

Bulletin of



IAISD

Institute of Allergic & Immunologic Skin Diseases

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Message from CODFI President

Derma-Allergologists: Role in Dermatological Care

Dr A K Bajaj, Allahabad

President, CODFI (Contact & Occupational Dermatoses Forum of India)

Skin is an important immunological organ and an open laboratory to study in-vivo allergic reactions. Skin participates or shows manifestations of allergic/immune reactions taking place in various internal organs or affecting only the skin. Such manifestations are protean in nature and require Sherlockian approach or Dr. Watson's keen eye and brain to critically analyze or evaluate them. Contact dermatitis, urticaria, atopic dermatitis and adverse

cutaneous drug reactions are the common allergic dermatological disorders accounting for a fairly large number of cases. Over and above these common disorders vasculitis, panniculitis, non-eczematous contact reactions etc. also come under the purview of derma-allergologists. It is unfortunate that almost all the dermatologists (who are small in number for an Indian population of billion plus), including those working in

tertiary care centers, are overburdened with the management of common skin diseases. With industrialization and tremendous change in the environment the number of allergic disorders are going up. The need of the hour is establishment of specialized centers exclusively engaged in the study and management of allergic disorders so that these are adequately treated and cured where the removal of causative agent is possible.

In a particular clinic the incidence of allergic contact dermatitis is determined by the interest the dermatologist takes in allergic contact dermatitis.

Hjorth and Tregat

Editorial

Can a Chemical Exposure in Localized Area Lead to Generalized Vitiliginous Process?

Dr Sanjay Ghosh
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We know that vitiligo originates as autoimmune response against melanocytes. However we are not certain about which triggers this autoimmune response and how this autoimmune process takes place. Recent research has emitted some sharp light to visualize this misty zone in the pathogenesis of vitiligo.

Overexposure to UV rays, mechanical stress (burns, cuts, scratch) [Koebner's phenomenon] and chemicals (phenolic compounds & others) are known initiating factors of vitiligo. Can this local induction lead to a generalized immune response?

In some recent experiments this puzzle has been attempted to be solved.

4-TBP (Tertiary butyl phenol), an alternate substrate for tyrosinase, at low concentration acts by competitive inhibition whereas at high concentration as cytotoxic (not depending on the pigmentation of melanocytes). 4-TBP induces oxidative stress upon melanocytes which in turn releases stress protein HSP 70 (Heat Shock Protein).

Stress proteins, also called "Chaperokines", protect the cellular proteins from premature degeneration.

Cells with increased stress proteins are protected from consequences of subsequent stress. Released stress proteins from the cells into the extracellular milieu produce immune

Contd to Page 4 Column 1

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Management Strategy of Plaque Psoriasis

Dr Susmit Haldar
IAISD, Kolkata

Psoriasis is a chronic inflammatory skin disease as yet of unknown specific etiology. Like all other diseases of unknown cause, various treatment modalities have been tried and developed empirically in the past several decades and are continued to evolve till now. It is bewildering to choose an appropriate one among this wide range of treatment options, unless a management strategy is followed. The problem is further compounded by the fact that efficacy of many of these therapeutic agents or modalities are not tested by prospective, randomized, double-blind, controlled studies.

In general, the factors which are important to remember in management are: a) extent of involvement, b) areas of involvement, c) patient's perception of the severity of the disease, d) patient's lifestyle, e) other associated health problems and medications, and f) potential side-effects of the specific therapy planning to start. It is also equally important to realize that management of chronic psoriasis needs individualization of therapy, even after accepting the need of a strategic approach. A management strategy is outlined below keeping a practical approach in view.

Localized Psoriasis (<5% BSA involvement)

Trunk and Extremities

The treating physician has a choice of a wide range of topical therapies such as topical corticosteroids (TCS), Vitamin D3 analogues (calcipotriol and others), tazarotene, anthralin and coal tar. Efficacy of all these agents is endorsed by various double blind, randomized, controlled studies. When compared, each of these agents has advantages and disadvantages. For an example, topical corticosteroids (TCS) are relatively more economic and devoid of any local irritation if compared with calcipotriol (Vitamin D3 analogue), calcipotriol is free from the local side effects of TCS like atrophy, telangiectasia. The chance of rebound flare of healed plaque after withdrawal is virtually absent in case of calcipotriol compared to TCS. If a single agent fails to control psoriasis, the strategy will be a combination therapy of two agents (e.g. calcipotriol or tazarotene can be combined with superpotent TCS). (Table 1)

Palms and Soles (Only Plaque Type)
Psoriasis in these regions, though limited in extent, is disabling, recalcitrant, and sometimes resistant to topical treatment. Strategy will be to try topically at first, either by monotherapy

(topical superpotent corticosteroids) or by combination (e.g. TCS +/- Salicylic acid or TCS +/- Coal tar or TCS +/- Anthralin). If it fails, phototherapy or systemic agents are required. The common practice is to combine Acitretin 25 mg daily with 'bath-PUVA'. Bath-PUVA is usually employed by soaking the hands at first in a water-filled basin with methoxsalen and then exposing to UVA irradiation. Other alternatives are methotrexate or cyclosporine, which are claimed to be highly effective. (Table 1.)

