASST) positivity. The pooled odds ratio (OR) for female versus men was 1.57 [95% confidence interval (CI) 0.80–3.09; $P = 0.168$], with substantial heterogeneity ($I^2 = 72.3\%$) and a 95% prediction interval of 0.23–10.53. Leave-one-out analyses did not materially change the estimate. A pediatric-inclusive sensitivity analysis also remained non-significant (OR 1.73, 95% CI 0.96–3.11). Individual studies suggested enrichment in females for selected findings related to thyroid autoimmunity, coagulation, platelets, basophil activation tests, and osteopontin, whereas inflammatory, eosinophil, and IgE results varied across populations and treatment settings.

Conclusion: Sex-related laboratory differences in CSU appear to be biomarker- and population-specific rather than components of a uniform female or male laboratory phenotype. The pooled ASST analysis did not demonstrate a statistically robust sex difference. Standardized sex-stratified reporting is required before sex-specific laboratory or treatment algorithms can be justified.

Keywords: Autoimmunity, autoreactivity, biomarkers, chronic spontaneous urticaria, sex differences

Introduction

Chronic spontaneous urticaria (CSU) is a mast cell-driven disease characterized by the recurrent occurrence of wheals, angioedema, or both for longer than 6 weeks in the absence of a specific external trigger.[1,2] Beyond visible skin lesions, CSU imposes a substantial burden through itch, sleep disruption, impaired quality of life, reduced work productivity, and repeated healthcare use.[2,3] Female are affected more frequently than men, and recent large-scale data suggest that the female predominance becomes particularly evident during adulthood and is accompanied by greater disease burden and poorer control in midlife.[4,5] These observations suggest that biological sex is not merely an epidemiological descriptor but may modify the inflammatory and autoimmune architecture of CSU.

Current concepts place type I autoallergy and type IIb autoimmunity at the center of CSU pathogenesis.[6] Type I autoallergy involves IgE antibodies against self-antigens,

Submission: 20-Jun-2026 **Epub:** 19-Oct-2026
Acceptance: 14-Sep-2026

Access this article online
Quick Response Code:
Website: www.turkjdermatol.com
DOI: 10.4274/tjd.galenos.2026.16779

Address for correspondence: Muhammed Burak Yücel, Clinic of Dermatology, Memorial Kayseri Hospital, Kayseri, Türkiye
Email: drmburakyucel@gmail.com
ORCID ID: 0000-0003-0130-8229

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given.

How to cite this article: Yücel MB, Ünal E. Sex-related differences in laboratory and autoreactivity biomarkers in chronic spontaneous urticaria. Turk J Dermatol. [Epub Ahead of Print]

 Copyright© 2026 The Author(s). Published by Galenos Publishing House on behalf of Turkish Society of Dermatology.

whereas type IIb autoimmune CSU is characterized by functional IgG antibodies that activate mast cells and basophils and are directed against IgE or its high-affinity receptor. The latter endotype is commonly associated with a more severe and treatment-refractory clinical course, basopenia and eosinopenia, low total IgE, elevated inflammatory markers, positive autologous serum skin testing, and positive basophil functional assays.[6-9] Because these tests reflect distinct biological domains, including systemic inflammation, coagulation activation, circulating granulocyte patterns, thyroid autoimmunity, immunoglobulin profiles, and functional serum autoreactivity, sex-specific evaluation may provide further valuable insights that cannot be inferred from clinical outcomes alone.

Sex-related immune regulation may be influenced by X-chromosome-linked immune genes, sex hormones, reproductive transitions, body composition, and differences in autoimmune susceptibility.[10,11] Kasperska-Zajac et al.[11] summarized experimental and clinical observations suggesting that estrogens may enhance mast-cell and basophil histamine release, whereas progesterone and testosterone may exert inhibitory effects on mast-cell secretion; they also highlighted possible modulation of urticaria during the menstrual cycle, pregnancy, menopause, and exposure to exogenous hormones. However, much of this evidence is experimental, case-based, or derived from small cohorts, and lower dehydroepiandrosterone sulfate concentrations have been reported in chronic urticaria irrespective of sex and autologous serum skin test (ASST) status.[11] Thus, biological plausibility has outpaced direct clinical evidence in supporting a reproducible female-versus-male laboratory phenotype. A broader systematic review identified 26 reports addressing sex and gender differences across epidemiology, clinical characteristics, comorbidities, treatment, and quality of life, but laboratory and functional autoreactivity findings were scattered and were not quantitatively synthesized.[4] The present study therefore aimed to identify and critically appraise evidence on female-versus-male differences in laboratory, inflammatory, autoimmune, and autoreactivity biomarkers among patients with CSU and, where data permitted, to quantitatively synthesize these differences.

Materials and Methods

Protocol and Reporting Framework

This evidence synthesis was conducted and reported in accordance with the PRISMA 2020 statement.[12] An operational protocol specifying eligibility criteria, outcome domains, deduplication rules, screening procedures, and sensitivity strata was finalized before independent title/abstract screening. The protocol was not prospectively registered.

Eligibility Criteria

We included cohort (observational), cross-sectional, case-control, registry, and interventional studies that provided baseline or pretreatment data for patients with CSU and reported a direct comparison between female and male patients for at least one laboratory, serological, hematological, inflammatory, coagulation, autoimmune, or functional autoreactivity outcome. Studies using the historical term chronic idiopathic urticaria were eligible when the population was clinically compatible with CSU and did not predominantly consist of inducible urticaria or urticarial vasculitis. Pediatric studies and treatment-selected cohorts were retained in prespecified sensitivity strata.

We excluded acute urticaria, isolated chronic inducible urticaria, urticarial vasculitis, hereditary or acquired bradykinin-mediated angioedema, case reports, narrative reviews, and studies that reported sex only as a covariate, without providing a direct female-versus-male laboratory estimate or sufficient data to calculate one. Studies comparing each sex with healthy controls, without a within-CSU female-versus-male comparison, did not contribute to the focused synthesis. When multiple reports described overlapping populations, the most complete report was retained and secondary reports were linked but not counted separately.

Information Sources and Search Strategy

MEDLINE/PubMed, Embase.com, Scopus, and Web of Science Core Collection were searched from database inception through 16 June 2026. The PubMed, Scopus, and Web of Science searches were run on 15 June 2026, and the Embase search was run on 16 June 2026. Search concepts combined chronic urticaria terminology and sex/gender terms with laboratory biomarkers, including inflammatory, autoimmune, coagulation, hematological, and functional autoreactivity biomarkers. No limits regarding date, language, human subjects, or publication type were applied. Full electronic search strategies for all databases, together with the search dates and result counts, are provided in Supplementary Table 1. Search screenshots and raw NBIB/RIS exports were retained for audit purposes. The 26 reports included in the previous systematic review were reconciled against the master database using DOI, PMID, author/year, and title. Fifteen were already represented in the database set, and 11 were unique citation-only records. The 11 unique records were screened using the same eligibility criteria; none advanced to full-text assessment.[4]

Study Selection and Data Extraction

The four database exports were merged and deduplicated using a prespecified Python-based workflow. Exact PMID matches were merged; exact normalized title plus first-author matches were merged when publication years were compatible; DOI matches were merged only when titles were compatible; and exact cross-database title matches were merged after checks of notice type and year. Retractions, corrections, and expressions of concern were kept separate from the original articles. Records from conference supplements that shared an issue-level DOI were not merged when their titles differed. The 6,760 raw database records were reduced to 4,072 unique publications, and the deduplicated library was exported to a structured screening workbook. Two investigators independently screened all 4,072 database records and the 11 unique citation-only records. Records considered potentially eligible by either investigator underwent full-text assessment. Differences at the title/abstract or full-text levels were resolved through discussion and consensus; no third adjudicator was required. One primary exclusion reason was recorded for each full-text exclusion, and companion publications were linked to prevent double-counting.

