

INTERNATIONAL JOURNAL OF UNANI AND INTEGRATIVE MEDICINE



E-ISSN: 2616-4558
P-ISSN: 2616-454X
IJUIM 2022; 6(1): 01-05
Impact Factor (RJIF): 6.3
Peer Reviewed Journal
Received: 17-10-2021
Accepted: 02-12-2021

Dr. Md. Gheyasuddin
Medical Officer (Unani),
APHC Kurisarai (under CHC
Belaganj), Gaya, Bihar, India

Dr. Khalid Eqbal
Assistant Professor,
Department of Moalajat,
Sufiya Unani Medical College
Hospital and Research Centre,
Bara Chakia East Champaran,
Bihar, India

Dr. Md. Najmuddin
Medical Officer, Patna Health
Clinic Subzibagh Patna, Bihar,
India

Corresponding Author:
Dr. Md. Gheyasuddin
Medical Officer (Unani),
APHC Kurisarai (under CHC
Belaganj), Gaya, Bihar, India

Understanding urticaria, and their management: An appraisal

Dr. Md. Gheyasuddin, Dr. Khalid Eqbal and Dr. Md. Najmuddin

Abstract

Urticaria, or hives, is a common skin and mucous membrane illness characterized by erythematous, edematous, irritating, and transitory plaques. It affects roughly 20% of people at some point in their lives. It can produce substantial discomfort, last for months to years, and seldom reflect a major systemic disease or life-threatening allergic reaction, despite being self-limited and benign. In the Unani system of medicine urticaria is labeled as *Shara*, *Avicenna*, defined that *Shara* is a *Damwi* (sanguineous) disease usually but it may also be due to *Safravi Khoon* (bilious blood) or *Balgham Boraqi* (acidic phlegm). Urticaria is induced by the release of histamine and other inflammatory mediators from mast cells and basophils mediated by immunoglobulin E and non-immunoglobulin E. Anaphylaxis must be ruled out before a diagnosis can be made. In 80 percent to 90 percent of instances, chronic urticaria is idiopathic. Treatment with H1 antihistamines and, in certain circumstances, short-term systemic corticosteroids is favored; H2 antagonists may be added in refractory cases, however additional treatment options, including omalizumab, cyclosporine, and leukotriene receptor antagonists, may be examined during missed occurrences. Unani Medicine has its own framework for treating patients based on treatment principles which include; *Ilja Bil Ghiza*, *Ilaj Bit Tadbeer*, and *Ilaj Bil Dawa*.

Keywords: Urticaria, hives, anti-histamines, *Safra*, *Shara*, unani medicine

Introduction

Hippocrates was the man who first introduced the concept of urticaria [1]. Urticaria refers to a group of disorders that are all different. All forms and subtypes of urticaria develop urticarial skin lesions and/or angioedema, which is a common and unique skin reaction [2]. Urticaria (hives) is a common ailment that is frequently accompanied by angioedema (swelling that occurs beneath the skin) [3]. Urticaria is a condition that causes wheals and/or angioedema and is divided into numerous kinds [4]. Adults are expected to have had at least one episode of acute urticaria at some point in their life, and the prevalence of chronic urticaria in adults is considered to be between 0.5 percent and 5% [5]. Previous research have estimated the prevalence of acute urticaria in children to range between 1% and 14.5 percent, however, there is no information on the prevalence of chronic urticaria in children [6]. Chronic urticaria is thought to be less common in children than in adults, and most research that estimates its incidence rely on extrapolation from adult data, hospital-based results, or general adult population surveys [7]. Around 15-20% of the world's population suffers from urticaria. Chronic spontaneous urticaria affects 8-10% of Europeans. A further 0.1-3 percent of diagnoses are chronic spontaneous urticaria with no recognized cause. Females are roughly twice as likely as males to be affected, with the peak incidence of chronic spontaneous urticaria occurring between the ages of 20 and 40 [8]. The appropriate treatment strategy is determined on the cause and whether the problem is acute or persistent [9].

