A STUDY OF 300 CASES OF ALLERGIC CONTACT DERMATITIS

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CERTIFICATE

Certified that this dissertation entitled ***"A STUDY OF 300 CASES OF ALLERGIC CONTACT DERMATITIS"*** is a bonafide work done by **DR. S. THILAK,** Post Graduate Student of the department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 600 003, during the academic year 2005 – 2008. This work has not previously formed the basis for the award of any degree.

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CONTENTS

ALLERGIC CONTACT DERMATITIS

Allergic Contact Dermatitis is a very common type of skin disorder seen among patients attending dermatology clinics. Allergic contact dermatitis occurs when the skin comes in contact with an allergen that the skin is sensitive or allergic to. Allergic contact dermatitis occurs more commonly in adults.

In other words Allergic contact dermatitis is caused by the body's reaction to something that directly contacts the skin. Many different substances can cause allergic contact dermatitis, which are called 'allergens'. like fragrances, small molecule preservatives, etc. Usually these substances cause no trouble for most people, and may not even be noticed the first time the person is exposed. But once the skin becomes sensitive or allergic to the substance, any exposure will produce a rash. Allergic contact dermatitis is the inflammation of the skin manifested by varying degrees of erythema, edema, and vesiculation. It is a delayed type of induced sensitivity (allergy) resulting from cutaneous contact with a specific allergen to which the patient has developed a specific sensitivity. Diagnosis of Allergic contact dermatitis is done by doing Patch tests.

REVIEW OF LITERATURE

ALLERGIC CONTACT DERMATITIS

HISTORY

The term "ALLERGIE" was first coined by the scientist Von Pirquet in 1906[1]. The word was derived from the Greek 'Allos' and 'ergon' meaning other or different work.

Allergic sensitization of skin was first proved by Bloch and Steiner-Woerlich's experimentally using Primula extract on humans[2]. Landsteiner and Jacobs showed that a simple chemical capable of causing contact dermatitis must be combined with proteins in order to sensitise[3]. Haxthausen's transplantation experiments finally proved that allergy was due to a factor supplied to the skin from within[4].

Josef Jadassohn is considered to be the founder of the Patch Testing technique in 1895 while working at Breslau university[5].

Bruno Bloch, a dermatological pioneer, expanded and enhanced Jadassohn's technique while working in Basel in 1911[5]. He introduced the concepts of standard series of allergens[6], cross sensitization and systemic allergic contact dermatitis[7].

Paul Bonnevie, Professor of Occupational medicine in Copenhagen, expanded the standard series to what could be considered the prototype of our present day series.

Allergic contact dermatitis is due to delayed type of hypersensitivity

reaction or cell meditated immunity. Here the induction of sensitivity is the primary event which has to take place before clinical expression of dermatitis can occur.

PATHOGENESIS

Two main processes occur

(i) SENSITISATION (induction or Afferent limb)

(ii) ELLICITATION (Efferent limb)[8]

SENSITIZATION

Main events occurring are

I BINDING OF ALLERGEN TO SKIN COMPONENTS:

An allergen penetrating the skin associates with major histocompatability complex (MHC) class II molecules either directly or via antigen-peptide binding sites in the groove of MHC class II molecule[9]. Epicutaneously applied antigen associates with these antigen presenting cells within 6 hrs[10]. This requires co-stimulatory factors such as Interleukin - 1β (IL-1β), Tumor Necrosis factor – α (TNF-α) and Granulocyte – macrophage colony – stimulating factor (GM-CSF)[11].

II RECOGNITION OF 'COMPLETE' OR CONJUGATED ANTIGEN:

This requires intact regional lymph nodes[12]. The Allergen carrying

Langerhan's cells travel via the afferent lymphatics to the paracortical areas of regional lymph nodes, where they become apposed to 'T' lymphocytes. This binding is assisted by physical factors and specialist Cellular Adhesion Molecules (CAMs). With Recognition of the Antigen, many mediators or cytokines are released such as IL-1, IL-2[13].

III PROLIFERATION AND DISSEMINATION OF SENSITIZED 'T' LYMPHOCYTES:

The cytokines cause blast formation in the lymph node and the proliferation of Antigen specific CD_8 and CD_4 cells[14]. The 'T' cells disseminate via the efferent lymphatics throughout the body and interact with Langerhan's cells and residual antigen in the skin[15]. Contact hypersensitivity is mediated through a subset of 'T' cells that express Cutaneous Lymphocyte Associated antigen (CLA). CLA-positive lymphocytes express CCR 10 – the receptor for the chemokine CCL 27 (produced by basal keratinocytes) which helps in localization[16]. Cytotoxic 'T' cells induce keratinocyte death through release of Fas ligand and Perforin mediated pathways. Allergen specific 'T' cells persist at site of original contact for some months[17].

ELLICITATION

If a sensitized person is re-exposed to a specific allergen in specific concentration, the clinical reaction subsequently develops much more quickly, usually within 24-48 hrs. Antigen presenting Langerhan cells pass to regional

lymph nodes and bind specific 'T' lymphocytes and also specific 'T' lymphocytes in epidermis[18]. IL1 secreting keratino cytes may acquire Ia/HLA-DR status and also present antigen to the specific 'T' lymphocytes, augmenting the cascade[19]. A delayed reaction (Late reaction) describes a delayed elicitation response following antigenic challenge in persons who are already sensitized.

PREDISPOSING FACTORS

I INDIVIDUAL

A. CONSTITUTION:

Sensitization presupposes individual susceptibility. This does not seem to follow Mendelian inheritance[20]. Capacity for sensitization varies from person to person. But certain individuals are more prone to developing sensitivity to a particular substance eg. Nickel[21].

B. SEX:

Women have stronger cell mediated immunity responses than men. Reason for female preponderance in clinical patch test studies is due to large number of metal sensitive females and greater exposure to fragrances, cosmetics and hair dyes[22].

C. HORMONES:

Response to DNCB is enhanced in women taking oral contraceptives. Pregnancy, menstrual cycle, use of gestagens may influence the results of patch tests[23].

D. RACE:

Racial differences exist but it is a reflection of exposure rather than predisposition[24].

E. AGE:

Age has little influence on capacity for sensitization. Number of positive patch test reaction tends to increase with age due to accumulation of allergies acquired over a lifetime[25]. Inflammatory response is diminished. Nickel, Fragrance, Thiomerosal, Medicaments, Rubber chemicals, Chromate are common allergens in children[26].

F. MEDICATION:

Antihistamines and Sodium chromoglicate have little effect on skin-test reactivity. Prednisolone more than 15 mg/day and potent topical steroids suppress allergic contact reactions[27]. Immunomodulators, UVB, PUVA therapy also reduce contact allergy reactions[28].

G. COINCIDENTAL DISEASES:

Patients with acute or debilitating diseases such as cancer, Hodgkin's disease, Mycosis fungoides and those with impaired T-lymphocyte function have impaired capacity for contact sensitisation.

H. LOCAL:

Pre-existing Irritant contact dermatitis affects barrier function of skin and increases allergen absorption. Nickel, chromate, cobalt sensitivity are increased with hand eczema[29]. Longer the duration of eczema, the greater the

chance of sensitization. No certain conclusion can be made about the relative risk of contact sensitization in atopic patients.

II ENVIRONMENTAL

A. CLIMATE:

Climate, by virtue of varying UV exposure, heat and relative humidity, may play a part in liability to contact allergy. UVB exposure has been shown to diminish the skin's immune response to contact allergens[30]. Chapping of skin during winter predisposes to irritant contact dermatitis and false positive patch test reactions.

B. FLORA AND FAUNA:

Allergenicity of Primula obconica vary with light and season. Allergenic plants of Compositae family are destroyed by cold and frosty weather and return during warmer months. Distribution of allergenic plant material will be facilitated by dry and windy climates. Fauna are not a major seasonal cause of contact allergy.