A standardized extraction form captured study design, country, setting, recruitment period, diagnostic terminology, age range, treatment status, inclusion and exclusion criteria, total sample size, female and male sample sizes, assay method, threshold definition, timing of sampling, and sex-stratified outcome data. For binary outcomes, event and total counts were extracted. For continuous outcomes, means and standard deviations were preferred; medians and measures of dispersion were retained without forced conversion when distributional assumptions were uncertain. Reconstructed denominators were explicitly labeled and restricted to sensitivity analyses. One investigator extracted all eligible data using the standardized form, and the second investigator independently verified each extracted entry against the source report before analysis. Discrepancies were resolved through discussion and consensus.

Outcomes

The prespecified outcome domains were: (i) systemic inflammatory and coagulation markers, including CRP, ESR, D-dimer, fibrinogen, and leukocyte indices; (ii) circulating eosinophil and basophil counts or categorical eosinopenia/basopenia; (iii) total IgE and immunoglobulin profiles; (iv) thyroid and other autoantibodies; and (v) functional or surrogate markers of autoreactivity, including the ASST, autologous plasma skin test (APST), basophil activation test (BAT), and basophil histamine release assay. Emerging biomarkers, such as osteopontin, were summarized in a separate exploratory domain. Laboratory abnormalities that are expected to differ physiologically by sex, such as hemoglobin levels or red blood cell counts, were not interpreted as CSU-specific signals unless supported by disease-specific evidence.

Risk-of-bias Assessment

Two investigators independently assessed risk of bias using the revised JBI analytical cross-sectional tool, with design-specific interpretation for cohort, registry, case-control, treatment-selected, and legacy-CU reports because the study synthesized baseline or pretreatment sex-stratified laboratory comparisons.[13] Overall study-level judgments were categorized as low-risk, some concerns, or high-risk using predefined decision rules, without calculating a numerical quality score. Disagreements were resolved through discussion and consensus.

Ethics Committee Approval and Informed Consent

This study was based exclusively on a secondary analysis of aggregate data extracted from previously published reports. No new data were collected from human participants, no intervention was performed, and no individual-level identifiable patient information was accessed. Therefore, ethics committee approval and informed consent were not required.

Statistical Analysis

Female constituted the index group, and men constituted the reference group. Binary outcomes were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). Continuous outcomes measured on the same scale were summarized as mean differences. Random-effects models were estimated using restricted maximum likelihood, and Hartung-Knapp CIs were applied when at least three clinically and methodologically compatible studies were available. Heterogeneity was reported using Cochran's Q, I^2, τ^2, and a 95% prediction interval. The stability of the primary synthesis was examined using leave-one-out analysis.

Planned sensitivity analyses examined the inclusion of pediatric cohorts in the ASST quantitative synthesis. Treatment-selected, phenotype-restricted, legacy-CU, and reconstructed-data studies were retained as contextual sensitivity strata and were interpreted separately when clinical or methodological compatibility was insufficient for pooling. For individual studies containing a zero cell, a continuity correction of 0.5 was added to all four cells of the corresponding 2 × 2 table. Small-study effects for the 10-study primary ASST analysis were assessed in an exploratory analysis using a funnel plot and Egger regression; these findings were interpreted

cautiously because only 10 studies were available. Outcomes represented by only two clinically heterogeneous studies were not considered confirmatory pooled analyses. Data checks were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Deduplication and quantitative synthesis computations, including the REML random-effects models, Hartung-Knapp inference, prediction intervals, leave-one-out analyses, and Egger regression, were performed in Python (version 3.13) using SciPy (version 1.17.0).

Results

Study Selection and Characteristics

The four database searches identified 6,760 records: 1,588 from MEDLINE/PubMed, 2,778 from Scopus, 666 from Web of Science, and 1,728 from Embase. Conservative within-database deduplication removed 2,688 records, leaving 4,072 unique database records. Reconciliation of the 26-report prior-review citation map identified 15 records already represented in the master database and 11 unique citation-only records; all 11 were excluded at the title/abstract stage because they did

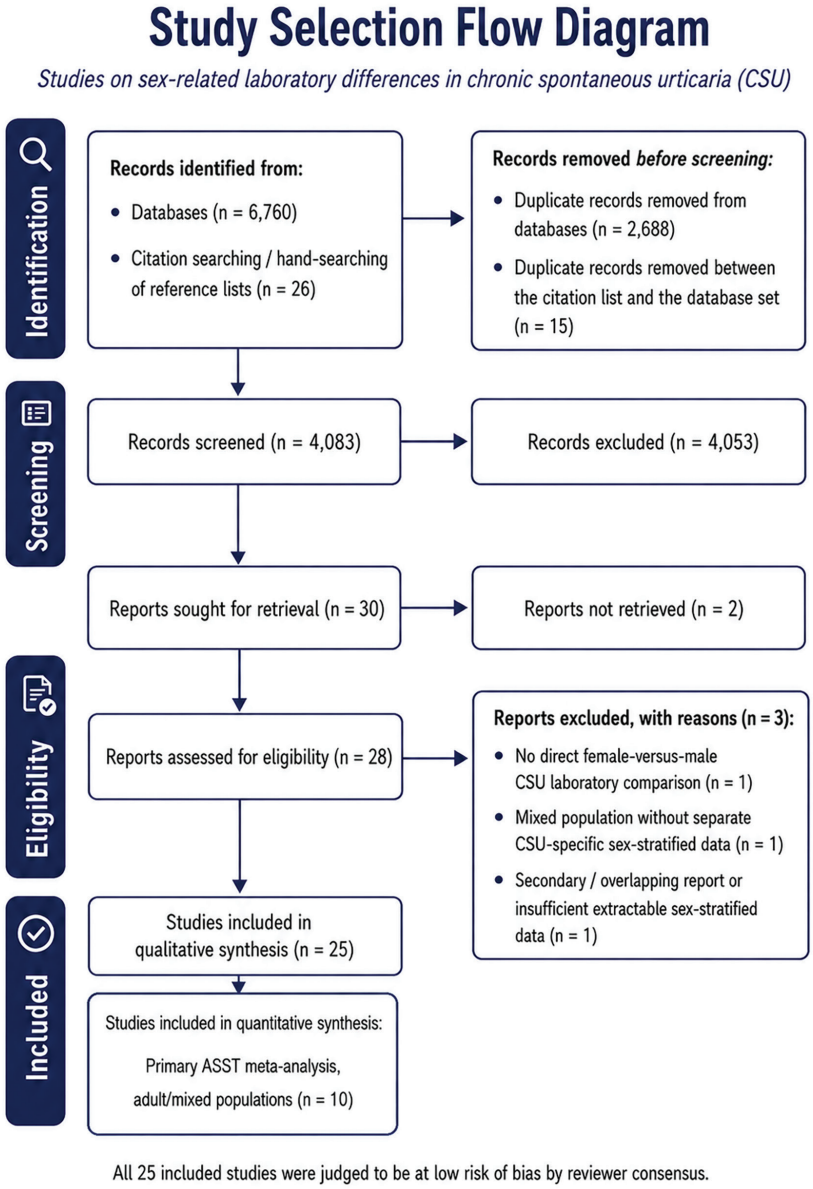


Figure 1. PRISMA study selection flow diagram. Flow of records through identification, deduplication, screening, eligibility assessment, and inclusion. Citation searching comprised the 26-report prior-review reference map and hand-searching. Two reports were not retrieved. Twenty-five studies were included in the qualitative synthesis, and 10 adult/mixed studies contributed to the primary ASST meta-analysis
CSU: Chronic spontaneous urticaria, ASST: Autologous serum skin test