should be diluted with petrolatum or used alternately with TCS. Other Vitamin D3 analogues (calcitriol and tacalcitol) are less irritating and thus can be used in face and flexors. Tazarotene is too irritating for intertriginous (crural) areas and should be avoided. It can be used effectively on facial psoriasis if its use is restricted only for 1-5 minutes. Such a short contact period is found to be less irritating on facial skin. Topical calcineurin inhibitors (Tacrolimus 0.1% ointment and Pimecrolimus 1% cream)

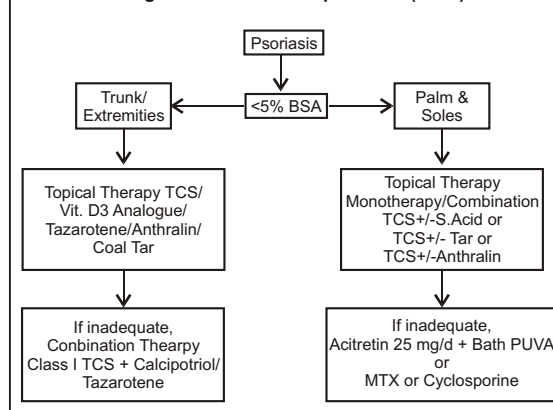
are used effectively nowadays and preferred for their safety and lack of side effects on continuous use compared to even low-potent TCS (Table 2).

Extensive Psoriasis (>5% BSA involvement)

Topical therapy may not be adequate to control, necessitating the use of systemic agents or phototherapy. Managing all the lesions of psoriasis involving more than 10% BSA

only by topical therapy is impractical, but it can be a useful adjunct to other treatment modality. UVB (BB-UVB) phototherapy is a preferred option to start with, especially for those patients who respond well to sun exposure. If

Table 1. Management of localized psoriasis (< 5%)



Scalp

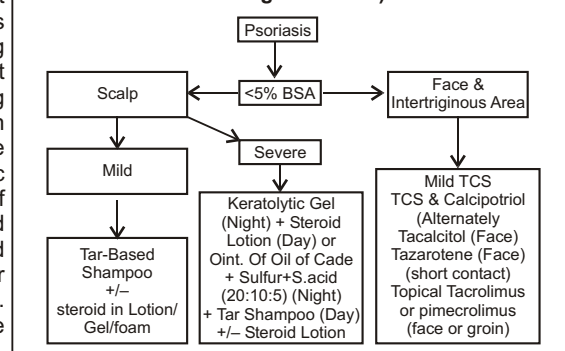
Mild involvement can be managed with a tar-based shampoo twice or thrice weekly with or without TCS in lotion, gel, and foam formulations. Messy preparations like cream or ointments are avoided for patients' dislike.

Severe scalp psoriasis, manifested clinically with very thick adherent scaly plaques, is managed by using keratolytic gel at night with TCS lotion during day. Alternatively, an ointment of oil of Cade with Sulfur and Salicylic acid (in a ratio of 20:10:5) can be applied and kept overnight and cleaned with a Tar shampoo next day. Steroid lotion can be applied during daytime. (Table 2.)

Face and Intertriginous Area

These areas respond very well to various topical agents, but are very much prone to acquire the side effects. Very low potent TCS is preferred to avoid the risk of atrophy, telangiectasia, striae and erythema. Calcipotriol is also irritating and, if at all to be used, it

Table 2. Management of localized psoriasis (scalp, face & intertriginous area)



failed, patients often respond to narrowband UVB (NB-UVB) phototherapy. PUVA has proved itself to be one of the most effective treatments of psoriasis. As an increased risk of cutaneous malignancy is associated with PUVA, it is usually kept reserved

Lichenoid Contact Dermatitis

Dr Dinesh Hawelia
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'Lichenoid' is the term used clinically to describe a flat-topped, shiny, papular eruption resembling lichen planus (LP) whereas histologically it connotes basal cell liquefaction and a band-like inflammatory cell infiltrate in the papillary dermis. Lichenoid contact dermatitis (LCD) implies lichen planus-like or lichenoid eruption resulting from contact with various chemicals and compounds.

LCD may present with features typical of classic lichen planus but papules are usually larger and scaly and they resolve with post-inflammatory brown pigmentation. Wickham's striae are usually absent unlike classic LP and mucous membrane is less involved in cutaneous LCD. Sometimes, the features of LCD may be atypical with localized or generalized eczematous papules and plaques and variable desquamation.

Lichenoid contact dermatitis may occur after contact with chemicals in colour film developer. Lesions begin in areas of contact with the developer, but sometimes extend beyond the site of contact. Lesions may persist for months and usually resolve with post-inflammatory hyperpigmentation. Substituted para-phenylene diamine (PPD)-A is usually responsible. Allergic patch test is positive to substituted PPD-A and is usually eczematous in nature. Temporary paint-on tattoos may lead to inflammatory skin reaction which on histology shows lichenoid dermatitis and allergic patch test is moderately to strongly positive to PPD (1% in petrolatum) and commercial black henna.