C. SOCIOECONOMIC AND CULTURAL:

Pattern of perfume, cosmetic and jewellery use and exposure might vary according to social class. Hair dyes are more commonly used by men in India[31]. Indian women become sensitized to dyes and adhesives in kumkum and bindi[32].

III CHEMICAL

Skin cells are composed of molecules that contain nucleophilic atoms whereas allergens contain electrophilic atoms. Interaction between these two result in strong covalent bonding to form a "Complete antigen"[33].

PATHOLOGY

Biopsies are of limited help in contact dermatitis. Most types of eczema shows identical pathological changes and allergic and primary irritant contact dermatitis cannot be distinguished with certainty[34]. Spongiosis is more marked than in irritant contact dermatitis. The role of Langerhan's ($CD1a^+$) cells in induction and ellicitation of allergic contact dermatitis is well established. Epidermal necrosis, acantholysis and pustulation are common with irritant contact dermatitis. For most episodes of ACD, the end result of the exquisitely orchestrated interplay of cytokines and adhesion molecules is the entrance into the skin of T-helper–1 cells secreting IL-2 and IFN-γ. The pathological findings of the non eczematous variants of ACD are nearly identical to the diseases they simulate.

CLINICAL FEATURES

The primary signs in contact dermatitis are erythema, swelling, papules and papulovesicles. In acute cases, vesicles and blisters can be seen. Dominant symptom is itching. If contact dermatitis persists due to repeated exposure to the allergen, skin becomes dry, scaly and thicker. Lichenification

and fissuring may develop later. The distribution of dermatitis is of diagnostic importance but its morphology is usually of no help.

I PRIMARY PATTERNS

Anatomical patterns of dermatitis often suggest a specific cause.

A. HANDS AND ARMS:

Hand eczema is multifactorial[35]. No pattern of hand eczema is characteristic of a particular etiology. Housewife's dermatitis and most occupational dermatitis remain confined to the hands. Rubber gloves may induce a clear pattern of dermatitis over the sites where they are worn. Chromate in cement can induce a palmar pattern of allergic dermatitis and a discoid pattern of allergy. Streaky dermatitis is mainly caused by plants.

B. FACE:

Fragrances, preservatives, skin care products and cosmetics are commonly implicated[36].

C. EYELIDS:

Allergens affecting face can cause eyelid dermatitis. Primula obconica and poison ivy can cause acute edema. Hair dye, eye drops and ointments, contact lens solutions are commonly implicated[37].

D. LIPS OR PERIORAL AREA:

Lipsticks, toothpaste, flavours, dentures and fillings are usually implicated.

E. EARS:

Medicaments, hairpins, hearing aids, spectacle frame, dyes, earrings are common causes.

F. SCALP:

Hair dyes are the commonest sensitizers. Bleaches, hair styling products, shampoos are other sensitizers.

G. NECK:

Textiles and necklaces can cause a collar like dermatitis. Airborne allergens, photo-sensitizers and perfumes are other causes.

H. AXILLAE:

Fragrances and antiseptics are common allergens.

I. TRUNK:

Clothing dermatitis involves trunk commonly. Photoallergic substances, detergents, fabric conditioners, perfumes are other causes.

J. ANOGENITAL:

Common site for medicament sensitization. Most toilet tissues, nylon, soaps, sprays are other causes[38].

K. THIGHS:

Nickel, rubber, textiles are common. Objects kept in pockets may provoke patch of dermatitis.

I. LOWER LEGS:

Medicaments predominate. Rubber, colophony, nylon are other sensitizers.

M. FEET:

Shoes, stockings, antiseptics are common allergens.

N. EXPOSED SITES:

Dust, sprays, pollens, volatile substances, plants, natural resins, woods are common allergens. Parthenium dermatitis is common in India[39].

O. MUCOUS MEMBRANES:

It is often secondary to skin sensitization. Symptoms are soreness and burning and itching is uncommon. Food additives, dentures, toothpastes, medicaments are common sensitizers.

II SECONDARY PATTERNS

Contact dermatitis may start at one site, but commonly other sites are subsequently involved. Heavily contaminated areas, or those that were exposed last, tend to be the ones to react first, other sites flaring later[40]. Regions close to the primary site of allergic contact dermatitis are easily contaminated by the allergen. Dissemination to distant regions has been termed an “id like” spread. The pattern of spread is largely determined by the primary site. Dermatitis of the hands commonly spreads to arms and face. Dermatitis of feet commonly spreads to legs and hands. Dissemination of leg

eczema commonly involves arms and shoulders in a patchy fashion, before becoming generalised. Severe Nickel allergy may induce extensive patchy eczema. Patch tests should be delayed until the acute eruption has settled.

SYSTEMICALLY REACTIVATED CONTACT DERMATITIS

Systemically reactivated allergic contact dermatitis, where ingestion or other systemic exposure to a contact allergen takes place in an already sensitized person, may result in a number of different patterns of skin eruption. The threshold of reaction varies in each individual case and depends on the dose given and the level of sensitivity. Reactions may occur not only after ingestion of the primary allergen but also after ingestion of secondary allergens[41]. Most frequent types of reaction are focal flares of previous patch tests and sites of previous dermatitis, vesicular hand eczema or wide spread eczema. All contact allergens can cause systemic reactions provided the patient has a sufficient degree of pre-existing sensitivity and the dose administered is sufficiently large[42]. Systemic contact dermatitis from medicaments is common. Neomycin, Quinolines, local anaesthetics, ethylenediamine and corticosteroids are the common culprits. Flares of vesicular hand eczema is caused by Nickel commonly[43], balsam of Peru, garlic, food colours, preservatives and antioxidants.

PHOTOALLERGIC CONTACT DERMATITIS

Certain substances are transformed into photosensitizers after irradiation with UV or short-wave visible radiation (280-600 nm). The wavelength required is usually the same as the absorption spectrum of the substance[44]. Photoallergic reactions are based on immunological mechanisms same as that of allergic contact dermatitis.

ALLERGENS

Common photoallergens are

1. UV filters – PABA, cinnamates, benzophenones
2. Perfumes – musk ambrette
3. Halogenated salicylanilides
4. Topical NSAIDs – ketoprofen
5. Phenothiazines
6. Sulphonamides
7. Others – Eosin, Quinines, Thiourea

CLINICAL FEATURES

They show the same spectrum of features seen with allergic contact dermatitis. The dermatitis is localised on the exposed areas of the skin. Area below chin is spared. The most distinctive sign is the exempt 'Wilkinson's triangle' behind earlobe[45]. Photoallergic reactions may progress to produce a light sensitivity that may persist a long time after the elimination of the sensitizer called as “Persistent light reaction”[46]. Combined airborne and photo aggrevated contact allergy is seen with Compositae. Chronic actinic

dermatitis may be associated with contact allergy to Compositae. Investigation of photo allergy is by Photopatch tests.

NON ECZEMATOUS RESPONSES:

Sl No.	TYPE	ALLERGENS
1.	Contact Urticaria	Natural rubber latex. Foodstuffs(Fruits, vegetables, potato, fish, eggs), silk, saliva, sweat, semen, food proteins
2.	Erythema multiforme like reactions	Quinones, primula, poison ivy, tree tea oil, rubber, clothing, hair dyes, topical medicaments
3.	PURPURIC reactions	Textile dyes, resins, hair dyes, balsam of Peru, rubber
4.	Lichenoid reactions	P-phenylenediamine, metals, tattoo pigments
5.	Pigmented dermatitis	Fragments, pigments, dyes, fungicides
6.	Depigmented lesions	P-phenylenediamine, perfumes, tattoos, primula, chloroxylenol, methacrylates
7.	Granulomatous lesions	Zirconium in deodarants, aluminium-absorbed vaccines, tattoo pigments
8.	Onycholysis	Hair dressing chemicals, nail varnish
9.	Systemic	Sulfones in lauryl ethyl sulphate

PATCH TESTING

The diagnosis of allergic contact dermatitis is made by patch testing and of photo allergic contact dermatitis by photopatch testing. Patch testing relies on the observation that primed antigen-specific 'T' lymphocytes will be present throughout the body.