Table 1. Characteristics of the included studies

Study	Design/setting	Population	Female/male	Primary relevant domain	Role in synthesis
Kulthanan et al.[22]	Single-center observational; Thailand	Chronic idiopathic urticaria, n = 85	69/16	ASST	Quantitative
Aktar et al.[23]	Single-center cross-sectional; Türkiye	Chronic urticaria, n = 50	32/18	ASST/APST; reaction diameters	Quantitative; legacy-CU sensitivity
Hossein Zadeh Attar et al.[21]	Retrospective cross-sectional; Germany	CSU, n = 87	56/31	CD63 BAT; total IgE	Quantitative + narrative
Asero[14]	Cross-sectional severe CSU; Italy	Antihistamine-refractory CSU, n = 132	88/44	D-dimer; CRP	Quantitative
Kolkhir et al.[18]	Two-center retrospective; Germany/Russia	CSU, n = 1,613	74% female	Eosinophil/basophil; CRP; IgE	Narrative/continuous
Lai and Sarkan[24]	Cross-sectional; Malaysia	CSU, n = 59	45/14	ASST; autoimmune antibodies	Quantitative
Sartorio et al.[15]	Multicenter retrospective; Italy	CSU with angioedema, n = 34	20/14	CRP/ESR; thyroid antibodies	Sensitivity only
Kocatürk et al.[5]	International registry baseline	CSU, n = 4,136	2,994/1,142	CRP; leukocytes; age strata	Quantitative/narrative
Liu et al.[19]	Cross-sectional biomarker study; China	Moderate-severe CSU, n = 36	18/18	Osteopontin; full laboratory panel	Quantitative
Sirufo et al.[25]	Prospective non-interventional; Italy	Omalizumab-treated refractory CSU, n = 42	26/16	ASST; total IgE	Quantitative; treatment-selected
Confino-Cohen et al.[16]	Retrospective database; Israel	Specialist-diagnosed chronic urticaria, n = 12,778	8,472/4,306	MPV; autoimmune serology	Quantitative; legacy-CU sensitivity
Chanprapaph et al.[26]	Retrospective single-center; Thailand	CSU with complete thyroid antibodies, n = 300	254/46	Thyroid autoimmunity; anti-TPO/anti-Tg; ASST/APST	Quantitative
Preis et al.[17]	Retrospective monocentric cohort; Germany	Omalizumab-treated CSU, n = 250	150/100	IgE; CRP; eosinophils; leukocytes; platelets	Quantitative; treatment-selected sensitivity
Curto-Barredo et al.[27]	Retrospective observational; Spain	Adult CSU, n = 549	402/147	ASST	Quantitative
Wan and Wu[36]	Prospective case-control; Taiwan	Legacy CIU, n = 60	Not reported	Anti-thyroid antibodies	Narrative only
Mohammed[28]	Prospective treatment cohort; Iraq	Chronic urticaria, n = 77	48/29	ASST	Quantitative; legacy-CU sensitivity
Safari et al.[29]	Cross-sectional; Iran	Idiopathic chronic urticaria, n = 38	25/13	ASST	Quantitative; legacy-CU sensitivity
Sahiner et al.[31]	Retrospective pediatric cohort; Türkiye	Pediatric CSU, n = 100; ASST n = 45	44/56; tested 24/21	ASST	Pediatric sensitivity
Moos et al.[20]	Retrospective; Poland	Adult CSU, n = 68	52/16	NEU; LYM; NLR; CRP	Narrative; figure-only dispersion
Özçeker et al.[33]	Retrospective pediatric cohort; Türkiye	Pediatric CSU, n = 87	38/49	ASST; IgE; eosinophils; autoantibodies	Narrative pediatric sensitivity
Najib et al.[35]	Retrospective cohort; USA	Legacy CIU, n = 236	Approximately 181/55 overall; tested subsets vary	BAT-CD203c; thyroid autoimmunity	Quantitative; full journal article; legacy-CU sensitivity
Collet et al.[34]	Cross-sectional clinical study; France	Legacy chronic urticaria, n = 45	16/29	Autoimmune thyroid disease; thyroid hormones/antibodies	Quantitative; legacy-CU sensitivity
Godse[32]	Cross-sectional pediatric study; India	Pediatric chronic urticaria, n = 17	9/8	ASST	Quantitative; pediatric sensitivity
Asero[10]	Comparative correspondence/short report; Italy	Legacy chronic urticaria, n = 257	188/69	ASST; thyroid autoantibodies	Quantitative; legacy-CU sensitivity
Caproni et al.[30]	Cross-sectional comparative study; Italy	Legacy CIU, n = 68	46/22	ASST; HRA; autoimmune laboratory panel	Quantitative; legacy-CIU sensitivity

ASST: Autologous serum skin test, APST: Autologous plasma skin test, BAT: Basophil activation test, CD63: Cluster of differentiation 63, CD203c: Cluster of differentiation 203c, CIU: Chronic idiopathic urticaria, CRP: C-reactive protein, CSU: Chronic spontaneous urticaria, CU: Chronic urticaria, CURE: Chronic Urticaria Registry, ESR: Erythrocyte sedimentation rate, HRA: Histamine-releasing activity, IgE: Immunoglobulin E, LYM: Lymphocyte count, MPV: Mean platelet volume, NEU: Neutrophil count, NLR: Neutrophil-to-lymphocyte ratio, anti-Tg: Anti-thyroglobulin antibody, anti-TPO: Anti-thyroid peroxidase antibody, N/n: Number of patients, USA: United States of America.

