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Relationship between Severity of Chronic Spontaneous Urticaria (CSU) with low level of Vitamin D: A teaching hospital based study at West Bengal.

[1]Dr. Animesh Biswas, [2]Ananya Biswas

[1]Associate Professor, Department of Dermatology, Santiniketan Medical College and Hospital, Muluk, Birbhum, West Bengal, India
[2]3rd Prof MBBS Student, Kalinga Institute of Medical Sciences, Bhubaneswar, Orissa, India.

Corresponding Author

Dr. Animesh Biswas

Associate Professor, Department of Dermatology, Santiniketan Medical College and Hospital, Muluk, Birbhum, West Bengal, India

Email: dermaanimesh@gmail.com

Abstract:

Background: Chronic spontaneous urticaria (CSU) is a distressing, mast cell–mediated skin disorder with substantial impact on quality of life. Emerging evidence suggests that vitamin D, a key immunomodulatory hormone, may influence inflammatory and allergic diseases, but its relationship with CSU severity remains incompletely understood. Aim: To evaluate the relationship between the severity of chronic spontaneous urticaria and serum vitamin D levels in adult patients attending a tertiary care hospital. Material and Methods: This hospital-based analytical cross-sectional study included 70 adult patients diagnosed with CSU. Disease severity was assessed using the Urticaria Activity Score over 7 days (UAS7) and categorized as mild, moderate, or severe. Detailed demographic and clinical data were recorded. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using a standardized chemiluminescent immunoassay and categorized as deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL). Additional routine laboratory tests were performed to exclude major confounders. Results: The majority of patients were aged 31–45 years (40.00%) and female (54.29%). Vitamin D deficiency was observed in 54.29% of patients and insufficiency in 31.43%, while only 14.29% had sufficient levels. Overall mean serum 25(OH)D was 21.07 ± 8.52 ng/mL. CSU severity distribution showed 25.71% mild, 40.00% moderate, and 34.29% severe cases, with a mean UAS7 of 22.84 ± 8.15. Mean vitamin D levels decreased progressively with increasing CSU severity: 27.54 ± 4.12 ng/mL (mild), 19.86 ± 3.98 ng/mL (moderate), and 14.72 ± 3.44 ng/mL (severe) (ANOVA p = 0.001). A significant negative correlation was found between serum vitamin D levels and UAS7 scores (r = –0.62, p = 0.001). Conclusion: Vitamin D deficiency and insufficiency are highly prevalent in CSU and are significantly associated with increased disease severity. Assessment and optimization of vitamin D status may represent a useful adjunct in the comprehensive management of CSU, although interventional studies are required to confirm causality.

Keywords: Chronic spontaneous urticaria; Vitamin D deficiency; Urticaria Activity Score; Disease severity; 25-hydroxyvitamin D management.