INDICATIONS[47]

1. Eczematous disorders where contact allergy is suspected or to be excluded.
2. Eczematous disorders failing to respond to treatment as expected.
3. Chronic hand and foot eczema.
4. Varicose eczema.
5. Persistent or intermittent eczema of the face, eyelids, ears and perineum.

METHODS:

The amount of allergen is defined by its concentration in the vehicle and amount applied. By testing the same allergens in parallel, the technique has been confirmed to be generally reproducible. Chambers or discs are used to ensure occluded contact with the skin. The fixing tape should be non-occlusive, non-allergenic and non-irritant. Ideally patch testing should not be carried out in patients with active eczema as it reduces the threshold of activity and cause non specific reactions. The procedure should be delayed until the test site has been clear of eczema for atleast a fortnight. Patch test should not be performed following sunbathing, the patches should not be exposed to the sun or other sources of UV light[48]. Corticosteroids and other

immunosuppressive drugs should be stopped before patch testing as they may reduce positive patch tests in sensitized subjects.

TEST MATERIALS:

The commonest system used to apply allergens is the Finn chamber on Scanpor tape. The chambers consist of small occlusive Aluminium discs. They are mounted on non-occlusive tape. Other systems consist of square plastic chambers (Vanderbend chambers), oval plastic chambers (Epicheck) and older AL test system (a filter paper mounted on aluminized paper). Of late, there is a new prepackaged ready to use patch-test system called TRUE (Thin layer Rapid Use Epicutaneous test), based on dispersion of allergen in a hydrophilic polymer.

VEHICLES:

The test substance should be soluble in the vehicle. Ideal vehicle used is petrolatum. Uniform dispersion and particle size are important. Irritant solvents such as chloroform and benzene must not be employed. Petrolatum is generally more reliable and has the advantage of being occlusive. Allergic reactions to petrolatum are rare. In hot climates, petrolatum may not be ideal as it melts too quickly. A modified Plastibase has been devised for Indian contact dermatitis group.

PATCH TEST CONCENTRATIONS:

The concentrations used for patch testing are usually much higher than those encountered during development of dermatitis. No chemical or substance should be applied to the skin until full details of its composition and potential irritancy or toxicity are known. If doubt about the optimum level of testing, it is advisable to start at a low and increase concentration gradually.

PATCH TEST DOSE:

If petrolatum is used as the vehicle and disposable syringes are the containers, a length of 5 mm of test substance will suffice. If vehicle is a fluid, a digital pipette should be used to deliver 15 μL to a filter paper in the chamber.

STORAGE OF ALLERGENS:

Shelf life is prolonged if test substances are stored in dark in a refrigerator at 4ºc. Many substances are unstable if exposed to light. Storage in small jars has drawbacks of oxidation, drying and evaporation of volatile test substances. Rubber pipette caps can contaminate the solutions. Homogeneity of patch test allergens may be lost in hot climates.

TEST SITE:

Upper back is the site commonly preferred. Both allergic and irritant

reactions are most easily provoked on the upper back[49] and the site is least likely to be disturbed. Other sites used are

1. Lateral aspect of upper arm
2. Abdomen
3. Thighs

MARKING:

Test site must be marked with indelible ink or stratum corneum stains. The patient should be instructed not to bathe or shower for the duration of the tests, and to avoid exercise or other activity likely to dislodge the patches.

EXPOSURE TIME:

Well established allergens, are conventionally tested in such concentration that a 48 hr exposure under an occlusive patch will generally allow penetration of an amount sufficient to produce a reaction. The ideal regimen is a 48 hr application time, with readings taken 1 hr after removal and again 48 hrs later i.e. at 2 days and 4 days[50], preferably with the same observer performing each reading.

Immediately after removal of the patch tests, there may be erythema from the stripping action of the tape, especially in dermographic subjects, and this must be allowed to settle. Some reactions may take upto 1 hr to develop once the pressure of the strips has been released and the infiltration allowed to swell the dermis.

READINGS AND INTERPRETATIONS:

Recording of patch-test reactions is done according to the International Contact Dermatitis Research Group (ICDRG)

-	Negative
?-	Doubtful reaction, Faint erythema only
+	Weakly positive reaction. Palpable erythema, infiltration, possibly papules
++	Strong positive reaction. Erythema, infiltration, papules and vesicles
+++	Extreme positive reaction. Intense erythema and infiltration and coalescing vesicles
IR	Irritant reaction
NT	Not tested

Patch test results should be recorded objectively, and the interpretation of the results should be recorded separately. Once developed, the positive allergic reactions often persist for several days. The strength of the reaction depend on barrier function, the presence or absence of sweating, the atmospheric humidity, test material, technique and reactivity of the individual. The infiltration causes a thickening of the dermis, which is palpable and can be distinguished from surface changes in the epidermis.

Sl No.	**Allergic Reaction**	**Irritant Reaction**
1.	Infiltration present	No infiltration
2.	Itching present	No itching

3.	Erythematous	Deep redness or Brown hue
4.	Reaction may extend beyond margins of the patch	Sharp delineation corresponding to margins of patch test

RELEVANCE OF PATCH TESTS

A positive reaction to a patch test commonly proves the cause of dermatitis. Other reactions may relate to previous attacks of dermatitis. 75% of '++' and '+++' patch test reactions are of current or past relevance. The relevance of '+' reactions is less certain[51]. A person may react to a patch test but still tolerate contact with the allergen. In a patient with dermatitis, a positive patch test must never be disregarded. If found in a healthy person, it may indicate a future risk of allergic dermatitis from that particular allergen.

FALSE POSITIVE REACTIONS

Common causes of false positive patch test reactions are listed below.

1. Excessive concentration of allergen applied
2. Impure substances
3. Irritant vehicle
4. Uneven dispersion
5. Current or recent dermatitis in patch test site or at distant sites
6. Pressure effects
7. Adhesive tape reactions
8. Angry back reaction
9. Artefact

Secondary non-specific reactions close to genuine positive ones have been termed 'ANGRY BACK' or the 'Excited skin syndrome'.

FALSE NEGATIVE REACTIONS

Common causes of false negative reactions are

1. Insufficient concentration
2. Insufficient amount applied
3. Poor adhesion of patches
4. Inappropriate vehicle
5. Readings performed too early
6. Pretreatment of patch test site with topical corticosteroids
7. UV irradiation of patch test site
8. Systemic treatment with immunosuppressants

COMPOUND ALLERGY

This concept was first proposed by Calnan[52]. Compound allergy occurs when a positive allergic patch test reaction is seen to a finished product, but tests with the ingredients are negative. This was elegantly demonstrated in Hirudoid cream, where a new allergen was formed as a reaction product of two preservatives in the medicament. The additive effect of multiple weak sensitizers or the additive effect of weak allergens and irritants should be considered. Commonly the reaction to a finished product is irritant. It is also an expression of the hydrophilic-hydrophobic balance of its ingredients.

QUENCHING

A combination of chemicals can also lead to a quenching effect. This phenomenon is commonly seen in fragrance material - aldehydes.

SELECTION OF TEST SUBSTANCES

STANDARD SERIES

The principle of screening all patients with a series of allergens commonly encountered in their environment is now well established. Standard series of allergens vary from one country to another. In general, a substance should be included in the standard series battery if it gives positive reactions in more than 1% of those tested.

ADDITIONAL SERIES

Main patch test allergen producers now market extra series, which have to be adapted to local habits or occupational exposures. This is true for investigation of dermatitis in certain sites liable to medicament allergy or sensitization from compounds of shoes or clothing.

OTHER MATERIALS

Patients may bring a wide variety of materials of their own from home or work for testing. They must be thoroughly assessed and diluted appropriately before being tested.

PHOTOPATCH TESTING

Photopatch testing is done to investigate patients with eczematous eruptions predominantly affecting light-exposed sites and who have worsening of lesions following sun exposure.