LP-like lesions have also been seen in persons exposed to dental restorative materials, aminoglycoside antibiotics, metals such as mercury, silver and gold. Oral mucosal LCD may be seen in persons coming in contact with dental devices.

Methacrylic acid esters used in car industry may lead to cutaneous LCD. As methacrylic acid esters are also present in dental devices, oral mucosal lichenoid dermatitis may also result.

LP-like contact dermatitis in atypical distribution has been seen in persons coming in contact with product containing chlorpheniramine maleate. There is a case report of a papular erythematous pruritic lesion on back of hand coming in contact with red inked pen writing. Other chemical inducers of

LCD reported are musk ambrette, nickel etc.

Histologically, features of LCD can be similar to that of LP. Certain histological features may help to diagnose lichenoid contact dermatitis or lichenoid tissue reaction and they are as follows: focal parakeratosis, focal hypogranulosis, greater degree of spongiosis, larger number of necrotic keratinocytes and cytooid bodies present higher in the stratum corneum and granulosum, more pleomorphic cellular infiltrate, abundant plasma cells and eosinophils in infiltrate, deeper perivascular infiltrate and less dense lymphocytic infiltrate which is not as band-like as in classic LP.

Avoidance of contactants usually resolves the LP-like eruption. Sometimes it may take many months for resolution. LCD should be suspected if LP-like lesions are present in atypical distribution and possibility of exposure to contactants should be explored. Allergic patch test may be highly rewarding in suspicious cases to solve the puzzle.

Source :

1. Pinkus H. Lichenoid tissue reactions. *Arch Dermatol* 1973;107:840-6
2. Schoel J, Tilgen W, Frosch RJ. Lichenoid contact eczema caused by colour film developer. *Hautarzt* 1991;42:251-3
3. Chung WH, Chang YC, Yang LJ et al. Clinico pathological features of skin reaction to temporary tattoos and analysis of possible causes. *Arch Dermatol* 2002;138:88-92
4. Breathnach SM, Black MM. Lichen planus and lichenoid disorders. In: Burns T, Breathnach SM, Cox N et al. Eds. *Rook's text book of dermatology*. 7th ed. Blackwell publishing, 2004;42.1-42.32
5. Lambo G, Belato N, Patruno C et al. Lichenoid contact dermatitis due to aminoglycoside antibiotic.
6. Pigatto PD, Guzzi G, Persichina P. Nummular lichenoid dermatitis from mercury dental amalgam. *Contact Dermatitis*. 2002;46:355-6
7. Kawamura T, Fukada S, Ohtake N et al. Lichen planus-like contact dermatitis due to methacrylic acid esters. *Br J Dermatol* 1996;134:358-60
8. Kurada K, Hisanaga Y. The diagnosis of lichen planus-like contact dermatitis to chlorpheniramine maleate. *Dermatology* 2002;205:281-4
9. Colin-Chamley N, Canbier MP, Barhoun K et al. Lichenoid contact dermatitis from the ink of a red pen. *Ann Dermatol Venereol* 1998;125:21-2.

Recent Research: Contact Dermatitis & Urticaria

Dr Nilendu Sarma
IAISD, Kolkata

1. Allergic Contact Dermatitis to Herbal Cosmetics

Allergic contact dermatitis from a natural deodorant: a report of 4 cases associated with lichen acid mix allergy. Sheu M, Simpson EL, Law SV, Storrs FJ *J Am Acad Dermatol*. 2006 Aug; 55(2):332-7.

Use of botanical ingredients in cosmetics and other personal care objects are thought to be safe and their use are on rise worldwide. In this study authors tried to evaluate the prevalence pattern of allergic sensitivity of a specific botanical deodorant in 4 referred patients. Allergens found in all cases were lichen acid mix and D-uscnic acid. The lichen acid mix were already established and common allergens in USA. However botanical ingredients in deodorants were rarely the reported source till date. Important lacunae in the study were the small sample size and lack of specificity regarding the importance of the individual lichens as the composite lichens were used.

2. Rubber Allergy : Which Component Responsible ?

Mercaptobenzothiazole or the mercapto-mix: which should be in the standard series? Diepgen TL, Bruynzeel DP, Andersen KE, et al. *Contact Dermatitis*. 2006 Jul; 55(1):36-8.

The European Environmental Contact Dermatitis Research Group (EECDRG) has conducted this study to analyze the relevance of using Mercaptobenzothiazole (MBT) and mercapto-mix, both being rubber allergens in European standard series. This large multicenter study on 32 475 consecutive tested patients among 11 centers in Europe proved that they should continue to be used simultaneously as omitting either MBT or mercapto-mix from the standard series will miss at least 20% or 22% of positive cases respectively.

3. Henna Dye : Chemical analysis

Quantification of para-phenylenediamine and heavy metals in henna dye. Kang JJ, Lee MH. *Contact Dermatitis*. 2006 Jul; 55(1):26-9.

