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Urticaria – A Complete Clinical Review

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Abstract :

Urticaria is an inflammatory skin disorder that affects up to 20 % of the world population at some point during their life. It presents with wheals, angioedema or both due to activation and degranulation of skin mast cells and the release of histamine and other mediators. Most cases of Urticaria are acute Urticaria, which lasts <6 weeks and can be associated with infections or intake of drugs or foods. Chronic Urticaria (CU) is either spontaneous or inducible, lasts >6 weeks and persists for >1 year in most patients. CU greatly affects patient quality of life, and is linked to psychiatric comorbidities and high healthcare costs. In contrast to chronic spontaneous Urticaria (CSU), chronic inducible Urticaria (CIndU) has definite and subtype-specific triggers that induce signs and symptoms.

Keywords *: Urticaria, Angioedema, Histamine, Cyclosporine, Coagulation Disease.*

Purpose of this clinical review

The purpose of this clinical update on urticaria is to facilitate earlier diagnosis and appropriate treatnent , minimise unneccessary investigations and facilitate early indentificatin of those who might benefit from a specialist refferal in response to and drawinf from this revision.

Definition :

The term Urticaria is defined as a transient eruption of circumscribed edematous and usually itchy swellings of the dermis. The other names given are nettle rash, hives or weals. These lesions last for a few hours but not exceeding 48 hours. The term angioneurotic oedema denotes similar but larger swellings of the deep dermal, sub-cutaneous and sub-mucosal tissues. The other names are angioedema, giant Urticaria and Quinke‟s oedema. Urticaria and angio-oedema are important components of systemic anaphylaxis which is an acute life threatening condition. Several well illustrated monographs are available on Urticaria which may be referred for details.

Classification :

Urticaria may be broadly classified on the basis of duration and trigger factors. It is classified into „acute‟ and „chronic‟ on the basis of duration. This is an arbitrary division and is between 6 weeks to 8 weeks. Acute Urticaria is diagnosed retrospectively with a history of less than 6-8 weeks duration. The causative factor is easily identified in acute urticarial whereas it is difficult in chronic urticaria. When there is no detectable cause in chronic urticarial it is also known as chronic idiopathic urticarial. Recently studies after thorough examination of patients with chronic idiopathic urticaria revealed that 30% had lg G autoantibodies against the lg E receptor.

Pathogenesis :

Activation of cutaneous mast cells liberates various mediators predominantly histamine which induces increased permeability of capillaries and venules which in turn produces urticarial. The clinical response of urticaria to antihistamines proves this hypothesis. Mast cells may be activated by allergic or non-allergic mechanisms. Allergic mast cell activation occurs as a result of linkage of two adjacent a sub units of high affinity eg.

penicillin. As a result of this, preformed histamines, proteases, prostaglandin D2, LT4 and interleukin 4, IL-8 and tumour necrosis factor are released. Non allergic cell activation occurs with a variety of substances like neuropeptides (substance P), drugs like morphine, codeine, vancomycin, radio contrast media and some foods such as strawberries.

Chronic idiopathic urticaria patients can develop lg G antibodies directed against the sub unit of the FCE RI or against the receptor bound lg. Immune modulating therapy like plsmapheresis and intravenous immunoglobulin were associated with remission in some patient. 8 causes neutrophil leukocyte accumulation.

Plasma derived mediators such as bradykinin and complement have no role in chronic urticaria. However, bradykinin plays a role in production of angioedema. Complement activatin occurs in urticarial vasculitis and immune complex urticaria. Lymphocytes, neutrophils and eosinophils also may release a variety of cytokines which may enhance or perpetuate the weal response. This mechanism is demonstrated by their presence in the venous.effluent from physical urticarias like cold, cholinergic and solar urticarias. Increased substance P and vasoactive intestinal peptide (VIP) have been demonstrated in cold and cholinergic urticarial lesins. Urticarial vasculitis, also called hypocomplementaemic vasculitis, resembles systemic lupus erythematosus in its pathogenesis. However, it may also involve abnormal genetic immunoregulation in some.

Clinical Features

Each episode starts with itching. Following this, erythematous macules and weals appear which are transient and disappear within a few hours to a maximum of 24 hours. The weals may vary in size ranging from a few millimeters to may centimeters. The weals appears on any part of the body and may be associated with angio-oedema. The skin lesions resolve without any trace. Eventhough many patients claim worsening of the lesions during full moon and new moon days there is no convincing evidence for this. Inspite of severe pruritus there are no scratch marks in urticaria as the patients tend to rub the skin. Urticaria occasionally may be associated with a few systemic symptoms such as vomiting, giddiness, malaise, headache, abdominal pain, diarrhea, dizziness and rarely anaphylaxis. Premenstrual exacerbation may be noticed in some.

