

CLINICO ETIOLOGICAL STUDY OF ALLERGIC CONTACT DERMATITIS WITH PATCH TESTING

*Dissertation submitted in partial fulfilment of the
Requirement for the award of the Degree of*

**M.D. DERMATO-VENEREO-LEPROLOGY
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CERTIFICATE

This is to certify that the dissertation entitled “**CLINICO ETIOLOGICAL STUDY OF ALLERGIC CONTACT DERMATITIS WITH PATCH TESTING**” submitted by **Dr. SANTHIYA VADHANA.A** to the Tamilnadu Dr. M.G.R Medical University, Chennai, is an original work done in the Department of Dermato-Venereo-Leprology, Tirunelveli Medical college for the award of the Degree of MD Dermato-Venereo-Leprology under our guidance and supervision during the academic period of 2013-2016.

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I solemnly declare that the dissertation titled “**CLINICO ETIOLOGICAL STUDY OF ALLERGIC CONTACT DERMATITIS WITH PATCH TESTING**” is done by me at the Department of Dermato-venereo-leprology, Tirunelveli Medical College, I also declare that this bonafide work or a part of this work was not submitted by me for any award, degree, or diploma to any other University, Board, either in India or abroad.

This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirements for the award of M.D Degree in Dermato-venereo-leprology.

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CONTENTS

S.No.	TITLE	Page No.
1.	INTRODUCTION	1
2.	REVIEW OF LITERATURE	3
3.	AIMS AND OBJECTIVES	60
4.	MATERIALS AND METHODOLOGY	61
5.	RESULTS	64
6.	DISCUSSION	83
7.	SUMMARY	92
8.	CONCLUSION	94
9.	BIBLIOGRAPHY	
10.	PROFORMA	
11.	MASTER CHART	
12.	ANNEXURES	



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CLINICO ETIOLOGICAL STUDY OF ALLERGIC CONTACT DERMATITIS WITH PATCH TESTING

INTRODUCTION

Allergic contact dermatitis is an inflammatory disorder which is T-cell mediated that occurs at the challenged site with a specific substance of low molecular weight in an already sensitized individual.¹ Contact dermatitis is one

PAGE: 1 OF 89

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ABBREVIATIONS

ACD	-	Allergic contact dermatitis
DTH	-	Delayed type hypersensitivity
ICDRG	-	International Contact Dermatitis Research Group
MHC	-	Major histocompatibility complex
IL	-	Interleukin
TNF	-	Tumor necrosis factor
GM-CSF	-	Granulocyte-macrophage colony-stimulating factor
APC	-	Antigen presenting cell
CAM	-	Cellular adhesion molecule
LFA	-	Leukocyte functional antigen
LC	-	Langerhans cell
CD	-	Cluster differentiation
CLA	-	Cutaneous lymphocyte-associated antigen
CCL	-	Chemokine (C-C motif) ligand
HLA	-	Human leukocyte antigen
NK cells	-	Natural killer cells
CHS	-	Contact hypersensitivity
UVA	-	Ultraviolet A
EDTA	-	Ethylene diamine tetraacetic acid
SQL	-	Sesquiterpene lactones
ABCD	-	Air-borne pattern contact dermatitis
CAD	-	Chronic actinic dermatitis
AD	-	Atopic dermatitis
PPD	-	Paraphenylenediamine

PABA	-	Para amino benzoic acid
PTBPFR	-	para-tert-butyl phenol formaldehyde resin
MBT	-	Mercaptobenzothiazole
PEG	-	Polyethylene glycol
TRUE	-	Thin layer rapid use epicutaneous test
ROAT	-	Repeat open application test

CLINICOETIOLOGICAL STUDY OF ALLERGIC CONTACT DERMATITIS WITH PATCH TESTING

ABSTRACT

Background:

Patch testing is a scientific tool to make a diagnosis of allergic contact dermatitis (ACD). It thus exposes the prevalence and current trends of allergic contact dermatitis in the community. As for now, there is no data on allergic contact dermatitis with patch testing in our area Tirunelveli.

Aim:

To study the incidence, clinical severity and morphological patterns of ACD in correlation with patch test results.

Methods:

This is an observational, prospective, single group, open labelled clinical study. 100 patients were recruited in the study during the period of June 2014 to August 2015. The Indian Standard Series was used from Systopic pharmaceuticals pvt limited, Newdelhi. The patch test readings were interpreted according to ICDRG criteria.

Results:

Of 100 patients(75 males ,25 females) , 89 patients showed one or more positive reactions to patch test. Cement dermatitis(38%) was commonest in our study followed by Parthenium dermatitis(25%). Potassium dichromate was the commonest allergen followed by Parthenium hysterophorus. Nickel sulphate was the commonest allergen in females. Hands and/or feet were the most common pattern observed in the study followed by contact site eczema. The clinical severity had no correlation with patch test severity.

Conclusion:

Our study revealed higher prevalence of cement and parthenium dermatitis in our area. Analysis with standard series is very significant to identify the cause of contact dermatitis and thus by avoiding the allergen, decrease the management cost and better quality of life.

Keywords: Allergic contact dermatitis, Patch testing, Cement, Parthenium, Nickel

INTRODUCTION

Allergic contact dermatitis(ACD) is an inflammatory disorder which is T-cell mediated that occurs at the challenged site with a specific substance of low molecular weight in an already sensitized individual.¹ Contact dermatitis is one of the most common skin disorders all over the world which accounts for 4-7% of all dermatological consultations.²

Substances responsible for contact dermatitis are haptens which are capable of triggering the type IV hypersensitivity reaction after single or multiple exposures. ACD occurs due to breakdown of cutaneous immune tolerance to haptens. Sensitization phase is the prime event, which takes place before elicitation phase occurs.

The clinical manifestations that occurs during the acute phase are erythema, edema, papulovesicular eruptions and secondary skin lesions like oozing and crusting. Lichenification, fissuring and pigmentation occurs in the chronic phase. The common allergens vary from place to place and from time to time. Parthenium dermatitis is common in India. And nickel in ornaments, potassium di chromate and cobalt in cement, paraphenylenediamine in hairdye, neomycin in topical medicaments, colophony in adhesive plaster, methyl isothiazolinone a preservative in baby wipes, mercaptomix in rubber gloves are few examples of contact sensitizers.

The gold standard method for identifying the causative allergen of ACD is the patch testing.^{3,4,5,6} Patch testing procedure can be cost effective only if

there is high index of clinical suspicion and if tested with chemicals pertinent to the clinical condition.

There are only few clinico etiological studies of ACD, which revealed linear streaky pattern due to plant allergens, fingertip pattern in garlic users, eye lid oedema due to dyes. In various Indian studies on patch testing done with Indian standard series, the five maximum frequent allergens were potassium dichromate, nickel, fragrance mix, cobalt chloride, mercaptobenzthiazole, even though percentage varies with different studies.

Through a prospective study we are focussing on determining the incidence of ACD and the causative allergen of ACD by patch testing and analysing the morphological patterns of presentation of various allergens. We did an analysis of clinicoetiologic correlation of ACD with Patch testing and the implications are herewith discussed.

REVIEW OF LITERATURE

DEFINITION:

Allergic Contact Dermatitis (ACD) is a, type IV, T cell mediated, delayed type hypersensitivity reaction (DTH) (i.e) an inflammatory reaction triggered by contact with specific exogenous allergen to which a person has established allergic sensitization. It is characterized in early stages by erythema, papules and vesicles, followed in late stages by lichenification, scaling, fissuring and xerotic skin.⁷

HISTORY:

Allergic contact dermatitis is most likely documented even in antiquity, because it has complemented menfolk all the way through the past.

In 1906 , the Scientist Von Pirquet coined from Greek words the term ‘allergie’, allos & ergon meaning other or different work.⁸ In 1840, Fuchs suggested that ‘dermatitis venenata’ was a manifestation of constitutional idiosyncrasy. Neisser used the word ‘Idiosyncrasy’ to describe iodoform dermatitis in 1884. Bloch and Steiner-Woerlich proved contact allergy of the skin, by using Primula extract on humans. Landsteiner and Jacobs substantiated that hapten molecules must combine with skin cells proteins to cause sensitization.

A standardized technique called ‘Patch test’ is used for the confirmation of the role of suspected causative agents in producing an allergic contact dermatitis. Patch test signifies a valuable diagnostic tool that unravels the mystery of many dermatoses in which etiology remains unknown.

Historical aspects of ACD in 20th century, is inseparable from patch testing, which is the diagnostic tool that unmasks the relevant allergens of ACD and the patch test is inseparable from the pioneer in the field, Josef Jadassohn (1860-1936).

In 1895, Josef Jadassohn introduced the patch test technique, while working at Breslau University, when he described the patch testing role in Dermatitis medicamentosa and He is considered the father of Patch testing. During the 17th, 18th, 19th centuries, some researchers made a replica of contact dermatitis, by applying the suspected allergen.

In 1847, Stadeler described the blotting paper strip technique. In 1889, Collins who was an ophthalmologist, tried atropine patches to the patients who manifested adverse reactions after atropine eyedrops instillation.

Bruno Bloch, a dermatological pioneer, upgraded Jadassohn's technique, and gave the grading system for patch testing, and introduced the concept of standard series of allergens, cross sensitization and systemic ACD.^{9,10}

Marion Sulzberger introduced the patch test technique in New World. Paul Bonnevie, Professor of Occupational Medicine in Copenhagen, expanded the standard series of allergens, the archetype of our current series.

In 1986, Fisher stated that 'Patch tests' are the only scientific proof of Allergic Contact Dermatitis, when properly applied and correctly interpreted. He also emphasized that learning the art of patch testing technique is as important as other diagnostic procedures.

Scandinavian dermatologists and other European members formed the International Contact Dermatitis Research Group (ICDRG) to formulate a standard protocol for patch testing and for international research in this field.¹¹

EPIDEMIOLOGY:

In India, allergic contact dermatitis has an incidence of 4-7%, which is one of the major occupational health problems.¹² The socio economic impact is also significant. 40-60% of industrial non-attendance is ascribed to some form of contact dermatitis. Incidence can vary depending on the degree of socioeconomic and industrial development in the area as well as the interest of the dermatologist in allergic contact dermatitis. The common allergens implicated to cause ACD varies from place to place and time to time.

Total population research works and scrutiny of random samples of people have revealed the incidence of contact dermatitis to be 1.5% to 6%. In definite professions like construction work and in biochemical and metallic industries, the frequency is predominantly increased.¹³

ETIOPATHOGENESIS:

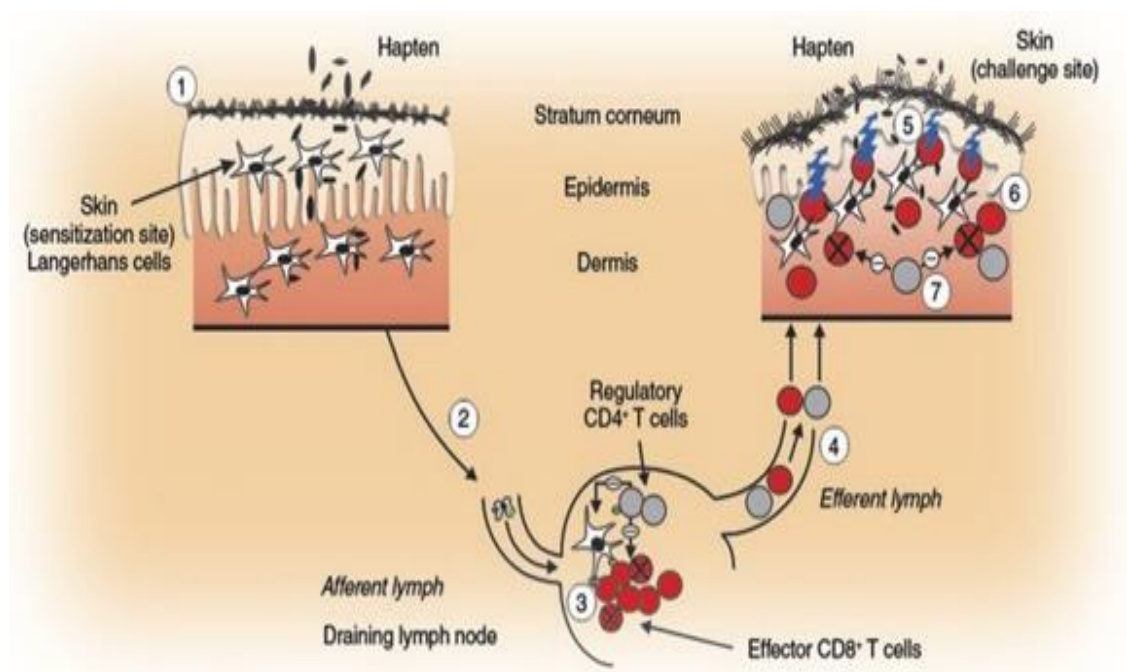
Allergic Contact dermatitis is an inflammatory skin condition that is hapten specific. Haptens are substances of low molecular weight which is less than 500 Daltons. These haptens penetrate the stratum corneum to the nucleated layers of epidermis to induce and elicit the contact sensitization. After single or multiple exposures, non-protein chemicals, i.e. haptens, induce ACD. ACD is well thought-out as an interruption of cutaneous immune tolerance to haptens.¹⁴ The pathophysiology of ACD consists of two different segments.

- ✚ Phase 1 - Sensitization phase (also referred to as afferent phase or induction phase)
- ✚ Phase 2 - Elicitation phase (also known as efferent or challenge phase)

I) Sensitization phase:

The prime events of this phase are

- The Allergen binding to components of skin
- The 'complete' or conjugated antigen recognition
- Sensitized T lymphocytes - Proliferation and dissemination



The Allergen binding to components of skin

Allergens that penetrate the skin bind covalently with skin peptides directly or alternatively to form a reaction product that binds with major histocompatibility complex (MHC) class II molecules which are present on the

surface of dendritic cells and Langerhans cells. Epicutaneously applied allergen allies with these antigen-presenting cells in 6 hours.

The ‘complete’ or conjugated antigen recognition

The APCs undergo a series of events activation, maturation and migration for which co-stimulatory factors like IL-1 β , TNF α and GM-CSF are required. In the absence of these co-factors, tolerance develops.

Within 24 hours of antigen exposure, APCs travel via the afferent lymphatics to the paracortical areas of the regional lymph nodes, where they are presented to T lymphocytes. This binding is strengthened by physical factors, the ruffled membrane and dendritic nature of the Langerhans’ cells and the intricate structure of the paracortical areas and also by specialist cellular adhesion molecules (CAMs). For example, leukocyte functional antigen-1 (LFA-1) on CD4 T helper cells interacts with intercellular adhesion molecule-1 (ICAM-1) on Langerhans’ cells. Subsequently, cytokines are released, IL-1 by LCs and IL-2 by T lymphocytes. An intact draining lymphatic system is required to induce a contact hypersensitivity reaction.¹⁵

Sensitized T lymphocytes- Proliferation and dissemination:

The blast formation in the lymph nodes and the multiplying of antigen-specific cytotoxic CD8⁺ (Tc1) and also CD4⁺ (Th1) lymphocytes is caused by the cytokines.¹⁶ The T cells disseminate into the blood stream and throughout the body via the efferent lymphatics vessel and thoracic duct and interact with Langerhans’ cells and residual antigen in the skin. Contact sensitization is mediated by a subset of T cells that express cutaneous lymphocyte-associated

antigen (CLA). Production of the chemokine CCL27 by basal keratinocytes is responsible for the localization of inflammation and binds to dermal glycoprotein; CLA-positive lymphocytes also express CCR10, the receptor for CCL27.¹⁷ CD8⁺ T cells induce apoptosis in these Keratinocytes and the skin is damaged which drives the inflammatory response. CD4⁺ Th1 & CD8⁺ T cells act as effectors on target cells. Sensitization phase lasts for 10 to 15 days.

II) Elicitation phase:

After sensitization has occurred, re exposure to the specific allergen causes eczematous dermatitis. On re-contact to the similar allergen, a clinically visible reaction occurs within 24–48 h, which is mediated via activated keratinocytes that express HLA-DR on their surface and can release IL-1, thus amplifying the function of LCs. Both types of cells present the antigen to specific T cells that are already present in the epidermis in small numbers, inducing a quick inflammatory response. This is responsible for the recruitment of leukocytes (including regulatory T cells) from the blood to the skin leading to the development of skin lesions.

The role of skin memory:

The mechanism for site specific allergen skin memory is related to chemokine CCL27 that causes retention of CCR10⁺ CD4⁺T cells perivascularly in the dermis at the site of Patch testing.

The role of Keratinocytes in all phases of ACD:

- ✚ In initiation phase- it secretes TNF alpha
- ✚ After Ag exposure- modulates APC migration & T cell trafficking

- ✚ In peak of inflammatory phase - interacts directly with epidermotropic T cells
- ✚ Resolution of ACD- produce anti-inflammatory cytokines IL10 & IL16 – recruits T Reg cells
- ✚ The cytokines produced by keratinocytes are
 - 1) IL 1: Enhances activation of accessory dendritic cells, which in turn activates T cells
 - 2) IL 5: Stimulates T cell proliferation
 - 3) IL 8: Has a strong chemotactic effect of T cells

Recent concepts in ACD:¹⁸

- Innate immune cells such as Natural Killer(NK) cells play a significant role in ACD
- NK T cell are necessary for initiation of ACD & it also presents in elicitation phase of ACD
- Studies from mice lacking Langerhans Cells also shows contact hypersensitivity , hence cells other than LCs also play a prime role in CHS
- Dermal Dendritic Cell also acts as Antigen Presenting Cell that complements the function of epidermal Langerhans Cell
- T regulatory cells (T- Reg) cells plays a critical role in control of ACD (i.e) resolution of T cell inflammation
- Loss of T- Reg cells cause chronic inflammation
- Mast cells determines the magnitude of inflammatory reaction

PREDISPOSING FACTORS FOR ACD:

I) INDIVIDUAL VARIATIONS

i) CONSTITUTION:

Sensitization depends on individual susceptibility.¹⁹ The role of atopy in ACD is a matter of debate. One study reported high prevalence of contact allergy in atopic individual but another study showed same prevalence and others reported decrease in the prevalence of contact allergy.²⁰

ii) ROLE OF SEX:

Women are supposed to have stronger cell mediated immunity responses than men.²¹ The reason for female preponderance is due to prior 'conditioning' exposure and subclinical sensitization to large number of metals, exposure to fragrances, cosmetics and hair dyes.²²

iii) HORMONES:

Pregnancy, menstrual cycle, use of gestagens either exacerbate or attenuate the readings of patch tests.^{23,24} Exacerbation has been reported during the premenstrual phase of menstrual cycle.²⁵

iv) RACE:

Racial differences exist but it is a reflection of exposure rather than tendency.^{26,27} Afro-Caribbeans are less susceptible than white people due to decreased exposure.²⁸ In another study black men were more sensitive than white men, may be because of exposure patterns or due to variations in N-acetylation.²⁹

v) AGE:

Age factor plays less significant role on ability for sensitization.³⁰ But positivity of patch test reactions increase with age due to allergen exposure that have acquired over a lifetime.³¹ Nickel, Fragrance, Thiomerosal, Medicaments, Rubber chemicals, Chromate are common allergens in children.³²

vi) MEDICATION:

Medication will affect the patch test results. Prednisolone (>15mg/day) and potent topical steroids will subdue the patch test reactions. Immunomodulators such as ciclosporin and azathioprine may reduce the intensity of patch test reactions.³³

vii) COINCIDENTAL DISEASES:

Patients with acute or debilitating diseases such as cancer, Hodgkin's disease and those with impaired T-lymphocyte function have impaired capacity for contact sensitisation.^{34,35}

viii) LOCAL FACTORS:

Preexisting or concomitant allergic or irritant dermatitis harms the skin, upsetting its barrier function and producing increased opportunities for allergen absorption. Occlusion promotes percutaneous absorption and contributes to the high incidence of medicament dermatitis in stasis dermatitis, leg ulcers and perianal dermatitis.

II) ENVIRONMENTAL FACTORS:

Certain important environmental factors predisposing to ACD are

1. Climate

2. Flora and fauna

3. Socio-economic and cultural factors

i) CLIMATE:

UV exposure, heat and relative humidity can influence the burden of contact allergy. UVB exposure shall diminish the skin's immune response to contact allergens, however decline in immune responsiveness by UVA exposure is transient due to adaptive mechanism.³⁶

ii) FLORA AND FAUNA:

Seasonal variations are most common in plant dermatitis. Many allergic plants especially compositae family plants are shattered by cold and frosty weather but reappearance occurs during spring and summer season. Geographical location plays an important influence. In India parthenium contact dermatitis is more common. Allergenicity of *Primula obconica* change with weather and sunlight. Fauna has only little influence.

iii) SOCIOECONOMIC AND CULTURAL FACTORS:

Exposure to cheap metals, various cosmetics and perfumes shall vary according to social class. In the Middle and Far East, the traditional herbal medicines and balms are commonly used to treat skin disorders. Hair dyes, kumkum and bindi are commonly used by Indian women.³⁷

III) CHEMICAL COMPOUNDS AND THEIR SENSITIVITY:

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”.

