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Review Article

Urticaria: Etiology, pathogenesis, diagnosis, and treatment

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ABSTRACT

Urticaria is the most common skin disease, it may be chronic or acute. Infections, medicines, psychogenic factors, food, and respiratory allergens such factors are accused of etiology. But sometimes it may be idiopathic. H1 antihistamines and short-term systemic corticosteroids are taken for the treatment. H2 antagonists may be preferred during resistant cases. Cyclosporine, omalizumab, and leukotriene receptor antagonists are other treatment options considered during missed events.

1. Introduction

Skin diseases are a typical form of infections occurring in people. Skin infections produced in all ages. There are so many type of skin diseases. skin infection is described by inflammation, redness, itching, pain. Urticaria is one of the skin disorder in which there is erythematous swelling in skin occurs.[1,2] This lesion is due to vasodilation of capillaries, small venules, and exudation of fluid into superficial dermis.[1] Urticaria is characterized by the sudden appearance of pruritic wheals.[3,4]

Urticaria mainly divided into two types.[5]

1. *Acute urticaria:* (symptoms present less than six week)
2. *Chronic urticaria:* (symptoms present longer time, more than six week)

Allergic reaction of food & drug or contact with chemicals, physical stimuli infection due to which urticaria occurs.[5]urticaria is an inflammatory disease described by itchy, redness of skin, edema also produced, increase in blood flow causes warmth.[5,6] The mast cell is the major affected cell in urticaria.[3,6,7] Antihistamines are most preferably for patient with urticaria.[1,3,4,6] H_1-antihistamins are first line drug therapy. Short term corticosteroid therapy may reduce urticaria, leukotriene receptor antagonist are also used in treatment of urticaria.[1,3,8] Urticaria is a skin disorder that pathologically producese itchy wheals, surrounded by a red halo or flare. Urticaria is also defined by swelling of skin i.e. edema. Angioedema is deep and localized edema which show with superficial wheals.[4,6,9]

Swelling usually in the face, lips, eyelids and genitalia see angioedema. Sometime the tongue and laryngopharynx are also affected, which is dangerous because it can block passage of air into lungs[3] Wheals consist of three different features, center swelling of variable size, almost invariably surrounded by a reflex erythema, associated itching or sometimes burning sensation and fleeting nature, along with the skin returning to its normal appearance within 1-24 hrs.

Angioedema is characterized by Sudden pronounced swelling of lower dermis and subcutis, sometimes pain rather than itching, frequent involvement of mucous

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membrane and resolution that is slower than wheals and can up to 72 hrs.[10]

2. Etiology

Many etiological factor related with onset of chronic urticaria but most cases are idiopathic.[4] Rate of identification of causes varies from 20-50% in patient with chronic urticaria.[3,4] most identified causes are inducible urticarial which cholinergic, symptomatic dermographism, cold, and pressure urticaria are most common forms.[4] In 80-95% cases of patient causes of chronic urticaria is unknown.[1,6]

Pathogenic condition should be considered for chronic urticaria cases as follows,

2.1. Infections

Urticaria can sometimes be brought on by infection. More than half of patients of acute urticaria had infections.[4] Infections caused by viruses include infectious hepatitis, infectious mononucleosis, and a number of others.[1] In comparison to bacterial infection, the hepatitis B virus accounts for 20–30% of cases. Anti-staphylolysis titer had increased in 15% of urticaria patients. In some cases of urticaria, chronic infections (such as those of the tonsils, teeth, sinuses, and gallbladder) are discovered. Viral infection in the upper respiratory tract is the cause of acute urticaria, which typically appears a few days before wheals occur.[6] Although they have been linked, fungi and parasites such ascaris, ancylostoma, strongyloides, filaria, echinococcus, schistosoma, trichinella, and fasciola rarely produce urticaria, even in infected areas.[1]

