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Clinico- Etiological Study of Urticaria in a Tertiary Care Hospital

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Abstract: *In spite of frequency of Acute&chronic urticaria there are few studies of its prevalence and causes, aetiology. Appropriate management of urticaria depends on the correct evaluation of clinical patterns and causes where these can be identified. Guidance for treatment is presented, based on the strength of evidence available at the time of preparation. As many of the recommendations relate to the off-licence use of drugs, it is particularly important that clinicians should be familiar with dosing and side-effects of treatment in the context of managing urticaria.*

Materials and Methods:*Patients who attended the Dermato-venereology out-patient department of S.V.R.R Govt.General Hospital,Tirupati with urticarial weals were examined and fifty patients who underwent all the preliminary investigations were included in this study.each patient interrogated about their H/O disease. Aboslute eosinophil count if DLC showed more than 10% eosinophils Peripherl blood smear for malaria and filarial parasites,urine for albumin, sugar,microscopic done Stool for Ova, cysts & intestinal parasites.*

Results:*Of all the associated diseases that we have observed, dermatophytosis and pityriasis versicolor appears to be co-incidental.Certain systemic diseases such as systemic lupus erythematosus, rheumatic fever and malignancy may be seen uncommonly in association with urticaria. , dermographism was elicited in 3 cases (6%) of urticaria. dermographism in 7.4% cases of urticaria compared to 2.7% in controls. Cold urticaria is a rare form of physical urticaria. One case with cold urticaria not showing positive response to cold test that some patients with cold urticaria respond to general body cooling or excessive. In a study wherein only the patients with physical urticaria were evaluated, cholinergic urticaria was observed in 10.8% In a total of 10 cases where evidence of infective foci were present, 4 cases responded to specific anti-infective treatment by clearing of urticaria and there was no recurrence of urticaria over a follow-up period from 2 to 4 months. In these four cases of urticaria, infective foci was considered as the probable cause of urticaria. The infective conditions included: caries tooth in one case, chronic tonsillitis in one case, bronchitis in one case pyoderma in one case, considering the response to antibiotics as the criteria for the presence of infection to be the cause of urticaria, reported 28 cases (5.7%). Although dental caries is incriminated in the causation of urticaria, Parasitic infestationsThe present study blames intestinal parasites as the probable cause of urticaria in 2 cases (4%) because, deworming had curative effect on urticaria only in 2 out of 6 cases of infestation.Eleven cases gave definite history of acute urticaria following drug intake(analgin)*

Conclusion*: CU is a complex disorder that has a substantial economic burden and a significant impacton patients' QoL . A complete history and physical examination will ensure the accurate diagnosis of CU and will determine the extent of laboratory studies needed for each individual patient.*

Key words: *Chronic urticaria; Idiopathic urticaria; itching; cold urticaria; chronic sponeous/idiopathic urticaria; physical urticaria; inducible urticaria; weals; hives; guidelines*

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I. Background

Urticaria (hives) is a relatively common condition, with a point prevalence of about 0.5–1%. The peak incidence is in the range of 20–40 years. Urticaria is the general term for a cutaneous response characterized by weals and swellings. A deeper localized swelling often associated with urticaria is called angioedema. Urticaria is mediated by mast cell degranulation. Mast cells can be activated by immunologic and non-immunologic mechanisms, which lead to degranulation of inflammatory mediators including histamine, leukotrienes, and prostaglandins.[Release of these mediators causes the characteristic pruritus, vascular permeability, and edema[1]

Classification: Urticaria traditionally classified in to acute and chronic. Urticaria present daily for less than 6 weeks called acute urticaria. If urticaria occurs continuously longer than this is called chronic urticaria[1]

There are several factors both immunologic and non- immunologic that cause the mast cells and basophils to release the mediators. A wide variety of drugs and compounds can release histamine by mechanisms not known to involve known immunologic processes. Histamine release by non-immunologic means can also result from the direct effect of physical agents and also by non-immunologic cholinergic effects as in cholinergic urticaria. The most common of immunological mechanisam is the type 1 hypersensitivity state mediated by igE.[Fig:1]

Ordinary urticarial: [Fig-3]

Clinical features

Itching erythematous macules develop into weals consisting of pale to pink, oedematous, raised areas of the skin often with a surrounding red flare. They occur anywhere on the body, including scalp, palms and soles, in variable numbers and sizes, ranging from a few millimetres to lesions covering large areas, and of varying shapes including rounded, annular, serpiginous and bizarre patterns due to confluence of adjacent lesions .Weals generally lasts few hours and resolve within 24 h, passing through a macular erythematous phase, leaving the skin with *a* normal appearance. They are generally very itchy.[1]

There may be associated angio-oedema in 50% of patients with ordinary urticaria. These deep swellings, which may be the same colour as normal skin, occur most frequently on the face, affecting the eyelids and lips, on the buccal mucosa, tongue and pharynx but laryngeal involvement is fortunately rare. Angio-oedema may be preceded by an itching or tingling sensation, but it is not always itchy and may be painful. The lesions may last for several days. Urticari may be preceded by vomiting, malaise loss of concentration, abdominal, pain, diarrhoea arthralgia, dizziness and syncope and, in its most severe acute form, with anaphylaxis**. [Chart-1&2].**

Urticaria in childhood: with a tendency to more cases of acute disease. Cow's-milk allergy is the commonest cause of urticaria in infants under 6 months old. In infants, there may be less itching and a greater tendency for wheals to become purpuric. Acute haemorrhagic oedema of infancy (purpura en cocarde) occurs in very young children and is borderline between urticaria and vasculitis

Aetiology of ordinary urticarial : Although some cases of acute urticaria can be ascribed to allergy, non-allergic causes or infections and around 50% of chronic urticaria may be attributed to autoimmunity, other cases will remain idiopathic after evaluation.