Types of Urticaria

Urticaria can be categorized based on the underlying cause or by a clinical classification. In most cases of chronic urticaria, the underlying cause cannot be identified, i.e. the rash is idiopathic; however, when seeing a patient, it is helpful to use a clinical classification, as it guides history-taking and ensures that all possible triggers for urticaria are identified in any given individual. Several variables may induce or aggravate the rash in some persons [10].

Physical Urticaria: Physical urticarias are a diverse subtype of chronic urticarias in which wheals can be caused by a variety of physical stimuli such as cold, heat, pressure, vibration, or sunshine.

Physical urticarias account for up to 25% of chronic urticarias and are more common in young individuals^[11, 12]. Dermographism, cold, hot, vibration, pressure, and sun variables are some of the etiological elements that cause it to appear. It accounts for 20–30% of chronic urticaria cases^[13]. Cholinergic urticaria is a type of urticaria that occurs when the basal body temperature rises as a result of physical exercise or exposure to heat^[14]. Cold (cold-induced urticaria), UV light (solar urticaria), water (aquagenic urticaria), and exercise are among physical stressors that can cause urticaria. These physical stimulation cause lesions that are normally limited to the stimulated area and disappear within 2 hours^[15-17].

Auto-reactive urticaria

Circulating histamine-releasing signals generate symptoms in patients with autoreactive urticaria (ArU), a subtype of chronic urticaria (CU). The autologous serum skin test, in which patients demonstrate inflammatory reactions to their serum following intracutaneous injection, is a simple way to identify ArU patients. Autoimmune urticaria is thought to affect CU patients who have functional autoantibodies against IgE and/or its high-affinity receptor Fc(epsilon)RI (Aiu). Other sub-forms of CU are pathogenetically and clinically distinct from ArU and AiU^[18]. The role of IgE in immunological reactions has been re-examined in recent years as a result of fresh studies. It appears to be more than merely a contributor to a form of allergic reaction that occurs right away. Monomeric IgE appears to increase mast cell activity without causing FcRI cross-linking by IgE-specific allergen or autoreactive IgG anti-IgE antibodies^[19]. Monomeric IgE molecules differ in their ability to stimulate mast cell survival and activation only by binding IgE to FcRI, but not in their ability to impact cell degranulation. It was also shown that IgE can react to autoantigens in the blood, which can occur not only in chronic spontaneous urticaria (CSU), but also in other autoimmune illnesses^[20].

Inducible Urticaria

Chronic inducible urticaria is a common inflammatory skin disorder marked by recurrent itchy wheals and/or angioedema lasting longer than 6 weeks and triggered by particular physical or environmental stressors (cold, heat, exercise, pressure, sunlight, vibration, water, etc.). Physical urticarias (dermographism, delayed-pressure urticaria, exercise-induced urticaria, cold urticaria, heat urticaria, solar urticaria, and vibratory urticaria) and non-physical urticarias (dermographism, delayed-pressure urticaria, exercise-induced urticaria, cold urticaria, heat urticaria, solar urticaria, and vibratory urticaria) (cholinergic urticaria, contact urticaria, and aquagenic urticaria)^[21]. Wheals, pruritus, and/or angioedema are all symptoms of inducible urticaria, which is a category of skin illnesses defined by the emergence of wheals, pruritus, and/or angioedema, occasionally accompanied by systemic symptoms brought on by harmless stimuli (cold, heat, pressure, etc.). This set of illnesses has a negative impact on people's quality of life^[22]. Chronic Inducible Urticaria symptoms can be moderate or severe, localised or disseminated (and occasionally extensive), and vary from one person to the next. It also depends on the type of stimulus that causes the body's physical reaction^[23].