A UV-A source is required. Dose is 5-10 J/cm^2. Application of the allergens is performed in an identical fashion to conventional patch tests, except that they must be applied in duplicate, one set is irradiated and the other (control) is not. Usually 2 sets of the tests are applied on either side of the midline on the upper back at the same level. The control site and the rest of the skin must be covered by a opaque material during irradiation. The common method followed is to apply the allergens on day 0. The patches are removed, results are read on day 2. On the same day allergens on one side are irradiated. Results are again read on day 4. If the same allergen provokes an equally strong reaction on both sides, it is an indication of contact allergy alone; if it is significantly strongly on the irradiated side, then combined allergy and photocontact allergy is occuring.

Photopatch Test Standard Series

1. Para amino benzoic acid (PABA)
2. Octyl dimethyl PABA
3. Octyl methoxycinnamate
4. Benzophenone 3
5. Butyl methoxydibenzoylmethane
6. Musk ambrette

COMPLICATIONS OF PATCH TESTS

1. Pruritis
2. Folliculitis
3. Leakage of materials on to clothing
4. Localized flare of dermatitis
5. Generalized flare of dermatitis
6. Irritant reactions
7. Active sensitization
8. Pigmentation or depigmentation
9. Scarring
10. Anaphylaxis

Quinones, phenols and acrylics can cause vitiligo[53]. Anaphylactic reactions are a potential risk when patch testing with some substances such as rubber latex and penicillin[54].

MULTIPLE PATCH TEST REACTIONS

Causes of multiple patch test reactions are

1. Non specific hyper reactivity
2. Multiple primary hypersensitivities
3. Cross reactions (true and false)

NON SPECIFIC HYPER REACTIVITY

The threshold at which false positive irritant reaction develops differs from individual to individual. During active dermatitis, uninvolved skin, even at distant body sites, exhibits increased susceptibility to irritant reactions. This

status eczematicus may lead to false positive patch test results. When this affects adjacent patch test sites, it is referred to as 'Excited skin' or 'Angry back'[55]. Rietschel has proposed that 'stochastic resonance' may be involved, that is signal amplification by immune mediated events.

MULTIPLE PRIMARY HYPERSENSITIVITIES

Multiple primary specific sensitivities to substances that are unrelated chemically are frequent among patients with contact dermatitis. Patients with a long history of dermatitis are those most likely to accumulate several primary sensitivities, because of the opportunities to encounter new allergens under conditions favourable for sensitization. This is commonly seen in leg ulcer patients[56] and with chronic actinic dermatitis. One sensitivity may predispose to the acquisition of another, and there may be a genetic or constitutional predisposition to acquire sensitivities. Sensitization is facilitated if allergen is applied on injured skin.

CROSS REACTIONS

It is a phenomenon where sensitization engendered by one compound, the primary allergen, extends to one or more other compounds, the secondary allergens as a result of structural similarity. The proposal is that the primary and secondary allergens are so closely related that sensitized 'T' cells are unable to distinguish between them. Enantio specificity or stereo specificity may lead to cross-reactivity with some isomers and not others.

SPOT TESTS

(i) DIMETHYL GLYOXIME TEST FOR NICKEL:

1% Dimethyl glyoxime and Ammonium hydroxide are stored in separate bottles. A few drops of each are put in separate clean white saucers, a cotton bud is then dipped in each of these and rubbed on the surface of test object. A pink colouration on the cotton bud denotes the presence of Nickel. This test is accurate to 10 ppm of nickel.

(ii) ACETYLACETONE METHOD FOR FORMALDEHYDE:

Reagent is prepared by dissolving 15 g of Ammonium acetate, 0.2 ml of acetylacetone and 0.3 ml of glacial acetic acid in 100 ml of distilled water. A sample of product to be tested is put in a disposable glass test tube and 2.5 ml of reagent is added. Mixture is shaken and stoppered and then placed in a water bath at 60^{o}c for 10 min. A yellow colour is produced in the presence of formaldehyde.

(iii) OTHERS

There are other spot tests for chromate and epoxy resin, but are not simple to perform.

ALLERGIC CONTACT DERMATITIS TO COMMON ALLERGENS

1. NICKEL:

Nickel is one of the most frequent source of sensitization. Commonly Nickel chloride ($Nicl_2$) and Nickel sulphate ($NiSo_4$) are readily soluble in water and sweat and have strong sensitizing properties. Nickel sensitivity is more frequent among women[59]. The usual prevalence of nickel sensitivity in a patch test clinic is 15-30%. The prevalence may be higher in some occupational groups such as hairdressers. The commonest source of metallic nickel are alloys and plated objects[60]. Sensitization is chiefly the result of frequent skin contact with corroded objects containing nickel. Jewellery and metal components of clothing are the usual sources of nickel in prolonged contact with skin. Other sources are coins, keys, scissors, knitting needles, thimbles, metallic tools and utensils. Systemic exposure may take place from the diet. Certain foods and plants contain much higher concentration of nickel.

Classic nickel allergy is seen at sites of contact with metal objects, most commonly ears from earrings, the wrists from watches and bracelets, the neck from necklaces, central back and upper chest from bra components, central abdomen from studs and zips in trousers, especially jeans and from

dorsa of feet from the shoe buckles. The eruption may be papular, nummular, diffuse or consist only of excoriated papules. Secondary spread of dermatitis to other areas can occur[61]. Nickel sensitive women do have a predilection for hand eczema[62]. A recurrent vesicular palmar dyshidrotic pattern of eczema has been related to dietary intake of nickel. Nickel sulphate 5% in petrolatum is used for patch tests. False negative tests are common with 2.5% nickel. Irritant false positive reactions can also occur.

2. CHROMIUM:

The metal itself is insoluble and seems to be non sensitizing unlike nickel. Hexavalent chromium (anion) is chromic acid or chromium trioxide and in chromates and dichromates of potassium, sodium and ammonium, is the commonest sensitizer. The less soluble lead chromate, barium chromate and zinc chromate are also allergenic. The prevalence of sensitivity is commoner in men. It is higher in clinics where men with occupational dermatitis predominate. The main source of hexavalent chromium is cement[63]. Other sources are antirust paints, plating salts, metal alloys, printing materials, cutting oils, foundry sand, matches, photographic chemicals, welding fumes, wood ashes, glazing enamels, cat gut, violin strings, textiles, tyre fitting solution and dental prosthesis. Trivalent agents are used as a tanning agent for leather. Acute weeping dermatitis is unusual and more commonly there is a dry insiduous eruption, which tends to fissure,

particularly the hands. Secondary lichenification is often a feature. There is frequently a concomitant irritant effect with wet cement. Contact with leather footwear, gloves, belts and other clothing and even hand bags and purses may produce dermatitis in areas of contact. Widespread eruptions can also occur.

Sensitivity is demonstrated by a closed patch test with potassium dichromate 0.5% in petrolatum. Chromate sensitivity tends to persist, and the prognosis of occupational dermatitis is poor as a result of its continuation and associated social and financial handicap. Chronicity and frequent relapses are the rule. Changing work to avoid contact with cement does not seem to improve the prognosis. Many chromate-sensitized cement workers develop hardening.

3. PLANTS:

Occupational dermatitis to plants is common in farmers, gardeners and florists. Patterns vary from country to country. Compositae, Anacardiaceae, Primulaceae, Alliaceae are the common plant families implicated in plant dermatitis. Of these, Compositae allergy is widely prevalent in India. More than 200 species of compositae have been reported to cause contact dermatitis. The allergens are Sesquiterpene lactones including dehydrocastus lactone, alantolactone, costunolide and parthenolide[64].

Six patterns of dermatitis are described which are generally worse during the summer months.

1. Pseudophytophotodermatitis
2. Atopic eczema like
3. Erythrodermatous exfoliative
4. Hand eczema
5. Localized dermatitis
6. Mucosal - Oral

Sesquiterpene lactone mix is used for patch testing consisting of alantolactone, costunolide, dehydrocostus lactone each 0.033%[65]. Seasonal compositae exposure may be difficult to avoid. Severe compositae allergy may necessitate changing occupation. Broad spectrum sunscreens are necessary for photosensitive patients.

