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Original Research Article

A STUDY ON THE ROLE OF AUTOLOGOUS SERUM SKIN TEST IN CHRONIC IDIOPATHIC URTICARIA AND ITS CORRELATION WITH THYROID DYSFUNCTION

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ABSTRACT

Background: The aim is to evaluate the prevalence of autoimmune urticaria in patients with chronic idiopathic urticaria (CIU) using the autologous serum skin test (ASST) and to assess its correlation with thyroid dysfunction, particularly anti-thyroid peroxidase (anti-TPO) antibodies. The study design is a prospective observational study conducted over a period of two years.

Materials and Methods: Sixty-five clinically diagnosed CIU patients attending a tertiary care dermatology outpatient department were enrolled. Patients underwent ASST and anti-TPO antibody testing. Total Severity Score (TSS) was calculated based on frequency, number, duration, and size of wheals, severity of pruritus, and antihistamine usage. Data were analyzed using chi-square and t-tests; p-values < 0.05 were considered statistically significant.

Results: ASST was positive in 52.3% of patients. Those who tested positive had significantly higher TSS, more frequent and prolonged wheals, severe pruritus, and increased antihistamine requirements. Anti-TPO antibodies were elevated in 50.8% of patients and significantly associated with ASST positivity ($p = 0.0372$). Hypothyroidism was observed in 44.6% overall and was more frequent among ASST-positive patients (58.8%) compared to ASST-negative patients (29%).

Conclusion: ASST is a simple, inexpensive, and effective screening tool to detect autoimmune urticaria in CIU patients. Its strong association with thyroid autoimmunity highlights the need for thyroid screening in such patients to guide comprehensive management strategies.

Keywords: Chronic idiopathic urticaria, ASST, autoimmune urticaria, anti-TPO antibodies, thyroid dysfunction.

INTRODUCTION

Chronic urticaria (CU) represents a pervasive and often debilitating dermatological condition, characterized by the persistent and spontaneous emergence of transient wheals, angioedema, or a combination of both, enduring for a period of six weeks or longer. These hallmark cutaneous lesions, known as wheals, are typically intensely pruritic, erythematous, and elevated, frequently accompanied by an uncomfortable sensation of burning or stinging. In some instances, patients may also develop angioedema, a deeper and less circumscribed form of swelling that commonly affects areas with loose connective tissue, such as the lips, eyelids, and extremities. A defining characteristic of CU is the ephemeral nature of individual lesions; each wheal typically resolves within 24 hours without leaving any residual skin changes, such as bruising or scarring. However, the disease itself is chronic, capable of persisting for many months or even several years, with a substantial proportion of affected individuals experiencing symptoms for over a decade, profoundly impacting their daily lives.

The burden of CU extends far beyond mere physical discomfort, exerting a significant negative impact on the patient's overall quality of life.[1] The relentless itching and unpredictable appearance of lesions lead to considerable physical distress, frequently disrupting sleep patterns and contributing to chronic fatigue. This persistent physical discomfort often culminates in significant emotional distress, fostering feelings of frustration, embarrassment, and isolation. Consequently, CU is frequently associated with psychiatric comorbidities, most notably anxiety and depression, which can further exacerbate the patient's suffering and complicate disease management. Epidemiological data suggest that CU affects approximately 0.5% to 1% of the general population worldwide, exhibiting a marked female predominance and a peak incidence observed between the ages of 20 and 40 years. While acute urticaria often has identifiable triggers such as specific allergens, infections, or drug reactions, chronic forms are distinguished by their protracted duration and, crucially, the frequent absence of clear, easily identifiable precipitating factors, making diagnosis and management particularly challenging.

Classification and Nomenclature

For clarity in clinical practice and research, CU is broadly categorized into two primary clinical subtypes based on the presence or absence of an identifiable trigger:

- **Chronic Spontaneous Urticaria (CSU):** Formerly known as chronic idiopathic urticaria (CIU), this subtype is characterized by the spontaneous occurrence of wheals and/or angioedema without any obvious external provocation. The shift in nomenclature from "idiopathic" to "spontaneous" reflects an evolving understanding that many cases previously deemed idiopathic are, in fact, driven by underlying endogenous mechanisms, particularly autoimmune processes, rather than being truly without a discernible cause.
- **Chronic Inducible Urticaria (CIndU):** In contrast, CIndU encompasses forms of urticaria where symptoms are consistently triggered by specific physical stimuli. These triggers can be diverse and include temperature changes (e.g., cold urticaria, heat urticaria), pressure (e.g., delayed pressure urticaria), sunlight (solar urticaria), vibration (vibratory angioedema), or water (aquagenic urticaria). The ability to classify CU into these distinct subtypes is critical for guiding diagnostic workups and tailoring therapeutic strategies.[2]