Skin cell molecules: Contain nucleophilic atoms

Hapten molecules: Contain electrophilic atoms (positively charged, electron deficit) covalent bonding

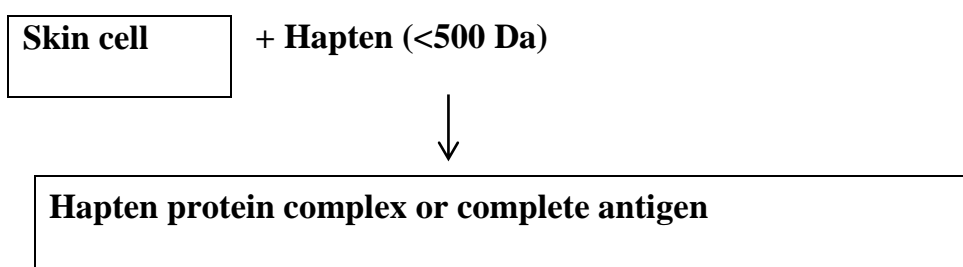


Table: Classification of haptens based on functional grouping³⁸

1.Acids	7.Amines
2.Aldehydes	8.Esters
3.Diazo compounds	9.Epoxides
4.Metals	10.Halogenated compound
5.Ethers	11.Quinones
6.Unsaturated compound	

Sensitization Potential

It is the capability of a given allergen to induce sensitization in a group of humans.

Various test procedures to assess the sensitization³⁹

1. Maximization test(described by Kligman and Epstein)
2. Buehler test
3. Open epicutaneous test
4. The Draize test
5. Freud's complete adjuvant test
6. The local lymph node assay
7. The mouse ear swelling test⁴⁰

Factors that can enhance the risk of sensitization:

1. Increased allergen absorption due to barrier disrupted skin.
2. Recruitment of immune competent cells and cytokines which leads to priming of immunological response
3. Accumulation of mononuclear cells.

Matzinger's 'danger model' concept for sensitization⁴¹

Contact allergy may develop in the presence of cytokine release from the keratinocytes which is provoked by a coexisting irritant or trauma. If there is no irritancy then tolerance will develop.

HISTOPATHOLOGY OF ACD

Histopathologic assessment of ACD is mainly helpful to eliminate other conditions that clinically simulates ACD. But other types of spongiotic dermatitis cannot be differentiated.

Cutaneous changes seen by light microscopy depends on two factors⁴²

- Severity of response to allergen
- Time of biopsy taken after exposure to allergen

Early lesions of ACD are acute spongiotic dermatitis. If vesicles develop, they may contain clusters of Langerhans cells. There is superficial dermal infiltrate of lymphocytes, macrophages and Langerhans cells with accentuation around the small vessels. Eosinophils may be present in the dermal infiltrate as well as within areas of spongiosis. In patients with continued exposure to the antigen, the biopsy may show a subacute or later a chronic spongiotic dermatitis.

HISTOPATHOLOGY OF ICD:

The histopathologic picture differs from widespread ulceration, to simply diffuse hyperkeratosis or parakeratosis with congestion and ectasia, to a spongiotic pattern essentially identical to allergic contact dermatitis. In some instances, there is significant necrosis with nuclear karyorrhexis and cytoplasmic pallor (Bandmann's achromia). In severe reactions, the necrosis may extend into the dermis.

Clinical approach to a case of ACD:

The Key symptom of ACD is Pruritus. The morphological pattern and distribution of dermatitis must raise the index of suspicion of ACD. One must consider contact allergy in patients of any types of dermatitis (eg. Atopy). Patients with stasis dermatitis have increased risk of ACD to topical medicaments. ACD is not always B/L even though Ag exposure is B/L (glove/shoe). Even when exposure to allergen is uniform, eczematous lesions are often patchy. ACD does affects palms and soles

In acute phase, ACD is characterized by erythema, oedema, followed by appearance of papules, vesicles, oozing & crusting.

In chronic stages, skin becomes lichenified, fissured, pigmented and scaling.

ACD can be classified as

- 1) Eczematous CD
 - a. Primary pattern
 - b. Secondary pattern
- 2) Non-eczematous CD
- 3) Photo allergic CD

I) ECZEMATOUS CONTACT DERMATITIS

PRIMARY PATTERNS:

Anatomical patterns of dermatitis often suggest a specific cause

Hands & arms:

There are multifactorial reasons for hand eczema. Housewives dermatitis and many of the occupational dermatitis mostly are confined to the hands. Chromate in cement, N-isopropyl-N'-phenyl-p-phenylenediamine (IPPD) and 1, 2-benzisothiazolin-3-one are the most common allergens that causes palmar pattern of allergic dermatitis. Discoid patterns of eczema occurs with chromate allergy. Allergy to nickel, chromate and p-tertiary-butyl phenol formaldehyde resin also develop at the wrists because of sensitivity to the metal, leather and glue, respectively, in watchstraps. Rubber gloves cause a clear pattern of dermatitis at the site of contact. Streaky dermatitis on the fingers and dorsa of the hands is caused by plants. Dust (exotic woods, cement), nickel and textiles induces dermatitis in the flexural aspect of elbows.

The morphological patterns of hand eczema described are pompholyx, recurrent focal palmar peeling, ring eczema, fingertip eczema, hyperkeratotic eczema, apron eczema, chronic acral dermatitis, and gut eczema.

Face:

Facial allergic contact dermatitis occurs due to fragrances, hair dyes, preservatives and other ingredients of cosmetics and skincare products, including nail varnish. Dermatitis caused by a cosmetic presents with dryness, tightness and itching⁴⁵. 'Hair dyes' might be a reason for acute oedema and intense pruritus. Spectacle frames containing nickel or plastics may be the reason for dermatitis on areas of contact with the cheeks, nose, eyelids and ears.

“Status cosmeticus” due to “stinging” compounds in cosmetics

The recently available cosmetics are free of compounds that cause allergic hypersensitivity. But, nonspecific irritation from cosmetics occurs. Thus, some persons appear to be in a condition of “status cosmeticus” in which every cosmetic or soap applied to the face produces itching, burning, or stinging sensations.⁴³

Eyelids:

The skin of the eyelids is thin, sensitive and often fiddled by the fingers causing eyelid dermatitis due to airborne droplets (e.g. fragrance sprays) or volatile substances (e.g. epoxy resin). Nickel and/or rubber Eye creams present in eye shadows, mascara and makeup applicators cause contact allergy at the site of contact.^{44,45} Eye drops and contact lens solutions also contain preservatives (benzalkonium chloride, EDTA, mercurials), which sensitizes. Common sensitizers in eye drops and ointments are neomycin, framycetin, gentamycin, tobramycin, local anaesthetics, β -blockers and sympathomimetics.

Lips and perioral area:

ACD can occur due to lipsticks, nickel, medicaments, flavourings, garlic, shellac and cosmetic excipients.^{46,47} Lipstick dermatitis does not extend beyond the vermilion border and manifests as dry, scaling or cracked lip. Cheilitis and perioral eczema occurs due to allergy to toothpaste and due to flavours like cinnamic aldehyde, spearmint oil and l-carvone.^{48,49} Allergic contact cheilitis may occur due to Colophony and derivatives present in chewing gum and food

additives such as sodium metabisulphite, preservatives, colours and antioxidants.⁵⁰

Ears:

External otitis always have a chronic relapsing course. Earrings cause dermatitis on the ear lobes which is mostly due nickel and gold. Piercing of the ear lobe may be the sensitizing event in nickel dermatitis.

Earplugs for noise protection contain antiseptics, dyes, rubber and plastic chemicals, Hearing-aids contain acrylates and stabilizing chemicals, Headsets contain urea and phenol-formaldehyde resins, Earphones has rubber, plastic components, including epoxy resins and acrylates, all these cause ear dermatitis.⁵¹

Spectacle-frame dermatitis may occur due to metals, particularly nickel and palladium, in some frames.^{52,53} Granulomatous contact allergy after ear piercing can occur due to nickel, palladium and gold.⁵⁴

“Sugarcane” ears, resembling cauliflower ears, occur in workers who carry bundles of burned stalks of sugar and the lesions are unilateral, depending on whether the worker is right or left-handed. This was described by Arnold.⁵⁵

Scalp:

Scalp dermatitis is caused by Hair-styling products such as mousses, gels and holding sprays, fragrances and preservatives p-phenylenediamine and related semi-permanent dyes and amphoteric detergents in hair cosmetics and manifests as persistent itching of the scalp and gradually spread to the ears, neck and face. Medicated shampoos that contain tar extracts, zinc pyrithione, formaldehyde,

isothiazolinones added as preservative cause scalp dermatitis. Topical minoxidil lotion applied over scalp to promote hair growth is also a sensitizer.⁵⁶

The Hindu practice of wearing a central forehead dot of color known as a bindi cause leukoderma associated with paratertiary butylphenol resin in the adhesive, with or without a related dermatitis or positive patch test.^{57,58}

Neck:

Nickel in the clasps of necklaces or zip fasteners cause dermatitis on the nape of the neck. Nail varnish from fingertips cause patchy allergic dermatitis. Textiles (finishes in collars, dyes) and necklaces (nickel, exotic wood) produce collar like dermatitis, or eruptions on the neck. Dermatitis from photosensitizers and airborne allergens is sharply limited by the collar to the 'V' of the neck if blouses or open-necked shirts are worn.

Axillae:

Dermatitis in axillary region occurs due to sweating, occlusion and the use of antiperspirants which contain aluminium salts. Allergic sensitivity occurs due to fragrances that are used to mask odour. Textiles cause periaxillary dermatitis. Dermatitis from dresses, blouses and sweaters affects folds of the axilla and the allergens are usually textile dyes.

Trunk / Torso:

Nickel buttons and zip fasteners are the reason for the contact dermatitis. Truncal eczema is due to chromate sensitivity from leather and rubber. Detergents and fabric conditioners cause truncal skin eruptions.⁵⁹ Open-air

workers sensitized to Compositae plants manifests as dermatitis which affects the sun exposed areas. Diffuse papular eczema occurs due to medicament sensitivity.

Anogenital region:

Medicament sensitization is the most common reason for ACD in anogenital region and the common medications implicated are neomycin, hydroxyquinolones, ethylenediamine and topical antifungals. Over-the-counter medicaments causes dermatitis in anogenital region. Ectopic contact dermatitis occurs due to nail varnish.⁶⁰ Moist toilet tissues contain preservatives that cause allergic hyper sensitivity.⁶¹ Cashew nut oil in butter causes perianal dermatitis.⁶² Rubber accelerators in condoms cause genital eczema or pruritus vulvae. Delayed hypersensitivity can occur to semen.⁶³

Gluteal region:

A follicular-type of dermatitis occur on the buttocks due to long time contact with wet bathing suits. By swimmers it is commonly called as “bikini bottom” and scientifically called as occlusive folliculitis characterized by annoying wet blisters all over the buttocks which is due to wearing swimsuit all day.

Thighs:

Textile dermatitis occurs at the contact site of the underclothing worn. Nickel coins and keys or boxes of matches may cause dermatitis on the underlying skin, since allergens passes through the clothes worn.

Lower legs:

Allergic contact dermatitis of the legs occurs due to application of sensitizing medications and dressings to stasis eczemas and ulcers. In a study of venous leg ulcer patients, the sensitizers implicated were fragrances in 30%, antimicrobials in 20%, vehicle ingredients in 20%, rubber accelerators in 13%, and topical corticosteroids in 8%.⁶⁴ Rubber allergy occurs due to compression bandaging.

Feet:

Dermatitis may occur due to shoe materials including leather, rubber, glues and nickel, stockings, topical medicaments, antiseptics and antiperspirants.

Nails:

Allergens produce onychia and nail discoloration. Thinness, fragility, splitting, separation into layers, detachment from the nail bed and long-standing infections are various manifestations of ACD that occurs to jewelers, weavers, metal platers, and printers. Occupational chemicals and trauma are common causes of onychia, koilonychia, nail dystrophy and discoloration of the nail.

Generalized:

Generalized erythroderma occurs due to chronic contact dermatitis because of continued exposure to many allergens.

Mucous membranes:

The constitutional make up of skin and mucous membranes have many differences. The cause for the rareness of allergic contact reactions in mucous

membranes is because of absent stratum corneum layer, absent lipid secretion and washaway of substances by saliva.⁶⁵

ACD in mucous membranes is uncommon and is often secondary to skin sensitization. Intraoral blistering occurs from cinnamon allergy.⁶⁶ Orofacial granulomatosis occurs with contact allergy to food additives.⁶⁷ Lichenoid reactions occur due to mercury from amalgam fillings.^{68,69} Generalized skin eruptions and perioral dermatitis may occur after dental filling.⁷⁰ Gingivitis occurs due to eugenol in dental cement.⁷¹

Secondary pattern:

The primary site pattern determines the secondary pattern. The dermatitis of the hands have a tendency to spread to forearms, arms and face. Similarly, dermatitis of feet spreads to legs and hands. Sensitization presenting as 'id' reaction occurs in stasis eczema.

II) NON ECZEMATOUS CONTACT DERMATITIS

Non-eczematous responses in ACD includes

- i) Contact urticarial eruptions
- ii) Lichen planus like lesions
- iii) Lichenoid eruptions
- iv) Lymphomatoid eruptions
- v) Erythema multiforme-like reactions
- vi) Purpuric lesions
- vii) Pigmented lesions
- viii) Leukoderma

- ix) Granulomatous reactions
- x) Systemic non eczematous reactions
- xi) Onycholysis

III) PHOTO ALLERGIC CONTACT DERMATITIS

Photosensitizers are the allergens which are transformed into irritants or sensitizers after irradiation with UV or short-wave visible radiation (280–600 nm). Photoactivated molecules are haptens. The etiopathogenesis is same as contact allergic reactions. The action spectrum for photoallergy is generally in the UVA range.

Photoallergens:

The most common photo allergens are UV filters including *p*-aminobenzoic acid and its derivatives, cinnamates, benzophenones and dibenzoylmethanes. Benzophenone 3 (oxybenzone) seems to be the most commonly identified photoallergen.

Other photocontact allergens are topical non-steroidal anti-inflammatory agents, especially Ketoprofen, Phenothiazines, Sulphonamides, Quinines, Perfumes and Halogenated salicylanilides in soaps and detergents.

Clinical features of photoallergic contact dermatitis

The sun exposed areas like the face, 'V' of the neck, back of the hands, dorsal forearms are the most common sites. The scalp, periorbital areas and the skin immediately under the chin are relatively spared. Sharply delineated areas appear below the sleeves. Cubital fossae spared commonly. The most distinctive

sign is the exempt 'Wilkinson's triangle' behind the earlobe. Some spread to covered sites.

ACD TO SPECIFIC ALLERGENS

PARTHENIUM ANTIGEN

Parthenium dermatitis is caused by *Parthenium hysterophorus*.⁷² It is caused by airborne dry and friable plant particles, and the most important allergens responsible for allergic contact dermatitis are sesquiterpene lactones (SQLs).⁷³ Among the SQLs, parthenin was found to be the major allergen, others being coronophillin, tetraeurin A, hymenin etc.

Patterns of parthenium dermatitis

Clinical features

- **Air-borne pattern contact dermatitis (ABCD):** Classical pattern affects eyelids and neck, V area of the chest and the cubital and popliteal fossae.
- **Chronic actinic dermatitis (CAD):** Lichenified lesions over the exposed areas
- **Mixed pattern dermatitis:** (Combination of air-borne and CAD)
- **Exfoliative dermatitis**
- **Hand and feet dermatitis**
- **Atopic dermatitis**
- Rare patterns like photosensitive lichenoid eruption, prurigo nodularis-like, perianal dermatitis, vesicular hand eczema and dermatitis simulating lichen nitidus.

Management includes avoiding contact with allergen, managing dermatitis with topical corticosteroids/tacrolimus, and other immunosuppressives like azathioprine.

POTASSIUM DI CHROMATE:

In Construction workers, potassium dichromate (hexavalent chromium) was the commonest allergen with the prevalence of sensitivity being more in men.

Levels of chromate in cement should be restricted to 2 ppm hexavalent chromium. The metal itself, if not dissolved in oil or acids or as a salt, seems to be non-sensitizing, unlike nickel and cobalt due to the insoluble monomolecular layer of chromium (III) oxide (Cr_2O_3) on the surface.

Source of potassium dichromate:

Cement, antirust paints (lead chromate and zinc chromate) painted metals, alloys, lithography/offset printing materials, anticorrosive oil, cutting oils, matches, photographic chemicals, chemicals for fat determination in milk, welding fumes, plating salts, wood preservatives and ashes, wood pulp, glazing enamels, catgut.

Clinical patterns caused by Potassium di chromate:

- i) Acral dermatitis
- ii) Hand dermatitis
- iii) Airborne contact dermatitis
- iv) Acro-facial dermatitis
- v) Feet dermatitis

- vi) Atopic eczema like
- vii) Discoid pattern like
- viii) Mixed pattern
 - a. Acrofacial and trunk dermatitis
 - b. Acrofacial and scalp dermatitis

Management:

- ✓ Advised to avoid contact with sources of chromate
- ✓ Ferrous sulphate added to cement changes soluble hexavalent chromate to insoluble trivalent chromate, and thereby preventing sensitization.
- ✓ Chelating compounds and ion exchangers.⁷⁴
- ✓ Dapsone tried, but studies are lacking.⁷⁵

NICKEL SULPHATE:

Nickel is the most common contact allergen of metal allergy and contact sensitization is more in females than males. Nickel allergy is a chronic and recurring skin problem. The nickel salts, nickel chloride (NiCl_2) and nickel sulphate (NiSO_4), are freely soluble in water and sweat and have strong sensitizing character.

The most common sources of metallic nickel are fashion jewellery, coins, machinery parts, utensils, stainless steel items etc.

Role of diet in causing dermatitis:

Nickel dermatitis can occur in a nickel sensitized person if the diet contains excess amount of nickel. And the foods that have increased nickel content are fresh and dried legumes, soya beans, oatmeal, chocolates, nuts etc.

Pattern of nickel allergy:

- Dermatitis at the site of contact
- A “secondary rash” due to spread of dermatitis to distant regions is rarely observed.⁷⁶
- Hand eczema pattern ; Vesicular type after consumption of nickel in diet.⁷⁷
- As baboon syndrome - a generalized rash involving gluteal region, anogenital area, flexural areas and eyelids.⁷⁸
- Erythema multiforme and vasculitis – rare patterns.^{79,80}
- Chronic urticaria⁸¹

Nickel is patch tested at 5% conc in aqueous form

Therapy

- ✓ Barrier creams and cleansers can be tried
- ✓ Combination creams containing clioquinol and steroids
- ✓ Low-nickel diet in recurrent palmar vesicular eczema can be advised
- ✓ Disulfiram (Antabuse), which has nickel chelating property

Prevention

The most effective means of preventing nickel sensitization would be to reduce exposure to nickel from costume jewelry, particularly earrings. The European Union has banned nickel objects, that release nickel in excess of 0.5 mg/cm² per week.

Spot test:

The dimethylglyoxime test is an easy technique to find nickel release from metal objects. A cotton swab is dipped in two drops each of a 1% solution of dimethylglyoxime in alcohol and a 10% solution of ammonium hydroxide in water, and is wiped regularly over test item for 30 seconds. If the cotton swab turns light pink to red, it confirms the release of nickel.

COBALT:

Cobalt metal, its oxides and salts (e.g. CoCl_2 and CoSO_4) are sensitizers. Cobalt is tested at 1% concentration in petrolatum.

Sources:

Cobalt is present in magnets and jewellery, as a contaminant in nickel, in alloys, in dentures and in nails for pinning fractures, glass and ceramics, crayons, multivitamin pills, textile dyes, tattoos, soaps, dyes and detergents.

PARAPHENYLENEDIAMINE (PPD):

PPD is used for permanent hair coloring. It is patch tested at a 1% concentration in petrolatum. In ACD due to cosmetics, the allergen implicated commonly are first fragrances, 2nd preservatives and 3rd is PPD.

Clinical aspects:

PPD causes weeping dermatitis of scalp, eyelids, face, hairline and spread to involve the neck, upper portion of the trunk and arms, hands with generalization.

Dyed hair does not cause dermatitis as oxidized PPD, is not allergenic.

Sources of PPD:

PPD is present in permanent hair dyes, cosmetics, leather dyes, rubber and plastics industry, lithography, oils, greases, epoxy resin hardeners and temporary tattoo, photographic developers etc

COLOPHONY:

Colophony (Rosin) is a yellow, complex, natural residue left after distilling off the volatile oil from oleoresin obtained from the coniferous trees *Pinus palustris*.

Colophony is patch tested at 20% concentration in petrolatum. The most potent allergen has been shown to be 15- hydroperoxyabietic acid.⁸²

Source of colophony:

It is used in a wide range of cosmetics, topical medications, industrial products like paper and paper products, printing inks, adhesives, tapes, bandages, waxes, varnishes, polishes, paints, dental cements.

BLACK RUBBER MIX :

It is composed of the following:

- ❖ N-isopropyl-N-phenyl-4-phenylenediamine - 0.1 %
- ❖ N-cyclohexyl-N-phenyl-4-phenylenediamine - 0.25%
- ❖ N-N-diphenyl-4-phenylenediamine - 0.25%

The preceding amines are used as antioxidants and antiozonants in the production of rubber and are the most effective and commonly used of available agents. The compounds prevent drying and cracking of the final rubber products. Since they discolour and stain, they are used primarily in black rubber.

The most potent sensitizer in the mix has been shown to be IPPD. The mix ingredients are chemically related to the hair dye base p-phenylenediamine, and cross-reactivity can occur.

Sources :

The three p-phenylenediamine compounds are extensively used in rubber manufacture. Since these agents discolour the final product, most finished products are dark, either gray or black.

These include tires, heavy black rubber gloves and boots, shoes (especially soles), cushions, earphones, and walking-stick handles.

CAINE MIX (BENZOCAINE):

Caines are local anesthetics that are used primarily in non-prescription topical medicaments, which are designed to ease pain and pruritus.

Benzocaine is tested at 5% concentration in petrolatum.

Sources:

Sources include over-the-counter medicines used to treat sunburns, dermatitis, athlete's foot and calluses, otic preparations for earaches, enemas and anal suppositories for hemorrhoidal discomfort, oral mucosal products for teeth pain and canker sores

Benzocaine- and tetracaine-sensitive individuals may also have to avoid PABA and PABA esters containing sunscreens, permanent hair dye (p-phenylenediamine, certain diuretics or fluid pills (hydrochlorothiazide), oral antidiabetic medications (sulfonylureas), certain antibiotics including sulfa drugs

(and PASI, azo and aniline dyes, and an important cardiac medication, procainamide.