4. RUBBER:

Latex is an aqueous dispersion of rubber. Natural latex is derived from sap of the tree Hevea brasiliensis. Rubber dermatitis is usually caused by accelerators, antioxidants and other chemicals used in its manufacture. Incidence is equal among both sexes. Common sources are rubber industries, revulcanization shops, rubber gloves, electric cords, tubes, masks, rubber bands, shoes, gloves, clothing, condoms, etc. Rubber sensitivity may be the primary cause of dermatitis or it may be superimposed on an existing dermatitis as with rubber gloves. Dermatitis from rubber gloves may be diffuse or localised. Shoe dermatitis commonly occurs on the dorsum of foot, soles or toes, usually with sparing of web spaces and instep. Sites of dermatitis often provides a clue to dermatitis. Most standard series for patch

testing contain rubber chemicals in the form of mixes. Some mixes are Mercapto mix, Thiuram mix, Black rubber mix, Carba mix, etc.

5. P-PHENYLENEDIAMINE AND RELATED DYES:

PPD is an aniline derivative whose main use is for dyeing hair. Oxidised PPD is not allergic. It has structural similarity to AZO dyes used for dyeing clothes. PPD is also seen in rubber antioxidants, photography developing solution, petrol, oils, greases and printing ink. PPD and related hair dye allergy can result in extremely severe skin reactions. Scalp is often relatively spared, but edema and weeping of the scalp margin, ears, eyes with extensive secondary eruptions can be seen. Lichen planus like presentations have been reported from India[66]. Hair dressers can also be sensitized by the dyeing process, resulting in hand dermatitis. Cross sensitivity to PPD has also been reported. Patch testing is done with a concentration of PPD in 1% petrolatum.

6. COSMETICS:

Contact dermatitis to ingredients of cosmetics and toiletries is common accounting for 10% of patients attending patch test clinics. Commonest allergens are fragrances and preservatives[67]. Also of importance are p-phenylenediamine, UV filters, nail varnish, lanolin and cocamidopropyl betadiene. More common presentation is erythematous scaling patches or a

more diffuse erythema. Eyelids, face and neck are the sites commonly involved in cosmetic allergy. 'Leave on' products are more likely to sensitize. Cheilitis can be seen with lipstick, lipsalve and toothpaste allergy. Hair cosmetic allergy may cause a scalp margin pattern. Nail varnish allergy is often ectopic. The allergen is usually tosylamide formaldehyde resin. Standard series for cosmetics testing includes fragrance mix, balsam of Peru, Parabens mix, Quarternium-15, formaldehyde, p-phenylenediamine and colophony. False negative and marginal irritant reactions are common when testing with cosmetics. If cosmetic allergy is still suspected despite negative patch tests, the possibility of photo-allergy should be considered, and if clinically indicated, photopatch tests should be undertaken.

7. CLOTHING:

Common allergens in clothing include textile dyes, formaldehyde, resins, rubber, chromate and nickel. Disperse dyes are the class of dye most likely to sensitize. Disperse dyes are used to colour artificial fibres such as polyester, acetate, acrylic and nylon. Both azo and anthraquinone dyes may cause dermatitis. The common sites of distribution of clothing dermatitis are areas of sweating and friction. The eruption typically starts in the axillae sparing the hairy part of the vault and forms a crescentic patch on the anterior chest wall sharply limited by the underwear. The dermatitis is located at the inner posterior thighs, popliteal fossae and lower legs may be involved when

trousers and pants are responsible garments. Some fabrics provoke a purpuric, sometimes lichenoid dermatitis in areas of contact as seen with uniforms. Cross sensitivity to substances is seen with textile dermatitis. Formaldehyde is a standard series allergen. Standard series screening with four textile disperse dye allergens is advocated.

8. SHOES:

The commoner identified allergens in shoes are rubber chemicals, chromate (in leather), nickel in buckles and p-tertiary-butylphenol formaldehyde resin (PTBPFR)[68]. Some other allergens are vegetable tanning agents, dyes, colophony, leather preservatives and polyurethane compounds. Prevalence of shoe allergy has ranged between 3 and 11% in patients attending patch test clinics. Rubber is the commonest allergen from various studies. Sweating causes allergens in shoes to leach out and migrate. Dermatitis from the upper commonly starts over the dorsal surface of big toes and spreads to dorsa of feet and other toes. Interdigital spaces are normally spared. Adhesives and rubber components may cause localised areas of dermatitis limited to toecap. Indian sandal dermatitis has a characteristic pattern, is often severe and affects mainly first toe web and adjacent toes and dorsum of foot[69]. Involvement of sole usually affects only the weight bearing areas and instep is spared. Hyperhidrosis is associated with shoe dermatitis. Shoe allergens found in standard series are dichromate, colophony, nickel, rubber accelerators and PTBPFR.

9. FRAGRANCES, BALSAMS, FLAVOURING AGENTS AND SPICES:

Perfumes are mixtures of essential oils and synthetic compounds. Balsams include balsam of Peru, balsam of Toru, balsam of spruce, gum benzoin and storax. Flavours may be natural or synthetic. Spices include nutmeg, mustard, cinnamon, cloves and oil of juniper. Fragrances are one of the commonest allergens. Analysis of common patterns of perfume dermatitis has shown to involve hands, face and neck in men and axillae in both sexes[70]. Substances used in the standard test series include balsam of Peru (25%) and fragrance mix.

10. OTHER ALLERGENS:

Sl. No	ALLERGEN	SUBSTANCES	FEATURES
1	Cobalt	Magnets, dentures, cements, paints, glass, ceramics, enamels, dyes, detergents, varnishes	Identical to nickel allergy, Cobalt chloride 1% in petrolatum for patch testing
2.	Palladium	Dental alloys, prosthesis, electrical components	Palladium chloride 1% in petrolatum for patch testing
3.	Gold	Jewellery, gold dental work	Gold, Sodium thiosulphate 0.5% in petrolatum for patch testing
4.	Mercury	Tooth filling, insecticides, fungicides,tattoos, medicaments	Mercury is tested at 0.5% in petrolatum

5.	Aluminium	Vaccines, eardrops, antiperspirants	As film chambers are aluminium, they are tested as such
6.	Neomycin, Clioquinol Benzocaine	Topical medicaments	Neomycin 20%, Clioquinol 3%, Benzocaine 5% petrolatum for testing
7.	Formaldehyde	Cosmetics, glues, hardness, tanning subs, fertilizers, polishes, preservatives, paints, printing chemicals, etc	Formaldehyde 1%. Aqueous is used for patch testing
8.	Quaternium-15	Cosmetic products, hand creams	Quaternium-15 1% in petrolatum for patch testing
9.	Parabens	Preservatives, cosmetics, foods, paste bandages, medicaments	Parabens mix 16% in petrolatum for patch testing
10.	Chloroxylenol	Disinfectant, powders, soaps and cleansers, coolant oils, ECG pastes	Chloroxylenol is patch tested at 1% in petrolatum
11.	Organic mercurials	Vaccine preservatives, eye drops and contact lens solutions	Phenylmercurie salts at 0.01% and Thiomersal at 0.1% in petrolatum for patch testing
12.	Lanolin	Medicaments, cosmetics, water, inks, adhesive tapes, bandages, cutting oil	Wood alcohols 30% in petrolatum
13.	Ethylenediamine dihydrochloride	Stabilizer in creams, lubricants, antifreeze, waxes, paints, dyes	Tested at 1% concentration in petrolatum
14.	Epoxy resins	Paints, varnishes, metals, fibreglass, rubber, ceramics, dental fillings, glues, plastics	Tested at 1% concentration in petrolatum
15.	Acrylic resins	Plastics, dentures, hearing aids, spectacle frames, nail cosmetics, bone cement, glues	Tested at 2% concentration in petrolatum