Cold Urticaria

Cold urticaria is a type of physical urticaria that causes

papules to appear on the skin when exposed to cold air, liquids, and/or objects. Angioedema and anaphylaxis may also occur in some circumstances. Cold urticaria symptoms can have a detrimental impact on a patient's quality of life. In cold urticaria, second-generation H1 antihistamines are the primary line of treatment; however, patients who do not respond to H1 antihistamines may require additional management alternatives. The most effective preventive measure is to avoid cold exposure. The major goal of therapy in mild to moderate cases is to improve the patient's quality of life^[24].

Cold urticaria is a type of urticaria that can occur in conjunction with other physical urticarias. The frequency is usually assumed to be two or three per 100 people. In the majority of cases, the triggering impact of cold is discovered during history taking. The urticaria is usually superficial, with deep and/or mucosal urticaria occurring rather infrequently. The diagnosis is made after a thorough examination of the patient's medical history and the results of the ice cube test^[25]. In cold urticaria, second-generation H1 antihistamines are the primary line of treatment; however, patients who do not respond to H1 antihistamines may require additional management alternatives. The most effective preventive measure is to avoid cold exposure. The major goal of therapy in mild to moderate cases is to improve the patient's quality of life. Treatment procedures to safeguard the patient's airway, breathing, and circulation may be required in more severe situations^[26].

Heat Contact Urticaria

Heat contact urticaria is an uncommon condition in which the development of wheal is limited to the areas of heat contact^[27]. Localized heat contact urticaria is a cutaneous disorder, one of the rarest kinds of urticaria, in which itching and whealing develop within minutes of contact with heat from any source, lasting up to an hour. Heat contact urticaria is an uncommon condition in which the development of wheal is limited to the areas of heat contact. If heat contact urticaria is suspected, skin testing with metal or glass cylinders filled with warm water or a warm water bath is the preferred technique of diagnosis^[28-30].

Delayed Pressure Urticaria

Delayed pressure urticaria (DPU) is a type of urticaria that causes recurring erythematous and typically painful swelling when prolonged pressure is applied to the skin. Treatment is difficult. Antihistamines, the sole FDA-approved treatment, are frequently ineffective^[31]. Delayed pressure urticaria is a type of physical urticaria in which erythematous, often painful swellings appear after many hours at areas of sustained pressure on the skin. It is present to variable degrees in up to 40% of people with conventional chronic idiopathic urticaria if it is sought. Pressure-induced urticarias have the greatest impact on patients' quality of life when compared to other urticarias. The pathophysiology is unknown, but whealing is caused by mast cell activation, and histology of lesions reveals a deep dermal inflammatory infiltration of neutrophils and eosinophils, but no vasculitis^[32]. The treatment of delayed pressure is often ineffective, and it is frequently resistant to antihistamines and a variety of anti-inflammatory drugs. Although oral steroids are the most effective treatment, they are not suited for long-term use. Delayed pressure urticaria can last for years, and new or improved treatment options are being researched^[33].

Solar Urticaria

Urticaria with solar urticaria is a rare urticaria type. It is responsible for fewer than 0.5 percent of all urticaria cases and 7% of all photo dermatoses. The condition usually starts in early adulthood (median age 35 years), but it has also been recorded in new-borns and the elderly. There is a female majority, yet there is no racial divide. A history of atopy is discovered in less than 30% of patients in the largest series of patients. Solar urticaria can be found in up to 16% of people with other kinds of chronic urticarial [34, 35]. Solar urticaria is caused by skin exposure to the sun. The spectrum of action for solar urticaria varies from patient to patient and runs from ultraviolet B to visible light (wavelengths of 300 nanometers to 500 nanometers) [36]. There are no treatment guidelines for solar urticaria. Various treatments have been tried, with varying degrees of effectiveness. Sun avoidance, as well as photoprotection using broad-spectrum sunscreens and dark clothes, are obvious recommendations [37, 38].