Acute urticaria: Most acute urticaria is ordinary and some will become chronic. Serum sickness due to injection of therapeutic animal antisera (e.g. to snake venoms) is now rare. Physical urticarias will go through an acute phase but most will last more than 6 weeks. Contact urticaria does not usually present as acute urticaria because it tends to be localized, intermittent and short-lived. The incidence in the community is not well defined.

Idiopathic:

This form of acute urticaria, in which no cause can be identified, was found in more than 50% of patients with acute urticaria common preceding event was an upper respiratory tract infection, followed by drug ingestion.

Allergic:

Allergic urticaria is due to interaction of an allergen with specific preformed IgE bound to the mast cell. It is commoner in people who are atopic and have elevated IgE levels, Although it is unusual to find an allergic cause for acute urticaria[1] any drug, food, foreign substance from blood transfusion, injection, implant, contactant and inhalant should be considered as a potential allergen

Acute urticarial reactions to food are believed to be common reactions to other ingredients, such as seeds or spices. Development of bee-sting allergy usually requires multiple exposures, and is commonly seen in beekeepers..

Here, prodrornal symptoms include itching or tingling of the mouth, palms, soles and genital area. Subsequent signs include widespread erythema, urticaria, lacrimation, nasal stuffiness, bronchospasm, laryngeal oedema, nausea, diarrhoea, vomiting, hypotension or cardiac arrhythmias and can rarely lead to death.

Infections

Urticaria may follow non-specific viral infections Epstein-Barr or acute hepatitis B viral infections,

Inhalants:
Grass pollens, mould spores, animal danders and house dust mite have been implicated as triggers of acute or chronic urticaria

Chronic urticaria:
Chronic urticaria of at least 6 weeks' duration may be ordinary, physical or vasculitic but the term is often considered to be synonymous with the ordinary presentation of chronic urticaria.

Idiopathic
Most patients were considered to have 'idiopathic' urticaria before autoimmunity was recognized as a cause, autoantibodies with the basophil histamine release assay. Degranulate hyper-releasable mast cells in chronic urticaria but not the normal basophils of healthy donors..

Autoimmune
There is increasing acceptance that histamine releasing autoantibodies define an autoimmune aetiology in an important subset of patients.sera from chronic urticaria patients with a positive ASST were histamine releasers from a panel of basophils and mast cells from different donors

Pseudoallergy
However, dietary additives and salicylates may be one of many factors that aggravate existing chronic urticaria and this should be explored in the medical history

Infections and infestations
The incidence of bacterial infections, such as dental sepsis, sinusitis, urinary tract and gallbladder infections, in chronic urticaria varies in different series.

Aggravating factors: Even though a specific cause of urticaria may not be identified in individual patients it is often possible to identify non-specific aggravating factors in chronic urticaria, such as heat and clothing pressure in ordinary utticaria,

Drugs:
Salicylates and other related NSAIDs, such as diclofenac, can aggravate urticaria and asthma by non-allergic mechanisms. Reactions are usually not life-threatening. ACEls can provoke arigio-oedema and may, perhaps, aggravate urticaria. The relationship between penicillin and chronic urticaria is a complex one, but the amount present in dairy products seems unlikely to perpetuate chronic urticaria except in people with extreme penicillin sensitivity, or if penicillin is present in food above permitted levels.

Foods and food additives
Numerous foods have been blamed as a cause of urticaria. There are many reports that food additives aggravate chronic urticaria, Menstrual cycle and pregnancy Urticaria may occur in pregnancy.. It should be distinguished from pruritic urticarial papules and plaques of pregnancy (PUPPP

Physical and cholinergic urticarias: physical stimulus induces reproducible whealing. Cholinergic urticaria occurs in response to sweating caused by an increase in core temperature, but it may also be triggered by emotional and gustatory sweating.

Urticaria due to mechanical forces
Dermographism[1]
Immediate symptomatic dermographism[Fig-3] (factitious urticaria) This involves the triple response which may arise from firm stroking of the skin This response consists of local erythema due to capillary vasodilatation, followed by oedema and a surrounding flare due to axon reflex-induced dilatation of arterioles. Symptomatic dermographism may have an immunological basis.

Vibratory angio-oedema
Vibratory urticaria is a very rare form of urticaria, Any vibratory stimulus such as jogging, vigorous towelling or using lawnmowers induces a localized, red, itchy swelling within minutes and lasting less than a few hours, vibratory, angio-oedema may occur in an acquired form

Temperature-dependent urticaria
Heat urticaria
Cholinergic urticaria

Cholinergic urticaria small weals appear in association with sweating. Wealing occurson stimulation of sweating.

Cold-Urticaria: Cold induces urticarial reaction

Solar urticarial, Aquagenic urticaria, Urticarial vasculitis (Type III urticarial reaction).

Contact urticaria: classified in to allergic urticaria, Oral allergic syndrome**,** non-allergic urticaria**,** Angio-oedema (without weals), Idiopathic angio-oedema, ACEI-induced angio-oedema ,Hereditary angio-oedema, Acquired C1 esterase inhibitor deficiency angio-oedema, Episodic angio-oedema with eosinophilia[1]

Other syndromes resembling urticarial or angio-oedema**,**[Fig-3] or with urticarial as one component**:**
1) Papular urticaria: Injection of foreign protein by biting insects in to skin of sensitive subjects cause immediate IgE -mediated reaction consisting of weals.
2) Systemic capillary leak syndrome:
3) Schnitzler's syndrome:
4) Herditary periodic fever syndromes: Cryopyrin-associated periodic syndrome.
5) Familial Mediterranean fever:
6) Hyper-IgD Syndrome:
7) TNF-receptor-associated periodic syndrome(TRAPS):

How to diagnose CsU caused by food intolerance: The current guide lines recommended a 3 week pseudoallergen–free elimination diet to test for CsU due to intolerance.Response to diet should be assessed by urticaria activity score(UAS**)[Table-III]**