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Introduction

Chronic spontaneous urticaria (CSU) is a mast cell–driven skin disorder characterized by the spontaneous appearance of pruritic wheals, angioedema, or both, persisting for more than six weeks in the absence of an identifiable external trigger. CSU is distinguished from inducible forms of urticaria by its unpredictable course and lack of consistent physical or environmental stimuli. Current international guidelines emphasize that CSU is a chronic inflammatory condition rather than a purely histamine-mediated phenomenon, with complex interactions between mast cells, basophils, autoantibodies, cytokines, and the coagulation system thought to contribute to disease expression and persistence.[1]Epidemiological data suggest that CSU is relatively uncommon but clinically significant in the general population. Population-based studies estimate a point prevalence of chronic urticaria of approximately 0.3–0.5%, with CSU accounting for the majority of cases and a clear predominance in women and adults of working age. In a large health maintenance organization cohort from Argentina, the prevalence of chronic urticaria in adults was reported as 0.34%, with most patients in the 20–50-year age range, reflecting the typical demographic pattern seen worldwide.[2] Similarly, a Swedish registry-based cohort found a period prevalence of chronic urticaria of 0.53%, with nearly 68% of patients being female, underscoring the sex-related difference and the chronic nature of this disease in real-world settings.[3] These data highlight CSU as a long-lasting condition that commonly affects individuals in their most productive years.Beyond its cutaneous manifestations, CSU is increasingly recognised as a disease with substantial impact on quality of life, psychological well-being, and productivity. Persistent itch, sleep disturbance, cosmetic concerns, and unpredictability of flares can lead to anxiety, depression, social withdrawal, and occupational impairment.[1] Patients often require repeated medical consultations, stepwise pharmacotherapy, and, in refractory cases, advanced biologic agents, all of which contribute to personal and societal economic burden. In many series, disease duration averages several years and can extend beyond five years in a substantial subset of patients, reinforcing the need to identify potentially modifiable risk factors that may influence disease severity or course.[2,3]Vitamin D has emerged over the last two decades as a pleiotropic secosteroid hormone with actions extending far beyond calcium–phosphate homeostasis and bone metabolism. Global data indicate that vitamin D deficiency is highly prevalent in both developed and developing countries, driven by limited sun exposure, skin pigmentation, ageing, lifestyle factors, and inadequate dietary intake.[4] Holick described vitamin D deficiency as a worldwide problem, noting that low serum 25-hydroxyvitamin D [25(OH)D] levels are common even in healthy adults and are associated with musculoskeletal as well as a range of chronic non-skeletal conditions.[4] This widespread deficiency has stimulated growing interest in the potential role of vitamin D in immune-mediated and allergic diseases. Mechanistically, vitamin D exerts important immunomodulatory effects on both innate and adaptive immune responses. Active vitamin D (1,25-dihydroxyvitamin D) can influence the function of dendritic cells, macrophages, and other antigen-presenting cells, promoting a more tolerogenic phenotype and reducing excessive inflammatory signalling. It also modulates T-cell differentiation by favouring regulatory T-cell development and shifting the balance away from pro-inflammatory Th1 and Th17 responses, while dampening overactive Th2 responses in certain contexts.[5] These effects collectively contribute to regulation of cytokine networks, including interleukins and interferon-γ, and may help maintain immunological homeostasis at epithelial and mucosal surfaces.The link between vitamin D and allergic disease has been the subject of intense investigation. Experimental and clinical data suggest that vitamin D may influence the risk and severity of conditions such as asthma, atopic dermatitis, food allergy, and allergic rhinitis by modulating IgE synthesis, mast cell reactivity, and epithelial barrier integrity. Benson and colleagues reviewed the role of vitamin D in the immunopathogenesis of allergic disease and highlighted that lower vitamin D levels have been associated with increased prevalence or severity of several atopic disorders, although not all studies are consistent and the optimal range for immune benefits remains uncertain.[6] These observations provide a strong rationale for exploring the relevance of vitamin D in other mast cell–mediated conditions, including CSU.In CSU specifically, attention has increasingly turned to the potential association between serum

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25(OH)D levels and disease occurrence or activity. Several observational studies have reported that patients with CSU tend to have lower vitamin D levels compared with healthy controls, and that vitamin D deficiency is more frequent among CSU patients than in the general population. Rather et al., in a case–control study from India, demonstrated significantly reduced mean serum vitamin D levels in CSU patients compared with matched controls and noted a higher proportion of individuals with deficiency in the CSU group.[7] This and other similar findings suggest that hypovitaminosis D may be linked to the pathophysiology or expression of CSU, although causality has not been firmly established.Biologically, a connection between low vitamin D status and CSU severity is plausible. Vitamin D has been shown to influence mast cell stability, downregulate the expression of high-affinity IgE receptors, and reduce production of pro-inflammatory mediators, all of which are central to the development of wheals and angioedema.[5,6] Inadequate vitamin D could therefore favour a more reactive mast cell phenotype, lower the threshold for degranulation, or amplify ongoing inflammation in the skin. Additionally, vitamin D's effects on regulatory T cells and autoantibody production may be relevant in the subset of CSU patients with autoimmune mechanisms, where autoantibodies against IgE or its high-affinity receptor have been implicated.[1]

Material and methods

This study was designed as a hospital-based analytical cross-sectional investigation conducted at a tertiary care hospital. The research aimed to evaluate the association between the severity of Chronic Spontaneous Urticaria (CSU) and serum vitamin D levels among adult patients diagnosed with CSU. The study environment included outpatient and inpatient dermatology units, where standardized clinical assessments and laboratory investigations were performed by trained clinicians and laboratory personnel.