Vibratory Urticaria

Vibratory urticaria is a rare type of chronic inducible urticaria in which urticarial weals appear in response to a vibrational stimulus to the skin. Previously, vibratory urticaria and vibratory angioedema (angioedema caused by a vibratory stimulus) were thought to be the same thing, but they are now regarded as separate clinical diseases [39, 40]. Vibratory urticaria is an allergic reaction that causes hives (urticaria), swelling (angioedema), redness (erythema), and itching (pruritus) in the afflicted area when the skin is exposed to vibration, repetitive stretching, or friction [41]. Towel drying, hand-clapping, sprinting, a bumpy car ride, or other repetitive stimulation can trigger the reaction. Headaches, weariness, dizziness, blurred vision, a metallic taste in the mouth, facial flushing, and more widespread edoema (especially of the face) can also occur during these episodes, particularly if the stimulation is intense or protracted. The reaction takes place within minutes of stimulation and can last up to an hour. Affected people can suffer multiple bouts each day [42].

Cholinergic Urticaria

Despite the fact that cholinergic urticaria (CU) has well-defined clinical manifestations, the exact pathogenic mechanism is still unknown. There have been various pathogeneses proposed, implying that there are several clinical subtypes [43]. Cholinergic urticaria is a kind of urticaria that is produced by an increase in core body temperature as a result of exercise, spicy meals, or stress [44]. Several mechanisms have been shown to have a role in the development of cholinergic urticaria. The pathophysiology and clinical characteristics of each subtype should be used to classify this condition. A classification system like this would help better manage this resistant illness [45]. The first-line treatment is antihistamines, but they are generally ineffective. Methantheliniumbromide is an anticholinergic drug that reduces perspiration [46].

Contact Urticaria

Contact urticaria (CU) is a transient wheal and flare reaction that occurs at the site of contact with the offending agent and disappears fully within 24 hours [47]. When the stratum corneum is disrupted by filaggrin gene mutations or skin irritants, the chance of getting CU increases. The common

agents linked to the development of CU are discussed in the sections below. Healthcare, lab work, agriculture, hairdressers, cosmetic industry, chemical industry, cleaning, building, catering, cooking, electronics, manufacturing, and metal products are the jobs with the highest risk of acquiring CU [48, 49]. Due to reduced disease diagnostic rates, the prevalence in the general population is undetected. Occupational CU affects 0.4 percent of workers and accounts for 30 percent of all occupational skin disorders. Latex-related CU has a prevalence of 5% to 10%, according to research [50]. The cornerstone of management is avoiding the offending substance. Cross-reactive proteins should be explained to patients, and they should be counselled to avoid them if they experience symptoms after being exposed to them. When exposure at work is inevitable, protective equipment such as gloves, skin conditioning lotions or emollients, and cotton liners are required [51, 52]. In the ICU, second-generation H1-receptor blockers (such as diphenhydramine, hydroxyzine, loratadine, and desloratadine) are the first-line therapy. Montelukast, zafirlukast, and zileuton are leukotriene inhibitors that reduce inflammation [53].