16.	Woods	Trees, wood, sawdusts	Tested at 10% concentration in petrolatum
17.	Colophony	Trees, wood, adhesives, glues, shoes, printing inks, varnishes, cosmetics, soaps	Colophony is tested at 20% concentration in petrolatum

PREVENTION

Principles of prevention can be related to two categories, individual and collective, and further divided into primary, secondary and tertiary. Primary prevention focusses on the induction of contact sensitization and control of exposure. Secondary prevention relates to elicitation and tertiary to measures for established and continuing dermatitis. Following steps can be taken

1. Allergen containment and replacement
2. Legal and regulatory measures
3. Corporate responsibility
4. Domestic precautions and hygiene
5. Barrier method for preventing contact
6. Proper education

PROGNOSIS

Prognosis of allergic contact dermatitis depends on its cause and feasibility of avoiding repeated or continued exposure of the causative allergen. The prognosis is poor for those allergic to nickel and chromate as a result of their ubiquity in the environment. There is a better outlook for those

allergic to materials that are easy to identify and avoid. As the skin integrity is compromised, there are enhanced opportunities for new sensitivities to develop. Once acquired, contact sensitivity tends to persist[71]. The degree of sensitivity may decline unless boosted by repeated exposure. There is no difference in prognosis between irritant and allergic dermatitis. Chronicity of contact dermatitis is attributed to the following factors.

1. Impaired barrier function of skin
2. In appropriate treatment
3. Ingestion of allergens
4. Secondary infection
5. Autosensitization
6. Stress
7. Constitutional factors
8. Inherent tendency of eczemas to become chronic
9. Atopy

TREATMENT

I AVOIDANCE ADVICE:

Once diagnosis of allergic contact dermatitis is made, possible sources of exposure to the causative allergen should be identified and avoidance advice given. Avoidance must be tailored to the individual. Some examples are plastic instead of rubber gloves, cosmetics and medicaments free of the identified allergen, clothing free of nickel-containing studs, zips, etc. Written information on the allergen sources may be helpful. In work related problems, appropriate protective clothing or change in handling technique may be

advised. Patients must be advised that the risk of relapse after further contact with the allergen persists throughout life.

II ACTIVE TREATMENT:

Topical corticosteroids will be required in most instances to control the disorder. In acute, severe, localised allergic contact dermatitis, a potent topical corticosteroid should be used. In more chronic or widespread contact allergies, the potency may need to be reduced.

1. Emollients and soap substitutes to be used.
2. Fissures of fingers, palms and soles can be covered with hypoallergenic tape.
3. For acute weeping forms, wet dressings with saline, Aluminium acetate or Silver nitrate can be given.
4. Topical Tacrolimus and Pimecrolimus can be tried.
5. Secondary infection is treated with antibiotics.
6. Antihistamine for pruritis.
7. In severe or widespread eruption, systemic steroids may be necessary[72].
8. Recalcitrant disabling cases require immunosuppresive therapy such as Azathioprine and Ciclosporin[73].

HYPOSENSITIZATION

Oral hyposensitization is not routinely recommended. The degree of hyposensitization achieved by oral doses of allergens is limited and transient. Some success has been claimed in India for hyposensitization against Parthenium hysterophorus.

AIMS OF STUDY

1. To study the incidence of various allergens in 300 patch test positive cases for that allergens.

2. To study the age incidence among patients of contact dermatitis to various allergens.

3. To study the sex incidence among patients of contact dermatitis to various allergens.

4. To study the association of Allergic contact dermatitis and Atopy.

5. To study the association between the duration of exposure of an antigen required for clinical manifestation of allergic contact dermatitis.

6. To find the incidence of occupational and non occupational causes of allergic contact dermatitis.

7. To study the association between Diabetes mellitus and allergic contact dermatitis.

8. To study the relationship between CD_4 cell counts and allergic contact dermatitis.

MATERIALS AND METHODS

STUDY

Prospective observational study

SAMPLE

300 cases of Allergic contact dermatitis who attended the dermatology OPD, from Govt. General Hospital, Chennai-3, Sep 2005 to Oct 2007, who were patch test positive were included in the study.

METHOD

A detailed history of the patients included in the study was taken. Duration and the type of occupation were noted for occupational cases of ACD. Morphology of the lesions and the sites of involvement were noted down. History, symptoms and signs suggestive of Atopy were noted down. Past history of the patient for similar complaints were asked for. History of any drug intake prior and after onset of lesions is noted down. All the patients were subjected to blood investigations namely routine hemogram and blood sugar. Based on the type and nature of exposure to a specific occupation or antigen, the patients were patch tested with the appropriate antigens. The patch test allergens used were approved by the Contact and Occupational Dermatoses Forum of India (CODFI). The following are included in that

Sl. No.	Allergen	Concentration in %
1.	Petrolatum	
2.	Potassium dichromate	0.5%
3.	Neomycin sulphate	20%
4.	Cobalt chloride – hexahydrate	1%
5.	Benzocaine	5%
6.	4-Phenylenediamine (PPD)	1%
7.	Parabens	15%
8.	Nickel sulphate – hexahydrate	5%
9.	Colophony	20%
10.	Gentamycin sulphate	20%
11.	Mercaptomix	2%
12.	Epoxy resin	1%

13.	Fragrance mix	8%
14.	Mercaptobenzothiazole	2%
15.	Nitrofurazone	1%
16.	4-Chloro-3-cresol	1%
17.	Wood alcohol	30%
18.	Balsam of Peru	25%
19.	Thiuram mix	1%
20.	Chinoform	3%
21.	Black rubber mix	0.6%
22.	P-tert-butylphenol formaldehyde resin	1%
23.	Formaldehyde	1.1%
24.	Polyethylene glycol	100%
25.	Plant antigens a) Parthenium hysterophorus b) Chrysanthemum c) Xanthium strumarium	

Patch testing was done as follows

1. Allergens were stored in a refrigerator at 4 degree C to 8 degree C. The allergens were taken out from the refrigerator 15 minutes before testing.
2. The patch test unit was marked with indelible ink - the names of the antigens to be tested.
3. The protective foil was removed and the patch test unit was placed on the table with the Aluminium chambers facing up.
4. 2-3 mm length of the allergens ointment from the syringe was put in the center of the Aluminium chambers.
5. Alcohol or aqueous based allergens were applied using a filter paper disc.
6. The upper back of the patient was gently cleaned with sterile gauze before application of antigens.
7. Allergens were applied on the patch test unit with first allergens in the top right hand corner and then downwards in the region of upper back.
8. The control is applied on the left side of vertebral column in parallel to the allergens on the right side.
9. Patches were removed after 48 hrs (2 days).
10. Reading was taken after 45-60 mm.
11. A second reading was taken on day 4 after application to confirm the presence of allergic reaction.

INSTRUCTIONS TO THE PATIENT

Following instructions were given to the patients.

1. Patch test must be left in place for two days and two nights.
2. Not to take bath or wash or wet the back during the period.
3. To avoid tight garments.
4. To avoid exercise or any other activity causing sweating.
5. To avoid friction or rubbing and lying on back.
6. To avoid scratching the patch test site.
7. To avoid exposure to sunlight/UV light.
8. To report immediately if there is severe itching or irritation.
9. To come after 48 hrs and 96 hrs for patch test reading.

PLANT ANTIGENS

As the plant antigens cause Phytophoto-dermatitis, a photopatch test is done. Two sets of antigens were applied one on either side of the midline in the upper back. The patients were instructed to come after 48 hrs. The plant antigen strip consists of non allergenic adhesive tape on which 4 paper discs have been fixed at appropriate distances. The content of each disc is indicated on the back of the strip. The polythene sheet protecting the antigen impregnated discs is separated. The antigen impregnated discs were wetted with a drop of distilled water and then applied. The strips are then removed and readings taken. Then one side is occluded and the other side is irradiated

with UVA in a dose of 5 J/cm^2 or sunlight. Then the patients were asked to come after 72 hrs or 96 hrs. The readings on both sides are then compared. Readings are then interpreted according to the guidelines devised by International Contact Dermatitis Research Group (ICDRG).