A total of **70 patients** diagnosed with Chronic Spontaneous Urticaria were included in the study. All participants were adults aged 18 years and above, presenting with recurrent wheals, angioedema, or both, persisting for at least six weeks without identifiable triggers. Eligibility was confirmed based on clinical history and dermatological evaluation. Detailed demographic information including age, sex, BMI, occupation, socioeconomic status, and residence was systematically recorded.

Inclusion and Exclusion Criteria

Patients were included if they met the clinical criteria for CSU and provided informed consent. Exclusion criteria involved individuals with physical urticarias, autoimmune connective tissue diseases, acute urticaria, chronic infections, or known vitamin D supplementation within the previous three months. Patients with chronic renal failure, liver disease, pregnancy, lactation, or those on immunomodulatory therapy were also excluded. These criteria ensured that the serum vitamin D level reflected the natural physiological state rather than pharmacological or disease-related alterations.

Methodology

The severity of CSU was assessed using the **Urticaria Activity Score (UAS7)**, a validated scoring system that quantifies symptom burden based on the number of wheals and intensity of pruritus over seven days. Patients were categorized into mild, moderate, and severe CSU groups based on standard UAS7 cutoffs. Additional clinical parameters included duration of disease, frequency of relapses, presence of angioedema, sleep disturbance, impact on daily activities, and requirement of antihistamines or systemic therapies. These variables provided a comprehensive evaluation of disease severity and burden.

Laboratory Investigations

Venous blood samples were collected under aseptic conditions from all participants after an overnight fast. Serum **25-hydroxyvitamin D [25(OH)D]** levels were measured using a standardized chemiluminescent immunoassay method in the hospital's central laboratory. Vitamin D status was classified as sufficient, insufficient, or deficient according to established clinical thresholds. Additional laboratory parameters—including complete blood count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), thyroid-stimulating hormone (TSH), total IgE level, fasting glucose, liver function tests, and renal function tests—were obtained to rule out

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confounding conditions and assess possible associations with CSU severity.

All patient information, clinical findings, laboratory results, and questionnaire-based assessments were recorded using a structured proforma specifically designed for this study. Data collection was performed by dermatology residents trained in standardized interviewing and examination techniques. To minimize observer bias, the clinical scoring process for CSU severity was independently verified by a senior consultant dermatologist. All laboratory investigations were processed under uniform protocols to ensure analytical accuracy and reproducibility.

Statistical Analysis

Data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables such as vitamin D levels and UAS7 scores were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The relationship between serum vitamin D levels and CSU severity was evaluated using Pearson or Spearman correlation tests, depending on data distribution. Group comparisons between different CSU severity categories were performed using independent t-tests, one-way ANOVA, or nonparametric alternatives as appropriate. A p-value of **<0.05** was considered statistically significant. Multivariate linear regression was conducted to adjust for confounding factors such as age, BMI, gender, and inflammatory markers.

Results

Table 1: Demographic Characteristics of CSU Patients

Table 1 presents the demographic distribution of the 70 patients included in the study. The majority of patients belonged to the 31–45-year age group, accounting for 40.00% of the sample, indicating that CSU is most commonly seen in middle-aged adults. Patients aged 18–30 years comprised 31.43%, while those older than 45 years represented 28.57%, suggesting that CSU affects a wide adult age range without extreme age predominance. Regarding gender distribution, females were slightly more affected than males, with 54.29% females compared to 45.71% males, indicating a mild female predominance, which aligns with known autoimmune tendencies in women. The BMI profile showed that nearly half of the patients (45.71%) were overweight, followed by 28.57% who were obese, whereas only 25.71% had normal BMI values. This pattern suggests a possible association between increased body weight and CSU occurrence or severity. Additionally, most patients (58.57%) resided in urban areas, while 41.43% were from rural settings, indicating higher healthcare access or higher disease reporting in urban populations.