Table 2. Key sex-related laboratory and autoreactivity findings

Domain/outcome	Study	Female	Male	Effect estimate	Interpretation/qualification
Elevated D-dimer	Asero[14]	50/88	16/44	OR 2.30 (95% CI 1.09–4.85)	Higher in female; severe refractory CSU
Elevated CRP, all ages	Kocatürk et al.[5]	498 (27.7%)	180 (24.5%)	Not directly reported	No significant overall sex difference; 27.7% in female versus 24.5% in men
Elevated CRP, age 51-65	Kocatürk et al.[5]	156/457 (34.1%)	45/178 (25.3%)	OR ≈1.53 (95% CI 1.03–2.25)	Sensitivity estimate based on a reconstructed female denominator
Elevated CRP and/or ESR	Sartorio et al.[15]	10/20	1/14	OR 13.00 (95% CI 1.42–119.08)	Angioedema-restricted; sparse data
Absolute eosinophils ($\times 10^9$/L)	Kolkhir et al.[18]	Median 0.14	Median 0.17	$P = 0.001$	Lower in female; dispersion unavailable
Absolute eosinophils ($\times 10^9$/L)	Liu et al.[19]	0.12 ± 0.09	0.14 ± 0.14	MD -0.02 (95% CI -0.10 to 0.06)	No significant difference
CD63 BAT positive	Hossein Zadeh Attar et al.[21]	41/56	15/31	OR 2.92 (95% CI 1.16–7.31)	Higher in female
ASST positive	Kulthanan et al.[22]	16/69	5/16	OR 0.66 (95% CI 0.20–2.20)	No female excess
ASST positive	Lai and Sarkan[24]	25/45	2/14	OR 7.50 (95% CI 1.50–37.46)	Higher in female; small male group
Total IgE (IU/mL)	Liu et al.[19]	94.21 ± 59.87	124.28 ± 72.54	MD -30.07 (95% CI -73.52 to 13.38)	Numerically higher in men; not significant
Osteopontin (ng/mL)	Liu et al.[19]	5.41 ± 1.58	4.39 ± 0.77	MD 1.02 (95% CI 0.21–1.83)	Higher in female; novel biomarker
ASST positive	Sirufo et al.[25]	8/26	8/16	OR 0.44 (95% CI 0.12–1.61)	No significant female excess; omalizumab-treated cohort
Total IgE	Sirufo et al.[25]	409 ± 76.1	355 ± 66.8	MD 54.00 (95% CI 10.10–97.90)	Higher in female; small selected cohort
High MPV	Confino-Cohen et al.[16]	2,652/8,472	1,010/4,306	OR 1.49 (95% CI 1.37–1.62)	Higher in female; legacy CU
Anti-TPO positive	Confino-Cohen et al.[16]	508/8,472	90/4,306	OR 2.99 (95% CI 2.38–3.75)	Higher in female; legacy CU
Anti-thyroglobulin positive	Confino-Cohen et al.[16]	117/8,472	21/4,306	OR 2.86 (95% CI 1.79–4.55)	Higher in female; legacy CU
ANA positive	Confino-Cohen et al.[16]	208/8,472	40/4,306	OR 2.68 (95% CI 1.91–3.77)	Higher in female; legacy CU
Rheumatoid factor positive	Confino-Cohen et al.[16]	180/8,472	53/4,306	OR 1.74 (95% CI 1.28–2.37)	Higher in female; legacy CU
ASST positive	Aktar et al.[23]	18/32	5/18	OR 3.34 (95% CI 0.96–11.62)	Historical CU; positive association reported
APST positive	Aktar et al.[23]	27/32	9/18	OR 5.40 (95% CI 1.43–20.38)	Historical CU; higher in female
Thyroid autoimmunity	Chanprapaph et al.[26]	69/254	3/46	OR 5.35 (95% CI 1.61–17.80)	Higher in female
Anti-TPO positive	Chanprapaph et al.[26]	51/254	2/46	OR 5.53 (95% CI 1.30–23.56)	Higher in female
Anti-thyroglobulin positive	Chanprapaph et al.[26]	52/254	3/46	OR 3.69 (95% CI 1.10–12.37)	Higher in female
ASST positive	Chanprapaph et al.[26]	78/108	13/17	OR 0.80 (95% CI 0.24–2.65)	No significant sex association; tested subset
APST positive	Chanprapaph et al.[26]	77/108	11/17	OR 1.35 (95% CI 0.46–3.98)	No significant sex association; tested subset
ASST positive	Curto-Barredo et al.[27]	181/402	44/147	OR 1.92 (95% CI 1.28–2.87)	Higher in female; adult CSU
ASST positive	Mohammed[28]	23/48	11/29	OR 1.51 (95% CI 0.59–3.85)	No significant association; legacy CU
ASST positive	Safari and Sayemiri[29]	10/25	5/13	OR 1.07 (95% CI 0.27–4.22)	No significant association; small legacy CIU

Table 2. Continued

Domain/outcome	Study	Female	Male	Effect estimate	Interpretation/qualification
ASST positive	Sahiner et al.[31]	15/24	6/21	OR 4.17 (95% CI 1.19–14.64)	Higher in girls; pediatric sensitivity
Total anti-thyroid antibodies	Wan and Wu[36]	25%	33%	Not calculable	Sex denominators omitted; narrative only
NEU, LYM, and NLR	Moos et al.[20]	Graphically reported	Graphically reported	Not effect-estimable	Significant sex-related differences were reported, but exact dispersion measures were unavailable
High total IgE (> 100 kU/L)	Preis et al.[17]	34/85	34/53	OR 0.37 (95% CI 0.18–0.76)	Less frequent in female; omalizumab-treated cohort
Elevated CRP (> 0.5 mg/dL)	Preis et al.[17]	47/119	22/77	OR 1.63 (95% CI 0.88–3.02)	No significant difference; treatment-selected
Eosinophils > 4%	Preis et al.[17]	7/114	7/72	OR 0.61 (95% CI 0.20–1.81)	Overall 3-category distribution $P = 0.609$
ASST positive	Özçeker et al.[33]	Not reported	Not reported	No significant association	Pediatric CSU; sex × ASST cells omitted
BAT-CD203c positive	Najib et al.[35]	19/50	1/8	OR 4.29 (95% CI 0.49–37.64)	Female-directed but imprecise; sparse tested subgroup in a full legacy-CIU cohort
Composite thyroid autoimmunity	Najib et al.[35]	50/146	7/42	OR 2.60 (95% CI 1.08–6.28)	Higher in female; anti-TPO and/or anti-Tg
Autoimmune thyroid disease	Collet et al.[34]	8/16	0/29	Corrected OR 59.0 (95% CI 3.08–1130.09)	Zero male events; Fisher exact $P < 0.001$; legacy CU
ASST positive	Godse[32]	4/9	2/8	OR 2.40 (95% CI 0.30–19.04)	Small pediatric chronic-urticaria cohort
ASST positive	Asero[10]	143/188	24/69	OR 5.96 (95% CI 3.28–10.84)	Markedly higher in female; legacy-CU short report
Thyroid autoantibodies among ASST-positive patients	Asero[10]	52/143	8/24	OR 1.14 (95% CI 0.46-2.85)	No significant sex difference; subgroup only
ASST positive	Caproni et al.[30]	14/46	9/22	OR 0.63 (95% CI 0.22-1.82)	No significant sex difference; legacy CIU

The female denominator for the CURE 51–65-year subgroup was reconstructed from the published data and was therefore used only in sensitivity interpretation. ANA: Antinuclear antibody, anti-Tg: Anti-thyroglobulin antibody, anti-TPO: Anti-thyroid peroxidase antibody, APST: Autologous plasma skin test, ASST: Autologous serum skin test, BAT: Basophil activation test, CD63: Cluster of differentiation 63, CD203c: Cluster of differentiation 203c, CI: Confidence interval, CIU: Chronic idiopathic urticaria, CRP: C-reactive protein, CSU: Chronic spontaneous urticaria, CU: Chronic urticaria, CURE: Chronic Urticaria Registry, ESR: Erythrocyte sedimentation rate, IgE: Immunoglobulin E, LYM: Lymphocyte count, MD: Mean difference, MPV: Mean platelet volume, NEU: Neutrophil count, NLR: Neutrophil-to-lymphocyte ratio, OR: Odds ratio

Table 3. Database search yield and all-source study selection

Database	Search date	Records	Status
MEDLINE/PubMed	15 June 2026	1,588	Complete
Scopus	15 June 2026	2,778	Complete
Web of Science Core Collection	15 June 2026	666	Complete
Embase.com	16 June 2026	1,728	Complete
Total before deduplication	-	6,760	Final database-identification count
Duplicates removed	-	2,688	Conservative deduplication
Unique publications for screening	-	4,072	4,072 unique database records + 11 unique citation-only records = 4,083 screened; 4,053 title/abstract exclusions; 30 reports sought; 28 assessed; 25 studies included; 3 reports excluded; 2 not retrieved

No date, language, human-only, or publication-type limits were applied. Citation searching identified 26 prior-review reports; 15 were duplicates of database records and 11 were unique additions. All 11 unique citation records were excluded at title/abstract stage.