EPOXY RESIN:

Epoxy resins are plastics that were synthesized for industrial purposes. They have been used extensively because of their versatility, chemical and electrical resistance, excellent adhesion, toughness, low shrinkage, and ability to be cured rapidly or slowly at various temperatures. Cured epoxy resin is nonsensitizing. Allergic contact dermatitis occurs with exposure to uncured resin.

The epoxy resin is a bisphenol A-based resin patch tested in a 1% concentration in petrolatum.

Sources:

Epoxy resins are used primarily in adhesives and glues, laminates, electrical encapsulators, surface coatings, paints and inks, eyeglass frames and vinyl gloves.

FORMALDEHYDE:

Formaldehyde (methanal) is a colorless gas that is readily soluble in water, alcohol, ether, and other polar solvents. It is the simplest member of the aldehyde series and is generally sold commercially as an aqueous solution, formalin.

Formaldehyde is patch tested as 1% in water. Formaldehyde was first used as a biologic preservative in 1868, and by 1889 it was being manufactured and marketed commercially.

Clinical aspects:

The adverse effects of this chemical, include mucous membrane and respiratory tract irritation, allergic and irritant contact dermatitis of the skin, contact urticaria, and potential carcinogenicity.

Textile dermatitis typically involves the peripheral parts of the axillae, the antecubital fossae, the neck, and upper parts of the trunk.

Because of partial combustion seen in cigarette smoke, automobile exhaust, and incineration products, formaldehyde is produced and released in the general environment

Sources:

Formaldehyde is a common chemical that is found in cosmetics, household products (disinfectants, Cleaners), medicated creams, leather tanning agent, photography, textiles, paper manufacturing, pathology fixative, rubber industry preservative, fertilizers, insulation and renal dialysis.

Clothing or avoidance measures such as a change in jobs to prevent dermatitis.

p-tert-BUTYLPHENOL FORMALDEHYDE RESIN:

p-tert-Butylphenol formaldehyde resin (PTBP formaldehyde resin) is one of a large group of synthetic polymers made by reacting formaldehyde with phenol or related alcohols to form network polymers. They are used primarily as adhesives and were the first synthetic polymers to be used commercially. The resin is tested at a 1% concentration in petrolatum

Clinical Aspects:

PTBP formaldehyde resin is a formaldehyde-based phenol resins. It is used exclusively as a glue or an adhesive. This usage depends on its superior qualities of rapid adhesion, durability, and pliability. It cures slowly without additional hardeners at room temperature. The pliability and flexibility make it particularly useful in the bonding of shoe components and parts of watch straps, handbags, hats, and belts. For this purpose it is frequently combined with natural or synthetic rubber.

Sources :

p-tert-Butylphenol (PTBP) formaldehyde resin is used primarily a component glue in leather shoes, handbags, and watch straps, plywood, boxes, insulation and automobiles.

PARABEN MIX:

The parabens are alkyl esters of p-hydroxybenzoic acid. They are the most commonly used preservatives in cosmetics and are usually patch tested as a paraben mix (16% in petrolatum) containing 4% each of methyl, ethyl, propyl, and butyl parabens.

Sources:

The parabens are the most frequently used preservative in cosmetics, medicines and medicated dressing, foods (marinated fish products) and textiles.

MERCAPTO MIX:

Mercapto mix is composed of the following thiazoles:

N-Cyclohexyl-2-benzothiazole-sulfenamide (CBS)

2,2 1-Benzothiazyl disulfide (MBTS)

4-Morpholinyl-2-benzothiazyl disulfide (MMBn)

Each thiazole is present at a 0.333% concentration in petrolatum (1% total) in the mercapto mix. The thiazoles are the most commonly used rubber accelerators in the world.

Clinical Aspects:

The thiazoles are frequently reported sensitizers in shoe and glove allergy but may also be responsible for dermatitis due to contact with rubberized fabric in undergarments, swimwear, and elastic bandages. Thiazole sensitivity is possible in the workplace in many industries involved in rubber manufacturing and the use of rubber in manufacturing other products.

Thiazoles may also be used in nonrubber products, including veterinary and pet products, cutting oils, antifreeze, disinfectants, adhesives, cements, greases, and photographic emulsion.

Sources:

Mercapto mix thiazoles are used primarily in the production of rubber or latex products. They are found in gloves, rubber shoes, leather shoes, rubber in elasticized clothings and other nonrubber sources like disinfectants, repellents, fungicides and insecticides used in agriculture.

MERCAPTOBENZOTHAZOLE:

Mercaptobenzothiazole (MBT) is a thiazole rubber accelerator.

Clinical Aspects:

MBT and other thiazoles are the frequently used accelerators in the production of rubber. Shoe contact dermatitis is mostly due to a rubber component allergy, usually MBT and next thiurams. Usually the dermatitis is limited to the area of contact. This may be primarily the soles of the feet bilaterally, but patients with such an allergy may also have unilateral involvement. MBT is second to the thiurams as the etiologic agent in allergic contact dermatitis to gloves.

Sources:

Used in cutting oils, antifreeze, industrial greases, anticorrosive agents, cements and adhesives, detergents, and fungicides. The most common sources are gloves and shoes.

THIURAM MIX:

Thiuram mix is composed of equal quantities of the following four chemicals:

- ❖ Tetra methyl thiuram disulfide (TMTD)
- ❖ Di penta methylene thiuram disulfide (PTD)
- ❖ Tetra methyl thiuram monosulfide (TMTM)
- ❖ Tetra ethyl thiuram disulfide (TETD)

Sources:

These four chemicals are used primarily as accelerators in the production of rubber and as disinfectants, germicides, and insecticides in agriculture; in adhesives; in soaps and shampoos etc.

Thiuram mix is patch tested at a total concentration of 1% (0.25% of each component) in petrolatum .

Clinical Aspects

The most common sources of thiuram exposure leading to the development of sensitivity appears to be in rubber gloves and shoes. In allergic contact dermatitis due to gloves, thiurams are found to be the most common sensitizer, whereas in shoe allergy, thiurams are found to be the second most common allergen following mercaptobenzothiazole.

Glove dermatitis is a particularly vexing problem, since gloves are frequently used as protection during wet work by people with hand dermatitis of various types.

Glove-induced rubber component allergy is likely to persist as health care workers continue their usage of gloves as a part of "universal precautions" for prevention of the transmission of human immunodeficiency virus (HIV) and hepatitis infections.

BALSAM OF PERU:

Balsam of Peru is a natural, viscous, dark brown, liquid mixture from *Myroxylon pereirae* (*Toluifera pereirae*), a tree that grows in Central America. It is patch tested at a 25% concentration in petrolatum.

Clinical Aspects:

Myroxylon pereirae (Balsam of Peru) is a naturally occurring mixture of resins

(20% to 40%) in the essential oil called cinnamein. It is an aromatic compound used in pharmaceuticals, fragrances, and flavourings and has antifungal, antibacterial and scabicial activities.

Balsam of Peru is incorporated in the standard tray as a screen for fragrance sensitivity.

The International Fragrance Association endorses that Balsam of Peru must not be used as an ingredient in fragrances. Flare-ups of dermatitis in balsam of Peru-sensitive patients have occasionally occurred after the ingestion of spices.

Sources:

Used in cosmetics, pharmaceuticals, tobacco and food industries, baby products, flavours, spices and medicated substances.

FRAGRANCE MIX:

The fragrance mix (8%) includes common fragrance allergens like Cinnamic Alcohol, Cinnamic aldehyde, Hydroxycitronellal, Amylcinnamaldehyde, Geraniol, Eugenol, Isoeugenol, Oakmoss absolute each constituting 1%.

Common sites affected include the hands, face, axillae.

Sources:

Fragrances are found in a wide variety of products to enhance odor or mask undesirable odours in cosmetics, household products, industrial exposure and medicated creams, ointment and traditional Chinese medicaments

LANOLIN:

Lanolin (Wool alcohols) is a complex, natural substance got from the sebum of sheep that constitutes 5% to 25% of the weight of sheared raw wool.

Patch testing is done with lanolin alcohol (wool alcohols) at a 30% concentration in petrolatum.

Sources:

Lanolin is predominantly found in cosmetics, medicated creams, polishes and waxes, paper and cutting oil emulsions.

NEOMYCIN SULFATE:

Neomycin is the most common sensitizer in topical antibacterial preparations. The patch test concentration is 20% in petrolatum. It is the active agent in creams and ointments designed for skin use as well as otic and ophthalmologic preparations. Neomycin is frequently used in combination with other antibacterials like polymyxin and bacitracin, antifungals, and corticosteroids. It is also infrequently used in deodorants, cosmetics, soaps, pet foods, and veterinary products.

Many reports document higher levels of sensitivity in individuals with atopic eczema, stasis dermatitis, and external otitis. In addition to acute localized contact eczema, neomycin sensitivity can also become evident as contact

urticaria with anaphylaxis; as "dermal" papular dermatitis, especially in atopic persons; and as a systemic eczematous dermatitis in sensitized patients receiving oral neomycin.

NITROFURAZONE:

Nitrofurazone (Furacin) is a topical antimicrobial agent that is used primarily to treat skin disease, burns, and injuries and is a potent sensitizer. It is tested at a concentration of 1% in petrolatum.

Clinical Aspects

Nitrofurazone is used as a topical antibiotic and available as ointment, cream and powder medications.

p-CHLORO-m-CRESOL:

p-Chloro-m-cresol is a substituted phenol that is used more commonly in medicaments than in cosmetics because of its bad smell. It is patch tested as a 1% concentration in petrolatum.

Clinical Uses

p-Chloro-m-cresol is a preservative that is widely used in medicated products, cosmetics, adhesives and glues.

POLYETHYLENE GLYCOLS:

Polyethylene glycols (PEGs) are clear, viscous liquids and white, solid polymers of ethylene oxide. They are used extensively in cosmetics and topical medicaments. They are patch tested "as is" at 100% concentration.

Clinical Uses

Polyethylene glycol is used as a solvent in cosmetics, medicines and industry and is in cosmetics, topical medicines, detergents, toothpaste, contraceptives, insect repellents, paper coating and polishes.

PATCH TESTING

Introduction

Patch testing is the gold standard method of choice in the diagnosis of ACD. It is a proof of hypersensitivity. It is used both as a Screening test & provocative test. Fisher stated that correctly applied and properly interpreted patch tests are, the only scientific ‘proof’ of allergic contact dermatitis.⁸³

The patch test is used to detect hypersensitivity to a substance that is in contact with the skin so that the allergen may be determined and corrective measures taken. So many allergens can cause allergic contact dermatitis that it is impossible to test a person for all of them. In addition, a good history and observation of the pattern of the dermatitis, its localization on the body, and its state of activity are all helpful in determining the cause. The patch test is confirmatory and diagnostic, but only within the background of the history and physical findings.

Indications of patch testing⁸⁴⁻⁸⁹

- Allergic contact dermatitis syndrome
- Highly suggestive history or distribution
- Specific antigen or substance suspected
- Non eczematous contact dermatitis

- Dermatitis that flare or do not respond to treatment
 - Highly suspected
 - Atopic dermatitis
 - Stasis dermatitis
 - Hand dermatitis
 - Irritant contact dermatitis
 - Dyshidrotic eczema or pompholyx
 - Pustulosis palmaris et plantaris
 - Psoriasis of palms and soles
 - Less likely
 - Seborrheic dermatitis
 - Chronic tinea pedis or manum
 - Nummular eczema
- Occupationally related dermatitis
- Undiagnosed cutaneous problem
- Erythroderma
- Urticaria
- Photodermatoses
- Systemic contact dermatitis

Contraindications

- Patients with Immune deficiencies
- Patients on Immuno suppressive treatment
- Auto immune diseases

Principles of patch testing

- It is based on provoking inflammation on a limited skin area $< 1 \text{ cm}^2$.
- Only known substances in “standard concentration” must be used. For unknown substances open or “use” tests with controls done.
- If the dermatitis is acute, test must not be done
- The patient is informed to leave the patches on for 48 hours
- Initial reading must be taken at 48 hours and next readings are taken between 72 and 120 hours.
- The patient is informed not to shower, get the back wet, or engage in sports. Heavywork have to be avoided.
- It is difficult to distinguish irritant reaction from allergic reaction. Itching is more common in allergic reaction

Methodology

The principle of patch testing is to induce a delayed type of hypersensitivity response by stimulating previously sensitized person to specific amount and concentration of allergen and the response is measured. For patch testing, chambers or discs are used. Chambers are aluminium chambered. A non-irritant, non-allergenic fixing tape is used. The test is repeated if the fixing tape is peeled off. A well informed consent must be obtained from the patient.

Patch testing is not done in patients with active dermatitis. The patch testing must be delayed for at least two weeks until the test site has been clear. Corticosteroids and other immunosuppressive drugs like methotrexate and azathioprine should be stopped prior to patch testing. It's mandatory because it

reduces the positive patch test reaction. But prednisolone less than 15 mg will not reduce the positive patch test reaction.

Patch testing could be delayed for 28 days following sun bathing. The patches should not be exposed to UV light including sun light. Patch tests can be done in infants, young children when indicated, but the number of allergens tested can be decreased. Pregnant patients should not be patch tested because of adverse effects.

Instructions to the patient

1. Patch should be left in place for two days and two nights
2. Patient should not take bath or wash or wet the back during this period
3. Patient should be instructed to avoid tight underclothes
4. To avoid exercise or any heavy physical activity which causes excessive sweating
5. To avoid friction or rubbing and lying on the back because patches will become loose
6. To avoid scratching the patch test site. Report immediately if there is severe itching or irritation
7. To avoid exposure to sunlight/UV light
8. To come after 48 hours and 72/96 hours for patch test reading.

Patch test vehicles

Certain allergens may be applied to the skin as they are. They are mixed or dissolved in a vehicle to avoid an irritant effect. The test substances should be soluble in the vehicle.

Petrolatum is the vehicle, most commonly used, because it is occlusive and it prevents oxidation. The shelf-life of the allergen is prolonged. Water, olive oil, methyl ethyl ketone, alcohol acetone are the other vehicles used. Irritants like chloroform and benzene must be avoided. Petrolatum may not be ideal in hot climates. Petrolatum allergic reactions are rare.⁹⁰ Recently, Modified Plastibase has been tried.

Test material

The list of CODFI antigens used are from Indian Standard battery Series. Finn chambers on Scanpor tape is commonly used to apply patch test allergens. They are available in strips of five and ten which consist of aluminium discs.

LIST OF CODFI ANTIGENS (INDIAN STANDARD BATTERY)			
S.NO	Compound	Conc.%	Veh
1	Control	100.0	pet
2	Potassium Dichromate	0.5	Pet
3	Neomycin Sulphate	20.0	pet
4	Cobalt Chloride	1.0	pet
5	Benzocaine	5.0	pet
6	4-Phenylenediamine base (PPD)	1.0	pet
7	Parabens	15.0	
	_Methyl-4-hydroxybenzoate	3.0	
	_Ethyl-4-Hydroxybenzoate	3.0	
	_Propyl-4--hydroxybenzoate	3.0	
	_Butyl-4-Hydroxybenzoate	3.0	
	_Benzyl-4-Hydroxybenzoate	3.0	
8	Nickle Sulphate	5.0	
9	Colophony	20.0	pet
10	Gentamicin	20.0	pet
11	Mercapto Mix	2.0	pet
	_N-cyclohexylbenzothiazyl sulfenamide	0.5	
	_Dibenzothiazyl disulfide	0.5	
	_MorpholinyImercaptobenzothiazole	0.5	

12	Epoxy resin	1.0	pet
13	Fragrance mix	8.0	pet
	_Cinnamic Alcohol	1.0	
	_Cinnamic aldehyde	1.0	
	_Hydroxycitronellal	1.0	
	_Amylcinnamaldehyde	1.0	
	_Geraniol	1.0	
	_Eugenol	1.0	
	__Isoeugenol	1.0	
	_Oakmoss absolute	1.0	
14	Mercaptobenzothiazole(MBT)	2.0	pet
15	Nitrofurazone	1.0	pet
16	Chlorocresol	1.0	pet
17	Wool Alcohols	30.0	pet
18	Balsam of Peru	25.0	pet
19	Thiuram Mix	1.0	pet
	_Tetramethylthiuram monosulfide (TMTM)	0.25	
	_Tetramethylthiuram disulfide (TMTD)	0.25	
	_Tetraethylthiuram disulfide(TETD)	0.25	
	_Dipentamethylenethiuram disulfide (DPTD)	0.25	
20	Chinoform	3.0	pet
21	Black rubber mix	0.6	

	_N_isopropyl-N-phenyl-4-phenylenediamine	0.1	
	_N--cyclohexyl-N-phenyl-4-phenylenediamine	0.25	
	_N-N-diphenyl-4-phenylenediamine	0.25	
22	P-tert Butylphenol formaldehyde resin	1.0	pet
23	Formaldehyde	1.1	aq.
24	Polyethylene Glycol 400(PEG 400)	100.0	aq.
25	Plant Antigens		
	(a)Parthenium hysterophorus		

Allergens Storage

Allergens are kept in the dark, at 4 degree C, because certain allergens on exposure to sunlight lose their stability. Expiry date is labelled in commercial preparations. If they are not refrigerated properly, homogeneity of allergens may be lost.

Patch test concentrations

Choice of the allergen is of fundamental importance because it is selected by exhaustive experience. The concentration of the allergen used for patch testing is always greater than the concentration that caused dermatitis.

Patch test dose

- Allergens are kept in a vehicle in disposable syringes of length 5 mm
- Finn chamber is of standard size

Patch Test site

Back is the preferred site. Both allergic and irritant responses are readily incited on the upper back. Stronger reactions occur on the lateral aspect of the upper arm than on the medial aspect.

Reactivity of various test sites⁹¹

Test site	Irritant reactions (%)	Allergic reactions (%)
Upper back	100	100
Upper arm	52	72
Lower back	50	95
Fore arm	38	74
Thigh	36	50

Patch tests reading

Patch test sites are marked with permanent ink or fluorescent pointers on dark skins. A 48 hour contact time in an occlusive patch is adequate to incite a reaction. 1st reading done at 48 hrs which is the optimum time to elicit positive reactions. 2nd reading done at Day 4 to 7 where immediate irritant reactions subside and reactions of most slow allergens fully develop.

Neomycin and corticosteroids particularly give late reactions. Readings taken at Day 5–7 will infer if the contact sensitization is weak or partially ‘forgotten’, or if the allergen absorbed is inadequate.

Interpretation of results

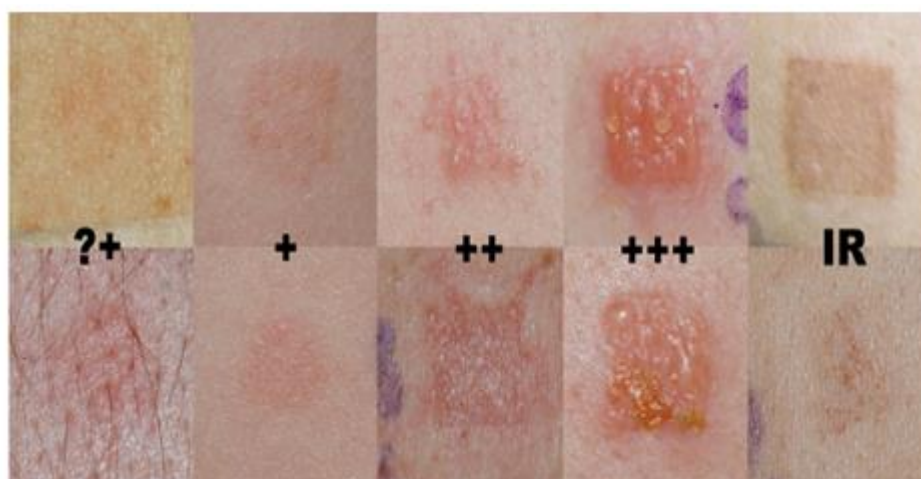
It is based on International Contact Dermatitis Research Group (ICDRG)

Criteria.⁹²

ICDRG

-Ve	No Reaction	Negative
?/+ve	Doubtful reaction	Faint erythema only
+	Weak positive reaction	Palpable erythema, infiltration, possibly papules
++	Strong positive reaction	Erythema, infiltration, papules, vesicles
+++	Extreme positive reaction	Intense erythema and infiltration and coalescing vesicles and bullae.
IR		Irritant reaction of different types

NOTATION OF POSITIVE RESULT (ICDRG)



Relevance of patch testing⁹³

A positive reaction of the patch test does not correspond with the diagnosis of ACD. Some patients with +ve patch test never experience clinical symptoms. Whether +ve patch test results really explain patient's symptoms is identified. COADEX classification system is very useful to assess the relevance.

COADEX

CODE	MEANING
CURRENT	Exposed currently to allergen before dermatitis developed, improvement after cessation of exposure
OLD	Past episodes of dermatitis after exposure to the allergen
ACTIVE SENSITIZATION	Presents with active sensitization reaction
DOUBTFUL	Relevance difficult to assess, no traceable relationship between positive test and disease
EXPOSED	H/O of previous exposures did not cause dermatitis
CROSS REACTION	Positive test is due to cross reaction with another hapten

If patient allergic has positive patch test, relevance established by carefully re-examining the patient's history, distribution of rash and materials with which there has been contact. If relevance clearly established, then avoidance advice can be given. If relevance is uncertain or impossible to

ascertain patients will need to be advised on the potential sources of all their allergies for future reference, and if necessary to reassess their exposures

Reasons for false-positive reactions

- Allergens in excessive concentration
- Allergen applied in increased amount
- If the allergen dispersed unevenly
- Impure patch test substance (contaminants)
- If the vehicle is irritant
- Adhesive tape reactions
- Pressure effect of hard materials
- ‘Angry back’ phenomenon
- Active dermatitis at patch-test site
- Active dermatitis at remote site
- Artefact

Reasons for false-negative reactions

- Allergens in Inadequate concentration
- Less quantity of allergen applied
- Inappropriate vehicle
- Reduced grip of patches
- Patches applied at incorrect site
- Readings taken too early
- If Patch test allergen had degraded
- Pre-treatment of patch-test site with topical corticosteroids

- UV irradiation of tested area
- On immunosuppressants

Potential complications of patch testing

- Irritant reactions to patch test substance

Irritation may be avoided by using standard procedures. Substances should be investigated toxicologically and allergologically.