Pathogenesis: Immune Mechanisms and Autoimmunity

The historical classification of CSU as "idiopathic" largely stemmed from a lack of understanding regarding its underlying etiology. However, significant advancements in immunology over recent decades have shed light on the complex immunopathogenesis of CSU, revealing a substantial autoimmune component in a considerable subset of patients.[3,4] Current estimates suggest that up to 30% to 50% of individuals with CSU harbor functional autoantibodies that play a pivotal role in disease perpetuation. These autoantibodies primarily target two key components:

1. The high-affinity IgE receptor (FcεRIα): Located on the surface of mast cells and basophils, this receptor is crucial for allergic responses. Autoantibodies directed against FcεRIα can mimic the action of IgE, leading to direct activation of these cells.[5]
2. IgE itself: Less commonly, autoantibodies may target IgE molecules directly, forming immune complexes that then bind to FcεRIα, subsequently activating mast cells and basophils.[6]

Regardless of their specific target, these autoantibodies induce non-IgE-mediated mast cell and basophil degranulation. This process results in the release of a myriad of pro-inflammatory mediators, most notably histamine, but also leukotrienes, prostaglandins, and cytokines. It is the release of histamine that primarily drives the characteristic symptoms of CU, including pruritus, erythema, and wheal formation due to increased vascular permeability and vasodilation. It is particularly noteworthy that autoimmune (type IIb) CSU, characterized by the presence of these functional autoantibodies, often presents as a more severe, persistent, and treatment-refractory form of the disease, necessitating more aggressive therapeutic interventions. Beyond autoimmunity, other pathophysiological contributors to CSU have been identified, including dysregulation of the coagulation cascade, deficiencies in vitamin D, chronic infections (e.g., Helicobacter pylori, viral infections), and various cofactors such such as psychological stress or the use of non-steroidal anti-inflammatory drugs (NSAIDs), which can exacerbate symptoms in predisposed individuals.

Diagnostic Challenges, ASST, and Autoantibody Testing

The multifactorial nature of CU, coupled with the often elusive triggers and the overlap with systemic autoimmune phenomena, significantly complicates the diagnostic approach. The direct identification of circulating functional autoantibodies, such as through sophisticated in vitro basophil histamine release assays (BHRA) or Western blot techniques, remains technically demanding, prohibitively costly, and typically accessible only in highly specialized academic or research centers. These limitations often preclude their routine use in general clinical practice, particularly in resource-constrained healthcare settings.

To circumvent these practical barriers, the Autologous Serum Skin Test (ASST) has emerged as a widely adopted, readily available, and cost-effective in vivo screening procedure. The ASST involves the intradermal injection of a small amount of the patient's own serum into their forearm, alongside saline and histamine controls. The

appearance of a wheal and flare response at the serum injection site, exceeding that of the saline control, suggests the presence of histamine-releasing factors in the patient's serum, including functional autoantibodies. Thus, ASST positivity is considered a valuable surrogate marker of underlying autoimmune activity in CU. While the ASST does not precisely identify the specific pathogenic antibody or its target, its simplicity, low cost, and reasonable sensitivity make it an attractive and practical tool for routine clinical assessment, especially in regions where advanced laboratory diagnostics are not readily available.[7] It serves as a useful indicator for clinicians to consider an autoimmune etiology.[8]

Association with Thyroid Autoimmunity

An increasingly recognized and clinically significant association exists between chronic urticaria and autoimmune thyroid diseases, particularly Hashimoto's thyroiditis (autoimmune hypothyroidism) and Graves' disease (autoimmune hyperthyroidism). Numerous studies across diverse populations have consistently documented an elevated prevalence of antithyroid antibodies—specifically anti-thyroid peroxidase (anti-TPO) antibodies and anti-thyroglobulin (anti-Tg) antibodies—in patients with CU, irrespective of their current thyroid hormone levels.[9] This compelling association strongly suggests shared underlying autoimmune mechanisms, implying that systemic thyroid autoimmunity may either directly contribute to or exacerbate the pathogenesis of CU in susceptible individuals.[10]

Some researchers have hypothesized that anti-TPO antibodies might directly participate in the activation or sensitization of cutaneous mast cells, thereby contributing to the degranulation process and subsequent wheal formation, although the precise molecular mechanisms underpinning this interaction are still subjects of ongoing investigation. The clinical presentation of this comorbidity is often complex and heterogeneous: some ASST-positive CU patients may be euthyroid (possessing normal thyroid function), while others may exhibit subclinical or overt thyroid dysfunction. This significant variability underscores the intricate and often bidirectional interplay between cutaneous and systemic autoimmunity within the chronic urticaria patient population, highlighting the need for a holistic diagnostic approach.