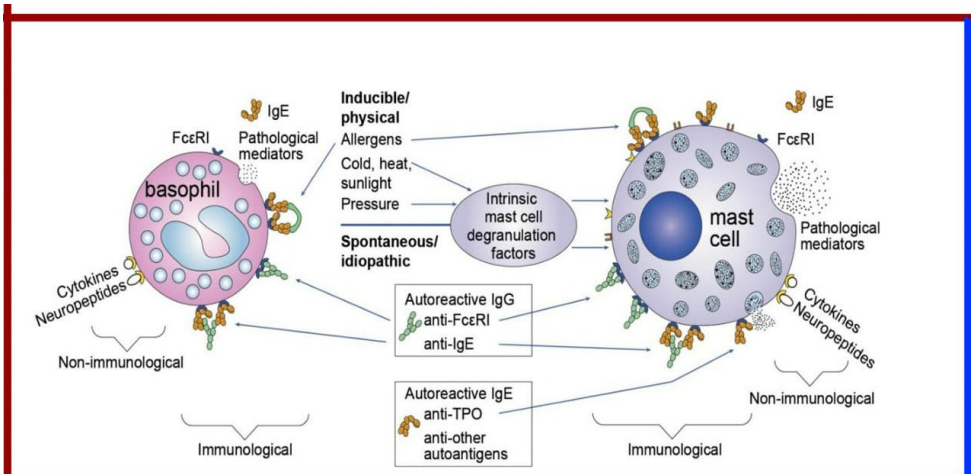
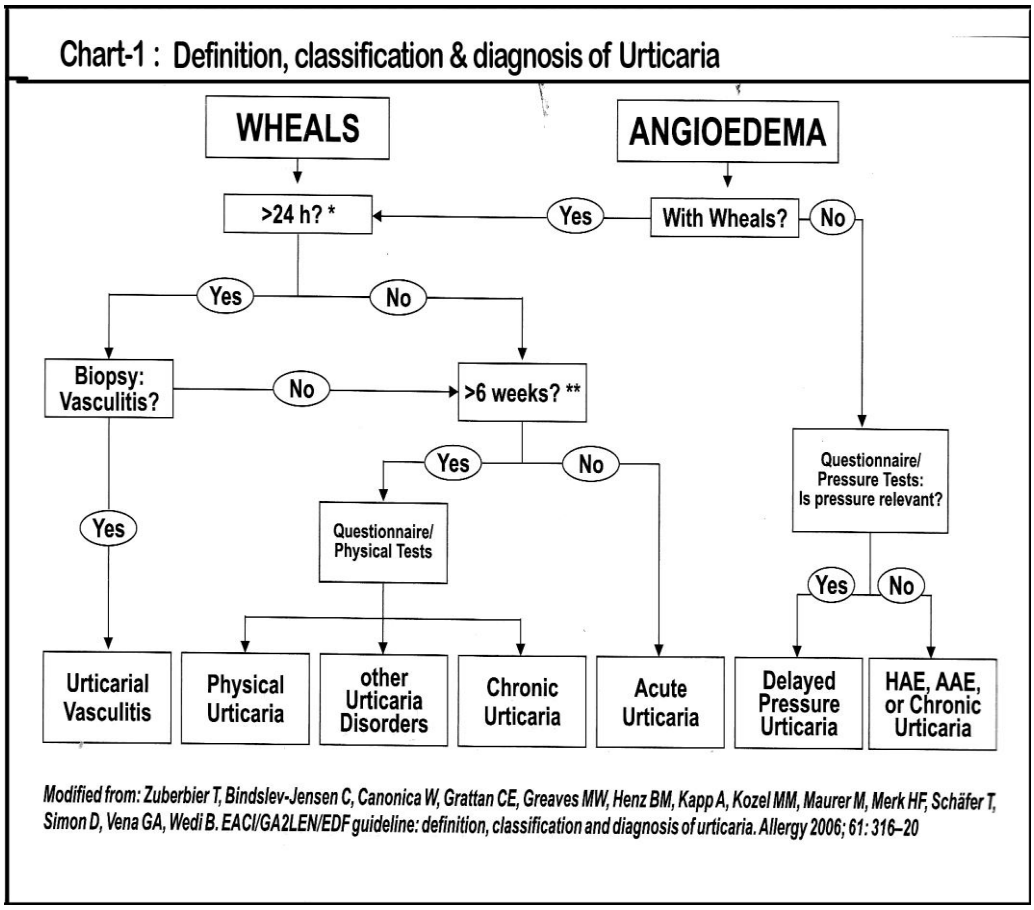


Fig. 1. Pathogenesis of chronic urticaria (CU). CU signs and symptoms develop when skin mast cells or basophils degranulate and release histamine and other proinflammatory mediators. In chronic spontaneous urticaria, the degranulation of these cells in some patients is thought to be due to the effects of autoantibodies directed against a subunit of the high-affinity IgE receptor, FcεRIa, or to IgE itself. Other mechanisms of mast cell or basophil activation that are potentially relevant to CSU involve autoantigens and IgE directed against these autoantigens, as well as complement components, cytokines and neuropeptides. TPO: thyroperoxidase.