- Edge Effect

The reaction is more at the periphery of the patch test but at the centre there is little or no reaction. This called as edge effect. Edge effect is due to increased concentration of liquid which act as an irritant. The edge effect will disappear following removal of the patch.

- Active sensitization

A risk of sensitization is there following patch testing. A patch test reaction occurring after 7 days of patch testing may denote delayed expression of a previous sensitivity. At about 3 weeks, active sensitization can occur as a strong positive test Cronin clearly states “active sensitization is a complication of patch test but it is not a hazard. It should not be used as an excuse by the indolent for eschewing this investigation. To reject patch testing is the greater disservice to the patient”.⁹⁴

- Koebner Phenomenon

In a patient who is having psoriasis or lichen planus, a positive test reaction may reproduce these dermatoses at the test site called the Koebner Phenomenon.

- Pruritus
- Folliculitis
- Allergen leakage on to clothing, particularly dyes
- Localized flare up of dermatitis
- Flare up of dermatitis at previous exposed sites
- Widespread flare up of dermatitis
- Pigmentation or depigmentation
- Scarring, Necrosis
- Anaphylaxis (very rare)

Irritant Patch Test Reaction

Causes for irritant patch test reactions

- ◆ Hyperirritability of the skin
- ◆ Application of an irritating concentration of a test substance

Spill over effect:

One positive test has influenced another test to appear positive.

Certain rules must be followed to avoid irritant reactions

- ◆ Patch testing should be carried out only on the normal skin
- ◆ Avoid patch testing with nonstandard substances other than standard series
- ◆ Irritating concentration of test materials should be avoided
- ◆ Cleansing the skin with soaps or solvents should be avoided

Janus reaction

It is a non papulovesicular patch test reaction consisting of palpable erythema and oedema. The significance of these reactions may be determined over time, based on patient's outcome. This mild reaction may or may not be relevant, so further correlation is needed to establish contact allergy. Irritant responses are held responsible to induce stronger reactions at 48 hours than at 96 hours. This is called crescendo-decrescendo effect.

Photo patch testing

Photocontact allergens, cause ACD, when exposed and triggered by sunlight. The UV rays in sunlight are responsible for photo contact dermatitis.

With photo-patch testing, duplicate sets of patches are placed on symmetrical sites of the back of trunk. One set of duplicate patches is irradiated with UVA light and the non-irradiated set serves as a control. . The skin is examined in the usual way (after two and four days) and if the irradiated site shows positive reaction and the non-irradiated site shows a negative one, contact photo allergy is present

Indications of photo patch testing

- ◆ If the eczema is present in sun light exposed areas
- ◆ If history of a reaction to sunscreens +

OTHER TESTS TO TEST ACD

TRUE TEST

Thin layer rapid use epicutaneous test. Allergens are available ready to use form coated onto polyester patches in a hydrophilic vehicle. It contains only

24 allergens of standard series. It is an expedient, expensive and manageable method.⁹⁵

Repeat Open Application test (ROAT)

The test substances are applied twice daily for 28 days or till an eczematous change occurs. The test site preferred is, the upper arm or flexor surface of the forearm. An area of five cm² should be employed.⁹⁶

Open use test or provocative use test

This test is mainly used for non-irritating substances such as cosmetics. The suspected substance is rubbed onto normal skin in the antecubital fossa. The test substance is applied two times daily for 1 week, over an area around 3 cm in diameter. If no reaction occurs, the test measured is negative. False negative reactions are common in this method. In contact urticaria, this test is advocated.

Usage Test

Usage test is performed when an open patch test or closed patch test is negative but patients history is reliable of contact sensitization.

Intradermal Tests

Intracutaneous tests have been done with simple chemicals, but it is primarily used for investigational issues. This procedure has evidence dependable for nickel and corticosteroid allergy.⁹⁷

PREVENTION of ACD

Principles of prevention can be categorized as

◆Primary ◆Secondary ◆Tertiary

Primary prevention emphasizes on the stimulation of contact sensitization and exposure control. Secondary prevention is interrelated to effluent phase. Tertiary prevention is steps taken for management of allergic contact dermatitis.

The measures taken are:

- ✓ Restraint and replacement of Allergen
- ✓ Legal and regulatory measures
- ✓ Corporate responsibility
- ✓ Domestic precautions and hygiene
- ✓ Barrier method for preventing contact to allergen
- ✓ Proper education

PROGNOSIS

The prognosis of ACD mainly relies on how the patients avoid repeated or continued exposure to the suspected allergens. In case of nickel/chromate allergy, there occurs poor prognosis, because of the omnipresence of the allergen in the environment. If the chances of avoiding the allergen is easy, the prognosis is good. If the barrier function of the skin is compromised, the chances of new sensitivities are increased. Contact sensitization persists, once acquired.

Chronicity of contact dermatitis is attributed to the following factors.

1. Impaired barrier function of skin
2. Secondary infection
3. Allergens ingestion
4. Inappropriate treatment

5. Auto sensitization
6. Constitutional factors
7. Inherent tendency of eczemas to become chronic
8. Stress
9. Atopy

TREATMENT

I) ADVICE REGARDING AVOIDING ALLERGEN

When the diagnosis of ACD is made, the possible exposure sources are explained to the patient and advice given regarding avoidance of the allergen. Patients can be suggested removal of plants (parthenium) from the close environment, not to use fashion jewelry in case of nickel dermatitis and substitutes with plastics can be tried. Occupational causes of dermatitis must be well explained to the patient and advice regarding to wear gloves and protective clothing recommended. Patients who are thiuram, black rubber mix, mercaptobenzthiazole sensitive and have hand/ foot dermatitis , rubber gloves are avoided and vinyl gloves suggested.

The allergen sources can be given in written information. Patients may be cautioned, on re-exposure, the dermatitis will relapse.

II)TREATMENT

The mainstay of treatment is topical corticosteroids. High potent topical corticosteroid should be used in acute, severe, localised dermatitis. Mild to moderate potent steroids given in chronic or widespread contact dermatitis.

1. Moisturizers/ Emollients /Soap substitutes given

2. Hypoallergenic tape for Fissures of fingers, palms and soles
3. Saline soaks with Aluminium acetate or Silver nitrate given for weeping dermatitis.
4. Topical Tacrolimus and Pimecrolimus can be tried.
5. Secondary infection is treated with antibiotics.
6. Antihistamine for pruritis.
7. Systemic steroids are inevitable in cases of sensitization / generalized dermatitis.
8. Immunosuppressive drugs like Azathioprine and Cyclosporin are tried for disabling cases.

AIMS AND OBJECTIVES

1. To study the incidence of ACD among patients of dermatology OPD, TVMCH
2. To study the various morphological patterns of ACD with different allergens
3. To determine the proportion of positive patch tests in adults with allergic contact dermatitis and thus determine the etiology of ACD
4. To assess the clinical severity of disease in correlation with patch tests grading
5. To find out the percentage of occupational contact dermatitis among patients with ACD

MATERIALS & METHODS

Study design

This is an observational, prospective, single group, open labelled clinical study.

Study population and Study period

A total of 100 patients clinically diagnosed as Allergic Contact Dermatitis who attended the Department of Dermatology, Tirunelveli Medical College Hospital during the period June 2014 to August 2015 were included in the study.

Inclusion criteria

1. Age : 18 years to 70 years
2. Sex: Both males and females
3. Patients with clinical diagnosis of ACD

Exclusion criteria

1. Patients with immune deficiency diseases
2. Patients who are on immunosuppressive treatment
3. Patients with autoimmune diseases
4. Patients with acute dermatitis
5. Pregnant & lactating mothers

Methodology

A total of 100 patients clinically diagnosed as ACD were recruited in the study. Both informed and written consent was obtained from patients to include them in the study, to do patch tests and to take clinical photographs.

A thorough clinical history was elicited, regarding the nature and duration of symptoms, contact with any specific allergen with respect to their occupation

and present clinical scenario. Also history about associated medical illness, personal & family history of atopy were obtained.

A detailed dermatological examination was carried out. The morphological pattern, extent of skin lesions and the presence of oozing, crusting and lichenification were noted down. Skin lesions other than ACD were also recorded.

Patients were subjected to routine blood investigations including Complete Hemogram, LFT, RFT & blood sugar. Patch test was performed for all 100 patients who were included in the study. For patients with acute eczema, patch test was done after 2 weeks when the lesions got cleared.

We did Patch test by using INDIAN STANDARD SERIES BATTERY, which was commercially available at Systopic laboratories, New Delhi. These allergens were applied on Finn chambers and strapped on the back of the patients with hypo allergenic tapes. The patches were kept undisturbed for 48 hours. Patients were advised to avoid strenuous hard work, showering and sunlight exposure. After 48 hours, the finn chambers were removed and the squares representing each chamber was marked using a marker pen. Reading was taken after half an hour. A second reading was taken after 72 hours to confirm the presence of allergic reaction.

Patch test results were interpreted according to International Contact Dermatitis Research Group (ICDRG) criteria.

Notation	Interpretation
--	Negative
?+	Doubtful reaction: faint erythema only
+	Weak positive reaction: palpable erythema, infiltration, papules
++	Strong positive reaction: erythema, infiltration, papules, vesicles
+++	Extreme positive reaction: intense erythema, and infiltration and coalescing vesicles
IR	Irritant reaction: no infiltration, lack of itching, sharp borders
NT	Not tested

Clinical photographs were taken at the time of clinical diagnosis of ACD, during patch tests procedure and at the time of reading patch tests.

The patients were treated with topical emollients, immune modulators, topical and systemic steroids. Patients were followed up periodically and they were advised to avoid exposure to the particular allergen(s) and the importance of changing their occupation if needed.

OBSERVATIONS & RESULTS

The results of the prospective study done in 100 patients of ACD during the study period from June 2014 to August 2015 is discussed below.

INCIDENCE OF ACD

During the study period between September 2014 to August 2015 the following observation was made.

Total OPD census in the dermatology department - 36569.

New Registration for adult patients - 16034.

Adult patients newly diagnosed as Allergic contact dermatitis - 792.

$$\text{Incidence} = \frac{\text{No. of cases newly diagnosed as ACD during study period} \times 100}{\text{Number of new registration for OPD during same study period}}$$

$$= 792 \times 100$$

$$\frac{\quad}{16034}$$

$$= 4.94 / 100 \text{ OPD cases.}$$

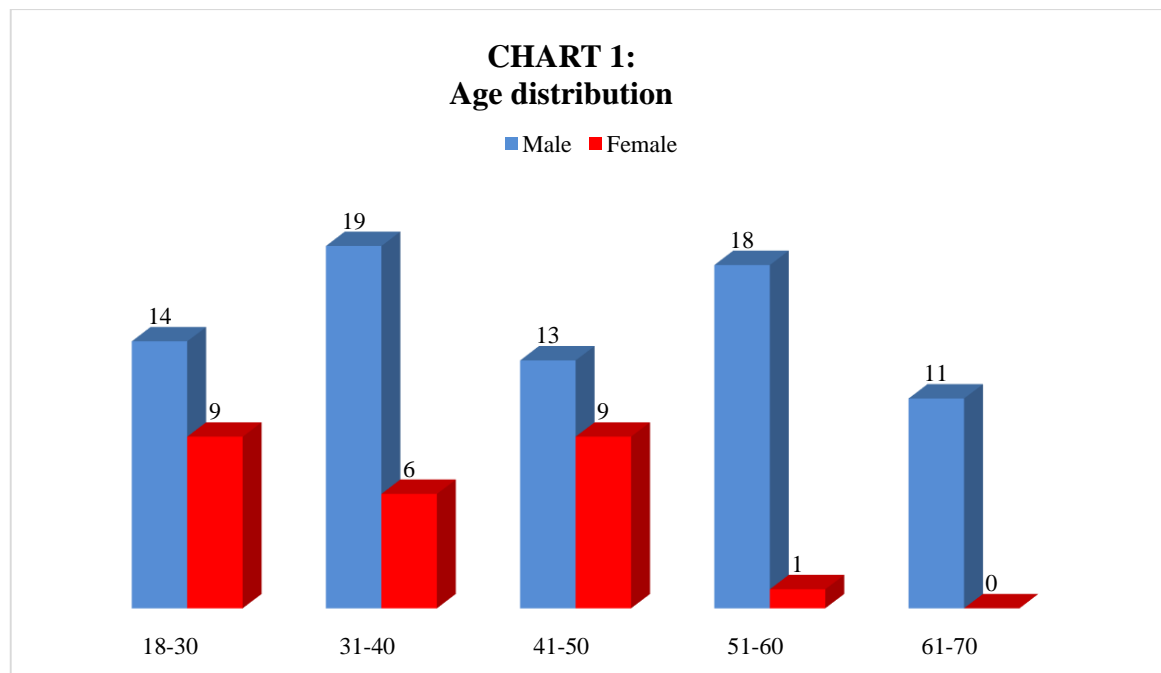
Hence in the present study the incidence of allergic contact dermatitis was estimated to be 4.94/100 newly registered adult OPD cases.

Study Participants

Newly diagnosed allergic contact dermatitis patients were recruited in the study by consecutive sampling method. Those participants who were diagnosed clinically as ACD during the month of June 2015 to August 2015 were recruited consecutively until the sample size of 100 was achieved.

TABLE 1: COMPARISON OF AGE DISTRIBUTION WITH GENDER

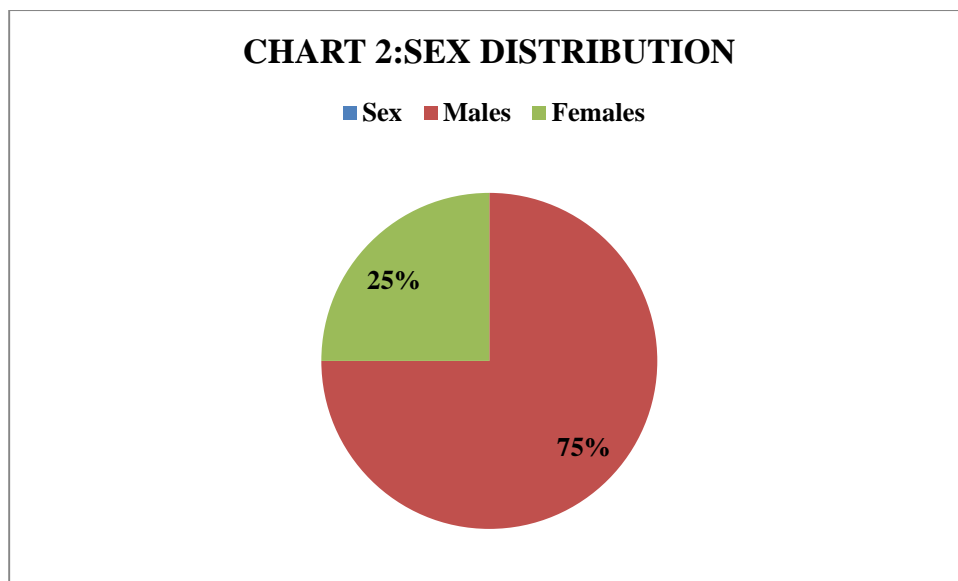
Age group	Males n=75	Females n=25	Total n=100
18-30	14	9	23
31-40	19	6	25
41-50	13	9	22
51-60	18	1	19
61-70	11	0	11
Total	75	25	100



ACD was observed highest in the age group of 31 to 40 years and second highest observed in age group <30 years (Table-1, Chart-1). The

youngest patient in the study was 18 years and the oldest was 70 years. The mean age observed in the study was 42.56 years with Standard deviation 13.94 years. ACD was found to be common in the fourth decade among males and in the third and fifth decade among females.

SEX DISTRIBUTION



As depicted in Chart 2, out of 100 cases in our study, 75 % were males and 25 % were females. The Male to Female ratio was 3:1.

TABLE 2: CLINICAL DIAGNOSIS OF ACD WITH GENDER WISE DISTRIBUTION

Clinical Diagnosis	M	F	Total (%)
ACD to Parthenium	18	7	25
ACD to Cement	35	3	38
ACD to Nickel	1	7	8
ACD to Hair Dye	6	2	8
ACD to Oils & Greases	5	0	5
ACD to Leather Foot wear	4	1	5
ACD to Rubber Foot wear	2	0	2
ACD to Paints	1	0	1
ACD to Textiles	0	2	2
ACD to Deodorant	1	1	2
ACD to Plaster	1	0	1
ACD to multiple etiologies(Plants / Dye / Nickel)	1	2	3
Total	75	25	100

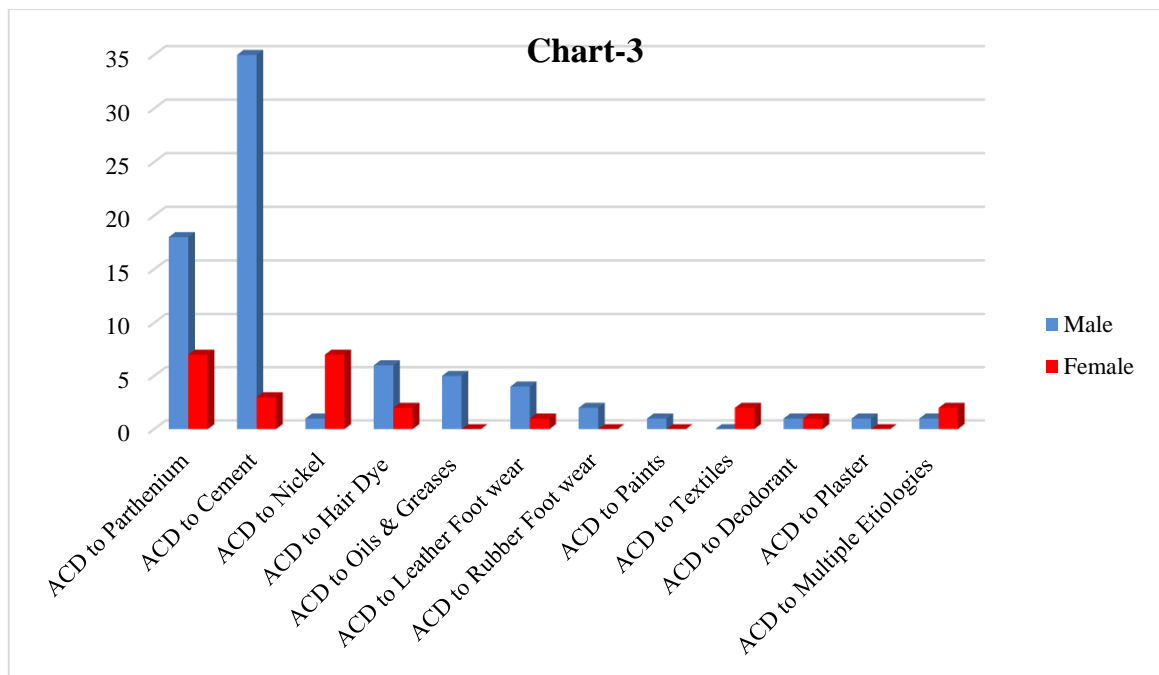


TABLE 3: AGE & SEX WISE DISTRIBUTION OF ACD TO MAIN ALLERGENS

Age group	ACD to Cement			ACD to Parthenium			ACD to Nickel			ACD to Hair Dye		
	M	F	T	M	F	T	M	F	T	M	F	T
18-30	7	1	8	0	1	1	1	4	5	1	0	1
31-40	10	1	11	1	0	1	0	3	3	1	1	2
41-50	9	1	10	2	5	7	0	0	0	1	1	2
51-60	6	0	6	7	1	8	0	0	0	3	0	3
61-70	3	0	3	8	0	8	0	0	0	0	0	0
Total	35	3	38	18	7	25	1	7	8	6	2	8

As depicted in the Table 2 & 3 and chart 3, Out of 100 cases diagnosed clinically, ACD to Cement tops the list with 38 cases (M=35; F=3) followed by ACD to Parthenium in 25 cases (M=18; F=7), ACD to Nickel in 8 cases (M=1;F=7), ACD to Hair dye in 8 cases (M=6; F=2) and other etiologies in remaining 21 cases.

Males were more commonly affected than females in ACD to Parthenium and Cement. Females were commonly affected in ACD to Nickel Group.