Unani Approach of Urticaria

Urticaria is known in Unani medicine as *Shara*. According to Avicenna, *Shra* is a *Damwi* (sanguineous) condition, but it can also be caused by *Safravi Khoon* (bilious blood) or *Balgham Boraqi* (acidic phlegm) [54]. *Shara* manifests as small, flat-topped eruptions that resemble vesicles (*nifatat*), are crimson in color, and cause acute itching and pricking sensations. *Shara* most typically occurs all over the body. He also went over the specific requirements and role of *fas'd* (venesection) in *Shara* treatment (Urticaria) [53]. According to Ahmad Al-Tabri, *Shara* (Urticaria) is caused by a small amount of *Har Hareef Dam* (Hot Saline Blood) mingling with *Ratoobat-e-Fasida Ghaliza* (morbid vicious senses of humor) or by a small amount of *Ratoobat-e-Raqiq Fasida* (morbid diluted touches of humor) becoming irritated owing to Malahat (salinity) [54]. This disease is caused by a mixture of *Akhlat Saudavia* (black bile). *Shara*, which is caused by the sanguine humor, manifests as crimson, tiny papules on the skin, causing significant discomfort [55]. *Shara* is represented by white, large maculae over the skin, which contain Warm and touches of humor inside it, due to morbid humors. Urticaria is categorized into two varieties by Ali Ibn Abbas Majoosi, the author of *Kamilus-Sana*: one is caused by blood combined with *Safra* (bile), characterized by reddish rashes and Hararat (Hotness) in his body, and the other is caused by mixing of *Balgham Shor* (acidic phlegm) in *Raqiq Khoon* (diluted blood) [56, 57]. *Shara* can arise when Sanguine, Phlegm, and Bile are mixed together. *Shara* is caused by *Kaseer Haad bukharat* (vapours emerging from excessive humor) that arise from blood, according to Abul Mansoor Al Hasan Al-Qamri [58]. These vapors are formed by *Kasrat-e-Dam* (excess blood) or by mixing of *Balgham Shor* (Acidic Phlegm) in blood. The stimulation of symptoms happens throughout the daytime if the cause is an excess of blood, and it is more common in young men and children [59, 60]. *Balgham Shor* (Acidic Phlegm) in the blood is the most common cause, and the stimulation of symptoms occurs at night, with the least sense of itching. He also went into great detail about how to treat urticaria [54-60].

Principles of Treatment

In Unani system of medicine, it is based on triad therapy; diet (*Jayyadul Kaimus, Sareeul Hazm*), regimes (Hamam, Ishal, Fasd, Hijama, and pharmacotherapy (Mufradat wa Murakkabat- Munzij Wa Mushil of Balgham, Wa Safra, along with Moaddilate dam Advia)^[54].

In western medicines, treatment approach- for 2-4 weeks, non-sedating H1-antihistamines are the first-line treatment; if urticaria persists, increasing the dose up to four times is indicated. The benefits of sedating first-generation antihistamines over non-sedating antihistamines have not been proved. Anti-IgE: omalizumab is the only strong evidence-based alternative regimen for CSU; however, because to its high cost, it may not be available in low-middle income countries. Non-pharmacotherapeutic approaches to reducing hyper-responsive skin, such as preventing skin from drying out, avoiding hot showers, washing, and excessive sun exposure, are also important and suggested^[61].

Conclusion

The first-line pharmacological treatment is second-generation H1-antihistamines (e.g., cetirizine, loratadine, fexofenadine) taken on a regular basis. If symptoms persist after 2 to 4 weeks, the dose can be increased to 4 times the usual dose. Supplemental therapeutic options include first-generation H1 antihistamines, H2 antihistamines, leukotriene receptor antagonists, high-potency antihistamines, and brief corticosteroid bursts. Patients with refractory chronic urticaria may be referred to a subspecialist for further therapy such as omalizumab or cyclosporine.