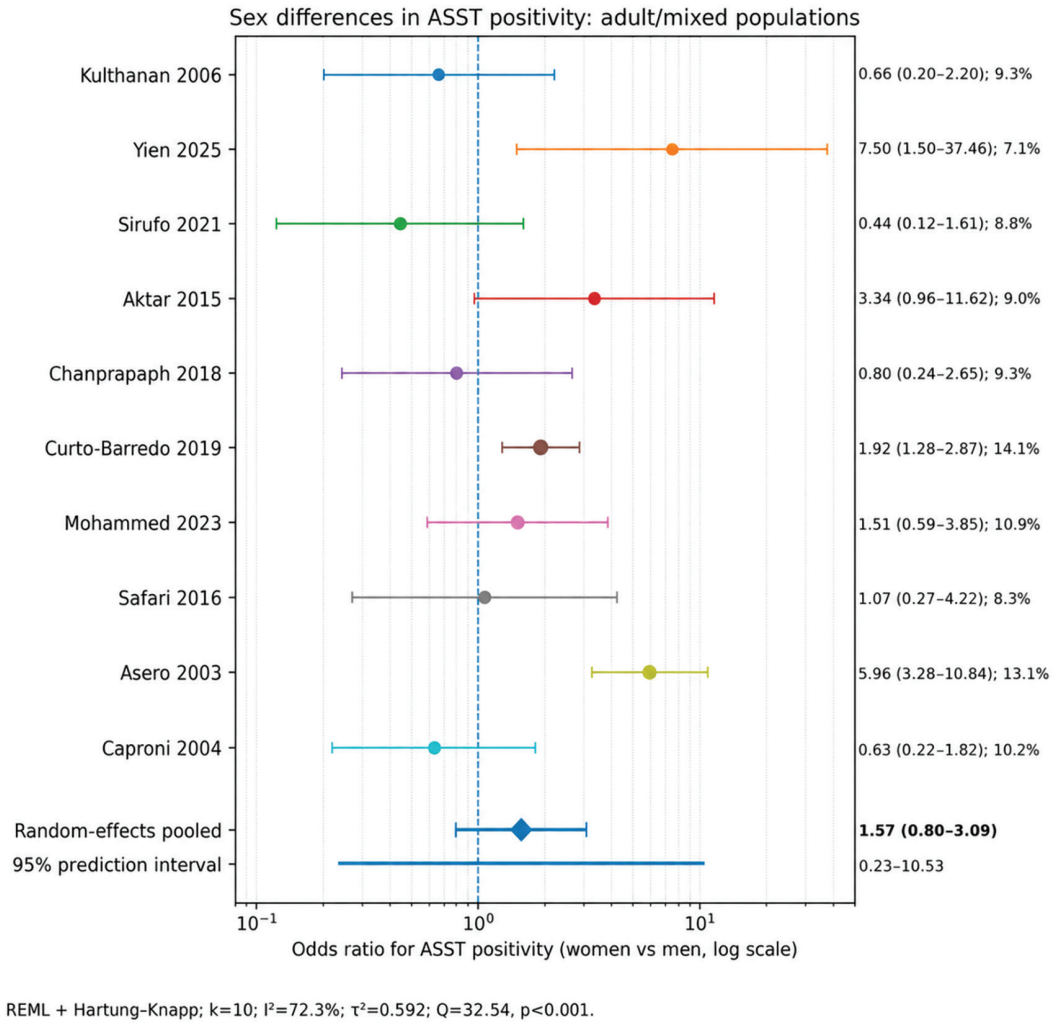


Figure 2. Forest plot of sex differences in ASST positivity in adult/mixed chronic urticaria populations. Study-specific and pooled odds ratios compare female with men; values greater than 1 indicate higher ASST positivity in female. The pooled estimate was calculated using a REML random-effects model with Hartung-Knapp confidence intervals. Marker size reflects random-effects weight, and the horizontal line beneath the pooled estimate represents the 95% prediction interval
ASST: Autologous serum skin test

not provide an eligible female-versus-male CSU laboratory comparison. Across all sources, 4,083 unique records were screened, 4,053 were excluded, and 30 reports were sought. Two reports were not retrieved; 28 were assessed; 3 were excluded; and 25 studies were included (Figure 1). Five reports from the previous broad review overlapped with the final evidence set, and no additional eligible study was identified through citation reconciliation.[4] Study characteristics, key sex-related laboratory and autoreactivity findings, and database-search and all-source study-selection yields are summarized in Tables 1-3, respectively.

Both reviewers independently judged all 25 included studies to be at low risk of bias, with no unresolved disagreements remaining after consensus. Study-level overall judgments and key methodological considerations are presented in Supplementary Table 2. No study was excluded on the basis of the risk-of-bias assessment.

Systemic Inflammatory, Coagulation, and Hematological Biomarkers

Signals of greater systemic inflammatory, coagulation, or hematological activation in female were not uniform across the CSU population. In severe antihistamine-refractory CSU, elevated D-dimer was more frequent in female (50/88 vs. 16/44; OR 2.30, 95% CI 1.09–4.85).[14] In the CURE registry, elevated CRP did not differ significantly between female and men overall, although a higher prevalence was reported among female aged 51–65 years. An approximate effect

estimate reconstructed from the reported age-stratified data was retained only as a sensitivity result.[5] In an angioedema-restricted cohort, elevated CRP and/or ESR occurred in 10/20 female and 1/14 men (OR 13.00, 95% CI 1.42–119.08), but sparse events and phenotype selection limited generalizability.[15] A large historical database showed elevated mean platelet volume in 2,652 of 8,472 female and in 1,010 of 4,306 men (OR 1.49, 95% CI 1.37–1.62).[16] Elevated CRP and eosinophil distributions were similar between sexes in the Preis cohort.[17] Other eosinophil, leukocyte, and neutrophil-to-lymphocyte findings were inconsistent or not fully effect-estimable.[18-20]

Autoreactivity and Autoimmune-Associated Biomarkers

CD63 BAT positivity was observed in 41/56 female and 15/31 men (OR 2.92, 95% CI 1.16-7.31).[21] Ten adult/mixed studies contributed directly calculable ASST data (10,22–30). The REML random-effects model yielded a pooled OR of 1.57 for female versus men (Hartung-Knapp 95% CI 0.80–3.09; P = 0.168), with substantial heterogeneity (I^2 = 72.3%; τ^2 = 0.592; Q = 32.54, df = 9, P < 0.001) (Figure 2). The 95% prediction interval was 0.23–10.53. Leave-one-out pooled ORs ranged from 1.30 to 1.78; each CI crossed unity (Supplementary Figure 1). Inclusion of the pediatric Sahiner and Godse cohorts produced a 12-study sensitivity estimate that also crossed unity (OR 1.73, Hartung-Knapp 95% CI 0.96–3.11; P = 0.064; I^2 = 67.6%);[31,32] Özçeker et al.[33] likewise reported no significant pediatric sex association but did not provide extractable sex-by-ASST cells. Exploratory Egger regression did not detect funnel-plot asymmetry (intercept -1.43; P = 0.312), although statistical power was limited (Supplementary Figure 2).

Emerging and Subgroup-Specific Biomarkers

In Liu et al.,[19] female had higher serum osteopontin than men (5.41 ± 1.58 vs. 4.39 ± 0.77 ng/mL; MD 1.02 ng/mL, 95% CI 0.21–1.83; P = 0.025), and osteopontin correlated with UAS7 in female but not men. Collet reported autoimmune thyroid disease in 8/16 female and none of 29 men (corrected OR 59.0, 95% CI 3.08–1130.09),[34] while Najib reported composite thyroid autoimmunity in 50/146 female and 7/42 men (OR 2.60, 95% CI 1.08–6.28).[35] Confino-Cohen et al.[16] and Chanprapaph et al.[26] showed female enrichment for several autoimmune serological markers, whereas Asero[10] found no significant sex difference in thyroid autoantibody prevalence within an ASST-positive subgroup.[10] Wan and Wu[36] reported anti-thyroid antibodies without extractable sex-specific denominators. In treatment-selected cohorts, IgE findings were inconsistent: high total IgE was more frequent in men in the Preis cohort, whereas mean total IgE was higher in female in the smaller Sirufo cohort.[17,25]

Discussion

Principal Findings

Across 25 studies, sex-related laboratory differences in CSU were not uniform. The primary quantitative synthesis of ASST positivity across 10 adult/mixed cohorts produced a female-directed, non-significant pooled estimate accompanied by substantial heterogeneity and a wide prediction interval. These findings indicate that sex-associated laboratory signals in CSU are likely to depend on the biomarker pathway, age, phenotype, and treatment context, rather than constituting a single reproducible female or male laboratory profile.