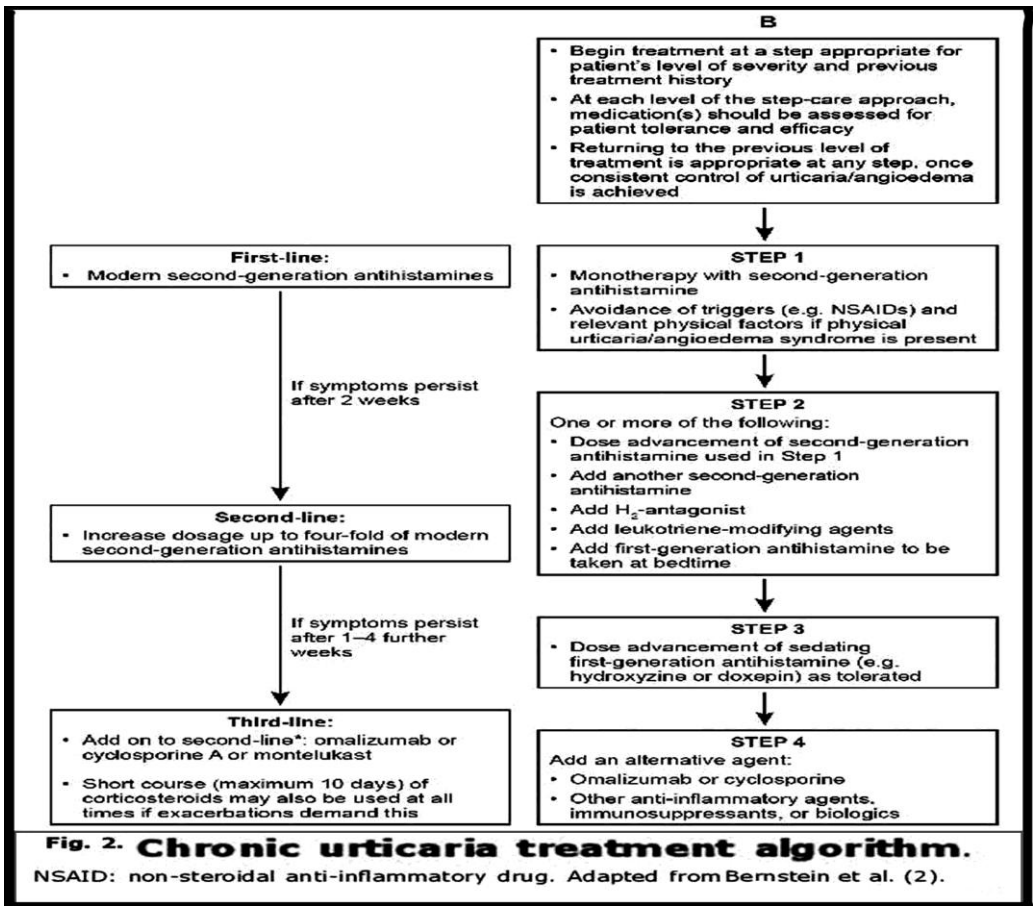


Fig. 2. Chronic urticaria treatment algorithm.

NSAID: non-steroidal anti-inflammatory drug. Adapted from Bernstein et al. (2).

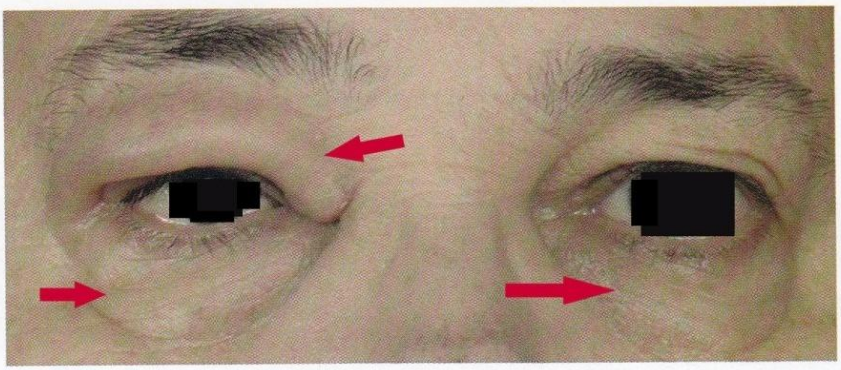


Fig 1 : Angioedema of eyes

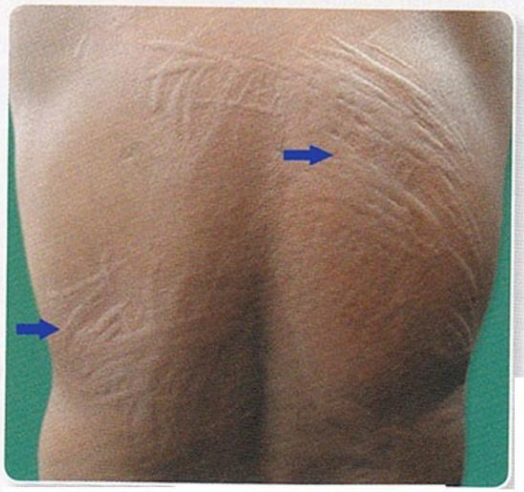


Fig- 2: Dermographism on the back

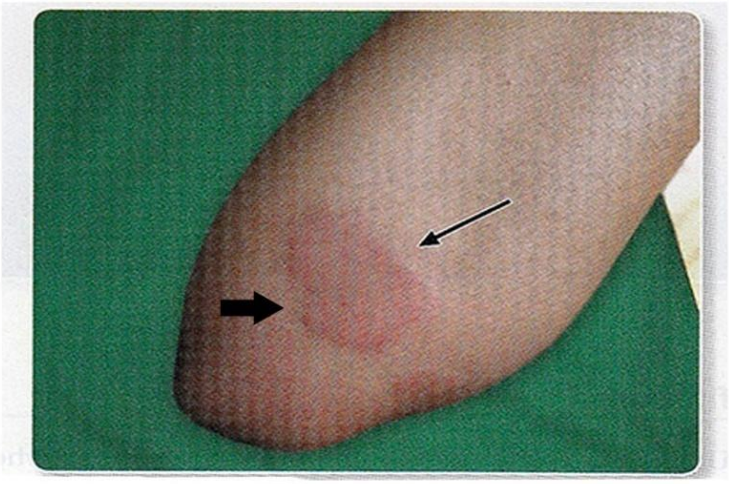


Figure 3 : A typical wheal on elbow

Fig-3: Various types of urticaria

Table II. Comparison of recommendations for confirming the relevance and threshold of triggers for inducible chronic urticaria (CU) in the EAACI/GA²LEN/EDF/WAO international guidelines and the US practice parameters for the diagnosis and management of CU