Conflict of Interest: Nil

Funding: Nil

References

- Ghosh S. What's New In Urticaria? *Indian J Dermatol.* 2009 Jul-Sep; 54(3):280-282.
- Amar SM, Dreskin SC. Urticaria *Prim Care.* 2008 Mar;35(1):141-57,
- Sachdeva S, Gupta V, Amin SS, Tahseen M. Chronic Urticaria. *Indian J Dermatol.* 2011 Nov-Dec;56(6):622-628.
- Zuberbier Classification of Urticaria. *Indian J Dermatol.* 2013 May-Jun;58(3):208-210.
- Lee SJ, *et al.* Prevalence and Risk Factors of Urticaria with a Focus on Chronic Urticaria in Children. *Allergy Asthma Immunol Res.* 2017 May;9(3):212-219.
- Khakoo G, Sofianou-Katsoulis A, Perkin MR, Lack G. Clinical features and natural history of physical urticaria in children. *Pediatr Allergy Immunol.* 2008 Jun;19(4):363-6.
- Zuberbier T, Balke M, Worm M, Edenharter G, Maurer M. Epidemiology of urticaria: a representative cross-sectional population survey. *Clin Exp Dermatol.* 2010 Dec;35(8):869-73.
- Raciborski F, *et al.* Epidemiology of urticaria in Poland - nationally representative survey results. *Postepy Dermatol Alergol.* 2018 Feb;35(1):67-73.
- Kayiran MA, Akdeniz N. Diagnosis and treatment of urticaria in primary care. *North Clin Istanbul.* 2019 Feb;6(1):93-99. doi: 10.14744/nci.2018.75010.
- Greaves MW. Pathology and classification of urticaria. *Immunol Allergy Clin North Am.* 2014 Feb;34(1):1-9.
- Abajian M, Mlynek A, Maurer M. Physical urticaria. *Curr Allergy Asthma Rep.* 2012 Aug;12(4):281-7.
- Deacock SJ. An approach to the patient with urticaria. *Clin Exp Immunol.* 2008 Aug;153(2):151-161.
- Önder M. Ürtiker ve anjioödem, genel yaklaşım ve sınıflandırma. *Turkiye Klinikleri J Dermatol-Special Topics.* 2008;1:6-11.
- Dice JP. Physical urticaria. *Immunol Allergy Clin North Am.* 2004 May;24(2):225-46.
- Grabbe J. Pathomechanisms in physical urticaria. In *Journal of Investigative Dermatology Symposium Proceedings.* 2001 Nov 1;6(2):135-136. Elsevier.
- Bal F, Kahveci M, Soyer O, Sekerel BE, Sahiner UM. Chronic inducible urticaria subtypes in children: Clinical features and prognosis. *Pediatric Allergy and Immunology.* 2021 Jan;32(1):146-52.
- Silpa-archa N, Kulthanan K, Pinkaew S. Physical urticaria: prevalence, type and natural course in a tropical country. *Journal of the European Academy of Dermatology and Venereology.* 2011 Oct;25(10):1194-9.
- Maurer M, Metz M, Magerl M, Siebenhaar F, Staubach P. Autoreaktive Urtikaria und Autoimmunurtikaria [Autoreactive urticaria and autoimmune urticaria]. *Hautarzt.* 2004 Apr;55(4):350-6. German. doi: 10.1007/s00105-004-0692-9.
- Panaszek B, Pawłowicz R, Grzegorzółka J, Obojski A. Autoreactive IgE in Chronic Spontaneous/Idiopathic Urticaria and Basophil/Mastocyte Priming Phenomenon, as a Feature of Autoimmune Nature of the Syndrome. *Arch Immunol Ther Exp (Warsz).* 2017 Apr;65(2):137-143. doi: 10.1007/s00005-016-0417-7.
- Maurer M, Eyerich K, Eyerich S, Ferrer M, Gutermuth J, Hartmann K, *et al.* Urticaria: Collegium Internationale Allergologicum (CIA) Update 2020. *Int Arch Allergy Immunol.* 2020;181(5):321-333. doi: 10.1159/000507218.
- Pozderac I, Lugović-Mihčić L, Artuković M, Stipičić-Marković A, Kuna M, Ferček I. Chronic inducible urticaria: classification and prominent features of physical and non-physical types. *Acta Dermatovenerol Alp Pannonica Adriat.* 2020 Sep;29(3):141-148.
- Amaya D, Sánchez A, Sánchez J. Inducible urticaria: Case series and literature review. *Biomedica.* 2016 Mar 3;36(1):10-21
- Maurer M, Fluhr JW, Khan DA. How to Approach Chronic Inducible Urticaria. *J Allergy Clin Immunol Pract.* 2018 Jul - Aug;6(4):1119-1130.
- Singleton R, Halverstam CP. Diagnosis and management of cold urticaria. *Cutis.* 2016 Jan;97(1):59-62.
- Claudy A. Cold urticaria. *J Invest Dermatol Symp Proc.* 2001 Nov;6(2):141-2.
- Kulthanan K, Hunnangkul S, Tuchinda P, Chularojanamontri L, Weerasubpong P, Subchookul C, Maurer M. Treatments of cold urticaria: A systematic review. *J Allergy Clin Immunol.* 2019 Apr;143(4):1311-1331. doi: 10.1016/j.jaci.2019.02.005.
- Chung HS, Lee KH, Ro JY. Heat contact urticaria--a case report. *Yonsei Med J.* 1996 Jun;37(3):230-5. doi: 10.3349/ymj.1996.37.3.230.
- Wise RD, Malkinson FD, Luskin A, Gewurz AT, Zeitz

