ORIGINAL RESEARCH PAPER
VOLUME-6 | ISSUE-5 | MAY - 2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A STUDY ON ETIOLOGICAL SPECTRUM OF CHRONIC URTICARIA IN AN URBAN CITY HOSPITAL

Dermatology

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ABSTRACT

Background: Urticaria is one of the most commonly encountered skin diseases which affect 15%-20% of the population once or more during life time. It is a heterogeneous group of disorder with a wide range of multifactorial etiology. In this study, we tried to find out the etiological spectrum of chronic urticaria in an urban city hospital.

Materials and Methods: This study is a cross sectional study which included 150 patients with urticaria who attended the skin outpatient department. A detailed history taking and complete physical examination was done for all these patients and the observations were tabulated.

Results: 150 cases of chronic urticaria were studied. 85 were female and 65 were male. The mean age was 35.5 years. The duration of chronic urticaria ranged from 2 months to 20 years. The mean duration of disease was significantly higher among females (mean duration of disease in female is 27.6 and male is 17.20). In the present study, 33 patients (22%) gave a personal history of atopy. One patient (0.7%) was found to have associated systemic disease (thyroid autoimmunity). 71 patients were subjected to ASST of which 50 of them were tested negative and 21(30%) of them were tested positive. In the study group, 31(43.7%) were males and 40(56.3%) were females.

KEYWORDS:

urticaria

INTRODUCTION:

Urticaria is defined as a transient, erythematous, edematous and itchy swelling of the skin. It is a quite common condition which sometimes can be extremely disabling and difficult to treat. Chronic urticaria is defined as persistence of urticaria, for a period of 6 weeks or more. As per literature, chronic urticaria constitutes a quarter of patients with urticaria.

Although, most of the urticaria is attributed to allergic reactions, only 20% cases have shown an identifiable allergic cause. The etiology of urticaria is multifactorial which includes infections (Helicobacter pylori, Candida), infestations (protozoans, helminthes) intolerance to food, drugs and autoimmune diseases. A subset of chronic urticaria called as "Autoimmune urticaria" has been described, with the advent of demonstration of auto antibodies against IgE and IgE receptor. Other causes attributed to the etiology of chronic urticaria are thyroid autoimmunity, connective tissue diseases, progesterone and estrogen sensitivity, psychological stress. Implants like dental amalgams, femur pin, etc can also trigger urticaria.

Despite being a common skin disease, the possible etiology of chronic urticaria remains unclear. Hence a study was undertaken to find out the possible triggering factors of chronic urticaria.

METHODS:

A total of 150 patients diagnosed with urticaria (all types), attending skin outpatient department were involved in this study. The patients were selected randomly, irrespective of the age, sex and socio-economic status. A pre-structured and pre-tested proforma was used to collect data, after obtaining consent from the patients. Baseline data including age and sex, a complete medical history including conventional risk factors, clinical examination and relevant investigations were recorded. The study was done after clearance from the ethical committee.

OBSERVATION & RESULTS:

Maximum number of patients 67.3% (101) belongs to < 40 years age group.

65 were males and 85 were females: 43.3 % and 56 % respectively. Mean duration of disease was significantly higher among females. Mean duration of disease was higher among those having the history of angioedema.

Among the study population, a maximum of 117 (78%) patients showed positive history of atopy. Out of 150 a maximum of 119 (79%) patients did not show the history of angioedema.

In a sample space of 150, 145 patients (97%) did not have any association with positive family history.

71 patients went through ASST test. 50 of them tested were negative and 21 of them tested positive. 31 (43.7 %) were males among this group. And 40 (56.3%) were females. Mean age of those who were tested negative was 37.62 and mean age of those who were tested positive was 34.24years. This difference was not statistically significant. Only 1 (0.7 %) patient had history of associated systemic disease like.

DISCUSSION:

Urticaria, even though it is a common skin disease, it is rarely life threatening. Approximately 15-20% of the population may experience at least one episode of urticaria in their lifetime, and about one quarter of these patients are likely to develop chronic urticaria. [1] The rational therapy for urticaria is the identification and avoidance of causative agents that directly or indirectly precipitate the eruption. [2] In chronic urticaria, the search for a cause is much more difficult. [3] Patients often seek medical attention with the hope that a reversible cause can be identified. Being able to efficiently apply a cost-effective work up for urticaria is challenging. Therefore, the challenge for the clinician is to try to identify a cause that could lead to a specific treatment or avoidance strategy. [4] The pathogenesis of urticaria is not fully known. There are many theories proposed to explain the pathophysiology like – humoral theory, toxin theory, nervous theory, angioneurotic theory, menstrual theory, microthrombus theory [5] and inflammation theory.