-	Negative
?-	Doubtful reaction, Faint erythema only
+	Weakly positive reaction. Palpable erythema, infiltration, possibly papules
++	Strong positive reaction. Erythema, infiltration, papules and vesicles
+++	Extreme positive reaction. Intense erythema and infiltration and coalescing vesicles
IR	Irritant reaction
NT	Not tested

RESULTS

300 patients with history of exposure to a specific substance and also who were patch test positive for the respective allergens were included in the study. Out of the total 300 cases, Allergic contact dermatitis to Cement tops the list with 130 cases (43.33%). Contact dermatitis to Nickel is the second common with 31 of a possible 300 cases (10.33%). Third common is Contact dermatitis to Plant antigens i.e. Phytophotodermatitis with a total of 27 cases (9%). Other substances are Paint – 20 cases (6.7%), Kumkum – 17 cases (5.7%), Rubber – 17 cases (5.7%), Leather – 14 cases (4.7%), Oil and Grease – 14 cases (4.7%), Turmeric – 11 cases (3.7%) and other miscellaneous substances – 19 cases (6.3%) (Table 1).

The commonest allergen to be tested positive was Potassium dichromate (positive in 167 cases), the second common being Nickel (positive in 31 cases). Formaldehyde was the third common allergen (positive in 16 cases). The next common were Cobalt chloride, Epoxy resin, Parabens, 4-Chloro 3-cresol, Black rubber mix (Table 2).

Among the miscellaneous cases 6 were cases of allergic contact dermatitis to hair dyes. Other cases were allergic contact dermatitis to Chrysanthemum, Neomycin, Polish, Lipstick, Tooth powder, Printing ink, Photographic film developing fluid and Eye ointment (Table 3).

Of the 300 cases, 214 patients were male (71.3%) and 86 patients were female (28.7%). Male to female ratio is 2.48:1. Female predominance was seen in allergic contact dermatitis to Nickel, Kumkum and Turmeric (Table 4).

Most of the patients fall into the age category between 41 and 50 years of age (94 cases – 31.33%). Second most common age category was 31 to 40 years of age (65 cases – 21.67%). Third most common age group was between 51 to 60 years (59 cases – 19.67%). The youngest patient in the study was 13 years of age and the oldest was 65 years of age (Table 5).

The results were read according to the ICDRG scoring system. Out of the 300 cases, 182 cases were 1+ (60.67%), 102 cases were 2+ (34%) and 16 cases were 3+ (5.33%) - (Table 6).

Among the 300 cases of allergic contact dermatitis, 201 cases were of occupational in origin (67%) and 99 cases were non occupational in origin (33%). The ratio between occupational and non-occupational cases is 2.03:1 – (Table 7).

Of the 300 cases, 28 cases were Atopic individuals (9.33%) by Hanifin and Rajka's criteria. 21 cases out of the 28 cases had 2+ positivity (75%). 6 cases were 1+ positive and 1 case was 3+ positive. Nickel was the commonest allergen to be positive among atopics – 12 cases (42.9%). Cement (Potassium

dichromate) was the second common allergen with 10 cases (35.7%) - (Table 8).

32 cases (10.67%) showed Eosinophilia (Eosinophils > 5 in a differential count – Davidson's textbook of Internal medicine). Eosinophilia was most common amongst the patients who tested positive for Nickel – 17 cases (53.12%) - (Table 9).

Out of the 300 cases, 24 had Diabetes mellitus (8%). 22 of the 24 patients had 1+ positivity (91.67%) - (Table 10).

The time interval between the duration of exposure of the allergen and the development of allergic contact dermatitis was studied. In case of allergic contact dermatitis to cement, the most common time interval was 2 to 5 years (38 cases out of 130 cases – 29.23%). Second most common time interval was >10 years (28 cases – 21.5%). Next comes 5 to 10 years time interval (23 cases – 17.7%). Similar results were obtained in the cases of allergic contact dermatitis to paint and oil and grease with maximum number of cases in 2 to 5 years group – (Table 11 and 12).

Footwear contact dermatitis cases were 24 in number out of 300 cases of which contact dermatitis to rubber was the most common (12 cases – 50%) followed by leather (10 cases – 41.67%) and lastly plastics (2 cases – 8.33%) - (Table 13).

CD_4 cell count was done for 30 cases (10 cases of 1+ positivity, 10 cases of 2+ positivity and 10 cases of 3+ positivity). 5 patients of Cement dermatitis, 3 patients of Nickel dermatitis and 2 patients of Plant dermatitis were included in each group. Average CD_4 count in the 1+ group was 830 cells/mm^3. Average CD_4 count in the 2+ group was 894 cells/mm^3. Average CD_4 count in the 3+ group was 972 cells/mm^3 – (Table 14).

Out of the 300 cases, 138 cases presented with Hand eczema (46%). Allergic contact dermatitis to cement was found to be the commonest cause followed by paint – (Table 15)

DISCUSSION

Allergic contact dermatitis to cement was found to be the commonest in the study (43.33%). Hexavalent chromium is the most common alllergen in the cement. The higher incidence of allergic contact dermatitis to cement is due to more people being employed in construction working in Chennai - Tamilnadu. Sensitivity to chromium was demonstrated by a closed patch test with 0.5% Potassium dichromate in the Indian standard series. In a similar study conducted in Mangalore, allergic contact dermatitis to cement tops the list. The most common sites to be involved were hands, forearms, feet and face, i.e. the exposed sites. With increasing industrialization, the construction industry provides employment to a large number of skilled and unskilled workers leading to increased incidence of allergic contact dermatitis.

Contact dermatitis to Nickel was the second commonest in the study (10.33%). Nickel sensitivity was tested with 5% Nickel sulphate. Nickel in general is the most common metal causing sensitization. Nickel sensitivity was found to be more common in females compared to males with the male female ratio of 1:3.4. This is in accordance to the studies done by Nielson in a group of Danish population. Jewellery and metal components of clothing were the frequent sources of Nickel in the study due to prolonged contact with the skin. Nickel salts being soluble in water and sweat easily cause sensitization. Most common substances causing Nickel sensitization in the

study were necklaces, other jewellery, watches and studs in clothing. 6 out of 31 sensitive patients showed evidence of hand eczema. Studies conducted by Meding and Swanbeck support a connection between hand eczema and Nickel allergy. European union Nickel directive has passed certain legislation with the intention of controlling the use of Nickel releasing objects in contact with the skin. No such legislations have been passed in India.

Phytophoto dermatitis to plant allergens was the third common. Photosensitivity commonly co-exists with Compositae family allergy. Compositae plant allergy show a wide geographical variation. From India, Parthenium hysterophorus has been reported to be the main cause of Compositae contact dermatitis. The same finding was seen in the study too. All the patients patch tested were uniformly sensitive to Parthenium hysterophorus both before and after phototesting. None were sensitive to either Chrysanthemum or Xanthium. Most common pattern seen was that of airborne contact dermatitis. This pattern was also the most common in the study conducted by Sharma S.C., Kaur.S.

Next in the list was allergic contact dermatitis to paint. It was tested with the allergens Potassium dichromate (0.5%), Epoxy resins, Formaldehyde and Colophony. Potassium dichromate was found to be the frequent sensitizer in paints. It was in accordance to the studies done by Mathias CGT. Increasing number of cases is due to the fact that more people are being

employed in construction industry in Chennai - Tamilnadu. Cross reactivity to dichromate in cement was observed in 2 patients.

Allergic contact dermatitis to Kumkum was seen in 5.7% cases and allergic contact dermatitis to Turmeric was seen in 3.7% cases. Kumkum was found to be the commonest cause of cosmetic dermatitis. Common allergens in kumkum are Brilliant lake red R, Sudan I, Canaga oil and Aminoazobenzene as separated by thin layer chromotography. Patch test was done with commercial kumkum as such. Due to traditional use of turmeric and kumkum by south Indian women, there is an increasing incidence of contact dermatitis.