Evidence Base and Methodological Quality

The present study addressed a more focused laboratory question than the previous broad review of sex and gender differences in CSU. Consequently, only five of its 26 reports overlapped with the final 25-study evidence set, and citation reconciliation identified no additional eligible studies.[4] This distinction is important because studies focusing on epidemiology, clinical burden, comorbidities, or treatment response cannot substitute for direct sex-stratified laboratory data. Although all included studies were judged to be at low risk of bias, these judgments do not eliminate limitations of the wider evidence base, including historical disease terminology, selective laboratory testing, underrepresentation of men, and incomplete sex-specific reporting.

Systemic Inflammatory, Coagulation, and Hematological Biomarkers

Inflammatory and hematological differences were conditional rather than universal. The female-associated D-dimer signal arose in severe treatment-refractory CSU, whereas CRP was similar overall in the much larger CURE registry and differed only in midlife female.[5,14] The large CRP/ESR effect in angioedema-restricted disease is biologically plausible, but remains hypothesis-generating because of sparse events and phenotype selection.[15] The robust MPV association in the Confino-Cohen database supports greater platelet activation in female, yet MPV is nonspecific and may reflect background sex differences, comorbidity, or testing patterns rather than a CSU-specific mechanism.[16] Discordant eosinophil, leukocyte, and NLR findings further suggest modification by disease activity, endotype enrichment, treatment exposure, assay timing, and age.[17-20] Taken together, these results argue against a fixed sex effect on systemic inflammation and instead support an interaction between sex and clinical context.

Autoreactivity and Autoimmune-Associated Biomarkers

Functional autoreactivity and autoimmune-associated biomarkers varied markedly across studies. The pooled ASST estimate was directionally female-associated, but the CI crossed unity, substantial heterogeneity was observed, and the prediction interval encompassed effects favoring either sex. The opposing estimates from large or informative studies, including Asero[10] and Caproni et al.[30] explain why a simple universal sex effect is not credible.[10,30] Leave-one-out analysis confirmed that no single study determined the conclusion, and pediatric inclusion did not materially change it.[31-33] By contrast, CD63 BAT positivity was higher in female in one directly stratified cohort.[21] This apparent difference is not contradictory: ASST, APST, BAT, HRA, and BHRA capture overlapping but non-equivalent biological processes and should not be treated as interchangeable measures. The current evidence therefore supports possible sex enrichment in selected autoimmune or autoreactive endotypes rather than a general female excess in autoreactivity.

Emerging and Subgroup-Specific Biomarkers

As a novel biomarker, osteopontin was higher in female and correlated with disease activity only in female, providing a biologically coherent link among immune-cell recruitment, tissue remodeling, and mast-cell-associated inflammation, but this finding currently rests on a single small study.[19] Female enrichment for clinical thyroid autoimmunity and autoimmune serology was observed across legacy and contemporary cohorts,[16,26,34,35] although Asero[10] and Wan and Wu[36] illustrate that not all thyroid-related comparisons are concordant or quantitatively complete.[10,36] These findings align with broader evidence that type IIb autoimmune CSU and autoimmune comorbidity cluster in selected patients and may influence treatment behavior.[37,38] Hormonal mechanisms remain plausible context rather than proof: experimental and clinical observations suggest that estrogens may enhance mast-cell or basophil activation, whereas progesterone and testosterone may be inhibitory, but available CSU data rarely account for menstrual phase, pregnancy, menopause, or exogenous hormone exposure.[11] The opposing IgE findings in the Preis and Sirufo treatment-selected cohorts further demonstrate that sex-related IgE patterns are not uniform and may depend on cohort selection, laboratory definitions, and treatment context.[17,25] Taken together, these findings indicate pathway- and population-specific rather than uniform sex effects.

Sex, Treatment Response, and Prognosis

Evidence that sex independently modifies treatment response or prognosis remains limited and internally inconsistent. In a 250-patient real-world omalizumab cohort, men achieved higher UCT scores at early follow-up, but 12-month outcomes were comparable between sexes, suggesting a difference in response kinetics rather than sustained efficacy.[17] A smaller 42-patient study found clinical remission in both sexes during treatment but reported a sex-associated difference in recurrence after omalizumab withdrawal; the small sample and limited adjustment preclude firm conclusions.[25] At the broader disease level, the CURE registry documented greater burden and poorer control in female, particularly in midlife.[5] In addition, thyroid autoimmunity was more frequent in female, while anti-TPO positivity predicted a longer time to remission and APST positivity was linked to greater treatment difficulty; these biomarker-outcome associations should not be interpreted as proof of a direct sex effect.[26] Taken together, these data do not justify sex-specific treatment selection or dosing. Prospective studies should test sex as an independent predictor and an interaction term while jointly accounting for age, hormonal status, baseline disease activity, autoimmune endotype, and biomarker profile.

Study Limitations

Strengths of this study include a comprehensive four-database search; formal reconciliation of the prior citation map; independent screening and quality appraisal; source-level verification of sex-stratified data; and a planned distinction between the primary adult/mixed synthesis and the pediatric, treatment-selected, phenotype-restricted, and legacy-CU sensitivity strata. The primary quantitative synthesis used REML random effects, Hartung-Knapp inference, a prediction interval, leave-one-out analysis, and an exploratory assessment of small-study effects. Limitations are equally important: male subgroups were often small; assay thresholds and disease definitions varied; two conference reports were unavailable; several outcomes were represented by only one or two studies; favorable within-study risk-of-bias judgments could not overcome the indirectness, selective testing, or clinical heterogeneity of the wider evidence base. Future cohorts should prespecify sex-stratified analyses, report complete denominators and dispersion measures, standardize biomarker thresholds, and capture age, menopausal status, hormonal exposure, autoimmune endotype, disease activity, and treatment status.

Conclusion

Current evidence does not support different routine laboratory panels or treatment algorithms solely on the basis of sex. Clinicians should continue guideline-based, clinically directed testing, while recognizing that female with severe, midlife, or autoimmune-featured CSU may be enriched for selected inflammatory, coagulation, platelet, thyroid, or autoreactivity

signals, whereas IgE findings remain inconsistent across treatment-selected settings. For research, sex should be treated as a prespecified biological and analytical variable rather than merely adjusted away. In summary, the available literature supports sex-aware phenotyping and standardized reporting, but not sex-specific diagnostic or therapeutic algorithms; larger prospectively designed studies are required to determine whether any of these signals can improve endotyping, treatment selection, or prediction of remission.

Ethics

Ethics Committee Approval: Ethics committee approval was not required because this study involved only a secondary analysis of previously published aggregate data. No new data were collected from human participants, no intervention was performed, and no individual-level identifiable patient information was accessed.

Informed Consent: Informed consent was not required because no new data were collected from human participants and no individual-level identifiable patient information was accessed.

Authorship Contributions

Concept: E.Ü., Design: E.Ü., Data Collection or Processing: E.Ü., Literature Search: M.B.Y., Writing: M.B.Y.

Footnotes

Conflict of Interest: The authors declared that they have no conflict of interest.

Financial Disclosure: The authors declared that this study received no financial support.