Various types of urticaria & and their induction	EAACI/GA²LEN/EDF/WAO international guidelines	US practice parameters for the diagnosis and management of CU (2)
1) Cold urticaria	Cold provocation and threshold test *Extended:* CBC with differential, ESR and/or CRP level, cryoproteins	Apply cold stimulus (e.g. ice cube on forearm) and observe for wheal-and-flare reaction during skin rewarming
2) Delayed pressure urticaria/ angioedema	Pressure and threshold test	Challenge with a 15 lb (6.8 kg) weight suspended over shoulder for 10–15 min and monitor for angioedema development
3) Heat urticaria	Heat provocation and threshold test	*Does not include as a separate subtype; patients with lesions in response to heat are categorized as having cholinergic urticaria*
4) Solar urticaria test	UV and visible light of different wavelengths and threshold *Extended:* Rule out other light-induced dermatoses	Phototest to various wavelengths of light
5) Symptomatic dermatographism	Elicit dermographism and threshold test (dermographometer) *Extended:* CBC with differential, ESR and/or CRP level	Stroke skin with firm object (e.g. tongue blade or other instrument with a firm edge) or a dermographometer
6) Vibratory angioedema	Test with vortex	Expose to a vortex mixer
7) Aquagenic urticaria	Wet cloth (body temperature) for 20 min	Water compress (35°C) applied to the upper body for 30 min
8) Cholinergic urticaria	Exercise and hot bath provocation	Provocative challenges that increase core body temperature (e.g. exercise, hot water immersion, or methacholine intradermal challenge)
9) Exercise-induced urticaria	*Considered a form of anaphylaxis, not urticaria*	Exercise challenge in a setting prepared for anaphylaxis management
10) Contact urticaria for	Cutaneous provocation test. Skin tests with immediate readings, example prick test	Cutaneous provocation test, skin test with immediate readings such as prick test

CBC: complete blood count; CRP: C-reactive protein; EAACI: European Academy of Allergy and Clinical Immunology; EDF: European Dermatology Forum; ESR: erythrocyte sedimentation rate; GA²LEN: Global Allergy and Asthma European Network; UV: ultraviolet; WAO: World Allergy Organization.

Table – III : Urticaria activity score (UAS)[1]

How many wheals have appeared during the last 24 hours?	Scoring
None	0
Mild (<20 wheals/24 hours)	1
Moderate (20-50 wheals/24 hours)	2
Intense (>50 wheals/24 hours)	3
How severe was the itching during the last 24 hours?	**Scoring**
None	0
Mild (present but not annoying or troublesome)	1
Moderate (troublesome but does not interfere with normal daily activity or sleep)	2
Intense (severe itch, which is sufficiently troublesome to interfere with normal daily activity or sleep)	3

The above urticarial activity score helps dermatologist how to plan the treatment modalities (Table:4)

TREATMENT: [Fig:2]

Approved doses of second-generation **H1**-antihistamines are the universally recommended first-line therapy for Chronic urticaia. A progressive increase to up to 4-fold the standard dose is recommended for patients who do not respond to approved doses [2]

First-generation antihistamines have similar efficacy, but greater sedation and impairment compared with second-generation antihistamines, and should therefore be used with caution.H_2-antihistamines, specifically cimetidine, used in combination with H_1-antihistamines have shown a limited additive effect.

Oral corticosteroids are frequently used in patients with CU not adequately controlled with antihistamine therapy. A large retrospective study found that 50% of patients with antihistamine-resistant CU treated with a single course of prednisone (25 mg/day for 3 days, de-escalated to 12.5 mg/day for 3 days and 6.25mg/day for 4 days) had a remission, and an additional 9% responded after a second course. The main concern with the use of corticosteroids is the risk of adverse effects. Leukotriene-modifying agents (LTMAs) such as montelukast and zafirluast, are reportedly effective for the treatment of CU as monotherapy or in combination with H_1-antihistamines, with the strongest evidence for montelukast (10 mg/day), although the treatment effect observed was small. [2]

Agents with $\mathbf{H_1}$- and/or $\mathbf{H_2}$-antagonist activity such as hydroxyzine, cyproheptadine, or doxepin are also options for patients whose symptoms do not respond to prior antihistamine therapy, but they have considerable sedating effects. Compared with other antidepressants .If symptoms persist after 1-4 further weeks such as amitriptyline, nortriptyline, and mirtazapine, clinical evidence is strongest for doxepin (at doses from 10 mg to 25 mg 3 times daily).

For patients with refractory CU, omalizumab, an anti-IgE antibody, has the most robust data supporting its use. Although omalizumab (administered as subcutaneous injections every 4 weeks at doses of 150 mg, or 300 mg) has a favourable risk /benefit ratio and was well tolerated in clinical studies it has infrequently been associated with anaphylaxis. Omalizumab has also been shown to be an efficacious treatment alone or as an add-on therapy to H_i-antihistamine plus H_2-antihistamine or LTMA, or a combination of these for patients with CIU refractory to antihistamine treatment in 3 Phase 3 studies. However, the cost of treatment, the requirement for subcutaneous administration in a physician's office and anaphylaxis concerns may limit its use. [2]