- HJ. Localized heat urticaria. *Archives of dermatology*. 1978 Jul 1;114(7):1079-80.
29. Maibach HI, Johnson HL. Contact urticaria syndrome: contact urticaria to diethyltoluamide (immediate-type hypersensitivity). *Archives of dermatology*. 1975 Jun 1;111(6):726-30.
 30. Porcel S, León F, Cumplido J, Cuevas M, Guimaraens D, Condé-Salazar L. Contact urticaria caused by heat-sensitive raw fish allergens. *Contact Dermatitis*. 2001 Sep;45(3):139-42.
 31. Kulthanan K, Ungprasert P, Tuchinda P, Chularojanamontri L, Charoenpipatsin N, Maurer M. Delayed Pressure Urticaria: A Systematic Review of Treatment Options. *J Allergy Clin Immunol Pract*. 2020 Jun;8(6):2035-2049.e5. doi: 10.1016/j.jaip.2020.03.004.
 32. Kobza-Black A. Delayed pressure urticaria. *J Investig Dermatol Symp Proc*. 2001 Nov;6(2):148-9.
 33. Lawlor F, Black AK. Delayed pressure urticaria. *Immunol Allergy Clin North Am*. 2004 May;24(2):247-58.
 34. Fityan A, McGibbon D, Fassihi H, Sarkany RS. Paediatric solar urticaria: a case series. *Br J Dermatol*. 2018 Jun;178(6):1453-1454.
 35. Raigosa M, Toro Y, Sánchez J. [Solar urticaria. Case report and literature review]. *Rev Alerg Mex*. 2017 Jul-Sep;64(3):371-375.
 36. Snyder M, Turrentine JE, Cruz PD. Photocontact Dermatitis and Its Clinical Mimics: an Overview for the Allergist. *Clin Rev Allergy Immunol*. 2019 Feb;56(1):32-40.
 37. Maurer M, Fluhr JW, Khan DA. How to Approach Chronic Inducible Urticaria. *J Allergy Clin Immunol Pract*. 2018 Jul - Aug;6(4):1119-1130.
 38. Morgado-Carrasco D, Fustà-Novell X, Podlipnik S, Combalia A, Aguilera P. Clinical and photobiological response in eight patients with solar urticaria under treatment with omalizumab, and review of the literature. *Photodermatol Photoimmunol Photomed*. 2018 May;34(3):194-199.
 39. Boyden SE, Desai A, Cruse G, Young ML, Bolan HC, Scott LM, *et al*. Vibratory Urticaria Associated with a Missense Variant in ADGRE2. *N Engl J Med*. 2016 Feb 18;374(7):656-63. doi: 10.1056/NEJMoa1500611.
 40. Pastor-Nieto MA, Gatica-Ortega ME, Vergara-de-la-Campa L, Giménez-Arnau AM. Proposal for a new classification of vibratory urticaria/angioedema. *J Allergy Clin Immunol Pract*. 2021 Jun;9(6):2542-2543. doi: 10.1016/j.jaip.2021.02.036.
 41. Kaplan AP, Greenberger PA, Geller M. Vibratory Urticaria and ADGRE2. *N Engl J Med*. 2016 Jul 7;375(1):94-5.
 42. Boyden SE *et al*. Vibratory Urticaria Associated with a Missense Variant in ADGRE2. *N Engl J Med*. 2016 Feb 18;374(7):656-63.
 43. Nakamizo S, Egawa G, Miyachi Y, Kabashima K. Cholinergic urticaria: pathogenesis-based categorization and its treatment options. *J Eur Acad Dermatol Venereol*. 2012 Jan;26(1):114-6. doi: 10.1111/j.1468-3083.2011.04017.x.