Supplementary Figures Link: https://d2v96fxpocvxx.cloudfront.net/cf9d60d6-523c-458a-a2e6-78728d3ffbb0/content-images/88d1b0f3-c3d7-4484-bc06-ecb686e040ed.pdf

Supplementary Tables Link: https://d2v96fxpocvxx.cloudfront.net/cf9d60d6-523c-458a-a2e6-78728d3ffbb0/content-images/50e17e2d-4800-4809-b082-5f28b7f9f02c.pdf

References

1. Zuberbier T, Ansari ZA, Abdul Latiff AH, Abuzakouk MM, Agcaoili-De Jesus MS, Agondi RC, Al-Ahmad M, Alangari AA, Alhameli H, Alonso Bello CD, Alshareef S, Al-Tamemi S, Altrichter S, Al Wahshi H, Aquilina S, Araújo M, Arnaout R, Asero R, Ballmer-Weber B, Bangert C, Bauer A, Ben-Shoshan M, Bernstein JA, Bindslev-Jensen C, Bizjak M, Boccon-Gibod I, Bonnekoh H, Bouillet L, Brockow K, Brzoza Z, Bulatović Ćalasan M, Bulkhi A, Buttgereit T, Bygum A, Caballero T, Calderon O, Campos R, Cancian M, Carne E, Castor MA, Cerecedo I, Çetinarslan T, Cherrez-Ojeda I, Chkhikvadze N, Chong-Neto HJ, Choo K, Christoff G, Chu CY, Ciupka K, Conlon N, Costa C, Craig TJ, Criado P, Danilycheva I, Darlenski R, De Arruda Chaves E, de Montjoye L, Doutre MS, Du-Thanh A, Ebo D, Elkhalifa S, Elmariah S, El-Shanawany T, Ensina LF, Ertaş R, Fachini Jardim Criado R, Ferrer M, Ferrucci S, Fok JS, Fomina D, Fonacier L, Fouda G, Francescantonio I, Fukunaga A, Galvan Calle CA, Garcia E, Gáspár K, Gelincik A, Geng S, Godse K, Gonçalo M, Gotua M, Grattan C, Grosber M, Guidos Fogelbach G, Guilarte M, Guillod R, Hamelmann E, Hawkes J, Hayama K, Heuer R, Hide M, Hoetzenecker W, Inomata N, Kang HR, Kaplan AP, Kapp A, Karam M, Kasperska-Zajac A, Katelaris CH, Kessel A, Khoshkhui M, Kim B, Kinaciyan T, Kocatürk E, Kolacinska-Flont M, Kolkhir P, Konstantinou GN, Kosnik M, Krasowska D, Kulthanan K, Kumaran MS, Kuprys-Lipinska I, Labrador M, Larco JI, Larenas-Linnemann D, Latysheva E, Lazaridou E, Li PH, Lima H, Lippert U, Magerl M, Makris M, Alves Marcelino J, Marzano AV, Medina I, Meshkova R, Micallef D, Mohammed Ali R, Mortz CG, Munoz M, Oude Elberink HNG, Nakonechna A, Nasr I, Nast A, Netchiporouk E, Nettis E, Nieto S, Ogueta Canales I, Okas TL, Orfali RL, Özkaya E, Parisi C, Pennitz A, Pawankar R, Pereira MP, Peter J, Petkova E, Pigatto PD, Podder I, Popov T, Porebski G, Pyatilova P, Ramon GD, Ratti Sisa HA, Recto MS, Ress K, Ridge K, Riedl M, Ritchie C, Rosario Filho N, Rosmaninho I, Rudenko M, Rukhadze M, Rutkowski K, Sabato V, Sahiner UM, Saini S, Saleh Al Sabbagh F, Salman A, Salvo F, Sanchez J, Santucci A, Schliemann S, Schmid-Grendelmeier P, Sekerel BE, Serpa F, Sheikh F, Sheikh J, Shendi H, Siebenhaar F, Sonomjamts M, Soria A, Sousa Pinto B, Staevska M, Staubach P, Stephan M, Stevanovic K, Stingeni L, Stobiecki M, Su Küçük Ö, Sussman G, Szegedi A, Takahagi S, Tanaka A, Teovska Mitrevska N, Thomsen SF, Toubi E, Tsatsou F, Turk M, Vadasz Z, Valerieva A, Valle S, Doorn MV, Veleiro Perez B, Vera Ayala CE, Vestergaard C, Vieira RJ, Maruta CW, Wedi B, Werner RN, Yap EWY, Xepapadaki P, Xiang YK, Ye YM, Yong P, Yosipovitch G, Zalewska-Janowska A, Zeyen C, Zhao Z, Metz M, Giménez-Arnau AM. The International Guideline for the Definition, Classification, Diagnosis and Management of Urticaria. Allergy. 2026;81(8):2582-2632.
2. Kolkhir P, Bonnekoh H, Metz M, Maurer M. Chronic spontaneous urticaria: a review. JAMA. 2024;332(17):1464-1477.
3. Gonçalo M, Gimenéz-Arnau A, Al-Ahmad M, Ben-Shoshan M, Bernstein JA, Ensina LF, Fomina D, Galvàn CA, Godse K, Grattan C, Hide M, Katelaris CH, Khoshkhui M, Kocatürk E, Kulthanan K, Medina I, Nasr I, Peter J, Staubach P, Wang L, Weller K, Maurer M. The global burden of chronic urticaria for the patient and society. Br J Dermatol. 2021;184(2):226-236.
4. Preis S, Claussen C, Ziehfreund S, Biedermann T, Horster S, Zink A. Is there a difference between female and men in chronic spontaneous urticaria? A systematic review on gender and sex differences in CSU patients. World Allergy Organ J. 2024;17(11):100974.
5. Kocatürk E, Salameh P, Asero R, Bizjak M, Gimenez-Arnau A, Grattan C, Pesqué D, Planella-Fontanillas N, Herzog LS, Buttgereit T, Bonnekoh H, Fomina D, Kovalkova E, Lebedkina M, Kasperska-Zajac A, Zając M, Zamłyński M, Kulthanan K, Tuchinda P, Khoshkhui M, Hassanpour Z, Peter J, Du-Thanh A, Meshkova R, Abuzakouk M, Makris M, Bouillet L, Bocquet A, Gregoriou S, Thomsen SF, Dissemond J, Staubach P, Bauer A, Danilycheva I, van Doorn M, Parisi C, Metz M, Fluhr JW, Zuberbier T, Weller K, Kolkhir P. Sex matters in CSU: Female face greater burden and poorer urticaria control, especially in midlife-CURE insights. J Eur Acad Dermatol Venereol. 2026;40(1):67-78.
6. Kolkhir P, Muñoz M, Asero R, Ferrer M, Kocatürk E, Metz M, Xiang YK, Maurer M. Autoimmune chronic spontaneous urticaria. J Allergy Clin Immunol. 2022;149(6):1819-1831.
7. Larenas-Linnemann D. Biomarkers of autoimmune chronic spontaneous urticaria. Curr Allergy Asthma Rep. 2023;23(12):655-664.
8. Schoepke N, Asero R, Ellrich A, Ferrer M, Gimenez-Arnau A, E H Grattan C, Jakob T, Konstantinou GN, Raap U, Skov PS, Staubach P, Kromminga A, Zhang K, Bindslev-Jensen C, Daschner A, Kinaciyan T, Knol EF, Makris M, Marrouche N, Schmid-Grendelmeier P, Sussman G, Toubi E, Church MK, Maurer M. Biomarkers and clinical characteristics

of autoimmune chronic spontaneous urticaria: Results of the PURIST Study. Allergy. 2019;74(12):2427-2436.