Henna (*Lawsonia inermis*, family Lythraceae) is a plant extract (dried leaf extract) and is found in India, Sri Lanka and North Africa. The active ingredient or lawsone is structurally 2-hydroxy-1, 4-naphthoquinone. Sensitization potential of henna is extremely rare in comparison to PPD so it is used

The greatest abuse of patch testing is failure to use the test.

Colman 1982

Contd to Page 5 Column 1

Ready Reckoner

Cutaneous Adverse Reactions to Hypoglycemic Agents

Dr. Manas Sen
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- | | |
|--|--|
| <p>A) Sulphonylureas</p> <p>1) Chlorpropamide : maculopapular rash
Steven-Johnson syndrome
erythema nodosum
lichenoid eruption
exfoliative dermatitis</p> <p>2) Glibenclamide : allergic skin rash, bullae</p> <p>3) Gliclazide : erythema</p> <p>4) Glimpiride : pruritus, erythema, urticaria,
morbilliform rash,
maculopapular eruption</p> <p>5) Glipizide : allergic skin rash</p> <p>6) Tolbutamide : exanthematous skin eruption</p> <p>B) Biguanide</p> <p>1) Metformin : urticaria</p> <p>2) Phenformin : yet not found</p> | <p>C) Insulin : Local reactions Arthus like reaction with
urticarial lesion at injection site;
lipodystrophy with decreased
adipose tissue at site of
subcutaneous injection</p> <p>Systemic reacting : urticaria, serum
sickness like reaction</p> <p>D) Others</p> <p>1) Acarbose : erythema, exanthema, urticaria (rare)</p> <p>2) Glucomanan/Guargum (food) : no skin adverse
reaction</p> <p>3) Nateglinide (non-sulfonylurea) : yet not found.</p> <p>4) Pioglitazone (Thiazolidinedione) : yet not found.</p> <p>5) Repaglinide (non-sulfonylurea) : rash</p> <p>6) Rosiglitazone (Thiazolidinedione) : yet not found</p> |
|--|--|

Can a Chemical Exposure in Localized Area Lead to Generalized Vitiliginous Process?

From Page 1

response to the very cell from which they were derived. Thus extracellular stress proteins are not at all protective rather damaging for the cells! Stress proteins can serve as antigens in certain auto-immune diseases. Stress proteins also enhance an immune response by inducing phagocytosis & antigen processing by dendritic cells.

HSP 70 induces TRAIL (TNF-related apoptosis-induced ligand) expression and activates dendritic cells (DC) effector functions. Dendritic cells (DC) can specifically kill tumor cells by leaving the surrounding healthy cells re

untouched. DCs are equally capable of killing stressed melanocytes to initiate an autoimmune response resulting in progressive depigmentation of skin. Melanocytes exposed to 4-TBP show elevated TRAIL death receptor expression. TRAIL is a major player in DC-mediated cytotoxicity towards stressed melanocytes. DC effector functions are partially inhibited by blocking antibodies to TRAIL. TRAIL expression and infiltration by CD11c+ cells are abundant in perilesional vitiligo skin.

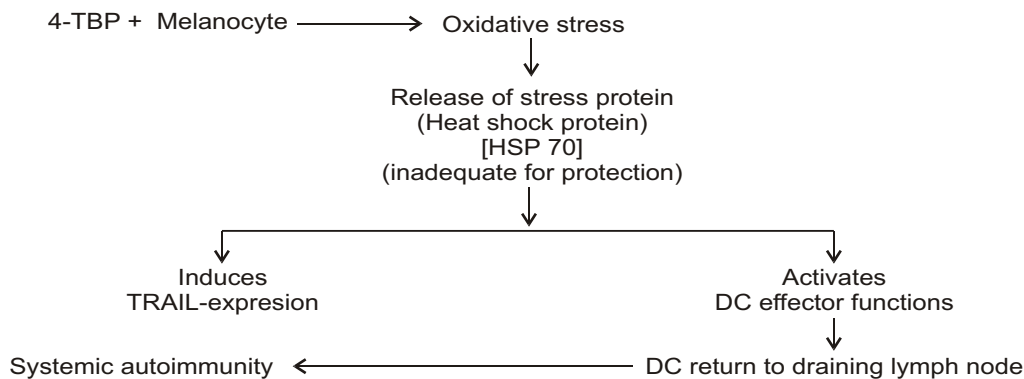
TRAIL expressing DC can be cytotoxic towards stressed yet untransformed tissue cells.

4-TBP induced epidermal stress can lead to selective killing of epidermal melanocytes by DC. This may instigate a systemic autoimmune response to melanocytes when the same DC return to draining lymph nodes, recruiting melanocyte-reactive cytotoxic T cells to the skin (Fig. 1).

Source :

1. Kroll TM, Bommasamy H, Boissy RE et al. J Investigative Dermatology 2005; 124:798-806.
2. Le Poole C, Yong F et al. J. Invest Derm. 1997, 113; 5:725-30.
3. Boissy RE, Marzu P. Pig Cell Res. 2004; 17 : 208-14.