TABLE 4: MORPHOLOGICAL PATTERNS OF ACD WITH DIFFERENT ALLERGENS

Allergens		Hands and/or Feet	ABCD	Mixed	Contact site	Others	T
1	Cement	27	-	4	-	7	38
2	Parthenium	3	9	5	-	8	25
3	Nickel	-	-	-	7	1	8
4	Hair dye	-	-	-	8	0	8
5	Rubber footwear	-	-	-	2	-	2
6	Leather footwear	-	-	-	5	-	5
7	Others	5	-	1	6	2	14
	Total	35	9	10	28	18	100

As per Table 4, the various morphological patterns of ACD due to various allergens observed in our study were: Hands and/ or Feet pattern was most frequently noted in 35 cases, and among these cases, ACD to Cement top the list with 27 (77%) cases. ABCD Pattern observed in 9 cases and all were in the ACD to Parthenium Group. Mixed Pattern i.e ABCD with CAD[3 cases], CAD with AD [1 case], ABCD with AD[1 case], Hands & Feet with trunk involvement[3 cases], Hands & Feet with Atopic dermatitis pattern[2 cases] were observed in the study thus attributing to 10% of the total study cases. Contact site eczema observed in the study was 28 cases. Hair dye dermatitis dominated the list with 8(28%) cases. Nickel dermatitis had 7(25%) cases with allergy to saree pin(2 cases), watch strap(2 cases), jewels (3 cases). Other morphological patterns were observed in 20 cases. Patterns like Prurigo Simplex (1 case), Follicular pattern (2 cases), Lichenoid pattern (1 case) was seen among cement dermatitis cases. One case of Pompholyx pattern was observed in ACD to Paints. 3 cases of Parthenium dermatitis and 1 case of Cement dermatitis had Exfoliative dermatitis

pattern. 4 cases of Parthenium dermatitis had CAD pattern. AD pattern seen in 2 cases of Parthenium dermatitis and 1 case of Cement dermatitis.

OCCUPATIONAL VS NON OCCUPATIONAL CAUSES IN RELATION TO ACD

CHART-4: OCCUPATIONAL VS NON OCCUPATIONAL CAUSES IN RELATION TO ACD

■ OCCUPATIONAL ■ NON-OCCUPATIONAL

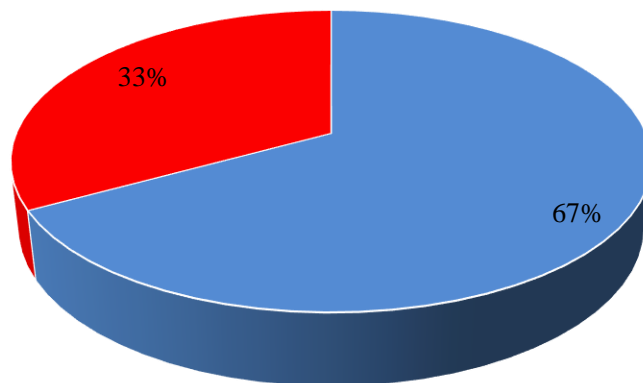
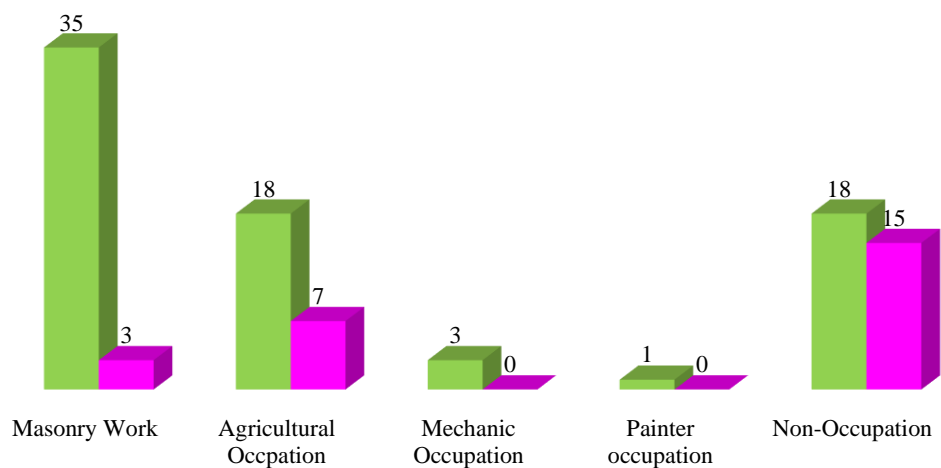


CHART 5: OCCUPATIONAL VS NON OCCUPATIONAL CAUSES IN RELATION TO ACD

■ Male ■ Female

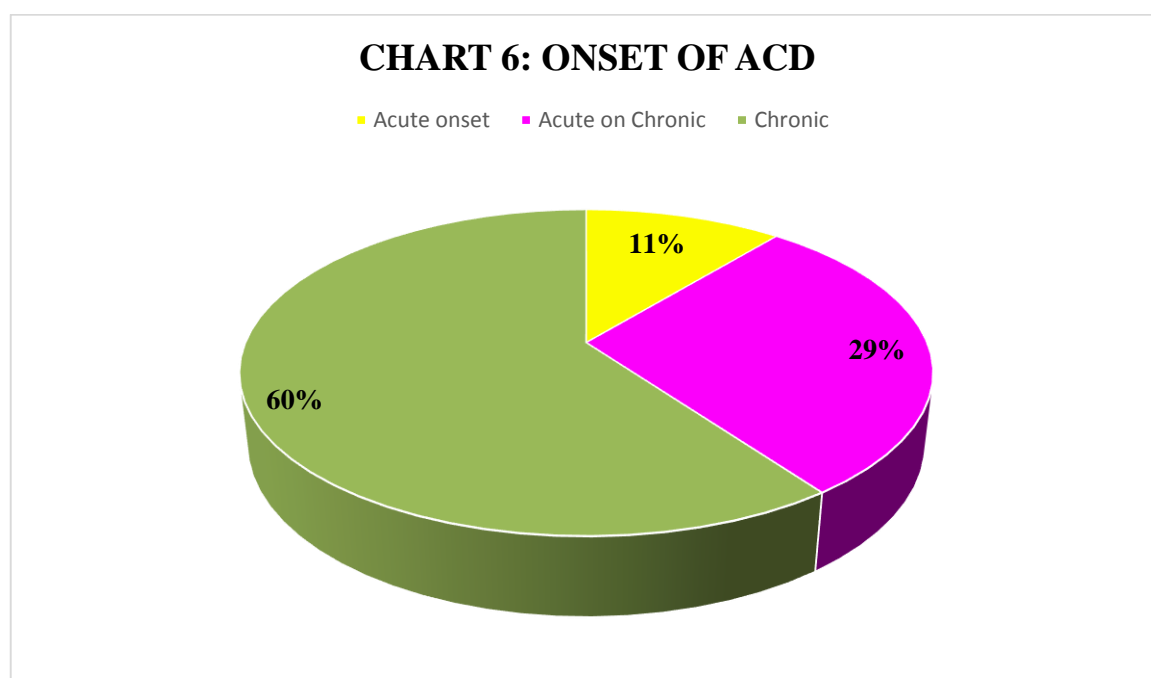


As seen in chart 4&5, out of 100 cases of ACD , 67% of cases were of occupational in origin and 33% were not related to their occupation but due to the substances they handle or come into contact with their daily life.

Out of 67 cases of OCD, 38 (56%) of them (Males=35 & Females=3) were related to masonry work. Majority of the cases were between 31 to 40 years age group. And 25 (37%) cases, who come under ACD to Parthenium group, were related to agricultural work and gardening and among these 25 (37%), 18 were males and 7 were females .Maximum number of cases were concentrated between 50 to 70 years age group. And 3 male cases were mechanic, and they had ACD to Oils & Greases. And one male patient was painter by occupation and he was clinically diagnosed to have ACD to paints which were also confirmed by patch testing.

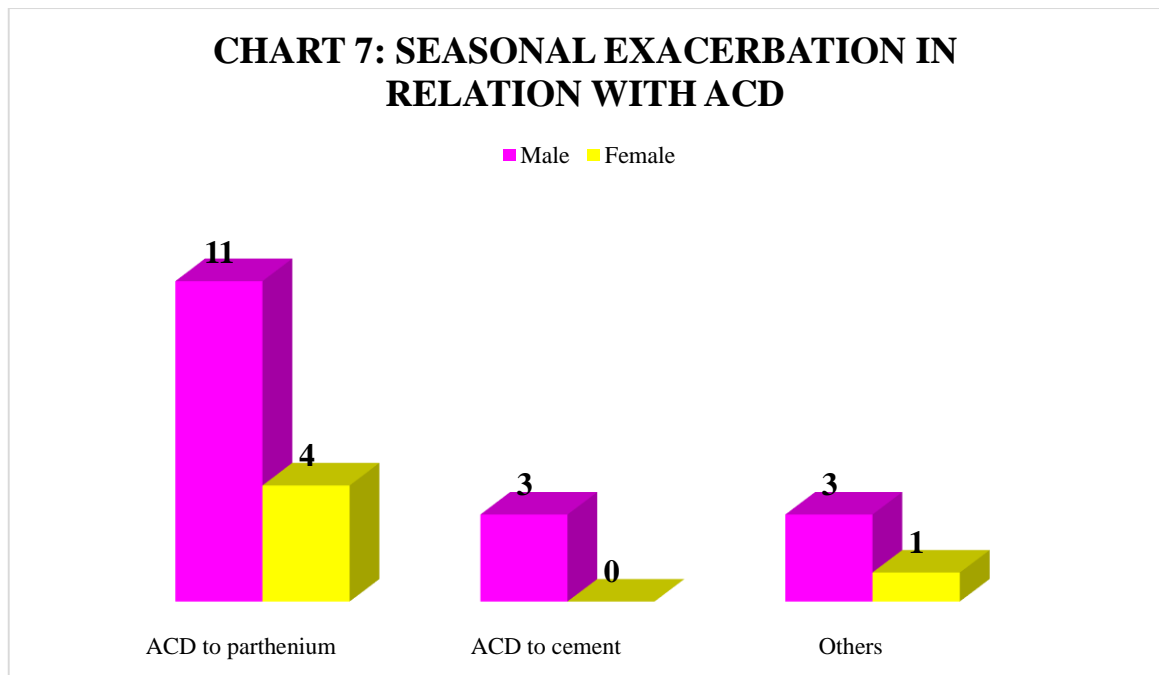
33 cases of Non occupational group had nickel, hair dye, foot wear and textile dermatitis and they were house wives, students, clerk, lab technician and medical representative.

ONSET OF ACD

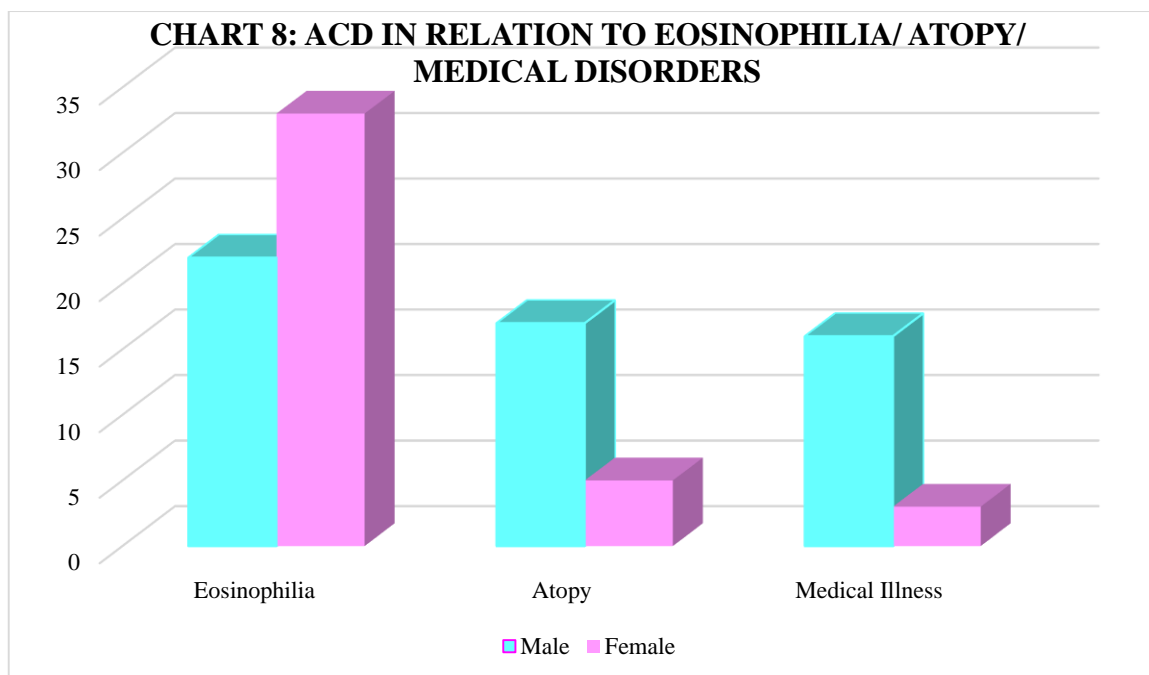


As per chart-6, out of 100 patients, 11% had acute onset of illness; 29% had acute on chronic illness and 60% had chronic illness. Among the acute onset group, 8 cases were to Hairdye. Nickel, Footwear and Plaster had one each. In 29 % of ACD cases who had acute on chronic onset, 19 cases were (M-17 & F-2) to Cement, 6 to Parthenium. ACD to Leather footwear, Rubber footwear, Paints and Oils & greases had one each. Among the chronic onset group, which constitutes 60%, ACD to Parthenium and Cement had 19 cases each and remaining 22 cases falls under other groups.

Seasonal exacerbation in relation with ACD:



Out of 100 cases (chart 7), seasonal variation was encountered in 22% of cases, out of which 15 cases in ACD to parthenium group had summer/spring exacerbation. Three cases of ACD to Cement, 2 cases of ACD to Nickel and single cases of ACD to Footwear and Paints had summer exacerbation.



As summarised in chart-8, Out of 46 cases who had eosinophilia, 68% of cases were ACD to Parthenium, followed by ACD to Cement in 47% , ACD to Nickel in 37.5 % and others amounting to 29% of total cases studied. Atopy was seen in 22% of cases. It was commonly observed in ACD to Parthenium cases amounting to 11% followed by ACD to Cement accounted for 9% of the cases. Out of 100 cases studied , Medical disorders were noted in 19% of cases and increased prevalence was encountered in the age group 51-60 years.

TABLE 5: PROPORTION OF POSITIVE PATCH TESTS IN COMPARISON WITH CLINICAL DIAGNOSIS

Allergens	Provisional Diagnosis	Patch test Allergens +ve	No reaction	Proportion
Cement	38	35	3	92%
Parthenium	25	23	2	92%
Nickel	8	4	4	50%
Rubber footwear	2	2	0	100%
Leather footwear	5	5	0	100%
Hair dye	8	6	2	75%
Textile	2	2	0	100%
Plaster	1	1	0	100%
Paints	1	1	0	100%
Oils/greases	5	5	0	100%
Deodorant	2	2	0	100%

In our study, out of 100 cases of ACD, the proportion of positive patch test reading was found to be 92% both in ACD to Cement as well as ACD to Parthenium group. The lowest positive patch test i.e 50% was encountered in ACD to Nickel. ACD due to other allergens showed 100% positivity with patch tests.

TABLE 6: CLINICAL SEVERITY & PATCH TEST SEVERITY CORRELATION

		Patch Test Severity		Total
		Mild	Severe	
Clinical severity	Mild	64	8	72
	Severe	12	5	17
Total		76	13	89

CHI-SQUARE TESTS

	Value	Exact Sig (2-sided)
McNemar Test		.503 ^a
N of Valid Cases	89	

*a Binomial distribution used.

SYMMETRIC MEASURES

	Value	Asymp. Std. Error ^a	Approx. T ^b	Approx. Sig
Measure of Agreement Kappa	.201	.125	1.922	.055
N of Valid Cases	89			

As inferred from the above tables, when the clinical severity of the disease was assessed with the severity of patch test reading, there was no correlation between both. And this was also statically proved by kappa agreement.

Indian Standard Series Battery- Patch test Allergens positivity

Out of the 100 cases in the study population, single allergen positivity was seen in 64%, multiple allergens positivity was seen in 25% and no reaction observed in 11% of cases.

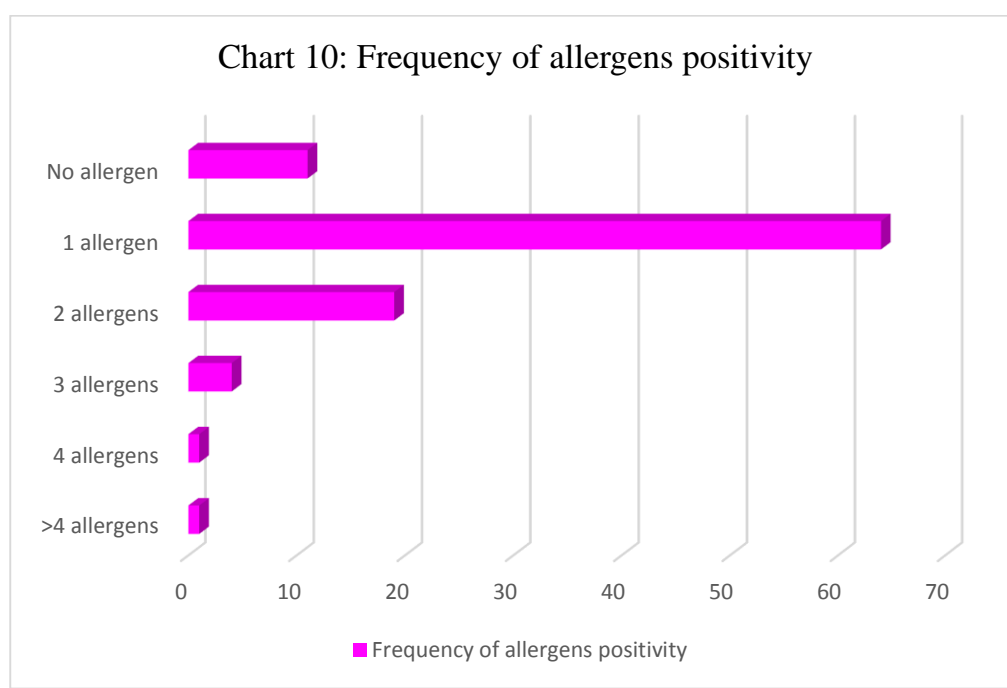
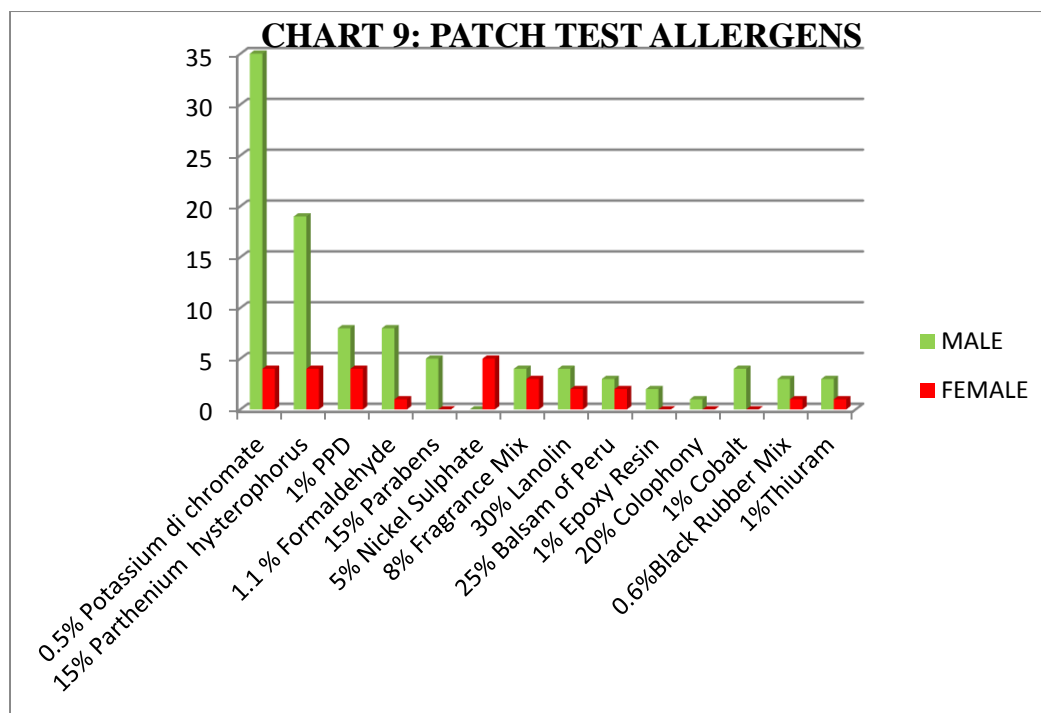


TABLE 7: PATCH TEST POSITIVITY OF ALLERGENS

Clinical diagnosis	PATCH TEST POSITIVITY OF ALLERGENS									Total
	Single allergen	Allergen	Two allergen	Allergen	Three allergen	Allergen	Four allergen	Allergen	No reaction	
ACD to Cement	27	KCr(26) Co(1)	7	KCr+Co(3), KCr +PPD(3) , KCr + parthenium(1)	-		-		3	37
ACD to Parthenium	22	Parthenium	-	-	-		-		3	25
ACD to Nickel	4	Ni			-		-		3	8
ACD to Hairdye	6	PPD			-		-		2	8
ACD to Oils & Greases	1	Parabens	4	Parabens + Formaldehyde	-		-		-	5
ACD to Clothing	2	Lanolin	-		-		-		-	2
ACD to Plaster	1	Colophony	-		-		-		-	1
ACD to Deodorant	-		2	FM+BP	-		-		-	2
ACD to Footwear	-		4	KCr+Formaldehyde(2), KCr+Lanolin(2)	2	BR/ Thiuram / PPD(1) KCr +Lanolin +Formaldehyde(1)	1	Epoxy/ MBT/ Thiuram/ BR	-	7
ACD to Paints	-		-		1	KCr + formaldehyde+ epoxy resin	-		-	1
ACD to multiple etiologies	1	PPD	2	FM+BP, Ni+ FM	1		-		-	
Total	64		19		4		1		11	99
KCr: Potassium dichromate Co: Cobalt FM: Fragrance mix BP: Balsum of Peru BR: Black Rubber Mix Ni: Nickel										

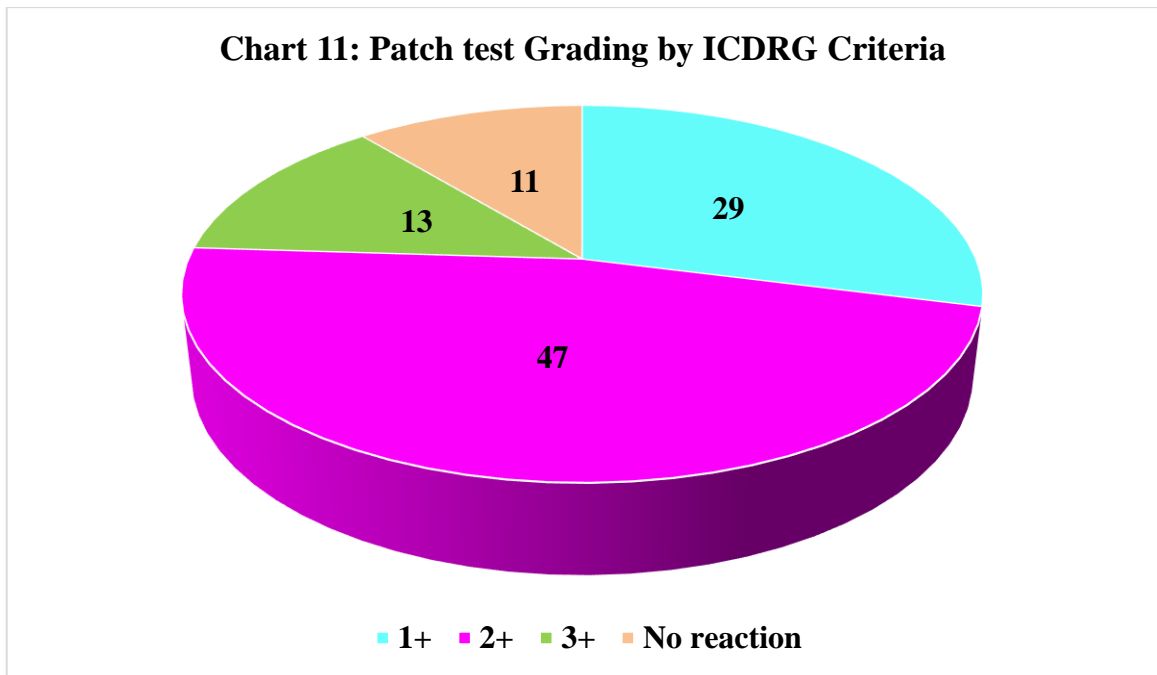
As inferred from the table 7 and chart 9, in the study population, the allergen 0.5% Potassium di chromate was positive in 39 cases which was highest in the study, followed by 15% Parthenium hysterophorus in 23 cases, 1% Paraphenylenediamine base in 12 cases, 1.1% Formaldehyde in 9 cases, 15% Parabens in 5 cases, 5% Nickel sulphate in 5 cases, 1% Cobalt chloride in 4

cases, 1% Epoxy Resin in 2 cases and 20% Colophony in 1 case. Colophony (1 Case) was the least observed in the study.