Table 2: CSU Severity Distribution Based on UAS7 Scores

Table 2 outlines the severity of Chronic Spontaneous Urticaria based on UAS7 scoring. A substantial proportion of patients (40.00%) had moderate CSU, indicating that most individuals experience a significant but not extreme burden of symptoms. Mild CSU was observed in 25.71% of patients, while severe CSU was present in 34.29%, demonstrating that over one-third of the cohort experienced high disease severity with frequent wheals and intense itching. The mean UAS7 score of 22.84 ± 8.15 further supports the observation that the majority of the population falls into the moderate severity category.

Table 3: Vitamin D Status Among CSU Patients

Table 3 describes the distribution of vitamin D levels among the study participants. More than half of the patients, 54.29%, were found to have vitamin D deficiency (<20 ng/mL), with a mean level of 13.42 ± 3.21 ng/mL, indicating substantially low vitamin D status in this subgroup. Another 31.43% exhibited insufficient vitamin D levels (20–29 ng/mL), with a mean value of 24.18 ± 2.56 ng/mL. Only a small proportion, 14.29%, had sufficient vitamin D levels (≥30 ng/mL), with a mean of 34.67 ± 3.09 ng/mL. These findings reveal that vitamin D inadequacy is highly prevalent among CSU patients, with nearly 85% showing either deficiency or insufficiency.

Table 4: Relationship Between Vitamin D Status and CSU Severity

Table 4 evaluates the association between CSU severity categories and mean serum vitamin D levels. It clearly demonstrates a declining trend in vitamin D concentrations as CSU severity increases. Patients with

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mild CSU had the highest mean vitamin D levels (27.54 ± 4.12 ng/mL), followed by those with moderate CSU (19.86 ± 3.98 ng/mL). The lowest vitamin D levels were seen in patients with severe CSU (14.72 ± 3.44 ng/mL). The ANOVA test yielded a p-value of 0.001, indicating this association is statistically significant.

Table 5: Correlation Between Vitamin D Levels and UAS7 Scores

Table 5 provides further insight into the relationship between vitamin D levels and CSU severity through correlation analysis. The mean vitamin D level was 21.07 ± 8.52 ng/mL, and the mean UAS7 score was 22.84 ± 8.15. A significant negative correlation was found between vitamin D levels and UAS7 scores, with a correlation coefficient (r) of –0.62 and a p-value of 0.001. This indicates that as vitamin D levels decrease, the UAS7 scores rise, reflecting increased CSU severity. The strength of the correlation (moderate to strong) suggests that vitamin D may play a meaningful role in modulating disease activity.

Table 1. Distribution of Demographic Characteristics of CSU Patients (N = 70)

Variable	Category	Frequency (n)	Percentage (%)
Age Group (years)	18–30	22	31.43%
	31–45	28	40.00%
	>45	20	28.57%
Gender	Male	32	45.71%
	Female	38	54.29%
BMI Category	Normal	18	25.71%
	Overweight	32	45.71%
	Obese	20	28.57%
Residence	Urban	41	58.57%
	Rural	29	41.43%

Table 2. Distribution of CSU Severity Grades Based on UAS7 (N = 70)

Severity Category	Frequency (n)	Percentage (%)
Mild (UAS7 < 16)	18	25.71%
Moderate (UAS7 16–27)	28	40.00%
Severe (UAS7 > 27)	24	34.29%

Mean UAS7 score: 22.84 ± 8.15

Table 3. Vitamin D Status Among CSU Patients (N = 70)

Vitamin D Category	Frequency (n)	Percentage (%)	Mean 25(OH)D (ng/mL) ± SD
Deficient (<20 ng/mL)	38	54.29%	13.42 ± 3.21
Insufficient (20–29 ng/mL)	22	31.43%	24.18 ± 2.56
Sufficient (≥30 ng/mL)	10	14.29%	34.67 ± 3.09

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Table 4. Association Between Vitamin D Status and CSU Severity (N = 70)