Fig. 1. 4-TBP induces systemic vitiliginous process



Diagnostic Test

Autologous Serum Skin Test (ASST)

Dr Sudip Kumar Ghosh
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Basis

One third of the patients of chronic idiopathic urticaria have circulating functional autoantibodies against high affinity IgE receptor.

Autoantibodies in patients' serum can be detected by serum induced histamine release from the basophils of healthy donors utilizing methods :

1. ELISA, 2. Western blot assay. However, neither Western Blot nor ELISA can distinguish between functional histamine releasing autoantibody from non-functional autoantibody. Moreover, these tests are done in some specialized centers only as well as time consuming to perform.

So a rapid and reliable clinical test to differentiate between patients with or without circulating functional autoantibodies would be of value in initiating or evaluating the efficacy of immunomodulatory treatment.

Weal and flare response can be induced by the intradermal injection of autologous serum in some patients. These observations lead to the identification of circulating autoantibodies in chronic idiopathic urticaria and provides the basis of autologous serum skin test (ASST).

Sensitivity of ASST ranges between 65% to 71% and specificity between 78% to 81%.

Indications

Suspected cases of autoimmune urticaria.

Prerequisites

1. Withdrawal of antihistamines at least 2-3 days prior to the test
2. Doxepin and astemizole should be withdrawn 2-6 weeks beforehand

3. Patient should not take immunosuppressants within last 2 months
4. Ethical approval should be taken from the appropriate body
5. Age should be 18 years or more
6. Written consent from the patient or guardian
7. Test area (usually forearms) should be free of lesions



Causes of False Positive Results

1. Variations in injection technique e.g. depth or volume of injections.
2. Dermographic subjects

Procedure

1. 2ml venous blood taken from antecubital vein
2. Blood is allowed to undergo clotting at room temperature.
3. Serum is separated by centrifugation.
4. 0.05ml serum is injected intradermally into the volar aspect of forearm, avoiding the areas of wealing happened within the past 24 hours
5. Similar amount of normal saline is injected intradermally distance from saline injection site 3-5 cm at in the volar aspect of the same forearm.

6. Weal and flare responses are to be measured after 30 minutes. Redness of weal and flare reactions is difficult to perceive in pigmented skin types (e.g. Indian skin).

Criteria For Positive Response

A positive test is defined as a red serum induced weal response with a diameter of 1.5 mm or more than that of the saline induced response at 30 minutes.

Significance

Positive ASST denotes a subset of population who has an increased potential to develop urticaria due to endogenous causes than do patients without a positive test. The significance of a negative test remains unclear.

Source :

1. Hide M, Francis DM, Grattan CEH et al. Autoantibodies against the high affinity IgE receptors cause of histamine release in chronic urticaria. *N.Engl J Med* 1993; 328:1599-604.
2. Fiebiger E, Hammerschmid F, Stingl G et al. AntiFc RI autoantibodies in autoimmune-mediated disorders. Identification of a structure-function relationship. *J Clin Invest* 1998; 101:243-51.
3. Nimi N, Francis DM, Kermani F et al. Dermal mast cell activation by autoantibodies against the high affinity IgE receptor in chronic urticaria. *J Invest Dermatol* 1996; 106:1001-6
4. Sabroe RA, Grattan CEH, Francis DM et al. The autologous serum skin test: a screening test for autoantibodies in chronic idiopathic urticaria. *Br.J. Dermatol* 1999; 140:446-52
5. Grattan C.E.H. Autoimmune urticaria. *Immunol Allergy Clin N Am* 24 (2004) 163-81.

Recent Research : Contact Dermatitis & Urticaria

From Page 3

extensively in different parts of world including Korea. Rare occurrence of allergic reactions could be due to the chemical itself. However this Korean study found presence of impurities like PPD or some heavy metals like nickel, cobalt, chromium, lead and mercury could in different samples of henna. It was interesting to find the presence of PPD, nickel and cobalt were detected in 3, 11 and 4 samples among 15 henna samples.

4. PPD : Late Phase Reaction

Late reactions to the patch-test preparations para-phenylenediamine

and epoxy resin: a prospective multicentre investigation of the German Contact Dermatitis Research Group.

Hillen U, Jappe U, Frosch PJ, et al *Br J Dermatol.* 2006 Apr;154(4):665-70. Late phase reaction on or after day 7 has been reported after many allergens. However its reason is still elusive. Patch test induced sensitization is one of the theories. Authors report the trial result of a prospective multicentre investigation by the German Contact Dermatitis Research Group.

It was intended to examine the incidence and etiology of late phase reaction of Para-phenylenediamine (PPD-1% pet) and epoxy resins (ER-1% pet) among 1748 patients and nickel sulphate (5% pet.) among 812 patients. Surprisingly late reactions were not seen with nickel sulphate in any of the patients. A high percentage of patients produced elicited late reactions with PPD and most probable etiology was patch-test sensitization. The group made a drastic step based on the study report to stop this sensitization by de-listing the PPD 1%

Contd to Page 7 Column 2

Diagnostic Tests

Photopatch Test

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Introduction

Photopatch test is an useful tool to detect photo-allergic contact dermatitis.