9. Altrichter S, Fok JS, Jiao Q, Kolkhir P, Pyatilova P, Romero SM, Scheffel J, Siebenhaar F, Steinert C, Terhorst-Molawi D, Xiang YK, Church MK, Maurer M. Total IgE as a marker for chronic spontaneous urticaria. Allergy Asthma Immunol Res. 2021;13(2):206-218.
10. Asero R. Sex differences in the pathogenesis of chronic urticaria. J Allergy Clin Immunol. 2003;111(2):425-426.
11. Kasperska-Zajac A, Brzoza Z, Rogala B. Sex hormones and urticaria. J Dermatol Sci. 2008;52(2):79-86.
12. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, Shamseer L, Tetzlaff JM, Akl EA, Brennan SE, Chou R, Glanville J, Grimshaw JM, Hróbjartsson A, Lalu MM, Li T, Loder EW, Mayo-Wilson E, McDonald S, McGuinness LA, Stewart LA, Thomas J, Tricco AC, Welch VA, Whiting P, Moher D. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. Int J Surg. 2021;88:105906.
13. Barker TH, Hasanoff S, Aromataris E, Stone JC, Leonardi-Bee J, Sears K, Klugar M, Tufanaru C, Moola S, Liu XL, Munn Z. The revised JBI critical appraisal tool for the assessment of risk of bias for analytical cross-sectional studies. JBI Evid Synth. 2026;24(3):401-408.
14. Asero R. Severe CSU and activation of the coagulation/fibrinolysis system: clinical aspects. Eur Ann Allergy Clin Immunol. 2019;52(1):15-17.
15. Sartorio S, Rivolta F, Tedeschi A, Manzotti G, Piantanida M, Marra AM, Cappiello F, Yacoub MR, Nannipieri S, Maffeis L, Longoni V, Sangalli A, Pravettoni V, Asero R. Demographic and clinical characteristics of chronic histaminergic angioedema and chronic urticaria with angioedema: a multicenter Italian experience. Eur Ann Allergy Clin Immunol. 2026;58(4):173-179.
16. Confino-Cohen R, Chodick G, Shalev V, Leshno M, Kimhi O, Goldberg A. Chronic urticaria and autoimmunity: associations found in a large population study. J Allergy Clin Immunol. 2012;129(5):1307-1313.
17. Preis S, Lisiecki M, Biedermann T, Horster S, Zink A. Early control or gradual relief? Gender-specific real-world insights into omalizumab response in chronic spontaneous urticaria patients. World Allergy Organ J. 2026;19(1):101163.
18. Kolkhir P, Church MK, Altrichter S, Skov PS, Hawro T, Frischbutter S, Metz M, Maurer M. Eosinopenia, in chronic spontaneous urticaria, is associated with high disease activity, autoimmunity, and poor response to treatment. J Allergy Clin Immunol Pract. 2020;8(1):318-325.e5.
19. Liu H, Guo J, Yuan J, Ye D, Gao Z, Yang F, Zhang L, Geng S, Zeng W, Wang Z. Gender-dependent differences in serum osteopontin levels in chronic spontaneous urticaria: a link to disease activity in females.Clin Exp Allergy. 2026;56(9):1097-1100.
20. Moos Ł, Chodak W, Czyczerska M, Garbino K, Gleba O, Śnietka B, Brzoza Z. Relationship between cigarette smoking and chronic spontaneous urticaria. Is there a difference in neutrophil-lymphocyte ratio? Postepy Dermatol Alergol. 2024;41(6):617-621.
21. Hossein Zadeh Attar M, Merk HF, Kotliar K, Wurpts G, Röseler S, Moll-Slodowy S, Plange J, Baron JM, Balakirski G. The CD63 basophil activation test as a diagnostic tool for assessing autoimmunity in patients with chronic spontaneous urticaria. Eur J Dermatol. 2019;29:614-618.
22. Kulthanan K, Jiamton S, Gorvanich T, Pinkaew S. Autologous serum skin test in chronic idiopathic urticaria: prevalence, correlation and clinical implications. Asian Pac J Allergy Immunol. 2006;24(4):201-206.
23. Aktar S, Akdeniz N, Ozkol HU, Calka O, Karadag AS. The relation of autologous serum and plasma skin test results with urticarial activity score, sex and age in patients with chronic urticaria. Postepy Dermatol Alergol. 2015;32(3):173-178.
24. Lai FY, Sarkan MM. Autologous serum skin test in chronic spontaneous urticaria: Evaluation of the relationship with disease activity and autoimmune antibodies. Med J Malaysia. 2025;80(5):575-581.
25. Sirufo MM, Bassino EM, De Pietro F, Ginaldi L, De Martinis M. Sex differences in the efficacy of omalizumab in the treatment of chronic spontaneous urticaria. Int J Immunopathol Pharmacol. 2021;35:20587384211065870.
26. Chanprapaph K, Iamsumang W, Wattanakrai P, Vachiramon V. Thyroid autoimmunity and autoimmunity in chronic spontaneous urticaria linked to disease severity, therapeutic response, and time to remission in patients with chronic spontaneous urticaria. Biomed Res Int. 2018;2018:9856843.
27. Curto-Barredo L, Pujol RM, Roura-Vives G, Giménez-Arnau AM. Chronic urticaria phenotypes: clinical differences regarding triggers, activity, prognosis and therapeutic response. Eur J Dermatol. 2019;29(6):627-635.
28. Mohammed JQ. Autologous serum therapy reduces the symptoms and antihistamine burden in patients with chronic urticaria. Postepy Dermatol Alergol. 2023;40(3):398-401.
29. Safari M, Sayemiri H. The results of autologous skin test in patients with chronic urticaria in Hamadan, Iran. Electron Physician. 2016;8(6):2539-2542.
30. Caproni M, Volpi W, Giomi B, Cardinali C, Antiga E, Melani L, Dagata A, Fabbri P. Chronic idiopathic and chronic autoimmune urticaria: clinical and immunopathological features of 68 subjects. Acta Derm Venereol. 2004;84(4):288-290.
31. Sahiner UM, Civelek E, Tuncer A, Yavuz ST, Karabulut E, Sackesen C, Sekerel BE. Chronic urticaria: etiology and natural course in children. Int Arch Allergy Immunol. 2011;156(2):224-230.
32. Godse KV. Autologous serum skin test in children. Indian J Dermatol. 2008;53(2):61-63.
33. Özçeker D, Özek Yücel E, Sipahi Çimen S, Dilek F, Güler N, Tamay Z. Serum autologous skin test in children with chronic urticaria: is it related to the severity of the disease? J Curr Pediatr. 2020;18(1):85-94.
34. Collet E, Petit JM, Lacroix M, Bensa AF, Morvan C, Lambert D. Urticaire chronique et pathologie thyroïdienne auto-immune [Chronic urticaria and autoimmune thyroid diseases]. Ann Dermatol Venereol. 1995;122(6-7):413-416.
35. Najib U, Bajwa ZH, Ostro MG, Sheikh J. A retrospective review of clinical presentation, thyroid autoimmunity, laboratory characteristics, and therapies used in patients with chronic idiopathic urticaria. Ann Allergy Asthma Immunol. 2009;103(6):496-501.
36. Wan KS, Wu CS. The essential role of anti-thyroid antibodies in chronic idiopathic urticaria. Endocr Res. 2013;38(2):85-88.
37. Kolkhir P, Altrichter S, Asero R, Daschner A, Ferrer M, Giménez-Arnau A, Hawro T, Jakob T, Kinaciyan T, Kromminga A, Konstantinou GN, Makris M, Metz M, Skov PS, Staubach P, Sussman G, Zhang K, Maurer M. Autoimmune diseases are linked to type IIb autoimmune chronic spontaneous urticaria. Allergy Asthma Immunol Res. 2021;13(4):545-559.
38. Kocatürk E, Başkan EB, Küçük ÖS, Özdemir M, Örnek S, Can PK, Haşal E, Engin B, Atakan N, Alpsoy E. Omalizumab versus cyclosporin-A for the treatment of chronic spontaneous urticaria: can we define better-responding endotypes? An Bras Dermatol. 2022;97(5):592-600.