In addition to omalizumab, both the international guidelines and the US practice parameters recommend consideration of cyclosporine A (**CsA**) for patients with refractory CU (1, 2). cyclosporine A is an immunosuppressant that has been shown to be an effective treatment for CU (at dosages of 3-5 mg/kg/day for up to 4 weeks) in placebo-controlled studies as a solo treatment and in combination with second-generation H_1-antihistamines. Treatment with cyclosporine A is associated with a relatively high incidence of mild adverse effects including gastrointestinal disturbances, paresthesia and infections; retrospective study showed that adverse effects were generally mild and transient for patients with CU using low-dose CsA (<3 mg/kg/day) for up to 10 years. However, long-term, low-dose CsA treatment is known to be associated with nephrotoxicity.Anti-inflammatory agents, including dapsone, sulfasalazine, hydroxychloroquine and colchicine, have limited evidence for efficacy in CU . It remains to be confirmed whether these agents are more effective in patients with neutrophil-rich urticaria. An open study reported that among CU patients with neutrophilic skin inflammation, 8 of 9 treated with colchicine and 3 of 3 treated with dapsone showed a response. Other immunosuppressants to consider include tacrolimus, mycophenolate and methotrexate, but clinical evidence supporting their use is very low . Case reports suggest that the anti-CD20 biologic, rituximab, may also provide some benefit.[2]

Material & Methods: Patients who attended the Dermato-venereology out-patient department of S.V.Medical College/ S.V.R.R Govt. General Hospital,Tirupati.
From September,2017 to September, 2018 with urticarial weals were examined and fifty patients who underwent all the preliminary investigations were included in this study.each patient interrogated about their H/O disease.

Inclusive criteria: all cases attending to dermato-venereology o.p & patients having history of urticaria more than 6 weeks were included in study.

Exclusive Criteria: (a) Uncertain allergy history. (b) Non-IgE-mediated prior reaction. (c) Medication interfering with anaphylaxis therapy.(d) Skin condition that gives false positive reaction. (e) H/O severe exfoliative reaction (f) anaphylaxis less than 4 weeks prior (g) The patients on immunosuppressive therapy (h) patients with known immunological disease (i)patient on systemic corticosteroid for long duration were excluded from the study

INVESTIGATION OF URTICARIA

Acute urticaria: Usually none, except where suggested by the history Specific IgE (CAP fluaraimmunassay or skin-prick tint) Tests for upper respiratory viral or bacterial infection Epsodic urticaria: Pseudoalergy challenge capsules (if avalable) Food additives Non-steroidal ant-inflammatory drugs

Chronic urticana

Physical challenge provocation tests (where suggested by the history) Blood tests (ordinary urticaria, if unresponsive to H1-antihistamines) PBC, ESR, thyroid antibodies, thyroid function tests
C4 complement (angio-odema without weals and urtcarial vascultis) Autologous serum skin test (where facilities are available) Basophil histamine release assay (If available) Others: as determined by the history and physical examination Skin biopsy (12-16 h old lesions. if urticarial vasculitis suspected) Stool examination far parasites (if infection suspected) Testing for *Helicobacter pylori infection (if* peptic ulcer symptoms) Testing for coeliac disease (primarily symptomatic children) Imaging; none routinely

2) **CLINICAL TESTs**:[**Table-II**] The following clinical test were done in all cases which were called as provocation testing for physical and cholinergic urticarias:

Provocation testing for physical and cholinergic urticarias

Patient Information

Name:

Date of Birth:

Instructions:

Perform testing as indicated and document presence (+) or absence (-) of weal (W), erythema (E), prutitus (P) and/or angioedema (A) as well as date/ time of testing and who performed the test.

1. Symptomatic Dermographism (Urticaria factitia)

Testsite: Upper back/ Volar forearm
Test: Moderate stroking of the skin with a blunt smooth object (e.g. closed ballpoint pen tip, wooden spatula)/ dermographometer (36 g/mm²)
Reading time: 10 minutes after testing

W	P

Date./Time _____ Test done by _____
It weal and pruritus: Test threshold with dermographometer

2. Cold contact urticaria

Testsite: Volar forearm/abdomen
Test: Melting ice cube in thin plastic bag/Temp Test (4°C) for 5 minutes
Reading time: 10 minutes after testing

W	P

Date./Time _____ Test done by _____
It weal: Test cold stimulation time or temperature threshold

3. Heat contact urticaria

Testsite: Volar forearm
Test: Heat source/Temp Test (4°C) for 5 minutes
Reading time: 10 minutes after testing

W	P

Date./Time _____ Test done by _____
It weal: Test cold stimulation time or temperature threshold

4. Delayed pressure urticaria

Testsite: Shoulder/Upper Back/Thighs/Volar forearm
Test: Suspension of weights over should (7 kg, shoulder strap width: 3 cm) for 15 min or weighted rods (1.5 cm diameter: 2.5 kg); or 6.5 cm diameter: 5 kg) for 15 min. Dermographometer at 100 g/mm² for 70 sec
Reading time: = 6 hours after testing

A	E

Date./Time _____ Test done by _____
It angioedema: Test threshold

5. Solar urticaria

Testsite: Buttocks
Test: UVA 6 J/cm² & UVB 60mJ/ cm² irradition (e.g. Saalmann Multitester SBC LT 400) Visible light (projector)
Reading time: 10 minutes after testing

	W	P
UVA		
UVB		
Visible light		

Date./Time _____ Test done by _____
It weal: Test threshold

6. Vibratory urticaria/ angioedema

Testsite: Volar forearm
Test: Vortex vibrator for 10 minutes, 1000 rpm
Reading time: 10 minutes after testing

A	P

Date./Time _____ Test done by _____

7. Cholinergic urticaria

Test 1: Exercise using a machine, e.g. bicycle trainer or treadmill, to the point of sweating, then continue for 15 minutes

if positive test reaction:

Test 2: 42°C bath, monitor body temperature. Continue bath for 15 min. after body temperature has increased by ≥1°C over baseline

Reading times: Immediately and 10 minutes after end of test

	W	P
1. Exercise		

If positive reaction

	W	P
2. Hot bath		

TABLE NO: 1

Age and Sex Distribution			
Age (in years)	Male	Female	Total
0-10	4	3	7
1-20	6	7	13
21-30	7	6	13
31-40	6	4	10
41-50	4	1	5
51-60	1	0	1
61-70	1	0	1
	29	21	50