Indication

Patients with eczematous eruption predominantly affecting light exposed sites and in whom a history of worsening following such exposure obtained.

Time Period

Patient has to come thrice to the physician. On the 1st day (0), 2nd day (after 24 hrs.) and 4th days (after 72 hrs.)

Method

1st day : Initially, a portion of the lower back portion of the patient is marked and exposed to UVA light at a dose of 5J/cm².

The test site, usually the upper back portion is selected, where the photo allergens, in two sets, are placed on both sides. The common photo allergens (Table 1) with control are applied by Finn chambers.

2nd day : The patch on the left side is removed and the patch test reading is taken.

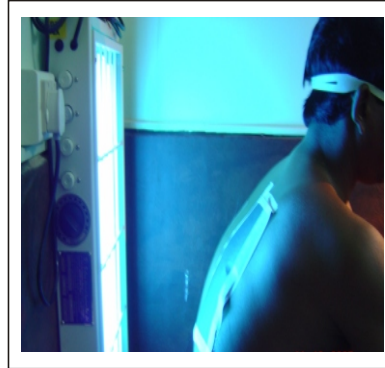
The right side (non irradiated side) is then covered with an opaque dark covering.

The left side is the exposed to UVA light at a dose of 4J/cm². (irradiated side). The patch on the right side is then removed.

Both the sides are marked accordingly with the marker pen.

3rd day : Reading on both the sides are noted. Offending allergens causing photocontact dermatitis or allergic contact dermatitis are identified.

Post-photo patch test counselling is done and the patients are then given



instructions to avoid individual allergens accordingly.

Instruction

Same as in patch test.

Contraindications

Photopatch test is otherwise a very safe test. The test is not done in patients suffering of SLE.

Source :

1. British photodermatology group : Workshop Report Photopatch Testing methods and indication, Brit J Dermatol, 1997; 136: 371-6.

Table 1. Common photoallergens

A. Sunscreen chemical	
1.	Para-aminobenzoic Acid (PABA)
2.	Padimate O & A
3.	Persol MCX
4.	Oxybenzone
5.	Parsol 1789
6.	Benzophenone
B. Fragrance Ingredients	
1.	Musk ambrette
2.	6-Methyl coumarin
3.	Sandalwood oil
C. Antibacterial agents	
1.	Buclosamide
2.	Hexachlorophene
3.	Halogenated salicylanilides
D. Miscellaneous compounds	
1.	Patients own product
2.	Benzocaine
3.	Chlorhexidine
4.	Chlorpromazine
5.	Diphenhydramine
6.	Hydrocortisone
7.	Promethazine
8.	Thiourea
9.	Paraphenyldiamine
E. Plants	
1.	<i>Parthenium hystorophorus</i>

Education in the technique of patch testing is as essential to physicians in training as the learning of most surgical procedures.

Fisher, 1986

Management Strategy of Plaque Psoriasis

From Page 2

achieve adequate remission to both BB-UVB and NB-UVB phototherapy. If PUVA or phototherapy fails to achieve satisfactory result, low dose acitretin (10-25 mg. daily) can be added to obtain improved response to UVB and to PUVA. In patients in whom UVB phototherapy and PUVA are contraindicated, methotrexate alone or combined with other treatments or Cyclosporine is highly effective option. However, a close monitoring of the toxic side effects of both these agents is mandatory. (Table 3)

Biologics : Recent strategic position in management

In recent years, an extensive research on pathogenesis-based treatment of psoriasis has developed producing a number of agents popularly named as 'Biologics'. Alefacept (antibody against T-lymphocyte surface molecule), Efalizumab (antibody against adhesion molecule), Infliximab and Etanercept (Anti-TNF alpha agents) are the agents showing their efficacy in prospective, randomized, double-blind, controlled t's

studies and are recommended in the management. However, the prohibitive cost may limit their use and the long-term safety of these agents is not known. Presently, expert's opinion is divided distinctly in two groups. Some are in opinion to use biologics as a first line therapy where the disease is too extensive to manage by topical agents, while others, because of high cost, want to try biologics only after trial of phototherapy or other systemic agents.

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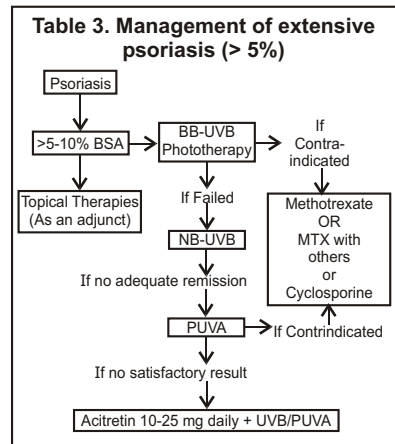
Management Strategy of Plaque Psoriasis

From Page 6

Other Strategic Concepts

Rotational Therapy

The primary aim of the rotational therapy is to minimize the cumulative toxicity of each of individual form of therapy. Thus, for example, the risk of carcinogenesis of PUVA, hepatic fibrosis and need for liver biopsy with methotrexate and the musculoskeletal toxicity of retinoids can be minimized if these three forms of treatment are used



in rotation. The different treatments used in rotational therapy are determined by patient response. Since biologics are not found to be associated with major organ toxicity, their long-term use obviates the need for rotation.