As per chart 10, single allergen was positive in 64 cases, 2 allergens were positive in 19 cases, 3 allergens were positive in 4 cases, 4 allergens were positive in 1 cases and >4 allergens positive in 1 case. Two allergens were positive in 19 cases. Out of 19 cases, two allergens positivity was seen in 7 cases of cement dermatitis and they were potassium di chromate with cobalt chloride in 3(7.8%) cases, potassium di chromate with paraphenylenediamine in 3 cases and potassium di chromate with parthenium in 1 case. Parabens and formaldehyde positivity was seen in 4 cases of ACD to Oils & greases. Potassium di chromate and formaldehyde positivity (2 cases), Potassium di chromate and lanolin (2 cases) were present in ACD to footwear. Three allergens positivity was seen to a combination of BR mix/thiuram mix/PPD in 2 cases and to potassium di chromate/lanolin/formaldehyde in one case of ACD to footwear cases and to potassium di chromate/formaldehyde/epoxy resin in ACD to paints. Positivity to a combination of four allergens (epoxy resin/MBT/thiuram/BR mix) was seen in a single case of ACD to rubber footwear. A single case had >7 allergens positivity possibly due to excited skin syndrome.

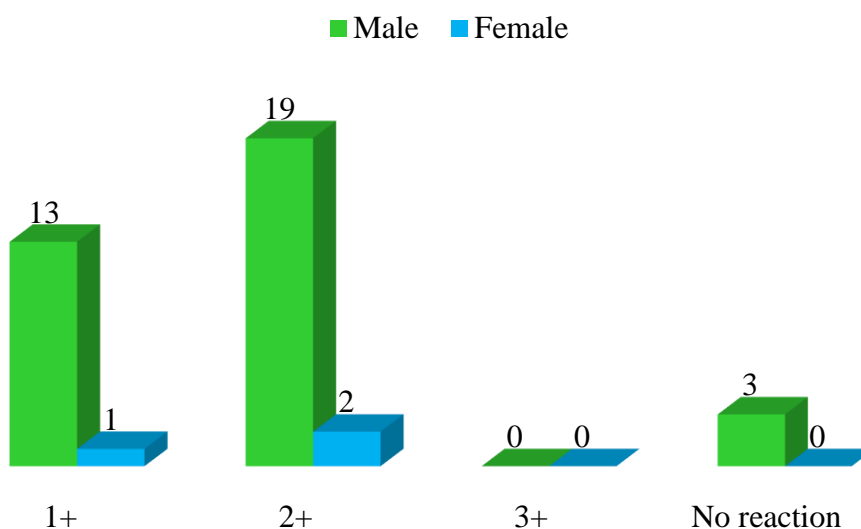
TABLE 10: PATCH TEST GRADING BY ICDRG CRITERIA:

ICDRG scoring	Cases
1+	29
2+	47
3+	13
No reaction	11



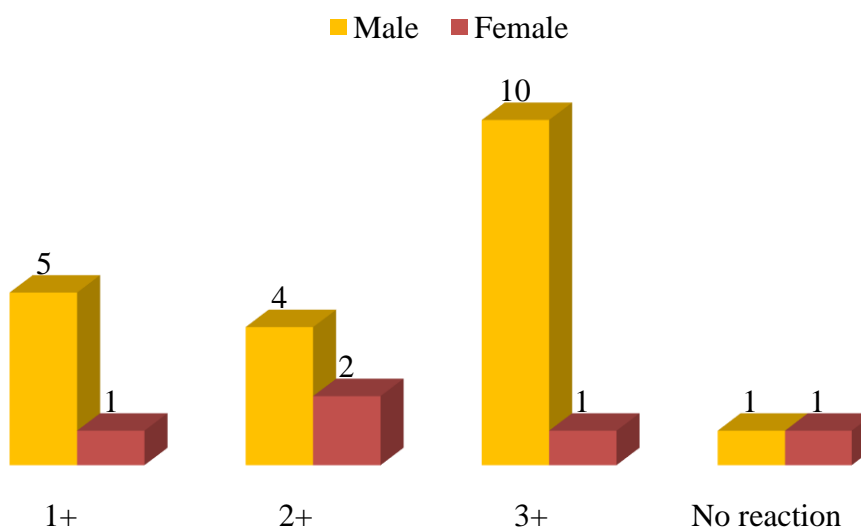
As depicted in Table 10 & chart 11, the patch test results were read according to ICDRG system. 29% had 1+ reading, 47% had 2+ reading, 13 % had 3+ reading and No reaction was observed in 11%.

CHART 12: PATCH TEST GRADING IN ACD TO CEMENT



Among 38 cases of ACD to Cement, 2+ reading was observed in 21(55 %) of cases, 1+ reading in 14(36.8%) cases and no reaction in 3(7.89%) cases.

CHART 13: PATCH TEST GRADING IN ACD TO PARTHENIUM



Among 25 cases of ACD to Parthenium, 3+ reading was observed in 11 (44%) cases, 2+ reading in 6(24%) cases, 1+ reading in 6(24 %) cases and no reaction in 2(8%) cases.

Patch test Grading in ACD to Nickel

Among 8 cases of ACD to Nickel, No reaction in 50% of cases, 2+ reading in 3(37.5%) cases and one case (12.5%) showed 1+ reading.

Patch test Grading in ACD to Hair Dye

Among 8 cases of ACD to Hair dye, 1+ reading in 4(50%) cases and 2+ reading in 1(25%) case and No reaction in 1(25%) case.

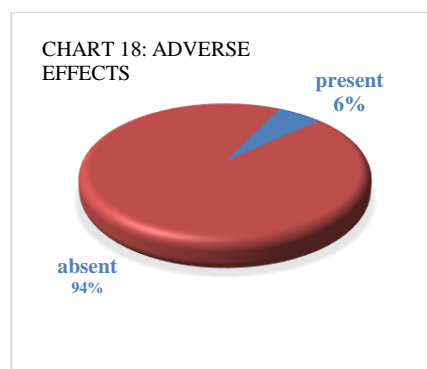
Patch test Grading in ACD to Oils & greases

Among 5 cases of ACD to Oils & Greases. 4(80%) cases showed 2+ reading and 1(20%) case showed 1+ reading.

Patch test Grading in ACD to leather footwear

Among 5 cases of ACD to Leather footwear, 2+ reading in 4(80%) cases and 1(20%) case showed 1+ reading.

ADVERSE EFFECTS:



Six cases (Chart 18) had adverse reaction to Patch testing. Miliaria rubra in 2 cases, excited skin syndrome, folliculitis, plaster site erythema and pustules at plaster site in 1 case each were present.

DISCUSSION

In our study, the incidence of ACD was found out to be 4.94%. This finding was similar to the study by Sudashree et al which showed incidence around 4 to 7%.⁹⁸

Sex incidence

In our study out of 100 patients, the male to female ratio was 3:1. A similar observation was also made by G.Narendra et al where men outnumbered women.⁹⁹ In another study by Kishore Nanda et al the male to female ratio was 1.27:1.¹⁰⁰ And the reason for this may be men are employed in preference to women. In patients with ACD to Nickel, females were more than males, this observation was also similar to another study done by Sharma AD.¹⁰¹ The reason observed was more usage of Nickel coated ornaments and dress accessories by female patients.

Age and Sex incidence

ACD was found to be highest in the age group of 31 to 40 years followed by <30 years age group. The youngest patient in the study was 18 years and the oldest was 70 years. The mean age observed in the study was 42.56 years with Standard deviation of 13.94 years. ACD was found to be common in the fourth decade among males. The age group observed was similar to the study done by Singhal V et al which showed the most common age group affected as 20-39 years.¹⁰² Our result was in contrast with the observation made by Sudhashree et al, where their mean age was 34.3 years, with a standard deviation of 11.8 years (range, 9 years to 67 years).

CLINICAL DIAGNOSIS OF ACD

ACD to Cement

ACD to Cement was found to be the commonest (38%) in our study. 0.5% Potassium di chromate was the most common allergen in the cement. This is more or less similar to the study done by Pillai et al where Cement dermatitis was found in 24%.¹⁰³ The higher incidence of ACD to Cement may be due to increased civilization and industrialisation which provides employment to skilled and unskilled workers in the construction industry. The barrier effect of the skin is compromised because of the irritant effect and the alkalinity of potassium di chromate/ cement which facilitates penetration and thus cause ACD.

In our study, the age group commonly involved was 31 to 40 years which had 11(28 %) cases. The number of persons between 25-50 years of age group was 27(71%) which was closely similar to the study done by Vikas et al where the results were 82%.¹⁰⁴ But the mean duration in our study was 6 years which was in contrast to the study done by Vikas et al where the mean duration was 14 years.

The most common pattern observed in our study was Hands and/or feet pattern (71 %) which was higher than the study done by Vikas et al(42%). Hands and feet pattern is anticipated to be common among construction workers due to direct contact throughout mixing, handling or spreading concrete and our population group is not well educated with the use of Gloves /Stockings to protect hands and arms. Single allergen, 0.5% Potassium di

chromate was positive in 26(68%) cases and 1% Cobalt chloride in 1 case. Two allergens positivity was seen in 7 cases and they were potassium di chromate with cobalt chloride in 3(7.8%) cases, potassium di chromate with paraphenylenediamine in 3 cases and potassium di chromate with parthenium in 1 case. A single case had >7 allergens positivity possibly due to excited skin syndrome.

ACD to Parthenium

ACD to Parthenium was the second most common (25%) dermatitis encountered in our study group. 15% Parthenium hysterophorus was the commonest allergen implicated. All the patients were related to agricultural occupation. Males outnumbered females(2.6:1) in our study population similar to the study done by Singh KK et al(5.5:1).¹⁰⁵ The most common age group involved was middle-aged or elderly males similar to the observation made by Sharma and Verma.¹⁰⁶ These patients were involved in open-air events like farming work and they were lightly-clothed.

ABCD was the most common pattern (36%) of Parthenium dermatitis observed in our study similar to the observation made by Sharma and Verma (81%). Even though ABCD was the most common pattern found in both studies, the difference in percentage may be due to the population group selected for the study. The mean duration noted in our study was 2.65 years but in the study done by Sharma and Verma the mean duration observed was 7.7 years.

ACD to Nickel

ACD to Nickel was the third most common, with 8% of cases observed in our study as seen in study by Kavitha et al (4-13.1%).¹⁰⁷ Nickel is the most common metal causing sensitization. 5% Nickel sulphate is the allergen implicated. In our study females outnumbered males (7:1), this is in accordance with study done by Steven A Smith et al (8.02:1).¹⁰⁸ Contact site eczema (87.5%) is the most common pattern observed in our study. Women are more usually sensitized by non-occupational contacts such as ear piercing and use of fashion jewellery that comprises free nickel.

ACD to Hairdye

ACD to Hairdye was also the third most common (8%) noted in our study. This was closely similar to the study done by Sharma VK et al which showed 11.5%.¹⁰⁹ The allergen implicated is 1% Paraphenylenediamine. The pattern most commonly observed was contact site acute eczema with weeping dermatitis over the moustache, beard, hairline site.

Other ACDs

Footwear dermatitis was observed in 7 cases. Among these, ACD to leather was seen in 5 (71%) cases followed by rubber in 2 (29%) cases. This was in contrast to 30.8% of footwear dermatitis seen in study done by Bajaj AK et al.¹¹⁰ The dissimilarity could be due to discrepancies in native culture, customs, occupational factors and environment and due to poor quality of tanning of the leather. ACD to Oils and greases were observed in 5% of cases and 3 patients were automobile mechanics. Deodorant dermatitis was

observed in 2 cases which was extremely low in the study and the reason may be that OPD population in a govt. hospital comprises mostly of low socio economic group and hail from villages and suburban areas.

Morphological patterns of ACD with different allergens

Hands and feet pattern was the commonest pattern (35%) observed in the study. The various morphological patterns due to various allergens observed in our study were highly statistically significant with p value - <0.0001. The hands and feet were the commonest sites involved in the study done by Singhal V et al (65.31%). Even though the percentage varies in both studies, the most common pattern observed was similar in both group.

Occupational vs non occupational causes in relation to ACD

In our study, two third of cases were of occupational (67%) in origin and remaining 33% were non-occupationally related. The majority were in cement related and agricultural occupation. This was in contrast to the study done by Brutti CS et al, where 29% were occupational in origin and 71% were non occupational in origin.¹¹¹ The reason for the difference noted is, in our study group, the population were labourers doing masonry and agricultural work. But in the study by Brutti CS et al, the majority of population included were non-occupational group and they had nickel and hair dye dermatitis because of their usage products.

33 cases of Non occupational group had nickel, hair dye, foot wear and textile dermatitis and they were house wives, students, clerical work, lab technician and medical representative.

Onset of ACD

In our study, 11% had acute onset of illness; 29% had acute on chronic illness and 60% had chronic illness. This was in contrast with the study done by Sudhashree et al, the symptoms were acute in 48 (56.5%) patients, chronic in 22 (28.2%) and acute on chronic in 13 (15.3%) patients. The difference noted between our study and Sudashree et al is, the majority of study population was contributed by housewives, teachers and business people in the later study group.

Seasonal variation of ACD

Seasonal variation was present in 22% of cases. Among the 22 cases, 15 cases were ACD to parthenium group which had summer/spring exacerbation. The reason for the seasonal variation, and the dermatitis increased during summer or autumn is, the pollens are destroyed in the months of winter, and the fauna grows well during the period of summer and spring and the dispersion of the pollen grains into the atmosphere. 2 cases of ACD to Nickel got summer exacerbation, due to increased sweating which increased the burden of contact allergy. Similar reason, for the seasonal exacerbation seen in a single case of Footwear dermatitis and another case of ACD to paints. This finding was similar to the study done by Shenoi et al and Lakshmi C et al.^{112,113}

ACD in relation to their Eosinophilia/ Atopy/ Medical disorders

In our study, 46 % of cases had eosinophilia, Atopy was seen in 22% of cases. The relation of atopy and eosinophilia to ACD has been explained in the studies done by Sharma AD and Silberg I et al.^{114,115} Medical disorders were observed in 19% of cases. And the patch tests grading observed was 1+ in 94% of cases. The reason for the low patch test Grading seen in cases who had medical disorders is because of the immunosuppression induced by the illness (Diabetes Mellitus). This relation is also emphasized in the study done by Grossman et al.¹¹⁶

Clinical severity & Patch test severity Correlation

Cases with weeping dermatitis, exfoliative dermatitis and acute clinical picture for that pattern were taken into clinically severe group and remaining in mild group. Cases with patch tests reading 1+, 2+ were taken in mild group of patch test severity and 3+ reading cases in severe group. When the clinical severity of the disease was assessed with the severity of patch test reading, there was no statistically significant correlation between both. The Kappa 'p' value score was 0.055 and this was statistically proved. A similar study done by Handa et al also showed the same results i.e no correlation between clinical severity and path test reading severity.¹¹⁷

Proportion of positive patch tests in adults with allergic contact dermatitis

In our study, 89% of patients showed one or more positive reactions in patch testing. This proportion was high when compared to study by Bajaj AK et al , where one or more positive reactions was observed in 60% of

patch-tested patients. Other clinical diagnosis of ACD cases showed 100% positivity rate, with respect to specific allergens like footwear, plaster, oils and greases, paints and as such the number of patients were less in the study group. Our tropical climate may be partly responsible for this phenomenon.

Indian Standard Series Battery- Contact sensitizers

In our study, Potassium di chromate(39 cases) was the most common sensitizer, followed by Parthenium(23), PPD(12), Formaldehyde(9), Parabens(5), Nickel sulphate(5), Fragrance mix (5), Lanolin (4), Cobalt (4), BR mix(3), Thiuram mix(3) Balsam of Peru(3), Epoxy resin(2) and Colophony(1 case). In another study by Singhal V et al, done with Indian Standard series, Parthenium (20%) is the most common contact sensitizer followed by potassium dichromate (16%), xanthium (13.33%), nickel sulphate (12%), chrysanthemum (8%), mercaptobenzothiazole, and garlic (6.66%). In a study by Narendra G and Srinivas CR, the frequent sensitizers observed were nickel sulphate-12 (15%), potassium dichromate-11 (13.75%), cobalt chloride and colophony-7 (8.75%) each, fragrance mix and thiuram mix-6 (7.5%) each. In our study, Potassium di chromate was the commonest sensitizer in men and Nickel sulphate was the commonest sensitizer in women which was similar to the study by Bajaj AK et al.

Indian Standard Series Battery- Patch test Allergens positivity

In our study, out of the 100 cases who were patch tested , Single allergen positivity was seen in 64%, Multiple allergens positivity was seen in 25% and No reaction observed in 11% of cases. In the study done by Sudashree et al, out

of 85 patients patch tested, 55(64.7%) patients were positive for one or more allergens, while (29)34.1% were negative and Out of the 55(64.7%) patients who were positive on patch testing, the majority, i.e., 41 (74.5%), were positive for multiple allergens and 14 (25.5%) were positive for single allergen. The study group of Sudashree et al comprised house wives, teachers and businessmen. The positivity rate found in our study, was higher than that obtained by Bajaj et al (58.6%).

Patch test Grading:

In our study, 29% had 1+ reading, 47% had 2+ reading, 13 % had 3+ reading and No reaction was observed in 11%. The patch tests readings were taken on day 2 and 4 and the results were same on both days.

Adverse effects to Patch testing:

Out of the 100 cases included in our study, 6 cases showed adverse reactions to Patch testing. And thus 94% did not show any adverse reactions in our study. This observation was closely similar to the results of the study done by Sudashree et al where 88.2% patients had no adverse reactions. From this we infer that Patch testing is a safe procedure.

SUMMARY

The inferences derived from this prospective study done on Allergic contact dermatitis patients are as follows:

- ✚ The incidence of ACD among the total new adult patients who attended our OPD during the study period was found to be 4.94%.
- ✚ The most common age group affected is 26-50 years, with a mean age of 42.56 years.
- ✚ There was a male preponderance in the study, with a male: female ratio of 3:1 except in the cases of ACD to Nickel.
- ✚ 11% had acute onset of illness; 29% had acute on chronic illness and 60% had chronic illness of the disease.
- ✚ Two third of cases were occupational in origin.
- ✚ Seasonal exacerbation was predominant in ACD to Parthenium group of patients.
- ✚ Medical disorders were noted in 19% of cases, Atopy was seen in 22% of cases, Eosinophilia present in 46% of cases.
- ✚ Hands and Feet pattern was most frequently noted in 35% of cases. ABCD pattern was most common among the patients of ACD to Parthenium.
- ✚ The proportion of positive patch test reading was found to be 92% both in ACD to Cement as well as ACD to Parthenium group. The lowest positive patch test i.e., 50% was encountered in ACD to Nickel.