Clinical and Therapeutic Implications

The accurate diagnosis of autoimmune subtypes of CU and the diligent assessment for associated comorbidities, such as thyroid autoimmunity, are paramount for optimizing patient management and prognosis. Standard initial therapeutic approaches for CU involve the use of second-generation antihistamines, often at up to four times the conventional dose, to control symptoms. However, a considerable fraction of patients remains symptomatic despite maximal antihistamine therapy, defining a group with refractory CU who necessitate alternative, more advanced treatments. These alternatives include omalizumab, a monoclonal antibody that targets IgE, thereby reducing free IgE levels and downregulating FcεRI receptors on mast cells, or systemic immunosuppressants like cyclosporine, which broadly suppress immune responses.[11,12]

The judicious combination of ASST with routine thyroid autoantibody testing offers a cost-effective and practical strategy to identify patients with underlying autoimmunity. This diagnostic approach not only guides further targeted investigations but also facilitates more precise risk stratification and, crucially, allows for the potential implementation of individualized therapeutic strategies. Recognizing and characterizing autoimmune urticaria subtypes is vital for tailoring interventions, especially in cases that prove refractory to conventional treatments or those associated with broader systemic manifestations, ultimately leading to improved patient outcomes and enhanced quality of life.

MATERIALS AND METHODS

Study Design and Setting: This was a prospective observational study conducted over a 2-year period. The institutional ethics committee approved the study protocol. Written informed consent was obtained from all participants.

Study Population: Sixty-five consecutive patients with clinically diagnosed chronic idiopathic urticaria, aged over 12 years, were enrolled based on inclusion and exclusion criteria.

Inclusion Criteria

- Diagnosis of chronic idiopathic urticaria persisting for more than 6 weeks
- Age >12 years
- Willingness to undergo ASST

Exclusion Criteria

- Physical urticarias (cold, pressure, cholinergic)
- Urticaria due to identifiable causes like food allergy, drugs, infections
- Hereditary angioedema
- Pregnancy and lactation

Data Collection: Detailed history and clinical examination were recorded, including duration, frequency, severity of pruritus, number and size of wheals, and requirement of antihistamines.

A Total Severity Score (TSS) was calculated using six parameters: number of wheals, pruritus severity, frequency, duration, antihistamine usage, and wheal size. Each parameter was scored from 0 to 3. Based on cumulative TSS:

- 0: Clear
- 1–6: Mild
- 7–12: Moderate
- 13–18: Severe

Procedure for ASST

Participants were instructed to stop medications as per standard washout periods (e.g., antihistamines ≥3 days, corticosteroids ≥30 days). Figure 1

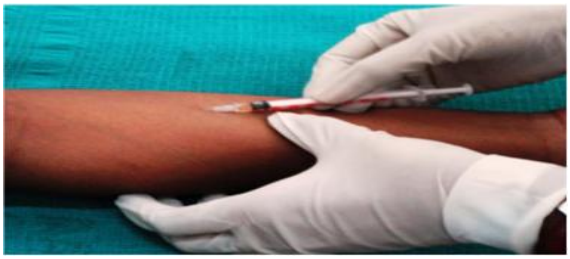

Figure 1: technique of autologous serum skin test

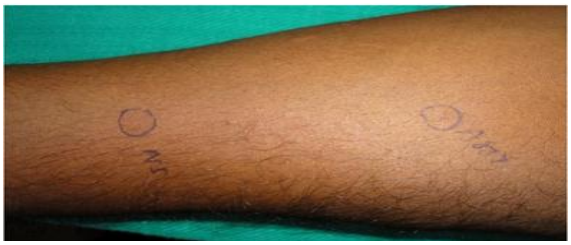

Figure 2: negative result of autologous serum skin test

Steps:

1. Collect 2 ml of venous blood from antecubital vein.
2. Allow clotting and centrifuge at 2000 rpm for 10 minutes.
3. Intradermally inject 0.1 ml autologous serum on one forearm and 0.1 ml normal saline as control on the same arm, 3-5 cm apart.
4. Measure wheal and flare at 30 minutes.

A negative ASST was defined as no visible flare or erythema beyond what is expected with normal saline injection. Figure 2.

A positive ASST was defined as a wheal ≥1.5 mm larger than the control. [Figure 3].