Combination Therapy

The goal of combination therapy is to achieve enhanced clinical response and to reduce the side effects of each

Table 4. Contraindicated Combinations in Psoriasis

Combinations	Effect
Acitretin + Cyclosporine	Accumu of Cyclosporine
Cyclosporine + UVB/PUVA	↑ PUVA Induced Cutaneous Malignancy
Methotrexate + UVB/PUVA	↑ PUVA Induced Cutaneous Malignancy
Tar + PUVA	↑ Phototoxic responses

agent often by reducing the individual dose in comparison to the treatment by them separately as monotherapy. Various topical agents are combined among themselves (e.g. calcipotriol + superpotent TCS) or with phototherapy/ photochemotherapy (e.g. calcipotriol + PUVA, tar + phototherapy) or systemic therapies (calcipotriol + acitretin). Phototherapy/ photochemotherapy and retinoids (UVB + retinoids; PUVA + retinoids) are also used effectively. Itnt

is also important to know the 'contraindicated' combinations, as the risk of toxicity or side-effects of one agent is increased in presence of other (e.g. accumulation of cyclosporine and thus its increased toxicity can be seen when acitretin is combined) (Table 4).

Sequential Therapy

The aim is to clear psoriasis by potent agent initially and then to maintain remission by safer, less effective agents. As for an example, cyclosporine is used initially to clear psoriasis and retinoids + UVB is used subsequently for maintenance.

Source :

1. Christophers E, Mroweitz U. Psoriasis. In: Freedberg IM, Eisen AZ, Wolff K, et al, eds. Fitzpatrick's Dermatology in General Medicine. vol.1, 6th ed. New York: McGraw Hill, 2003: 407-27.
2. Lebwohl MG, Kerkhof PCM. Psoriasis. In: Lebwohl MG, Heymann WR, Berth-Jones J, Coulson I, eds. Treatment of Skin Disease- Comprehensive Therapeutic Strategies. 2nd ed. Philadelphia, USA: Mosby, 2006: 550-59.
3. Arndt KA, Bowers KE. Manual of Dermatologic Therapeutics with Essentials of Diagnosis. 6th ed. Philadelphia, USA: Lippincott Williams & Wilkins, 2002: 173-184.
4. Kerkhof PCM. Psoriasis. In: Bologna JL, Jorizzo JL, Rapini RP, et al, eds. Dermatology, vol.1, 1st ed. London : Mosby, 2003:125-149.

Recent Research : Contact Dermatitis & Urticaria

From Page 5

pet from the standard patch test series.

5. Auto-immune Urticaria : Search for Biochemical Markers

a. Szegedi A, Irinyi B, Gal M et al, Significant correlation between the CD63 assay and the histamine release assay in chronic urticaria. *Brit J Dermat* 2006; 55(1):67-75

Autoimmune urticaria is a newly coined subset of chronic urticaria (CU) based on the development of wheal and flare reaction in simple clinical test called autologous serum skin test (ASST). In search of a reliable and confirmatory test authors developed a new method of CD63 expression assay by flow cytometry as a marker of serum-induced basophil activation and

compared its efficacy for this purpose with HR assay by ELISA, serum levels of soluble CD40 ligand (sCD40L) and also the ASST. Basophils were obtained from atopic (DA) and a nonatopic (DNA) donor and serum taken from of 72 patients with CU. Twenty normal people and 26 patients with systemic autoimmune diseases served as control. CD63 expression was found unregulated in 57% of DA and 28% of DNA. Authors concluded mentioning that the CD63 expression assay was a reliable functional test in the diagnosis of ACU especially when basophils were obtained from suitable donors.

b. Chronic urticaria sera increase basophil CD203c expression Yasnowsky KM,

Dreskin SC, Efaw B, J Allergy Clin Immunol. 2006; 117(6):1430-4.

CD203c expression on basophil by the sera of patients with CU was assessed by flow cytometry and the relationship examined between the size of ASST, a clinical parameter and level of CD203c expression. Results showed that patients with CU and positive ASST had a significant upregulation of CD203c. There was also a significant clinicomolecular correlation between the size of ASST and level of CD203c expression.

Invitation

Articles are invited from dermatologists and other physicians regarding various facets of allergic and immunologic skin diseases for contribution in this bulletin.

– Editorial Team

Case Report

Paraphenylenediamine (PPD) Simultaneously from Industrial and Personal Sources Causing Allergic Contact Dermatitis of Hands

Dr Sanjib Chowdhury
IAISD, Kolkata

Introduction

Many cases of hand dermatitis originate from contact dermatitis among which irritant contact dermatitis outnumbers allergic contact dermatitis. Allergic patch test remains the only sure diagnostic tool to differentiate between the two forms of contact dermatitis. Results of patch test should be properly interpreted in the light of information obtained from the pre- and post-patch test counselling of the patient. Various sources of the identified allergens should be highlighted to the patients for strict avoidance in future.