Classification:

Urticaria is a heterogeneous group of disorders that may be broadly classified by duration of disease, etiology and clinical features.

Etiological classification [6]

a) Immunological
1. Autoimmune
2. IgE mediated
3. Immune complex mediated
4. Complement and kinin mediated

b) Non Immunological
1. Direct mast cell releasing agent (e.g. Opiates)
2. Vasoactive stimuli (e.g. Nettle sting)
3. Abnormalities of arachidonic acid metabolism (Aspirin, other NSAIDS, dietary pseudo allergens)
4. Angiotensin converting enzyme inhibitors

Aetiology:

Ordinary urticaria

Genetics: HLA-DR4, HLA-DRB4 53 & HLA-DQ8 and DQA 3011/12 are associated with chronic idiopathic urticaria. Hereditary angioedema is transmitted in an autosomal dominant manner.

Atopy: A gene predisposing to atopy (as defined by hyper-IgE responsiveness) has been found on chromosome 11q13, and it is possible that it codes for the subunit of the high affinity IgE receptor (Fc RII). It may manifest as respiratory distress due to bronchospasm or oedema of the mucosa of the larynx and bronchi, cutaneous changes vary from erythema to angioedema and urticaria and intestinal spasm which are accompanied by vomiting or diarrhoea and shock. However various studies had stated that the prevalence of chronic urticaria and or angioedema is not greater in atopic persons. [7]

Autoimmune urticaria:

In some patients with chronic urticaria, the wheals are caused by circulating auto antibodies that have histamine releasing properties. The incidence of such autoimmune urticaria varies in different studies from 25% to 60%. It has been demonstrated that there are two distinct types of auto antibodies in the circulation of these patients. One is directed towards the α-subunit of the high affinity receptor for IgE [FcεR1] that is found on the surface of the mast cell and basophils. This autoantibody, IgG1 or IgG3 subtype forms cross-linking with this receptor, resulting in mast cell degranulation. The other type of autoantibody is directed against IgE itself rather than towards the receptor, leading to formation of IgE-Anti IgE complex that can activate mast cells. The presence of a circulating antibody directed against FcεRI can be detected by intradermal testing with autologous serum; a positive reaction leads to a wheal formation. Another test detects release of histamine from blood basophils in response to serum of patients with these circulating antibodies. These findings were subsequently proved by immunoblotting and immunoprecipitation tests. [8],[9]

Foods:

Food urticaria is suspected when patient has repeatedly noticed aggravation of symptoms following ingestion of certain foods. The agents responsible can be either food proteins or substances added to food for colour, preservatives and taste. Foods can cause urticaria by both IgE mediated and non IgE mediated mechanisms. However an allergic cause was found in less than 3.5% of all cases. [10]

Infections: Urticaria has been reported to occur coincidently with infections caused by bacteria, virus, parasites, yeast and molds. However temporal relationships between onset of infection and the development of urticaria as well as the resolution of urticaria following successful treatment of the infection have been described.

Viral: Viral associations include Epstein Barr virus, Herpes Simplex virus, Hepatitis A, B & C virus. In addition, urticaria has been reported with Coxsackie and Enteric Cytopathic Human Orphan (ECHO) viral infections. The serum sickness phase of viral hepatitis and infectious mononucleosis may develop into chronic urticaria. [11]

Bacterial Infections: Infections of the oral cavity, prostate, genitourinary tract, lungs, gall bladder, sinuses etc can cause chronic urticaria. More recently a possible role for Helicobacter pylori has been suggested by a number of studies.

Fungal infections and yeasts: Fungal infections have also been implicated as a possible cause of chronic urticaria. The association between tinea pedis and candida albicans has also been implicated as a cause of chronic urticaria.

Parasites: Parasites responsible for chronic urticaria include Ascaris, Toxocara, [12] Ancylostoma, Fasciola, Strongyloides, Filaria, Echinococcus, Schistosoma, Trichinella and malaria parasites. House dust mite has also been found to cause chronic urticaria.