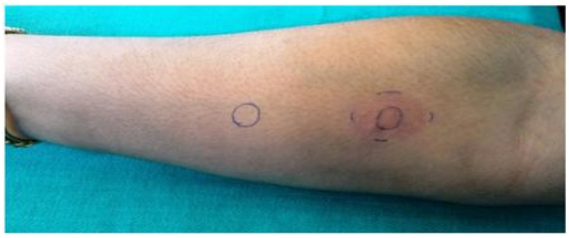

Figure 3: positive result of autologous serum skin test

Anti- TPO and Thyroid Function Testing

Serum anti- TPO levels were assessed using CLIA. Levels >34 IU/ml were considered abnormal. Thyroid function was evaluated via serum TSH, T3, and T4.[13]

Statistical Analysis: Data were analyzed using SPSS. Chi-square, independent t-tests, and paired t-tests were used. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 65 patients with chronic idiopathic urticaria were included in the study. The mean age of the participants was 35.63 ± 13.41 years, with the majority (41.5%) falling in the 21–40-year age group. Females predominated the study population (64.6%) compared to males (36.3%), with a female-to-male ratio of 1.8:1.

Table 1: Asst results

ASST	Frequency	Percent
Positive	35	53
Negative	31	47.7
Total	65	100.0

Out of 65 patients, 34 (52.3%) were ASST-positive, while 31 (47.7%) were ASST-negative. Table 1. ASST positivity was most frequent in the 41–60-year age group (63.6%), though age-wise and gender-wise differences in ASST results were not statistically significant ($p > 0.05$).

Patients who tested ASST-positive had a higher mean Total Severity Score (TSS), indicating significantly more severe urticaria ($p = 0.00278$). They also required more frequent antihistamine use and experienced greater wheal duration and pruritus severity, all with statistically significant differences compared to the ASST-negative group ($p < 0.001$). However, the number and size of wheals did not differ significantly between the two groups.

Angioedema was significantly more prevalent among ASST-positive patients (59.6%) compared to ASST-negative patients (33.9%) ($p = 0.002$). A history of atopy was reported in both groups (29.7% in ASST+ vs. 27.4% in ASST−), but this was not statistically significant.

Thyroid autoimmunity, assessed via anti-TPO antibody levels, was found in 50.8% of all patients. Among the ASST-positive group, 58.8% were anti-TPO positive, while 41.1% of ASST-negative patients also had elevated anti-TPO levels. The association between ASST positivity and anti-TPO antibody presence was statistically significant ($p = 0.0372$).

Regarding thyroid function, 44.6% of patients had hypothyroidism, 7.7% had hyperthyroidism, and 47.7% had normal thyroid profiles. The prevalence of hypothyroidism was notably higher among ASST-positive patients. [Table 2].

Table 2: Thyroid function test and asst

Asst results	TFT			Total
	Hypo	Hyper	Normal	
Positive	20	3	11	34
Negative	9	2	20	31
Total	29	5	31	65

In summary, ASST positivity was associated with increased urticaria severity, higher frequency of angioedema, greater antihistamine dependence, and significant correlation with anti-TPO positivity and thyroid dysfunction, suggesting an autoimmune basis for disease in these patients.

DISCUSSION

Chronic idiopathic urticaria (CIU), now commonly referred to as chronic spontaneous urticaria (CSU), continues to pose diagnostic and therapeutic challenges despite its high prevalence and impact on quality of life. A growing body of literature supports an autoimmune mechanism in a substantial subset of these patients, with a pivotal role played by autoantibodies against the high-affinity IgE receptor (FcεRIα) or IgE itself. These autoantibodies are capable of degranulating mast cells and basophils, resulting in histamine release and the clinical manifestations of urticaria. The Autologous Serum Skin Test (ASST) serves as a practical in vivo method for identifying such autoimmune mechanisms and remains a valuable tool, particularly in settings with limited access to sophisticated laboratory assays.

In our study, 52.3% of patients were ASST-positive, a proportion that aligns with findings from previous Indian and international studies. For instance, Bajaj et al. reported a positivity rate of 53%, while O'Donnell et al. observed rates between 27% and 61% in different cohorts. This reinforces the idea that nearly half of all patients with CIU could harbor functional autoantibodies, underscoring the autoimmune basis of disease in a significant subgroup.

Disease severity, as assessed by the Total Severity Score (TSS), was significantly greater in ASST-positive patients. These patients also experienced more intense pruritus, longer wheal duration, and increased reliance on antihistamines. These findings are consistent with prior research suggesting that autoimmune urticaria tends to be more severe, persistent, and less responsive to conventional therapy. The presence of frequent angioedema in ASST-positive patients (59.6% vs. 33.9% in ASST-negative) further supports this interpretation, indicating heightened mast cell activation and a more florid inflammatory response.