Causes of Urticaria :

1) **Drugs :-**
Many drugs cause urticaria through allergic or non allergic mechanisms. Drugs produce acute urticarial reactions usually within 36 hours of administration. Penicillins, cephalosporins, sulfonamides and tetracyclines are some examples of drug induced urticaria. Acetyl salicylic acid and non steroidal anti inflammatory drug are responsible to produce urticaria through allergic mechanism. Drug which is taken for a long time is unlikely to cause urticaria. On the contrary even a small quantity of penicillin present in the diary products may produce severe urticaria in a sensitive person.

2) **Infections :-**
Infections may produce either acute or chronic urticaria. Non specific infections may be responsible for acute urticaria. Hepatitis B viral infection,streptococcal throat infection, and Campylobacter jejuni are a few examples which can produce acute urticaria. Bacterial infections of the dental, throat, respiraroty, urinary tracts and gall bladder rarely may be responsible for chronic urticaria. The utticarial lesions do not alter with treatment of underlying chronic infection. Tebbi et al stressed the role of Helicobacter pylori in chronic urticaria.

3) **Infestations :-**
Gastro-intestinal parasites were thought to be commonly responsible to cause urticaria. Ankylostoma, strongyloides, echinococcus and Toxacare canis are few examples. However this has become a rare cause as shown in recent Indian studies. Ascariasis produces urticaria through allergic mechanism while the other intestinal parasites produce the lesions through non allergic mechanism.

4) **Inhalants :-**
Various substances like grass, pollens, mould, spores, animal danders, and house dust may cause acute or chronic urticaria.

5) **Ingestion :-**
Food and food additives can cause both acute and chronic urticaria. Fish, milk, peanut, beans, potato, rice, carrot and drumstick are responsible for acute urticaria mediated through lgE dependent mechanism. The reaction can occur in a few minutes to many hours. Though a variety of foods are attributed to produce urticaria a double blind challenge reproduced the lesions only in some. Among the alcoholic beverages red wine may cause urticaria in some.

6) **Insect bites :-**
Papular urticaria occurs through the bite of insects most commonly mosquitoes and bed bugs. These are chronic anda recurrent. On the contrary bee or wasp stings (hymenoptera) may produce severe acute urticaria or anaphylaxis through allergic mechanism which may be life threatening.

7) **Injections :-**
Parenteral injection of therapeutic sera produces serum sickness through x complement Type III reactions.

8) **Implant :-**
Dental prosthesis and metal pins used in orthopaedics practice may rarely cause urticaria.

9) **Systemic disease :-**
Systemic lupus erythematous produces urticarial vasculitis more commonly than chronic urticaria. Increased incidence of patient with thyroid auto antibodies have been reported and treatment with thyroxine can result in clinical remission in some.

10) **Others :-**
Psychological stress may favour urticaria whereas depression decreases the threshold for pruritus.

Differential Diagnosis

More often persistent erythemas and purpuras have to be differentiated from urticaria. These lesions last longer than 24 hours whereas urticarial weals resolve by that time. Erythema multiforme and early vesico bullous eruptions have also to be distinguished from urticaria.

Prognosis

In a follow up study by Champion at London in 554 patients 50 % still had active disease after 6 months. Of these patients 40 % continued to have intermittent symptoms 10 years later. The prognosis worsened in patients with angio-oedema.

Investigations

Routine hematological investigations are to be carried out in all patients. Erythrocyte sedimentation rate (ESR) if high may reflect on occult infection. Repeated microscopic examination of the stools may reveal an unusual intestinal parasite. Other investigations are done according to symptomatology. If history does not reveal a cause investigations rarely provide an answer either in acute or chronic urticaria. If weals persist and are painful, with the presence of systemic symptoms, a skin biopsy may be done to rule out urticarial vasculitis. Screening tests for thyroid functions may reveal an abnormality in 7 % of patients with chronic urticaria. Extensive bio- chemical tests, total IgE Levels and RAST are not indicated routinely.

Conclusion

The aim of review is to provide clinicians a useful tool for correctly suspecting and Indentifying the principal skin disorders that can be misdiadnosed as urticaria. These conditions must be taken in consideration in case of atypical wheals ,in morphology and duration (>24 hr) , association with other elementary skin lesions absence of angiodema and appearance of systemic symptoms. The presence of one or more of these features should prompt further investigations in order to allow an accurate diagnosis and ensure the best therapeutic choice for the patient.

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