- ✚ There was no correlation between the clinical severity of the disease and the severity of patch test reading.
- ✚ Single allergen positivity was seen in 64%, Multiple allergens positivity was seen in 25% and No reaction observed in 11% of cases.
- ✚ Potassium di chromate was the commonest sensitizer observed in the study, followed by Parthenium hysterophorus in males.
- ✚ Nickel sulphate was the commonest sensitizer in females.
- ✚ The patch test results were, 29% of cases had 1+ reading, 47% of cases had 2+ grading, 13 % of cases had 3+ grading and No reaction was observed in 11%.
- ✚ Adverse effects to patch tests were present in 6% of cases.

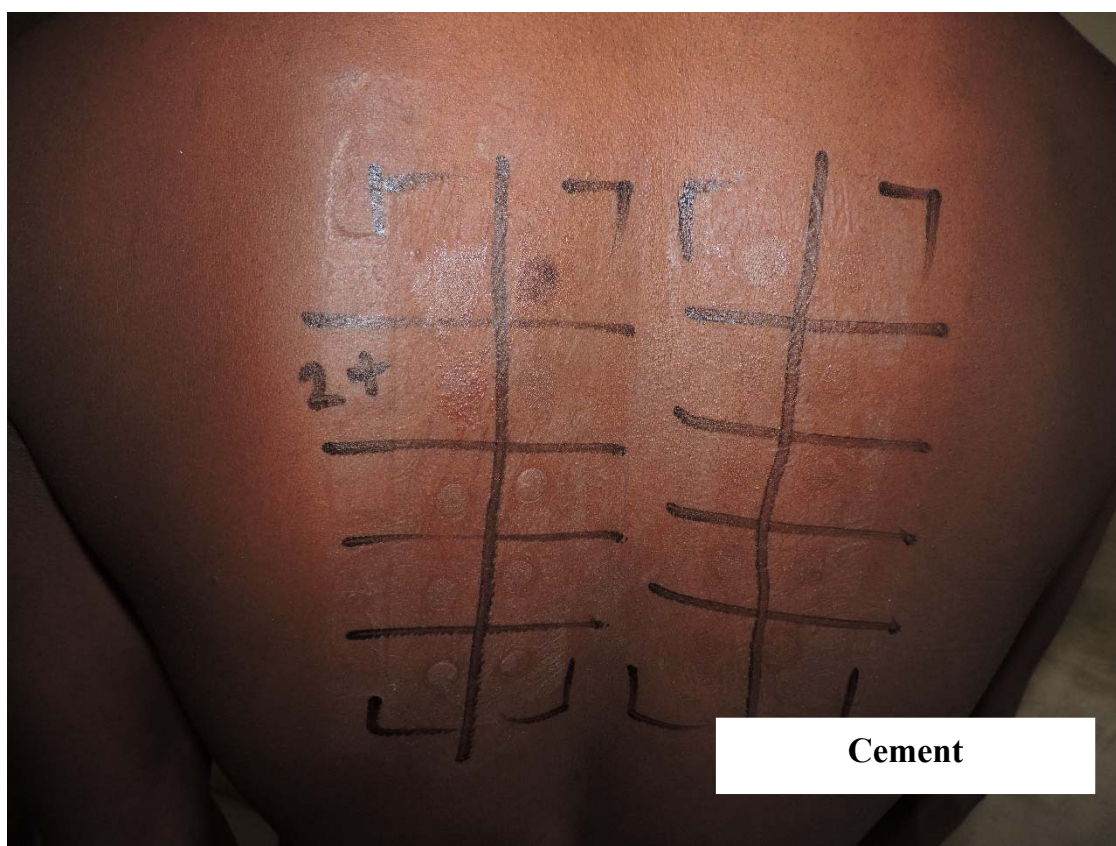
CONCLUSION

Allergic Contact Dermatitis is one of the most common diseases which has a great socio economic impact on the patients. Patch testing is a very useful scientific diagnostic tool that unravels the cause. And thus the advantages of patch testing in patients suspicious of ACD include finding the causative allergen and thus by avoiding the allergen, shortens the time lapse between first visit and final diagnosis, increases the chance for full remission, decrease the management cost and better quality of life. Apart, patch testing also reveals the current prevalence and pattern of contact dermatitis in the community. To conclude, in our centre Cement dermatitis dominated the list followed by Parthenium dermatitis. Potassium di chromate was the commonest allergen followed by 15% Parthenium hysterophorus in males. Nickel sulphate was the commonest allergen in females.