Interestingly, while the number and size of wheals did not differ significantly between ASST-positive and -negative patients, other factors such as pruritus severity and frequency of wheals did. This suggests that qualitative rather than quantitative aspects of the urticarial reaction are more prominently influenced by underlying autoimmunity.

One of the most significant observations in our study is the strong correlation between ASST positivity and anti-thyroid peroxidase (anti-TPO) antibody levels. Among ASST-positive patients, 58.8% had elevated anti-TPO, compared to 41.1% in ASST-negative individuals—a statistically significant difference ($p = 0.0372$). These findings are in line with multiple studies that have demonstrated an association between CIU and autoimmune thyroid disease. For example, Confino-Cohen et al. found that 21% of urticaria patients had thyroid autoimmunity compared to only 5.6% in controls. Turktas et al. and Aversano et al. also reported a high prevalence of anti-TPO antibodies in urticaria cohorts, with positive associations to disease activity.

The mechanistic link between thyroid autoimmunity and urticaria remains speculative, but several hypotheses have been proposed.[14,15] One theory is that anti-TPO antibodies may activate mast cells either directly or through complement-mediated pathways. Another possibility is that both thyroid autoimmunity and urticaria share a common autoimmune diathesis, possibly driven by genetic predispositions or environmental triggers. Regardless of the mechanism, our findings support the need for routine thyroid screening in patients with chronic urticaria, especially those with ASST positivity.

It is also noteworthy that a substantial number of ASST-negative patients (41.1%) exhibited anti-TPO positivity. This raises important questions. Firstly, it suggests that thyroid autoimmunity may influence urticaria through pathways independent of FcεRIα or IgE-binding autoantibodies, which ASST primarily detects. Secondly, it implies that ASST alone may not be sufficient to capture all forms of autoimmune urticaria. There may be other circulating factors—such as cytokines, chemokines, or different classes of autoantibodies—that contribute to the urticarial response but are not detected by ASST. Further research using more sophisticated immunological assays may be necessary to delineate these components.

Thyroid dysfunction, especially hypothyroidism, was also more common in the ASST-positive group. Overall, 44.6% of the entire study population had hypothyroidism, while 7.7% had hyperthyroidism. This prevalence is significantly higher than that in the general population and reinforces the potential need for long-term endocrine follow-up in patients with CIU, particularly those with autoimmune features. In many cases, appropriate management of underlying thyroid dysfunction—especially through levothyroxine replacement in hypothyroid patients—has been associated with an improvement in urticaria symptoms.

Our findings have both diagnostic and therapeutic implications. From a diagnostic perspective, ASST can be used as a quick and cost-effective screening tool to identify patients with possible autoimmune urticaria. This is particularly relevant in resource-constrained settings where in vitro basophil activation or histamine release assays are not readily available. A positive ASST can prompt further work-up, including thyroid antibody testing and thyroid function assessment. From a therapeutic standpoint, identifying autoimmune urticaria may help guide clinicians toward second-line treatments such as omalizumab, cyclosporine, or immunomodulatory

therapy, especially in cases that are antihistamine-refractory.

However, several limitations must be acknowledged. Firstly, our study was observational and conducted in a single center with a relatively small sample size. A larger, multicenter design would enhance generalizability. Secondly, although ASST is useful, it is not specific for IgG anti-FcεRIα autoantibodies and may also reflect the presence of other serum histamine-releasing factors. Thirdly, thyroid autoimmunity was assessed solely using anti-TPO antibodies. While anti-TPO is the most sensitive marker, other thyroid antibodies (such as anti-thyroglobulin) and imaging findings (e.g., thyroid ultrasound) might provide a more comprehensive view. Finally, we did not evaluate patient response to thyroid hormone therapy or immunomodulators, which could further clarify the clinical relevance of our findings.

Despite these limitations, the present study contributes meaningfully to the evolving understanding of autoimmune mechanisms in chronic idiopathic urticaria. By demonstrating a significant association between ASST positivity, increased urticaria severity, and thyroid autoimmunity, we underscore the need for a broader immunological evaluation in patients presenting with chronic urticaria, especially those with poor response to standard therapy. Moreover, integrating thyroid screening into the initial assessment protocol for CIU may lead to early diagnosis and management of subclinical thyroid disorders, which may in turn reduce urticaria burden.

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CONCLUSION

ASST is a simple, inexpensive, and effective screening tool to detect autoimmune urticaria in CIU patients. Its strong association with thyroid autoimmunity highlights the need for thyroid screening in such patients to guide comprehensive management strategies.

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