Table no-2	
Duration of urticaria	
Duration of Urticaria	No of cases
Up to 8 weeks (acute)	23
More than 8 weeks (Chronic)	27

Table No-3	
Duration of individual wheals	
Duration in hours	**No of cases**
Less than1	**5**
1-2	**16**
2-4	**17**
4-8	**6**
8-12	**4**
12-24	**2**

Table NO:4	
PRECIPITATING FACTORS	
Pricipitating factors	**No. of cases**
Drugs	**11**
Friction/Scrathing	**3**
Heat	**1**
cold	**1**
Exertion	**2**
Psychogenic factors	**2**
Foods	**3**
Liquor	**1**
Infections	**2**
Mensturation	**1**
No precipitated factors	**23**

Table-5	
Constitutonal symptoms	
Symptoms	**No of cases**
Fever	**7**
Wheezing	**1**

TABLE-6 ASSOCIATED DISEASES

Associated Diseases	No of Cases
Tinea cruris	**2**
Pityriasis versicolor	**1**
Pyoderma	**2**
Miliria	**1**
Non-gonococcal urethritis	**1**
Chronic cervicitis	**1**
Upper respiratory tract infection	**5**
Chronic tonsillitis	**1**
Bronchitis	**1**
Pulmonary tuberculosis	**1**
Caries teeth	**4**
Apical abcess	**1**
Hook worm infestations	**1**
Intestinal ameobiosis	**1**
Giardia	**2**

Table-7 FOCUS OF INFECTION		
Focus of infection	**No. of cases**	**No of cases cured of Urticaria**
Caries Tooth	**3**	**1**
Apical abscess	**1**	**-**
Chronic tonsillitis	**1**	**1**
Bronchitis	**1**	**1**
Pyoderma	**2**	**1**
Non-gonococcal urethritis	**1**	**-**
Chronic cervicitis	**1**	**-**

Table-8 DRUGS	
DRUGS	**No. of Cases**
Analgin	**4**
Aspirin	**2**
Ibuprofen	**1**
Procaine pencilin	**2**
Ampicillin	**1**
Isoniazid	**1**

II. Results & Discussion

A total of 50 cases of urticaria who attended the outpatient department of dermato-Venereology at S.V.Medical College/S.V.R.R.G.G.Hospital,TIRUPATI,were examined as per criteria that fulfil the cases of acte & chronic urticaria.The bellow following studies were compared.

1. Age and Sex [Table:1]

The age of the patients varied from 1 ½ years to 62 years. The pattern of age in the present study indicates that Urticaria can be seen in any age group. Maximum number of cases (72%) were found in the age group of 11-40 years. This is in agreement with the finding of Sarojini et al.[3] who found 82% of cases in the Same age group. Male predominance is seen in the present study (58%):which is similar to the observation by Sarojin et al.[3] But Juhlin[8] found a female predominance 0f 63% in a study of 330 cases.

Another study by P. Gaig, M. Olona et all[7] epidemiology of urticaria in Spain higher incidence in women than in men at a proportion of 4:1. We cannot explain this sex difference This sex difference also holds for acute urticaria, and it continues through all the age groups. One possible explanation could be the 35-40% autoimmune origin of chronic idiopathic urticaria since women have a higher prevalence of autoimmune diseases 2) Duration of urticaria:[Table:2]

In the present study, acute urticaria was seen in 46% and chronic urticaria was observed 54% of cases

Precipitting factors : [Table:4] like drugs, friction, heat, cold, exertion, psychogenic factors, foods, liquor, infections, mensturation seen in 27 cases i.e 54%, no precipitating factors seen in 23 cases i.e 46% Two patients in the present study related urticaria to psychogenic factors. In these patients, physical examination revealed caries tooth in one case and chronic tonsillitis in the other case. After dental extraction in case of caries tooth and antibiotic therapy in chronic tonsillitis, the urticarial lesions were cleared. Hence, we may assume that stress or psychogenic factors are not causally related to urticaria in these patients.[Table:6,7]

3. Constitutional symptoms: [Table:6,7]

patients who had fever were suffering from upper respiratory infection (5 cases), apical abscess (1 case) and pulmonary tuberculosis (1 case). In our cases, we did not observe causal relationship between fever and urticaria. In all the above cases, drugs were responsible for urticaria**.** In a recent report wherein fever was seen in combination with urticaria, angioedema and eosinophilia, the aetiology was obscure (Gleich et al.,).[9]

4. Personal or family history of atopy

History of atopy was found in 10% of the cases in the present study. According to Carr-et al [10] , the incidence of atopy in general population was about 20 percent**.** From these observations, we may assume there is no significant increase of atopy in urticaria patients, a finding which was previously observed by Sarojini et al.

5) Associated diseases:[Table-6,7]

Of all the associated diseases that we have observed, dermatophytosis and pityriasis versicolor appears to be co-incidental. A similar observation was made earlier (Monroe**).**[11]

Certain systemic diseases such as systemic lupus erythematosus, rheumatic fever and malignancy may be seen uncommonly in association with urticaria (Braverman). [12] But, we have not come across such systemic disorders.

6) Physical urticarias

In the present study, dermographism was elicited in 3 cases (6%) of urticaria. Ebken et al.[16]found dermographism in 7.4% cases of urticaria compared to 2.7% in controls. The increased incidence of dermographism can be explained by the presence of increased levels of histamine in the skin of urticaria patients (Phanuphak).[17] Sarojini et al. observed the same incidence of dermographism in patients with urticaria and in controls. Cold urticaria is a rare form of physical urticaria. One case with cold urticaria not showing positive response to cold test is in agreement with Velou[18] that some patients with cold urticaria respond to general body cooling or excessive

cooling with ice (super-cooling). With the typical history (urticaria following cold water bath), the case was diagnosed to be cold urticaria. Two cases of cholinergic urticaria was diagnosed. Both of them developed urticarial wheals following exercise.