CSU Severity	n	Mean Vitamin D (ng/mL) ± SD
Mild	18	27.54 ± 4.12
Moderate	28	19.86 ± 3.98
Severe	24	14.72 ± 3.44

ANOVA p-value = 0.001 (statistically significant)

Table 5. Correlation Between Serum Vitamin D Levels and UAS7 Scores

Variable	Mean ± SD	Correlation Coefficient (r)	p-value
Vitamin D (ng/mL)	21.07 ± 8.52	**–0.62**	**0.001**
UAS7 Score	22.84 ± 8.15	—	—

Discussion

In the present study, CSU predominantly affected adults between 31 and 45 years (40.00%), with a mild female predominance (54.29%) and a high proportion of overweight (45.71%) and obese (28.57%) patients, and most patients residing in urban areas (58.57%). This pattern is broadly compatible with large international CSU cohorts such as the ASSURE-CSU study by Maurer et al. (2017), which reported a mean age of 48.8 years and a stronger female predominance of 72.7%, indicating that CSU generally affects working-age adults and is more frequent in women, although our cohort was slightly younger and less skewed towards females.[8] Our higher burden of excess body weight may also point to a possible link between metabolic factors and CSU, which has been suggested in other real-world series, though not systematically quantified in ASSURE-CSU.

With respect to disease activity, most of our patients had moderate (40.00%) or severe (34.29%) CSU, with a mean UAS7 of 22.84 ± 8.15, placing the average patient clearly in the moderate range. This degree of activity is higher than that reported in the ASSURE-CSU UAS7 analysis by Hollis et al. (2018), where 16.5% of patients fell in the UAS7 0–6 band, 34.0% in 7–15, 30.1% in 16–27, and 19.3% in 28–42, such that roughly half (49.4%) had moderate-to-severe disease (UAS7 ≥ 16).[9]In contrast, our study shows 74.29% of patients in the moderate-to-severe categories (UAS7 ≥ 16), suggesting that patients presenting to our tertiary-care setting may represent a more symptomatic, referral-biased subset with heavier disease burden than those in broader multinational cohorts.

Vitamin D insufficiency and deficiency were strikingly common in our CSU cohort: 54.29% were frankly deficient (<20 ng/mL; mean 13.42 ± 3.21 ng/mL), 31.43% insufficient (20–29 ng/mL; mean 24.18 ± 2.56 ng/mL), and only 14.29% had sufficient levels (≥30 ng/mL; mean 34.67 ± 3.09 ng/mL), giving an overall mean 25(OH)D of 21.07 ± 8.52 ng/mL. These findings mirror those of Movahedi et al. (2015), who demonstrated significantly lower serum 25(OH)D levels in patients with chronic idiopathic urticaria compared with controls and concluded that vitamin D deficiency increased susceptibility to CIU.[10]Although Movahedi et al. did not categorise patients exactly as in our study, they similarly found a high frequency of suboptimal vitamin D status and a clear separation between urticaria patients and healthy individuals, supporting our observation that vitamin D insufficiency is the rule rather than the exception in CSU.

The proportion of our patients with vitamin D below sufficiency (deficient + insufficient: 85.72%) aligns closely with Indian data. In a hospital-based study from Jammu, Dass et al. (2022) reported vitamin D deficiency in 76.2% and insufficiency in 19.2% of CSU patients (total 95.4% below 30 ng/mL), compared with only 16.1% deficiency and 35.4% insufficiency in controls.[11]They also found a significant inverse correlation between serum vitamin D levels and UAS7 scores ($r = -0.538$, $p < 0.001$). Our cohort shows a slightly lower proportion of frankly deficient patients (54.29% vs 76.2%) but still a very similar overall burden of suboptimal vitamin D (85.72% vs 95.4%), and the correlation between vitamin D and UAS7 in our data ($r = -0.62$, $p = 0.001$) is even stronger in magnitude. Together, these findings suggest that the relationship between low vitamin D status and higher

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CSU activity is robust across different Indian tertiary-care settings.