2.2. Food and food additives

Some food products like shellfish, nuts and fruits in adult and eggs, fish, eggs, peanuts,milk (cow milk) and peanuts in childrenmay also responsible for causing reaction (IgE mediated).[1,3,6] Food additives are probably not IgE mediated and they include tartrazine, sulfites,benzoates and, perhaps, natural salicylates. Inhalant and contactant allergens may be IgE mediated. urticaria through IgE pathway may be induced by usual inhalant allergens.[1,6]

2.3. Drug's

Different types of drugs responsible for inducing urticaria. antibiotics such as penicillin, cephalosporin, macrolide's, vanomycin are responsible for urticaria with or without angioedema.[1,4] Sulfonamides and some group including sulfonylurea oral hypoglycemia agents, procaine-type local anesthetics, thiazide diuretics, furosemide, carbonic anhydrase inhibitors have been known to cause urticaria.[1] Aspirin and other non-steroidal anti-inflammatory drugs (NSAID) may play an important role in precipitating urticaria.[1,3,6] those Person who is sensitive to aspirin have an increased urticarial reaction rate.[6]

2.4. Insect's

Urticaria can cause due to insect bites and sting. It may be hymenopteasting from wasps, hornets bees, yellow jackets and fire ants.[1,3] Urticaria result from flea bites, swimmers and possibly mosquito bites and may confused with hives.[1]

2.5. Autoimmune disease

Autoimmune disease including systemic lupus erythematous, serum sickness, cutaneous Vasculitis rheumatiods arthritis, which may have urticaria like symptoms.[1,3] Malignancies rarely induce urticaria. Endocrine disease may be hyper and hypothyroidism or hyperparathyroidism can result in urticaria.[1]

2.6. Physical urticarial

Physical urticaria includes idiopathic cold urticaria with development of hives at local site cold contact.[3] Physical stimuli include cold, heat, scratchy, pressure, vibration.[1] Cold urticaria associated with cryglobulins, cold agglutinins, and cryfibrinogen. Cholinergic urticaria involves small punctate wheals with large flares induced by exercise. Hotshowers, sweating anxiety it may be associated with fall in lung fun.[1] Dermatographism is most common form of physical urticaria.[3,6] it is precipitated by gentle stroking of the skin. Which result in a large wheals and flare within 5 min heat urticaria is vary rate disorder characterized by wheal formation a circum scribedstimulis within temp range 45^0c to 50^0c.[6]

3. Pathogenesis

The mast cell known as major effector cell in most forms of urticaria, though other cell types may be involved. First step for formation of wheals is the degranulation of mast cells with release of histamine. Appearance of urticaria shows local increase in permeability of venles, and capillaries. activation of the cutaneous mast cells due to vascular changes, which contain a range of mediators mainly histamine. When introduced in skin they causes wheals-and-flare similar to urticarial lesions. Interaction of both H_1 and H_2 histamine receptors produces vascular permeability in skin. Due to activation of H_1 receptors in the skin induce flare, erythema, whealing, itching, contraction of smooth muscle in the respiratory and gastro-intestinal tract. Activation of H_2 receptors lead to erythema and whealing in the skin. The postulated effector mechanisms for immunological and non-immunological activation of mast cells is presented in [Figure 1].[3,10,11]

Immunological mechanism mediated by IgE antibody. IgE antibody binds to the specific receptor present on mast

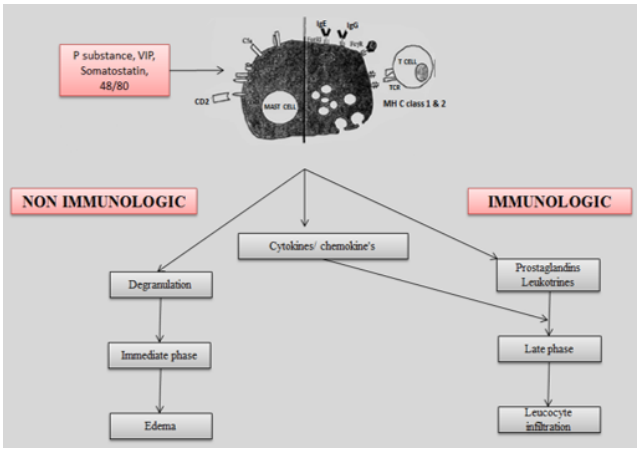

Fig. 1: Pathogenesis of urticarial [12]

cell. These causes histamine release from mast cell. Due to histamine release swelling and itch take place.