Contact dermatitis to Rubber constituted 5.7% cases. Sensitivity to rubber and its constituents was tested with black rubber mix, thiuram mix and 4-phenylene diamine in the Indian series. Rubber was found to be the commonest allergen in the footwear series. This is in contrast to the studies conducted by Choudhuri Sanjib where Leather was the commonest substance to cause allergy in footwear. Contact depigmentation in footwear series was due to rubber. This was also seen in studies conducted by Singh P Singh and Agarwal V.S.

Allergic contact dermatitis to Leather was seen in 4.7% cases. It is the second common cause of footwear dermatitis second only to rubber. Allergic contact dermatitis to leather was tested with Potassium dichromate,

Formadehyde, Wood alcohol and 4-Chloro 3-cresol. Footwear and watch straps were the common substances causing allergic contact dermatitis.

Allergic contact dermatitis to oil and grease were seen in 14 cases. Allergens in oil and grease are Parabens, 4-phenylene diamine, Mercaptobenzothiazole in the Indian series. Most of the patients in this group were automobile mechanics who were constantly in contact with oil and grease.

Allergic contact dermatitis to hair dye was seen in 6 cases. Paraphenylene diamine is the allergen implicated. PPD is an aniline derivative most commonly used for dyeing hair. Allergic contact dermatitis was commonly seen in beard areas and scalp was relatively spared. This was in accordance with the studies done by Foussereau.

Allergic contact dermatitis to plastics was tested with Formaldehyde and Epoxy resin. Allergic contact dermatitis to printing ink was tested with Colophony, Wood alcohol, 4-Chloro 3-cresol. Allergic contact dermatitis to polish was tested with Colophony, Polyethylene glycol, Formaldehyde, Potassium dichromate, Parabens. Allergic contact dermatitis to Photographic film developing fluid was tested with PPD, Formaldehyde and Mercaptobenzothiazole.

Potassium dichromate was found to be the commonest allergen in the Indian standard series. Next in the order of the frequency are Nickel, Formaldehyde, Cobalt chloride, Epoxy resin, Parabens, 4-Chloro 3-cresol. The Indian standard series differs from the European standard series by the inclusion of Propylene glycol, Nitrofurazone, Gentamycin, Chlorocresol, PEG 400 and Ethylene diamine chloride whereas Sesquiterpene lactone mix and Primin allergens are excluded. The study conducted by Srinivas C.R in P.S.G Institute of Medical Sciences and Research showed Nickel to be the most frequent sensitizer followed by Potassium dichromate, Cobalt chloride and Colophony in that order. The reason for Potassium dichromate to be the commonest allergen in the study is due to the increased number of patients with allergic contact dermatitis to cement in the study.

Male to female ratio in the study of 300 cases was 2.48:1. Reason for male predominance may be due to the fact that more cases were occupational in nature where men were employed in preference to women. In the study conducted by Srinvas C.R. In P.S.G. Institute of Medical Sciences and Research showed a male to female ratio of 1.8:1 and in the study conducted by Kishore Nanda et al in Mangalore the ratio was 1.27:1. Female predominance was specifically seen in cases of allergic contact dermatitis to Nickel, Kumkum and Turmeric. It has also been seen in the study conducted by Nielson et al. It is also due to common usage of Nickel and Kumkum by women in South India.

The most common age category of the patients was 41 to 50 years. In a similar study conducted in Iran, the mean age of the patients was found to be 43.6 years. Very young and extremes of ages were lease affected. This is due to the fact that people accumulate allergies acquired over a life time and that inflammatory response is diminished in elderly patients.

Occupational cases of allergic contact dermatitis was found to be twice common when compared to non-occupational cases. Construction industry and agriculture top the list of occupations causing contact dermatitis. This is in accordance to the studies conducted by Cherry N Meyer.

Nearly 10% of the patients were atopic individuals as diagnosed by Hanifin and Rajka criteria. In the study conducted by Sharma A.D. In Assam, allergic contact dermatitis was found not uncommon amongst atopic individuals. Patch test positivity was 2+ in most cases. This was also observed in that study. Nickel was the most common allergen among atopics in the study. Similar observation was found in the previous study.

Eosinophilia was observed in 10% cases. Eosinophilia was found to be more common among atopic individuals. It was also more common in patients of ACD to Nickel. Eosinophils gain more importance in cases of irritant contact dermatitis. Eosinophilia was found in 8.5% cases among 400 cases of allergic contact dermatitis in a study conducted by Sieberberg.

8% of patients were diabetics. 1+ positivity was most common among diabetic patients. It is due to immuno suppression induced by Diabetes mellitus. This has been shown by Grossman.

Average time duration between exposure of the allergen and development of allergic contact dermatitis was found to be 2 to 5 years for most allergens primarily to cement, paint, oil and grease. It was found to be an average of 2.6 years in a study conducted by Rajanna M.S in Bangalore.

46 % of 300 cases presented with hand eczema. It was the predominant presentation of allergic contact dermatitis in the study. Similar study conducted in Singapore showed hand eczema to be the predominant presentation of allergic contact dermatitis. Allergic contact dermatitis to cement was found to be the commonest cause of hand eczema.

CD_4 counts were done for 30 patients (10 each for 1+, 2+ and 3+). CD_4 counts in the 3+ group were found to be more than that of the other two groups. This is in accordance to the studies conducted by HOEFAKKER et al.

CONCLUSION

1. Allergic contact dermatitis to cement was found to be the commonest cause of ACD in the study. Potassium dichromate was found to be the most frequent allergen to be positive in the Indian standard series.
2. Most cases of allergic contact dermatitis fall in the 41 to 50 years age category.
3. Male to female ratio of the total cases was 2.48:1.
4. There was an increased incidence of ACD in atopic patients and more incidence of 2+ positivity. Nickel was the most common allergen causing ACD in atopic individuals. Eosinophilia was more common among atopics and in patients with ACD to Nickel.
5. The average duration between the exposure of the allergen and manifestation of ACD was commonly between 2 to 5 years.
6. Occupational cases of allergic contact dermatitis were twice common than non-occupational cases.
7. Patients with Diabetes mellitus have increased incidence of 1+ positivity due to immuno suppression.
8. The most common presentation of allergic contact dermatitis in the study was that of hand eczema.
9. CD_4 cell counts in the 3+ group were found to be more than 2+ and 1+ groups.

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PROFORMA

NAME	:	OCD No.	:
AGE	:	Address	:
SEX	:	Phone no.	:
OP No.	:	Occupation	: (Nature and duration)

H/O PRESENT ILLNESS

A. H/O contact with allergen (type and duration)
B. Onset
C. Progression
D. Exacerbating factors
E. Course of the disease

H/O ATOPY – Patient and among family members

PAST HISTORY

A. Similar complaints in the past.
B. Whether lesions occurred in the past due to exposure to the same allergen.
C. H/O any drug intake.
D. Diabetes / Hypertension / Tuberculosis / Bronchial asthma.

PERSONAL HISTORY

A. Diet
B. Habits
C. Occupation

TREATMENT HISTORY

A. Drug intake or topical application for the present condition.
B. Drug intake for any other disease – nature and duration.

GENERAL EXAMINATION

A. Anemia
B. Jaundice

C. Cyanosis / Clubbing
D. Pedal edema / Lymphadenopathy

VITALS

Temperature, Pulse, BP

SYSTEMS EXAMINATION

A. CVS
B. RS
C. Abdomen
D. CNS

DERMATOLOGICAL EXAMINATION

A. Morphology of the lesions.
B. Sites of involvement.

INVESTIGATIONS

I Hemogram

A. Hemoglobin
B. Total count
C. Differential count (for Eosinophils)
D. ESR

II Blood Sugar

III Skin Biopsy – for doubtful conditions

IV PATCH TESTING – Readings and Interpretation according to ICDRG

V CD_4 Count – for selected patients

DIAGNOSIS

- Categorization of the patient

TREATMENT

ADVICE

FOLLOW UP