The stepwise decline in mean vitamin D concentrations with increasing CSU severity in our study—27.54 ± 4.12 ng/mL in mild cases, 19.86 ± 3.98 ng/mL in moderate, and 14.72 ± 3.44 ng/mL in severe CSU (ANOVA $p = 0.001$)—is consistent with prior work exploring vitamin D as a marker of disease activity. Woo et al. (2015) evaluated 72 patients with chronic urticaria and found that serum 25(OH)D levels were significantly lower in chronic urticaria than in acute urticaria, atopic dermatitis, or healthy controls, and that the proportion of subjects with "critically low" vitamin D (<10 ng/mL) reached 49% in the chronic urticaria group versus only 8% in healthy controls.[12]They also demonstrated a significant negative trend between UAS and 25(OH)D ($p < 0.001$), with higher disease severity associated with progressively lower vitamin D levels. Our observation that patients with severe CSU had vitamin D levels nearly 13 ng/mL lower than those with mild disease fits well with this graded pattern and reinforces the concept that vitamin D status may reflect underlying inflammatory activity.

The overall negative correlation between serum vitamin D levels and UAS7 scores in our cohort ($r = −0.62$, $p = 0.001$) also sits comfortably within the broader quantitative evidence base. In a systematic review and meta-analysis of 17 observational studies and 6 supplementation trials, Li et al. (2021) reported that urticaria patients had serum 25(OH)D levels on average 9.35 ng/mL lower than controls (95% CI −12.27 to −6.44), and that vitamin D deficiency was consistently more frequent in adult chronic urticaria subgroups.[13] Moreover, vitamin D supplementation trials analysed by Li et al. showed significant reductions in urticaria severity scores, with pooled standardised mean differences of −3.63 in randomised controlled trials and −1.54 in repeated-measures studies. When considered alongside these pooled results, our correlation coefficient of −0.62 indicates a clinically meaningful association between lower vitamin D levels and worse CSU activity rather than a trivial or incidental relationship.

Although our study is observational, the strong inverse relationship between vitamin D and CSU severity dovetails with interventional data suggesting that vitamin D repletion may improve symptom control. In a randomised trial of 42 chronic urticaria patients, Rorie et al. (2014) compared low-dose (600 IU/day) and high-dose (4,000 IU/day) vitamin D_3 as an add-on to standard triple-drug therapy for 12 weeks. They observed an initial 33% reduction in Urticaria Symptom Severity (USS) scores with standard therapy in week 1, followed by an additional 40% reduction by week 12 in the high-dose vitamin D group, whereas the low-dose group did not show a comparable further decline.[14]High-dose vitamin D also tended to improve pruritus and sleep quality, with no major safety issues. Although that trial did not include UAS7 specifically and the correlation between change in 25(OH)D and symptom scores was not strong, the magnitude of clinical benefit is compatible with our observation that patients with higher vitamin D levels cluster in the milder UAS7 categories.

Taken together, our findings align well with the broader CSU–vitamin D literature summarised by Tuchinda et al. (2018), who systematically reviewed studies on chronic spontaneous urticaria and reported that vitamin D deficiency in CSU cohorts typically ranged from 34.3% to 89.7%, compared with 0.0% to 68.9% in control groups, and that most interventional studies showed symptomatic improvement after vitamin D supplementation.[15]Our figure of 54.29% deficiency and 31.43% insufficiency (total 85.72% below sufficiency) therefore lies within the upper part of this reported range, and our strong inverse correlation between vitamin D and UAS7 supports the hypothesis that vitamin D insufficiency may contribute to the burden of CSU rather than merely co-existing as a benign comorbidity.

Conclusion

The present study demonstrates a high prevalence of vitamin D deficiency and insufficiency among patients with chronic spontaneous urticaria, with nearly 85% having suboptimal 25(OH)D levels. A significant inverse relationship was observed between serum vitamin D levels and CSU severity, with lower vitamin D levels associated with higher UAS7 scores and more severe disease. These findings suggest that vitamin D status may play an important modulatory role in CSU activity and could be considered in the routine evaluation of such patients. However, as this was a cross-sectional study, longitudinal and interventional trials are needed to determine whether vitamin D

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supplementation can effectively reduce disease severity and improve clinical outcomes.

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