Urticaria caused by non-immune mechanism is found in physicalurticaria such as dermographism, delayed pressure urticaria, coldurticaria, solar urticaria, aquagenic urticaria and vibratory urticaria. Along with Mast cells, basophils also act as effector cells in this mechanism. These cells release vasoactive substances like histamine, bradykinin, leukotriene C4, prostaglandin D2, release of these substances and vasodilatation takes place and therefore increases vascular permeability. This produces erythema and edema dominant to urticaria lesions. Itching, an important feature of urticaria is mediated by histamine receptors as erythema and flushing. [1]

4. Mast Cell Mediators of Urticaria

4.1. Histamine

Binding of histamine H1 receptors on small cutaneous blood vessels mediates vasopermeability and vasodilatation. It also mediates itch through stimulation of cutaneous nociceptors and the surrounding flare by antidromic stimulation of local C-fiber networks. The flare response is interfere by substance P release from cutaneous nerve endings rather than histamine. Stimulation of H2 receptors on cutaneous blood vessels is also responsiblefor vasodilatation and vasopermeability within the weal but not itch or flare. Effects of histamine on the cellular immune system have been demonstrated, but their relevanceto urticaria is uncertain. [6,13]

4.2. Cysteinyl leukotrienes

The cysteinyl leukotrienes have secondary importance in urticaria. It may contribute to vasopermeability and vasodilatation in urticaria. Synthesis of LTC4, D4, E4 by mast cells at the time of degranulation and subsequently by infiltrating basophils and eosinophils may be a factor in the prolongation of urticarial weals in some types of urticaria, particularly aspirin sensitive urticaria, autoimmune urticaria and delayed pressure urticaria. It is thought that aspirin and other non-selective NSAIDs may activate mast cells indirectly by inhibiting formation of prostaglandin E2 (PGE2) via cyclo-oxygenase (COX). [13]

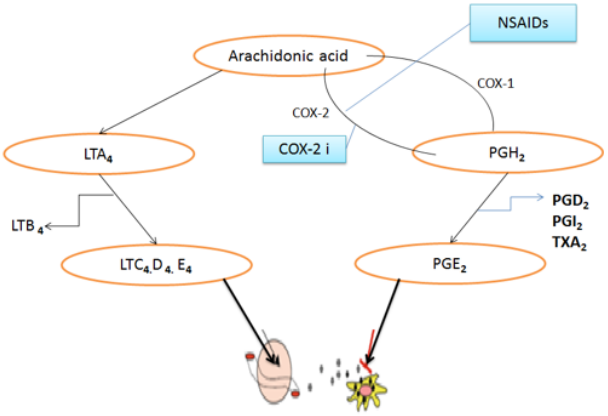

Fig. 2: Inhibition of the cyclo-oxygenase (COX) pathway [13]

5. Other Inflammatory Cells

5.1. Eosinophils

Eosinophils contain toxic granules including major basic protein (MBP) and eosinophil cationic protein (ECP) that are released on activation. MPB can degranulate mast cells non-immunologically. [13]

5.2. Basophils

Basophils are thought to migrate into weal of chronic urticaria and probably perpetuate the inflammatory edema by releasing histamine and leukotrienes. [13,14]

5.3. Neutrophils

There is no specific role for polymorphonuclear neutrophils have been identified but it is possible that they are involved with oxygen-free radical formation. [13,14]

6. Diagnosis

Urticaria and angioedema are diagnosed based on a thorough medical history, physical examination, and routine patient assessment. The most frequent causes are infections' signs and symptoms, atopic disease, drug use, and allergies. It's important to consider any family history of thyroid disease, autoimmune disorders, atopy (allergic rhinitis, asthma, and aspirin allergy), and angioedema. Knowing when symptoms first appear, as well as whether or not

Table 1: Recommended diagnostic tests in frequent urticaria subtypes [10]