 44. Godse K, Farooqui S, Nadkarni N, Patil S. Prevalence of cholinergic urticaria in Indian adults. *Indian Dermatol Online J*. 2013 Jan-Mar;4(1):62-63.
 45. Fukunaga A, Washio K, Hatakeyama M, Oda Y, Ogura K, Horikawa T, *et al*. Cholinergic urticaria: epidemiology, physiopathology, new categorization, and management. *Clin Auton Res*. 2018 Feb;28(1):103-113. doi: 10.1007/s10286-017-0418-6.
 46. Altrichter S, Wosny K, Maurer M. Successful treatment of cholinergic urticaria with methantheliniumbromide. *J Dermatol*. 2015 Apr;42(4):422-4.
 47. Maibach HI, Johnson HL. Contact urticaria syndrome: contact urticaria to diethyltoluamide (immediate-type hypersensitivity). *Archives of dermatology*. 1975 Jun 1;111(6):726-30.
 48. Orb Q, Millsop JW, Harris K, Powell D. Prevalence and interest in the practice of scratch testing for contact urticaria: a survey of the American contact dermatitis society members. *Dermatitis*. 2014 Nov-Dec;25(6):366-9.
 49. Amaro C, Goossens A. Immunological occupational contact urticaria and contact dermatitis from proteins: a review. *Contact Dermatitis*. 2008 Feb;58(2):67-75.
 50. Doutre MS. Occupational contact urticaria and protein contact dermatitis. *Eur J Dermatol*. 2005 Nov-Dec;15(6):419-24.
 51. Adishes A, Robinson E, Nicholson PJ, Sen D, Wilkinson M. Standards of Care Working Group. U.K. standards of care for occupational contact dermatitis and occupational contact urticaria. *Br J Dermatol*. 2013 Jun;168(6):1167-75.
 52. Nicholson PJ. Evidence-based guidelines: occupational contact dermatitis and urticaria. *Occup Med (Lond)*. 2010 Oct;60(7):502-4.
 53. Bhatia R, Alikhan A, Maibach HI. Contact urticaria: present scenario. *Indian J Dermatol*. 2009 Jul;54(3):264-8.
 54. Rifaqat, Faureen Baqi, Zamir Ahmad, Abdul Mannan. A Historical Review on Urticaria. *J. Res. Tradit. Med*. 2016;2(6):173-177.
 55. Razi AMBZ. *Al Hawi Fit Tib* (Urdu translation by Jafri SA, Siddiqui MY). Aligarh; Saba Publishers. 1994;23(2):448.
 56. Majusi AIA. *Kamil-us- Sana'a* (Urdu translation by Kanturi GH). New Delhi. Idara Kitab-us-Shifa. 2010;1:433-3413.
 57. Ibn-e-Sina AA. *Al Qanoon fit Tib* (urdu translation by Kanturi (GH). New Delhi; Idara Kitab-us-Shifa, YNM, 4, p.126214.
 58. Qamri AMH. *Ghina Muna*, (Urdu translation by CCRUM). New Delhi. Ministry of Health and Family Welfare. 2008;2:490-9111.
 59. Tabri AR, Firdausul Hikmat. (Urdu translation by Sambhali MAS). New Delhi; Idara Kitab-us-Shifa, 2010, p.297.
 60. Razi, AMBZ. *Kitabul Mansoori*. (Urdu translation by CCRUM). New Delhi. Ministry of Health and Family Welfare, 1991, p.203-410.
 61. Kulthanan K, Tuchinda P, Chularojanamontri L, Chanyachailert P, Korkij W, Chunharas A, *et al*. Clinical practice guideline for diagnosis and management of urticaria. *Asian Pac J Allergy Immunol*. 2016 Sep;34(3):190-200.