Case Report

A male patient aged 47 years reported at Institute of Allergic and Immunologic Skin Institute, Kolkata with itchy scaly rash over the finger tips (thumb, index, ring) of both hands as well as over palmar aspect. The lesions had developed about a year back. Painful crevices were also present over the finger tips. He was employed at the 'ORE & coat berth Division at Port where exposure to hydraulic oil is quite common. He had history of using hair dye for last 2 year.

On examination he had papulosquamous plaque lesions over the index finger, ring finger and thumb of both hands. A few discrete erythematous papular eruptions appeared over the palm. Other areas of body were spared. Routine investigations were within normal limits, including scraping for fungus which was negative. Systemic examination did not reveal anything.

Previous topical steroid applications, as advised by previous doctors, was stopped for 7 days and then patch test using Indian standard battery of allergens as designed by CODFI (Contact & Occupational Dermatoses Forum of India) and the brand of dye used by patient. Readings were taken after 24 hours and 72 hours respectively. The allergens which were

found positive were : (1) Paraphenylenediamine (PPD) 1+ (2) Personal hair dye brand used by patient 3+. The patient was advised to hold the personal hair dye strictly and managed by potent topical steroid, antihistamine



and using gloves during works. Even after 1 month the patient did not get appreciable improvement. Hence he was advised to strictly avoid his hydraulic oil exposure. After one month the patient get considerable improvement.

Discussion

Apart from hair dye, the allergen paraphenylenediamine is present in grease, mechanical oil, permanent hair, dye, rubber, plastic. He was advised to avoid the above allergens strictly, which ultimately solved the problem. Thus avoidance of all this sources of identified allergens became essential for proper management of the dermatitis. Hence in the pre- and post-test counselling of the patients about all this possible sources of allergens both industrial exposure as well as personal sources should be explored for out and out avoidance.

Conclusion

Paraphenylenediamine (PPD), an important and common allergen causing allergic contact dermatitis can origin from personal usage source as well as industrial or occupational sources.

Source :

1. Ket NS, Leok GC. The principles and practice of contact and Occupational Dermatology in the Asia-Pacific Region. New Jersey : World Scientific 2001; 47-70.
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IAISD NEWS 2005-2006

Academic Activities

● Celebration of World Allergy Day by IAISD on 8th July 2005 at Rotary Sadan, Kolkata involving dermatologists, chest specialist and allergologists of Kolkata and surroundings.

a) Prof A K Bajaj, Ex-National President of IADVL and eminent derma-allergologist of International repute delivered 'Mr Sukumar Ghosh Memorial Lecture' on chronic urticaria.

b) Eminent dermatologists of the State took part in a panel discussion on 'Derma-Allergy'.

c) Consultant dermatologists from IAISD presented an audio-visual symposium on 'Contact dermatitis'

● Inter-disciplinary and intra-disciplinary in-house seminars :

a) Psoriatic arthritis (among rheumatologists, orthopedicians and dermatologists).

b) Chicken pox (among physicians, pediatricians and dermatologists).

c) Epidemiology & statistics of skin diseases (among epidemiologists and dermatologists)

d) Photodermatology (among dermatologists)

e) Contact dermatitis (among dermatologists)

f) Urticaria (among dermatologists)

○ Scientific papers/Invited lectures from IAISD accepted in the following conferences :

a) Asian Congress of Dermatology, 2005, Kuala Lumpur (Guest Lecture)

b) Asia-Pacific Environment & Occupation Dermatology Symposium 2005, Manila

c) National Seminar on Pollution in Urban Industrial Environment Kolkata 2005 (Faculty)

d) State conferences of Dermatology, Goldcon, Cuticon 2005, Kolkata (awarded prize)

e) National conference of AIDS & STD, 2005 Kolkata.

f) Zonal conference of Dermatology, Dermozone 2005, Guahati.

g) National conference of Dermatology, Dermacon 2006, Hyderabad (5 papers).

h) Fungal Diseases : An Update – A Multi disciplinary seminar organised by Institute of Pulmocare and Research, Kolkata, 2006.

Social Activities

○ Free skin check up camp at Industrial area of Budge Budge (in collaboration with the Budge Budge College)

○ Free School health camp (at Kamala Chatterjee School for Girls, Kolkata)

○ Free Skin Camp at IAISD premises regularly

○ Social awareness programme :

● Invitation for essay competition among students from different schools of Kolkata on 'environmental pollution'. Three students awarded best prizes as recommended by a panel of judges comprising of eminent writer, critic and teacher.

● Participating in 'Ananda Mela' arranged by Presidency Girls' Educational Society for display of poster negating myth on skin diseases.

● Publication of books in 'Bengali' on 'Contact dermatitis' and vitiliginous for patients.