Group	Subgroup	Routine diagnostic tests	Extended diagnostic tests *
Spontaneous urticaria	Acute urticarial, Chronic urticarial	None+ Differential blood count & ESR/CRP# Omission of suspected drugs (e.g. NSAID)	None Test for a) Infectious diseases (e.g. *Helicobactor pylon*) b) Type 1 allergies c) Autoantibodies d) Thyroid hormones e) Physical tests f) Psudoallergen-free diet for 3 weeks & tryptase, biopsy
Physical urticaria	Cold contact urticarial	Cold provocation & threshold test (ice cube, cold water, cold wind)	Differential blood count & ESR/CRP# Cryoproteins rule out other diseases, especially infectious
	Delayed pressure urticarial	Pressure test (0.2-1.5 kg/cm^{2}for 10 min & 20 min)	None
	Heat contact urticaria	Heat provocation test & threshold test (warm water)	None
	Solar urticaria	UV & visible light of different wave-lengths	Rule out other light-induced dermatoses
	Dermographic urticarial/ urticaria factitia	Elicit dermographism	Differential blood count, ESR/CRP
	Aquagenic urticaria	Wet cloths at body temperature applied for 20 min	None
Other urticaria disorders	Cholinergic urticaria	Exercise & hot bath provocation	None
	Contact urticaria	Prick/ patch test read after 20 min	None
	Exercise-induced anaphylaxis/ urticaria	According to history exercise test with/ without food	None

ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; NSAID: nonsteroidal anti-inflammatory drugs. * Depending on suspected case # Unless strongly suggested by patient history, e.g. allergy. + As indication of severe systemic disease.

itchiness, pain, or heat are present, is crucial. Physical examination is yet another critical stage in the diagnosing process. Correct diagnosis is based on the urticaria lesions' distinctive appearance and development. Patients with suspected food allergies or inhalant allergies may find skin tests and IgE measurements helpful. In many instances of chronic urticaria with angioedema, the main goal of the examination is to rule out underlying illnesses. [3,4,10]

7. Treatment

Depending on how severe the symptoms are, urticaria can either be treated with or without angioedema. [3,6] The main and consistent objective of CU treatment is to obtain total symptom relief (treat the disease until it is gone). Elimination and prevention of pertinent triggers and symptomatic medication therapy make up the therapeutic approach. Complete symptom alleviation is the main objective of urticaria treatment. Identifying and eliminating urticaria causes comes first in management, followed by symptom relief. In patients with IgE-mediated urticaria and physical urticaria, avoidance of triggers and stimuli might be started. [3]

Fig. 3: Treatment for urticarial. [2]

7.1. First generation H_1-antihistamines

These are used as first line therapy for urticaria .they are more effective. Many symptoms of urticaria are mediated

by H1-receptors located on nerves and endothelium. H1-receptor antagonistsare most effective in treating urticaria although not all patients respond.[3,9] First generation antihistamines are used because of anticholinergic effectsand sedative actions are not recommended for the management of various kinds of urticaria.[3] First-generation antihistamines have similar efficacy, but greater sedation and impairment compared withsecond-generation antihistamines.[3,8,9] The US practice parameters recommend the use of first-generation antihistamines at bedtime for reduce daytime impairment; and they have been often lead to daytime somnolence, sedation, drowsiness, fatigue and impaired concentration and memory. H2-antihistamines, specifically cimetidine, used in combination with H1-antihistamines have shown a limited additive effect.[9] Diphenhydramine, Chlorpheniramine, Hydroxyzine are used as first generation H_1-antihistamines.[3,8,15]

7.2. Second-generation H_2-antihistamines

Second generation antihistamines shows dose-dependent effect and important aspect is that they do not produce side effects at high doses. Two other important features of second generation antihistamines are rapid onset and long duration of action. Patients with chronic urticariause second generation antihistamines in a single daily dose preferably given at bedtime. Patients prescribed with second-generation antihistamines has been shown to reduce the intensity of itching and wheals. The second-generation anti-histamines cetirizine, loratadine, and fexofenadine are much longeractingand cause less sedation than the first-generationagents. Cetirizine is the most likely to cause sedation butis usually well tolerated.[3,6,8,16]