Drugs: Penicillin, other β-lactam antibiotics [13], cephalosporin, carbapenems and monobactams NSAIDS and angiotensin converting enzyme inhibitors can induce urticaria and or angioedema

Systemic Diseases: Urticaria has been associated with diabetes mellitus, hypothyroidism, hyperthyroidism, hyperparathyroidism, malignancy (Hodgkin's disease, paraproteinemias and malignant tumors of colon, rectum, liver, lung and ovary), SLE, urticarial vasculitis, Henoch Schöinlein purpura, erythema elevatum diutinum, rheumatoid arthritis and pregnancy (auto antibodies to progesterone).

Physical urticaria:

The physical urticarias are a subgroup of urticarias induced by external stimuli and the wheals are present mostly over the stimulated areas. It can be induced by pressure, sweat, vibration and temperature changes.

Pathophysiology:

Urticaria is due to local increase in permeability of capillaries and venules. These changes are dependent on the presence of signal that causes release of mediators of cutaneous mast cells and the interaction between cytokines and adhesion molecules stored within endothelial cells. [14] Activation of the mast cell results in the release of preformed and newly generated mediators from the cytoplasmic granules. Histamine activates the endothelium, along with IL-1, TNF-α upon release from the mast cell. This causes the endothelial cell to express various adhesion molecules like endothelial leucocyte adhesion molecule -1 (ELAM-1) and intercellular adhesion molecule-1 (ICAM-1). These account for recruitment of neutrophils and lymphocytes in various skin disorders, including urticaria. Local cutaneous fibrinolytic activity by endothelium is also important in the pathogenesis of different forms of urticaria. Cutaneous fibrinolytic activity was found to be reduced in urticarial vasculitis. In contrast, it is enhanced in some non-vasculitic forms of urticaria. Hence it is suggested that non-vasculitic forms of urticaria may be due to pro-inflammatory properties of fibrinogen degradation products, which could activate release of synthesis of other mediators. [15] Secretagogues are triggers, which initiate the mast cells & basophils to release their mediators. The secretagogues that induce mast cell degranulation can act both by immunological and non-immunological ways.

Histopathology:

The histology of wheals are usually non-specific with vascular and lymphatic dilatation, dermal edema and a variable perivascular cellular dermal infiltrate consisting of lymphocytes, monocytes, neutrophils and eosinophils.

Clinical Features:

Urticaria is manifested by clinically evanescent, itchy, erythematous macules, which develop into wheals consisting of pale-pink,

edematous, raised skin areas often with a surrounding flare. When the edematous process extends deep into the dermis or subcutaneous and submucosal layers it is known as angioedema. Urticaria and angioedema can occur alone individually or both can occur together. The individual lesions of urticaria arises suddenly, usually persist for less than 24 hours. Wheals lasting more than 24 hours raise the possibility of other diagnosis, including urticarial vasculitis. Vomiting can precede urticaria and it can be associated with systemic symptoms of malaise, headache, vomiting, abdominal pain, diarrhoea, arthralgia, dizziness and syncope and in it is most severe acute form with anaphylaxis.

Investigations:

A detailed history is essential for diagnosis and elucidation of a causative factor in chronic urticaria. In acute urticaria, they are usually not required as it is usually self limiting. Complete blood count, erythrocyte sedimentation rate, LFT, RFT, thyroid function test, urinalysis, stool examination , autologous serum skin test, skin prick testing, skin biopsy, complement levels, ANA, Serum IgE levels, anti IgE auto antibodies and Vitamin B 12 assay are some of the test done in chronic utricaria.

CONCLUSION:

150 cases of chronic urticaria were studied.
85 were female and 65 were male. The mean age was 35.5 years. The duration of chronic urticaria ranged from 2 months to 20 years. The mean duration of disease was significantly higher among females

In the present study, 33 patients (22%) gave a personal history of Atopy.

In the present study, one patient (7%) was found to have associated systemic disease (thyroid autoimmunity).

In the present study, 71 patients was subjected to ASST. 50 of them were tested negative and 21(30%) of them were tested positive. 31(43.7%) were males among this group and 40(56.3%) were females.

ACKNOWLEDGEMENT:

Nil

CONFLICT OF INTEREST:

Nil

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