Cholinergic urticaria has been variably reported in 1.3% to 18% of patients. In a study wherein only the patients with physical urticaria were evaluated, cholinergic urticaria was observed in 10.8% (Mohan Singh).[19]

7). Focal infections: [Table-6,7]

In a total of 10 cases where evidence of infective foci were present, 4 cases responded to specific anti-infective treatment by clearing of urticaria and there was no recurrence of urticaria over a follow-up period from 2 to 4 months. In these four cases of urticaria, infective foci was considered as the probable cause of urticaria. The infective conditions included: caries tooth in one case (responded to tooth extraction), chronic tonsillitis in one case, bronchitis in one case pyoderma in one case

Considering the response to antibiotics as the criteria for the presence of infection to be the cause of urticaria, Pasricha and Kanwar[14] reported 28 cases (5.7%). Although dental caries is incriminated in the causation of urticaria, it appears that it may riot play a significant role (Surrinder Kaur).[15]

8) Parasitic infestations:[Table-6,7]

The present study blames intestinal parasites as the probable cause of urticaria in 2 cases (4%) because, deworming had curative effect on urticaria only in 2 out of 6 cases of infestation. The incidence of urticaria caused by intestinal parasites in the study by Pasricha and Kanwar[14] was 1.4%. The increased incidence in our study may be due to regional variation, variation in the educational status and hygiene of the people.

9) Drugs: [Table-8]

Eleven cases gave definite history of acute urticaria following drug intake (analgin 4 cases, aspirin 2 cases, ibuprofen 1 case, procaine penicillin 2 cases, ampicillin 1 case and isoniazid 1 case). Provocation with the drug blamed of producing urticaria was done in 5 cases, and all of the 5 cases gave positive results (analgin 2 cases, aspirin 1 case, ibuprofen 1 case and isoniazid 1 case). In the other 6 cases, urticaria disappeared after stopping the offending drug.

The incidence of drugs producing urticaria in the present study is much high compared to the findings of Pasricha and Kanwar [14] - 3.4%. However, this is comparable with the results of Sarojini et al. - 20%.

Any drug can cause urticaria. In our study, analgin is the commonest offender. In a recent study by Boonk and von Ketel[13] penicillin was the most common cause of drug-induced urticaria.

Withdrawal of drug generally stops the occurrence of urticarial eruption. But, with certain drugs such as salicylates, penicillin and ampicillin, the wheals continue to appear for 3 to 4 weeks even if the drug is not re-administered (Pasricha et al).

10). Diet: [Table-4]

Diet elimination and provocation test was done in 3 cases of urticaria. In one case, diet elimination showed improvement of the urticaria and the other 2 cases did not show any improvement.

III. Conclusion

CU is a complex disorder that has a substantial economic burden and a significant impact on patients' QoL . A complete history and physical examination will ensure the accurate diagnosis of CU and will determine the extent of laboratory studies needed for each individual patient. Studies related to acute& chronic urtiaria are very few only so far in literature i searched.

Abbreviations used
CIU: Chronic idiopathic urticaria
FcεRI: High-affinity receptor for IgE
UV: Urticarial vasculitis

References

[1]. Rook's Textbook of dermatology.8th .edition.2019.urticaria&mastocytosis.22.1-22.30.
[2]. L.A.Beck et al:A review of international recommendations for the diagnosis and management of acute and chronic urticaria:Acta derm Venereol 2017;97: 149-158.
[3]. Sarojijni PA, Gopinadhan T:Studien on 100 cases of urticaria and its etiology I.J.D.V.L 38: 137,1972.
[4]. Mohan singh,surrender kaur and kanvar AJ.Evolution of the causes of physical urticaria.Ind.J Dermatol venereal leprosy 56: 109-111, 1990
[5]. Monroe EW. Urticaria.Int J Dermatolo 20: 32-41,1981.
[6]. Pasricha JS,Gupta R. Urticaria & urinary tract infections: A case report.Indian J Dermatol venereal leprol : 277, 47:1981.
[7]. Moolani Y, Lynde C and Sussman G. Advances in Understanding and Managing Chronic Urticaria *F1000Research* 2016, **5**(F1000 Faculty Rev):177 (doi 16 Feb 2016,

[8]. P.Gaig,M.Olona,-Epidemiology of urticaria in Spain -*Invest Allergol Clin Immunol* 2004; Vol. 14(3): 214-220.
[9]. Juhlin.:clinical investigations of Recurrent urticaria patients.Br. J Dermatol 104:369,1981.
[10]. Gleich GJ,et all:Episodic angioedema associated with eosinophilia: New England journal of Medicine 310:1621-1626, 1999.
[11]. Carr SH et all:Incidence of atopy in general population:Arch Derma 89;27,1964.
[12]. Monroe EW:Urticaria:Int J Dermatol 20;32-41.,1981.
[13]. Brevarman IM; Urticaria sign of internal disease,post graduate medicine 41;450-454.
[14]. Boonk WJ et al.The role of pencillin in the pathogenesis of chronic urticaria;Br J Dermatology 106: 183-190;1982
[15]. Pasrich JS, Kanwar Aj;survey of the cause of urticaria;I.J.D.V.L 45;6-12.
[16]. Surrinder kaur et al; Incidence of dental caries in chronic urticaria:I.J.D.V.L57;276-278.
[17]. Ebken RK et al;Dermographisam:.Its definition,demonstration, and prevalence.J Allergy 41:338-343.
[18]. Phanuphak et al: vasculitis in chronic urticaria: J allergy clin immunol 65:436-444,1980
[19]. Velou A et al:cold urticaria in the tropics: J IND MED Association 43: 401,
[20]. Mohan Singh et al:Evolution of the causes of physical urticaria.:I.J.D.V.L 56: 109-111.

Dr. Jonnalagadda balachandrudu. "Clinico- Etiological Study of Urticaria in a Tertiary Care Hospital" IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 12, 2018, pp 76-75.