7.3. H2 antagonists

H2 antagonists are ineffective in hives when used alone. It has been demonstrated that H1 antagonists in combination with H2 antagonists, such as ranitidine and cimetidine, are more efficacious than an H1 antagonist alone. These medications are employed to reduce gastric acid production.[3,6]

7.4. Corticosteroids

Short-term use and the lowest effective dose of oral corticosteroids are both possible. Due to the potentially serious side effects of long-term therapy (diabetes, hypertension, osteoporosis, and gastrointestinal bleeding), The EAACI/GA2LEN/EDF/WAO management guideline advises against using corticosteroids except in the most extreme circumstances. Urticaria may be lessened with short-term corticosteroid therapy. It can be used to treat acute urticaria that doesn't respond to antihistamines as well as flare-ups of chronic spontaneous urticaria. In addition to easing symptoms like edoema and inflammation, these medications also lessen the risk of relapse.[3,6,8,9]

8. Leukotriene Receptor Antagonists

The cysteinyl-leukotriene antagonists as montelukast and zafirlukast are frequently used in CU. It can be used in combination with H1 antihistamines and were particularly effective in CU patients positive for autologous serum skin test (ASST) and with intolerance to aspirin or to food additives. However, not many studies have confirmed the efficacy of leukotriene antagonists when used as single. Existing evidence for leukotriene receptor antagonist's effectiveness is limited, and the grade of recommendation for their use is less. Nonetheless, these types of drugs may be tried inpatients unresponsive to antihistamines.[6,8,9,16]

8.1. Immunosuppressive drugs

Several immunosuppressive drugs have been used to treat severe, antihistamine resistant CU, although most experience relies on uncontrolled studies. Methotrexate 15mgin weekly mean dosage of seems effective and safe in most CU patients who are not responsive to conventional therapy. Sometimes reports have shown the efficacy of intravenous and oral cyclophosphamide and azathioprine in antihistamine resistant CU with positive autologous serum skin test. Recently, mycophenolatemofetil, a purine biosynthesis inhibitor, has emerged as a possible therapeutic option for CU patients who do not respond to antihistamines and/or corticosteroids.[6,8,9]

8.2. Anti-inflammatory drugs

The anti-inflammatory drugs includes dapsone, hydroxychloroquine and sulfasalazine. These drugs in the treatment of CU is often limited, but they are safe, cheap, and they could be tried before considering more expensive toxic agents.[6,8,9,16]

8.3. Dapsone

Dapsone has been shown to be useful in treating various forms of CU or angioedema, including spontaneous CU, when taken daily in doses between 25 and 50 mg. Dapsone is generally well tolerated, but it can also cause DRESS syndrome (drug reaction with eosinophilia and systemic symptoms), hepatotoxicity, gastrointestinal problems, skin rashes, anaemia, and methemoglobinemia. In patients who lack G6PD, it could cause severe hemolysis. Before beginning dapsone therapy, the G6PD phenotype should be assessed in each patient, and the administration of this medication necessitates meticulous patient monitoring.[6,8,9]

8.4. Sulfasalazine

The potency of sulfasalazine in CU patients is supported by some case reports and by a retrospective observational

study. Effective dose is up to 2 g per day and response occurs within 1 month of therapy. Side effects include dyspepsia, anorexia, headache, nausea, vomiting, and less frequently, hematologic abnormalities, hepatotoxicity and proteinuria.[6,8,9]

8.5. Hydroxychloroquine

Hydroxychloroquine notably improved the quality of life of CU patients in a randomized, blinded, placebo-controlled study. Although urticaria activity scores were hardly influenced. The most pertinent side effectvery rare, is retinopathy which is associated with a more than 5-year use of the drug.[8,9]

9. Biologicals

9.1. Omalizumab

Omalizumab is a recombinant, humanised monoclonal IgG antibody that inhibits mast cell activity by binding free IgE. The free IgE is bound by omalizumab, which lowers free IgE levels and, as a result, decreases the expression of FcRI receptors on mast cells, basophils, and dendritic cells. Mast cell survival and proliferation are thought to be dependent on IgE-FcRI-dependent pathways, hence this action may lead to a reduction in the number of mast cells. The anti-IgE drug omalizumab prevents mast cell activation by separating preattached IgE from its receptor. Omalizumab was first authorised for the treatment of moderate to severe persistent allergic asthma, but it soon discovered a market with CU. The signs and symptoms of CU were lessened with omalizumab, but over the course of around 10 weeks after the medicine was stopped, the symptoms persisted.[6,8,9,12]

9.2. Anticoagulants

CU patients generally show a definiterise of plasmatic markers of thrombin generation. fibrinolysis during severe exacerbations of the diseasemay be a consequence of tissue factor term by activated eosinophils. The activation of coagulation and fibrinolysis decreases previously full normalization during remission. This phenomenon (activation of coagulation/fibrinolysis) plays a major role in the disease pathophysiology. Simply it act as an amplification system is still to be defined. The fact that such activation parallels the activity of CU may provide the motive for the evaluation of anticoagulant and antifibrinolytic therapy in patients. The potency of anticoagulant therapy in some cases with refractory CU has been observed as long as aDecade ago by the use of both oral anticoagulants and heparin. Anticoagulant therapy cannot yet be recommended as a routine treatment for CU.[6,8]

9.3. Other treatment modalities

Calamine lotion, cooling powder and a refreshing napkin may be applied to the area affected by hives to relieve symptomatic itching.[17]

9.4. Treatment of urticaria in children

The long-term efficacy profiles of new generation H1 antihistamines make them the first recommendation for the treatment of urticaria. The use of first-generation H1 antihistamines is not advised due to their potent sedative effects and reduced psychomotor development in youngsters. In circumstances where typical doses of an antihistaminic are ineffective, the dose can be increased by up to two times while still being mindful of the child's body weight. The use of LTRAs, cyclosporine, and omalizumab in the management of pediatric urticaria is not supported by adequate information. Several medications may be added to antihistamines in the third step of treatment according on how adult urticaria is treated. There is growing evidence that omalizumab is reliable and safe in treating children older than 7 years old. The literature has described pediatric patients who very well tolerated monthly dosages of 150–300 mg. Cyclosporine has been discovered to be quite effective in treating youngsters who do not respond to antihistaminic medication. For just a maximum period of ten days, systemic corticosteroids may be administered to pediatric children who are suffering from angioedema episodes or chronic widespread urticaria.[18]

9.5. Treatment of urticaria during pregnancy and lactation

According to the most recent treatment recommendations, the traditional therapy strategy suggested for the treatment of urticaria during pregnancy can be used. Pregnancy category B is recommended for antihistamines such as chlorpheniramine, loratadine, cetirizine, and levocetirizine, while category C is recommended for all other antihistamines. First-generation H1 antihistamines with sedative effects shouldn't be used right away because doing so results in respiratory arrest in the baby. All current recommendations stress that new generation antihistamines are more effective at treating urticaria in people who are pregnant. LTRAs and cyclosporine are assigned pregnancy classifications B and C, correspondingly. Omalizumab hasn't been used in pregnancy before. 169 pregnancies in asthmatic patients were reported while taking omalizumab, however, there was no significant increase in serious abnormalities. Omalizumab was placed in pregnancy category B by the FDA. Since they are present in very small quantities in breast milk, loratadine and cetirizine may be favored even during lactating phase.[18]

10. Conclusion

Urticaria is skin disorder has a profound impact on the quality of life and causes immense distress to patients. This review includes definition, causes, diagnosis, and treatment of urticaria. Approach to manage urticaria is identification and elimination of underlying causes and eliciting triggers while second is treatment aimed at providing symptomatic relief. And use of second generation non-sedative H1 antihistamines as first line treatment. In some cases other drugs such as corticosteroids, anti-inflammatory, leukotriene receptor antagonist may use.

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None.

12. Conflict of Interest

None.

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