H&F

Figure 1: H&F – ACD to cement



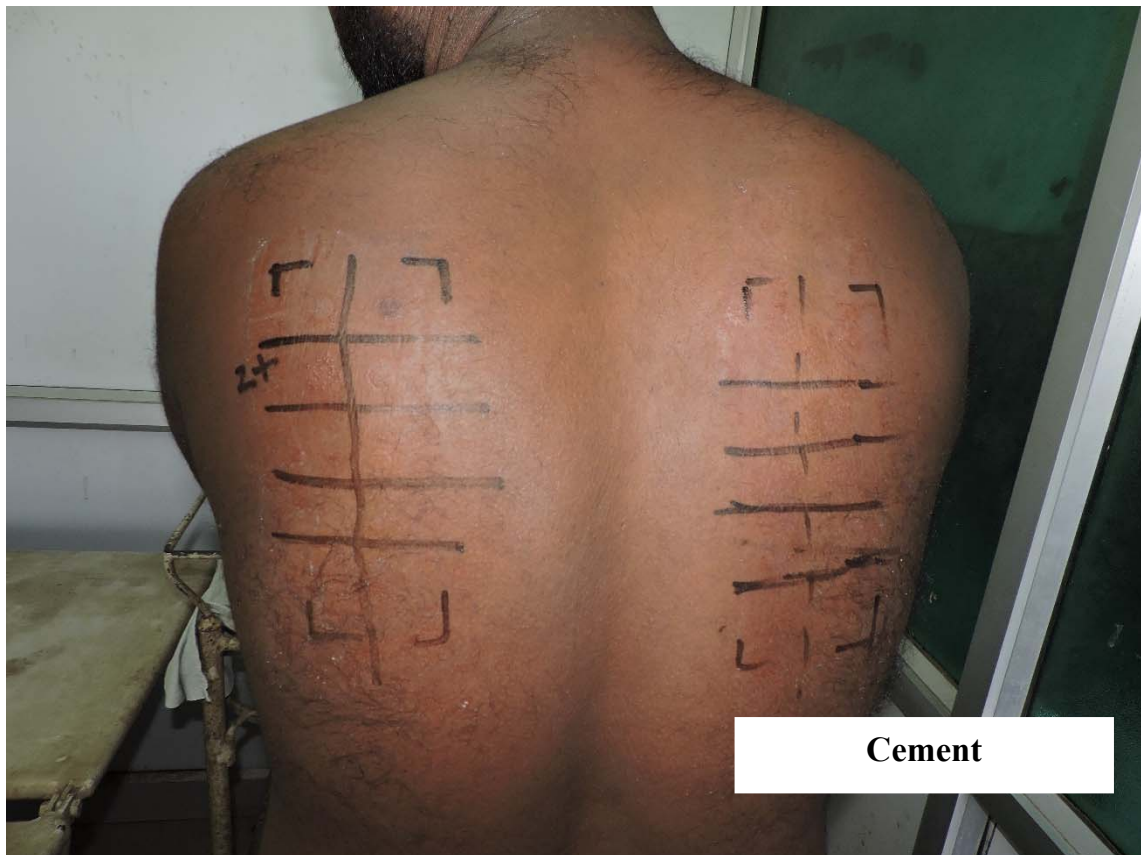
Cement

Figure 2: Potassium Dichromate



Feet eczema

Figure 3: Feet eczema – ACD to Cement



Cement

Figure 4: Potassium Dichromate



Figure 5: 70 years old male ABCD Pattern ACD to Parthenium

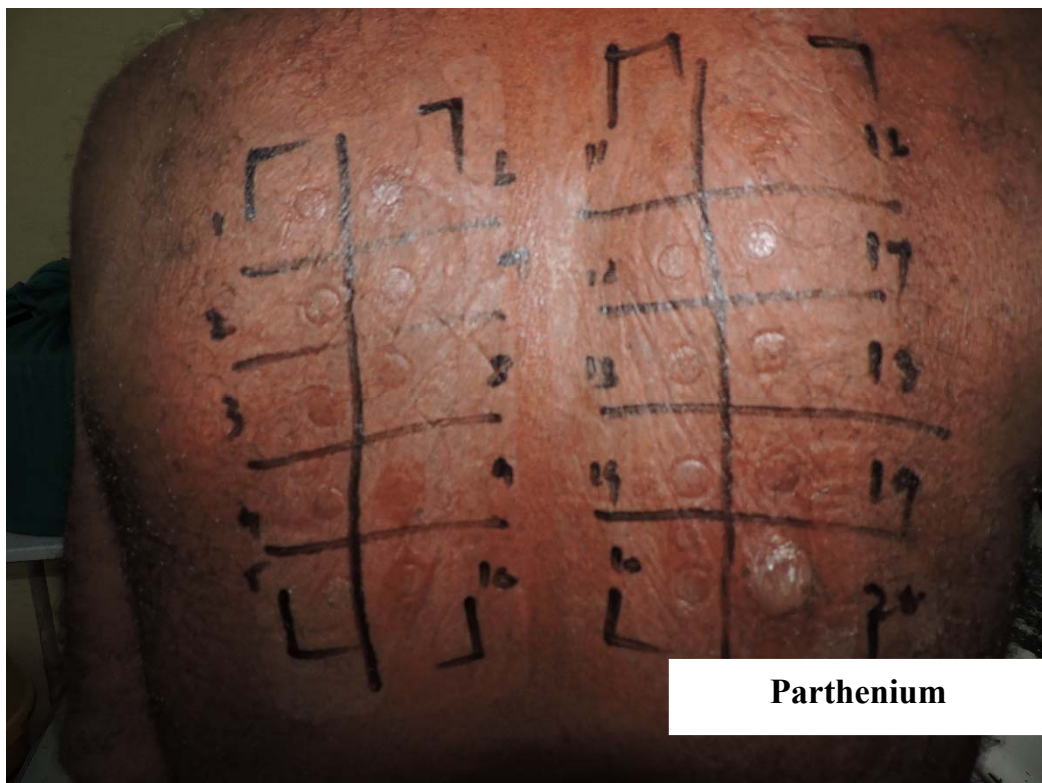


Figure 6: Parthenium 3+



Figure 7: ABCD pattern

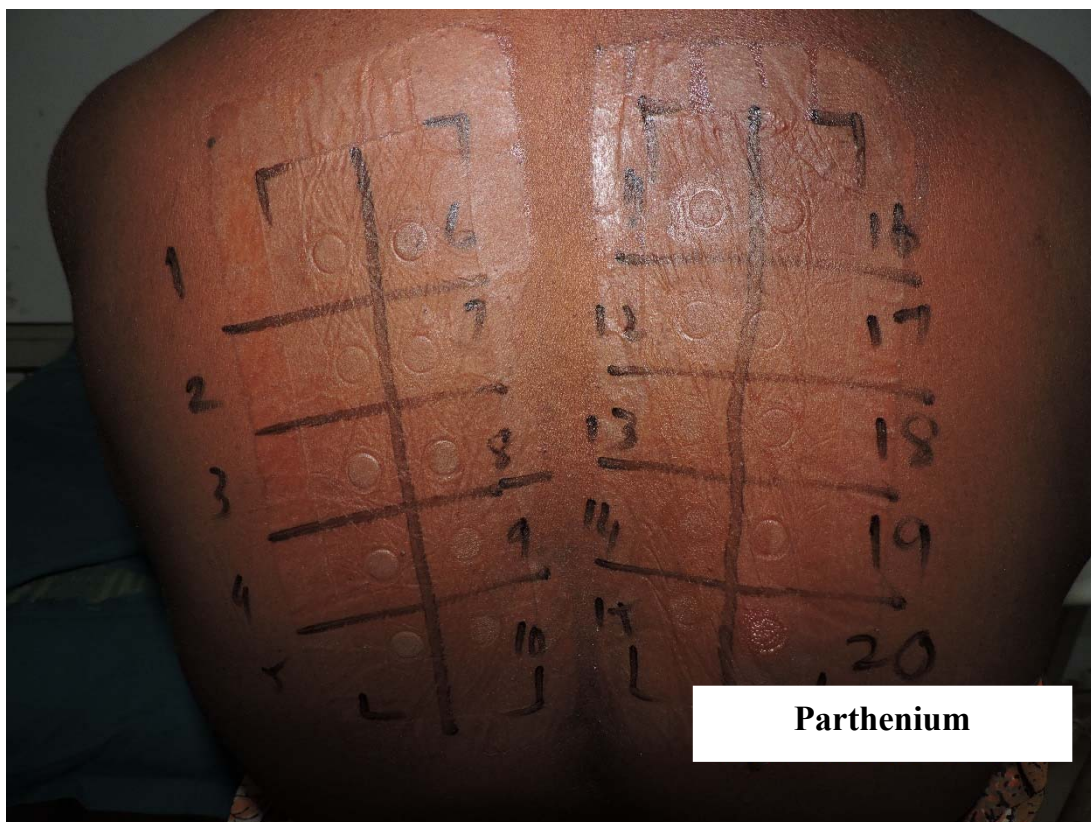


Figure 8: Parthenium 2+



Figure 9: Exfoliative dermatitis- ACD to parthenium



Figure 10: Atopic dermatitis pattern



Figure 11: ACD to Hairdye



Figure 12: ACD to Hairdye



Figure 13: ACD to Hairdye

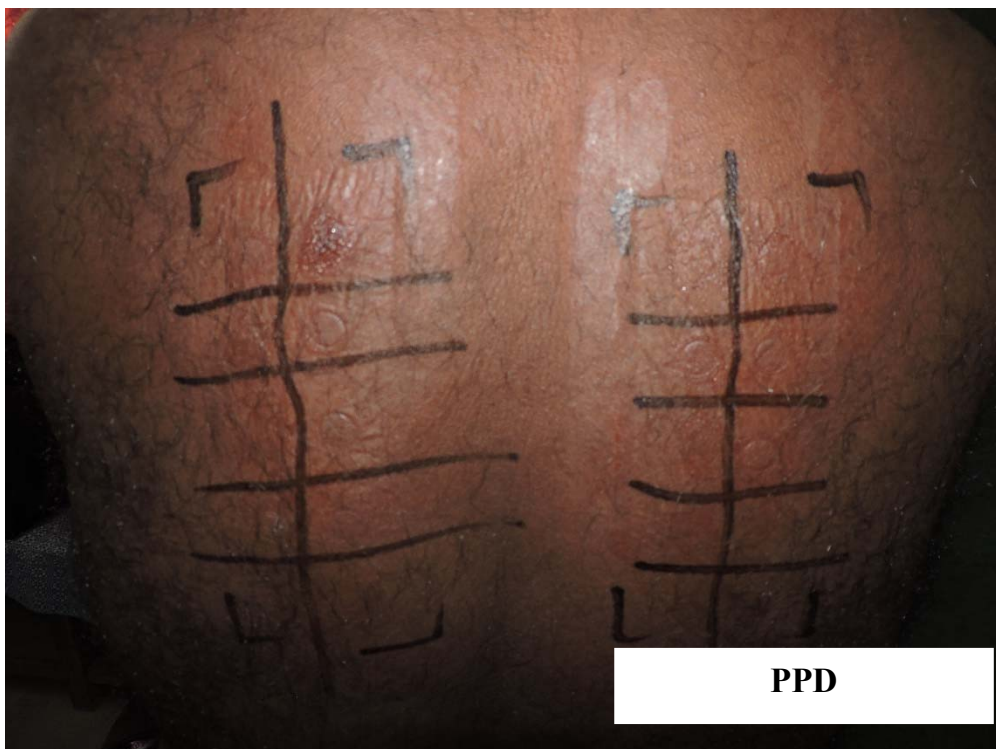


Figure 14: PPD 3+



Figure 15: ACD to Nickel

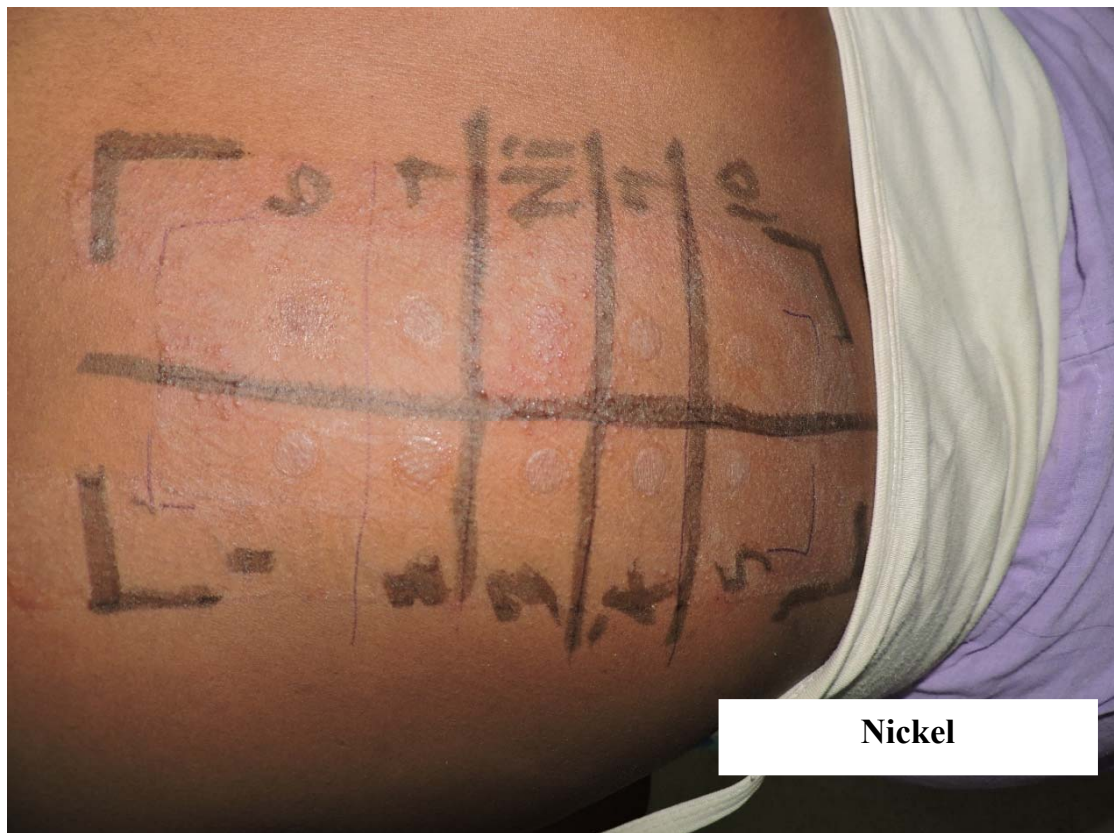


Figure 16: Nickel 3+



Figure 17: ACD to Plaster

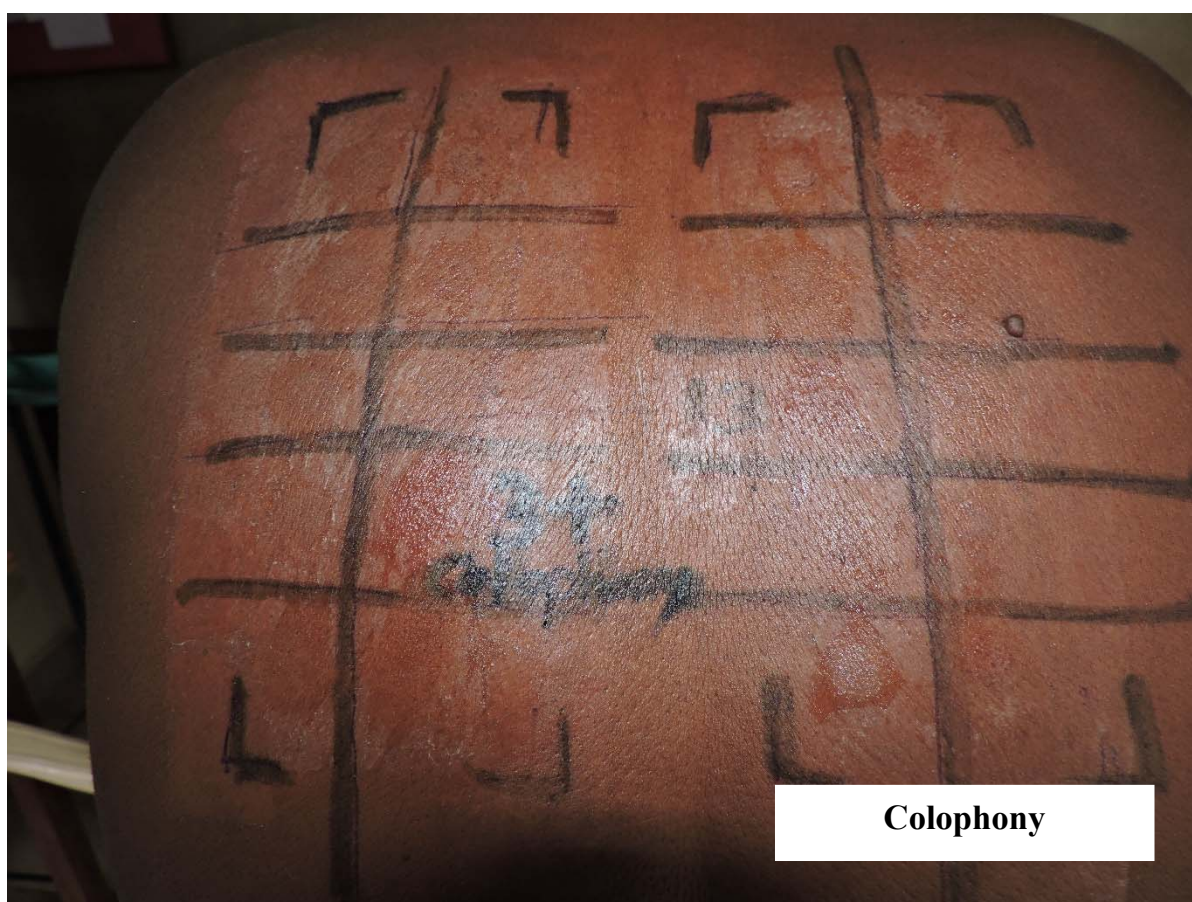


Figure 18: Colophony 3+



Figure 19: ACD to Leather footwear



Figure 20: ACD to Leather footwear



Figure 21: Pompholyx pattern

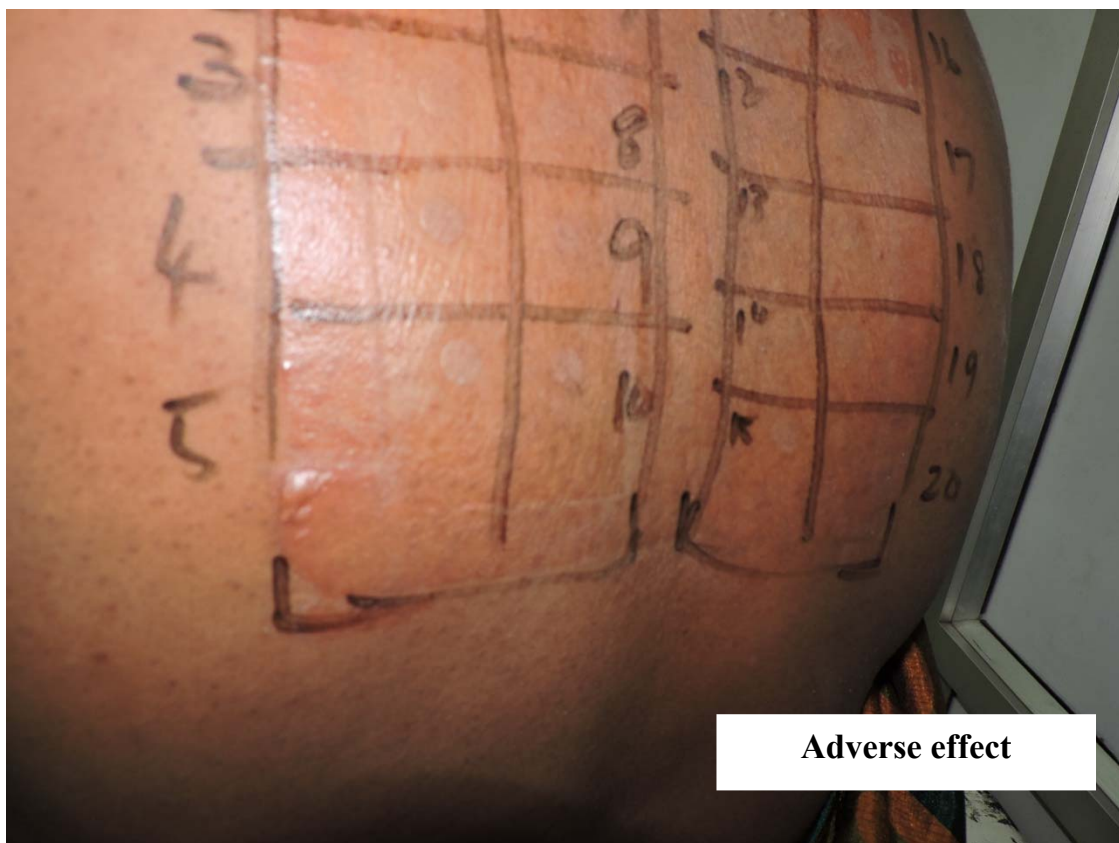


Figure 22: Erythema & blister at plaster site



Figure 23: ACD to Cement

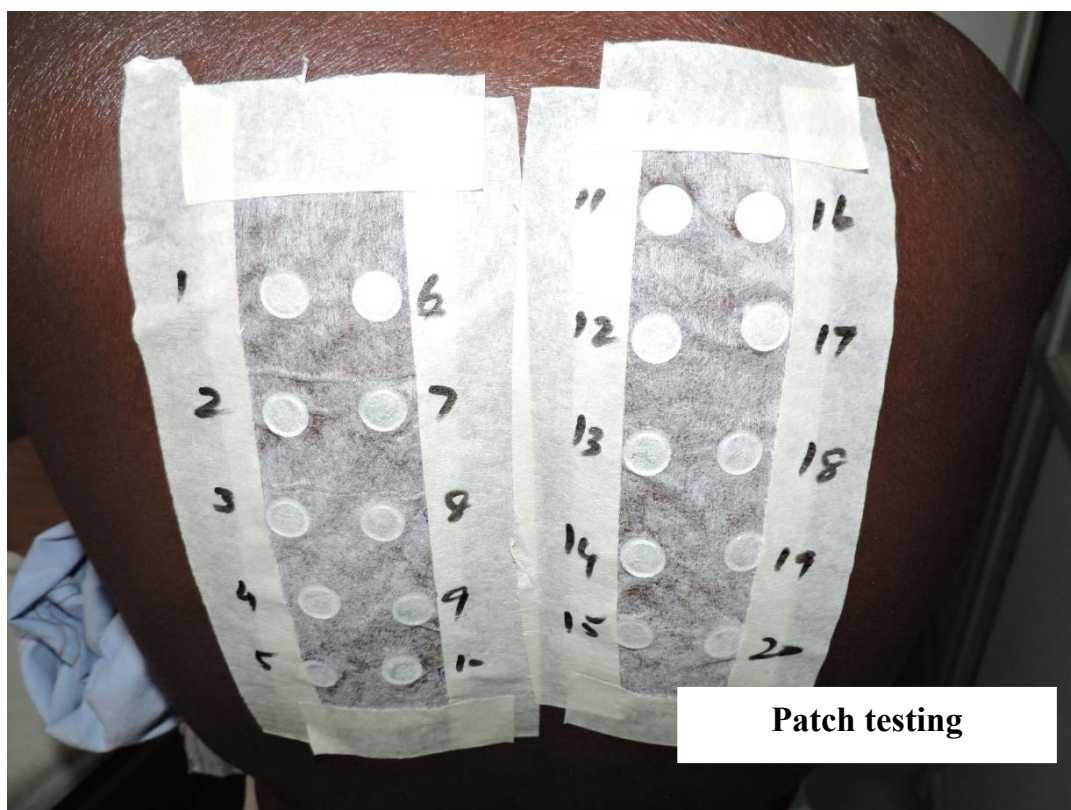


Figure 24: Patch testing

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Sl.no	Name	Age	Sex	Occupation	Duration	Onset	Pruritus	Oozing	Atopy	Seasonal variation	Contact alleged plants /cement /dye /nickel /etc	Asso medical disorders	Smoking/ alcohol	Areas involved	Pattern	Clinical Severity	Provisional diagnosis	Eosinophils	Patch test allergen	48 Hr reading	72 H	Adverse effect
1	shanmugam	60	M	farmer	9 M	A on C	Severe	P	P	S/A	Plants	BA	P	F/N/T/UL/LL	Ex D	Severe	ACD to parthenium	P	parthenium	3	3	N
2	dharmaraj nadar	70	M	gardener	36 M	C	Mild	N	P	S/A	Plants	No	P	F/N/V area/UB	ABCD	Mild	ACD to parthenium	P	parthenium	3	3	N
3	muthaiah	70	M	agriculture	12 M	C	Mild	N	N	S	Plants	No	P	F/N/V area/FA/UB	ABCD + CAD	Mild	ACD to parthenium	P	parthenium	3	3	N
4	velsamy	60	M	agriculture	4 M	C	Mild	N	P	S	Plants	No	N	F/N/V area/UL	ABCD	Mild	ACD to parthenium	P	parthenium	2	2	N
5	ayyar	64	M	farmer	96 M	A on C	Severe	P	P	No	Plants	HT	N	F/FA	ABCD	Mild	ACD to parthenium	P	parthenium	1	1	N
6	ayyammal	50	F	farmer	32 M	A on C	Severe	P	P	S/A	Plants	DM	N	F/N/V area/UL	ABCD	Mild	ACD to parthenium	P	parthenium	2	2	N
7	parvathy	55	F	farmer	48 M	A on C	Severe	P	N	S	Plants	DM	N	F/N/Flex	AD	Severe	ACD to parthenium	P	parthenium	3	3	N
8	periasamy	65	M	agriculture	24 M	C	Mild	N	N	S/A	Plants	No	P	F/N/T/UL/LL	ABCD + CAD	Mild	ACD to parthenium	A	parthenium	3	3	N
9	arumugam	54	M	agriculture	36 M	A on C	Severe	N	P	S/A	Plants	No	N	F/N/T/UL/LL	Ex D	Severe	ACD to parthenium	P	parthenium	3	3	N
10	narayanan	60	M	gardener	40 M	C	Mild	N	N	No	Plants	No	N	F/N	CAD	Mild	ACD to parthenium	A	parthenium	3	3	N
11	arumugam	70	M	farmer	60 M	C	Mild	N	N	No	Plants	No	N	Feet	H&F	Mild	ACD to parthenium	A	parthenium	3	3	N
12	petchiammal	49	F	farmer	15 M	C	Mild	N	N	No	Plants	No	N	N/Hands	CAD	Mild	ACD to parthenium	P	Balsam peru/ Fragrance mix	2	2	N
13	sornam	50	F	agriculture	24 M	C	Mild	N	N	No	Plants	No	N	N/Hands	CAD	Mild	ACD to parthenium	A	No rxn	0	0	P
14	kombiah	41	M	farmer	30 M	C	Mild	N	N	No	Plants	No	P	N/H&F	H&F	Mild	ACD to parthenium	A	parthenium	1	1	N
15	murugan	60	M	farmer	60 M	C	Mild	N	P	No	Plants	No	N	F/N/Flex	CAD+ AD	Severe	ACD to parthenium	P	parthenium	3	3	N
16	chellaiah	62	M	farmer	54 M	C	Mild	N	N	S/A	Plants	No	N	F/N	ABCD + CAD	Mild	ACD to parthenium	P	parthenium	3	3	N
17	murugan	62	M	farmer	24 M	A on C	Severe	P	P	S/A	Plants	HT	N	F/N/Flex	ABCD + AD	Severe	ACD to parthenium	P	parthenium	2	2	N
18	fathima	42	F	house wife	6 M	C	Mild	N	P	No	Plants	BA	N	F/N/T/H&F	ABCD	Mild	ACD to parthenium	P	No rxn	0	0	N
19	PONRAJ	65	M	farmer	18 M	C	Mild	N	N	No	Plants	No	N	F/N	CAD	Mild	ACD to parthenium	A	parthenium	1	1	N
20	perumal	55	M	mason	40 M	C	Severe	N	P	No	Plants	HT	P	F/N/T	ABCD	Mild	ACD to parthenium	P	Parthenium	3	3	N
21	Arockiam	45	M	farmer	60 M	C	Mild	N	N	S/A	Plants	No	N	N/Hands	Hand eczema	Mild	ACD to parthenium	P	Parthenium	2	2	N
22	velu	60	M	farmer	45 M	C	Severe	N	N	S	Plants	No	N	F/N/T	ABCD	Mild	ACD to parthenium	A	parthenium	1	1	N
23	Selvaraj	62	M	mason	48 M	C	Severe	N	N	No	Cement	M/H	N	T/H&F	Ex D	Severe	ACD to Cement	A	K+ di chr	2	2	N
24	thalavai	45	M	mason	36 M	C	Severe	N	P	S/A	Cement	No	N	H&F/Legs	Prurigo simplex like	Mild	ACD to Cement	P	K+ di chr	1	1	N

Sl.no	Name	Age	Sex	Occupation	Duration	Onset	Pruritus	Oozing	Atopy	Seasonal variation	Contact alleged plants /cement /dye /nickel /etc	Asso medical disorders	Smoking/ alcohol	Areas involved	Pattern	Clinical Severity	Provisional diagnosis	Eosinophils	Patch test allergen	48 Hr reading	72 H	Adverse effect
25	kumar	25	M	mason	12 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	2	2	N
26	subramanian	23	M	mason	10 M	C	Mild	N	P	S/A	Cement	No	N	H&F/FA/Legs	Lichenoid	Mild	ACD to Cement	P	K+ di chr	1	1	N
27	maniraj	30	M	mason	42 M	A on C	Severe	P	N	S	Cement	No	N	H&F/FA/Legs	H&F	Mild	ACD to Cement	A	K+ di chr	2	2	N
28	chandran	50	M	mason	96 M	A on C	Severe	P	N	No	Cement	No	P	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	1	1	N
29	chandraa	45	F	mason	180 M	C	Severe	N	N	No	Cement	No	N	N/H&F	H&F/Atopic	Severe	ACD to Cement	A	K+ di chr/PPD2+/epo xy resin2+	2	2	N
30	vinayaga sundari	25	F	mason	20 M	A on C	Severe	P	P	No	Cement	No	N	H&F/Legs	H&F	Mild	ACD to Cement	A	K+ di chr	1	1	N
31	ayyadurai	63	M	mason	120 M	A on C	Severe	P	N	No	Cement	DM	P	H&F/Legs	H&F	Mild	ACD to Cement	A	K+ di chr/ PPD/FM	2	2	N
32	shanmugasundaram	31	M	mason	96 M	A on C	Severe	P	P	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	> 7 allergens	2	2	P
33	murugan	47	M	mason	120 M	C	Mild	N	N	No	Cement	No	P	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	2	2	N
34	ramakrishnan	27	M	mason	84 M	C	Mild	N	N	No	Cement	No	P	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	1	1	N
35	murugan	40	M	mason	180 M	C	Mild	N	N	No	Cement	No	P	T/H&F	Mixed (H&F/ trunk)	Mild	ACD to Cement	A	K+ di chr/cobalt	2	2	N
36	mariappan	31	M	mason	72 M	A on C	Severe	P	P	No	Cement	No	P	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	2	2	P
37	jeyanthan	32	M	mason	80 M	A on C	Severe	P	N	No	Cement	No	P	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	1	1	N
38	ganesan	50	M	mason	120 M	C	Severe	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	K+ di chr/PPD/Parab ens	2	2	P
39	sivasubramanian	60	M	mason	150 M	A on C	Severe	P	N	No	Cement	BA	N	H&F/Flex	AD	Severe	ACD to Cement	A	K+ di chr	2	2	N

Sl.no	Name	Age	Sex	Occupation	Duration	Onset	Pruritus	Oozing	Atopy	Seasonal variation	Contact alleged plants /cement /dye /nickel /etc	Asso medical disorders	Smoking/ alcohol	Areas involved	Pattern	Clinical Severity	Provisional diagnosis	Eosinophils	Patch test allergen	48 Hr reading	72 H	Adverse effect
40	murugan	45	M	mason	60 M	A on C	Severe	P	N	No	Cement	No	N	H&F/FA/Legs	FOLLICULAR	Mild	ACD to Cement	A	K+ di chr	2	2	N
41	mahalingam	35	M	mason	60 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	Cobalt	1	1	N
42	sahayam	54	M	mason	96 M	C	Mild	N	N	No	Cement	No	N	T/H&F	Mixed (H&F/ trunk)	Mild	ACD to Cement	P	No rxn	0	0	N
43	hariguru	58	M	mason	120 M	A on C	Severe	P	N	No	Cement	M/H	N	H&F/Flex	Mixed (H&F/ trunk)	Mild	ACD to Cement	P	K+ di chr	2	2	N
44	marimuthu	52	M	mason	120 M	A on C	Severe	P	P	No	Cement	DM	P	H&F	H&F	Mild	ACD to Cement	P	K+ di chr/parthenium	2	2	N
45	aathinarayanan	25	M	mason	60 M	A on C	Severe	P	P	No	Cement	No	N	T/H&F/FA/Legs	follicular	Mild	ACD to Cement	P	No rxn	0	0	N
46	jesu	37	M	mason	84 M	C	Severe	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	2	2	N
47	lingam	35	M	mason	48 M	C	Mild	N	P	No	Cement	No	N	Finger	FINGER TIP	Mild	ACD to Cement	P	K+ di chr	2	2	N
48	madasamy	65	M	mason	80 M	C	Severe	N	N	No	Cement	DM	N	T/H&F	Ex D	Severe	ACD to Cement	A	K+ di chr	1	1	N
49	muthukrishnan	45	M	mason	96 M	A on C	Severe	P	P	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	1	1	N
50	sindhamathar	45	M	mason	60 M	A on C	Severe	P	N	No	Cement	No	N	H&F/FA/Legs	forearm/legs	Mild	ACD to Cement	P	K+ di chr/cobalt	1	1	N
51	thirumalai	55	M	mason	120 M	A on C	Severe	P	N	No	Cement	DM	N	H&F/FA/Legs	forearm/legs	Mild	ACD to Cement	A	K+ di chr/cobalt	2	2	N
52	sankarapandi	45	M	mason	180 M	A on C	Severe	P	N	No	Cement	No	N	F/H&F	acrofacial	Mild	ACD to Cement	A	No rxn	0	0	N
53	lalitha	22	F	lab tech	5 M	C	Mild	N	N	No	Nickel	No	N	Wrist	Subacute eczema/watchst rap site	Mild	ACD to Nickel	A	nickel	1	1	N
54	valli	36	F	teacher	6 M	C	Mild	N	N	No	Nickel	No	N	N/Wrist	POD/neck eczema	Mild	ACD to Nickel	A	No rxn	0	0	P
55	vijayalakshmi	28	F	house wife	8 M	C	Mild	N	N	S	per Footwear/ N	No	N	Feet	feet dermatitis	Mild	ACD to Footwear/Nick el	P	BR/THIU/PPD	2	2	N
56	lakshmi	49	F	house wife	3 M	C	Mild	N	N	No	leather Footwea	No	N	Feet	footwear site	Mild	ACD to Footwear	P	K+ di chr/ Formaldehyde	1	1	N
57	mariammal	29	F	house wife	4 M	C	Mild	N	N	S	Nickel	No	N	Shoulder	saree pin site ec	Mild	ACD to Nickel	P	nickel	2	2	N
58	agilandeswari	26	F	student	3 M	C	Mild	N	N	No	Nickel	No	N	Wrist	watch strap site	Mild	ACD to Nickel	A	nickel	2	2	N
59	sudhamathi	39	F	type writer	10 M	C	Severe	N	N	No	Nickel	No	N	N/Shoulder	Lichenoid	Mild	ACD to Nickel	P	No rxn	0	0	N
60	rajasekar	30	M	contract woker	2 M	C	Mild	N	N	No	Nickel	No	N	Finger	Ringwear site	Mild	ACD to Nickel	A	No rxn	0	0	N
61	vaajitha	40	F	house wife	12 M	C	Severe	N	P	No	Nickel	BA	N	H&F/FA/Legs/Flex	H&F/Atopic	Severe	ACD to Nickel/Plants/ Deodorants	A	nickel/partheni um/PPD/Fragr ance mix	2	2	N

Sl.no	Name	Age	Sex	Occupation	Duration	Onset	Pruritus	Oozing	Atopy	Seasonal variation	Contact alleged plants /cement /dye /nickel /etc	Asso medical disorders	Smoking/ alcohol	Areas involved	Pattern	Clinical Severity	Provisional diagnosis	Eosinophils	Patch test allergen	48 Hr reading	72 H	Adverse effect
62	megala	25	F	student	4 days	A	Severe	P	N	No	Nickel	No	N	Ear	Weeping dermatitis/ ear ring site	Severe	ACD to Nickel	P	No rxn	0	0	N
63	Valliammal	21	F	student	3 M	C	Mild	N	N	No	Textile	No	N	Axillae	Peri axillary dermatitis	Mild	ACD to Clothing	A	Lanolin	1	1	N
64	chellammal	45	F	house wife	12 M	C	Severe	N	N	No	Textile	BA	N	Axillae	Axillary / contact site back	Mild	ACD to Elastic	A	Lanolin	2	2	N
65	esther	34	F	teacher	24 M	C	Mild	N	N	S	Nickel	No	N	N/T	jewel contact site ecz	Mild	ACD to Nickel	A	nickel	2	2	N
66	madasamy53	53	M	farmer/hairdye user	48 M	C	Mild	N	N	No	Hair dye/ plants	BA	N	H&F	H&F	Mild	ACD to parthenium/ha irdye	P	PPD	3	3	N
67	kamala	40	F	housewife	7 days	A	Severe	P	N	No	Hair dye	No	N	Hairline/Ears	Acute eczema/ Hairline	Severe	ACD to Hairdye	A	PPD	1	1	N
68	arunchalam	52	M	clerk	3 days	A	Severe	P	N	No	Hair dye	No	N	Moustache	Acute eczema/ Moustache	Severe	ACD to Hairdye	A	No rxn	0	0	N
69	gunasekar	55	M	professor	4 days	A	Severe	P	N	No	Hair dye	No	N	Moustache	Acute eczema/ Moustache	Severe	ACD to Hairdye	A	PPD	1	1	N
70	jayarani	49	F	staff nurse	5 days	A	Severe	P	N	No	Hair dye	No	N	Hairline/Ears	Acute eczema/ Hairline	Severe	ACD to Hairdye	A	PPD	1	1	N
71	marimuthu	32	M	teacher	2 days	A	Severe	P	N	No	Hair dye	No	N	Face	Contact urticaria	Mild	ACD to Hairdye	A	PPD	2	2	N
72	paldurai	55	M	constable	2 days	A	Severe	P	N	No	Hair dye	No	P	Face	Contact urticaria	Mild	ACD to Hairdye	A	PPD	2	2	N
73	Muhammed Ali	35	M	Representative	2 M	C	Mild	N	N	No	Leather Footwea	No	N	Feet	Footwear site	Mild	ACD to Leather footwear	A	K+ di chr/Formaldeh yde	2	2	N
74	mageshwaran	32	M	business	2 M	A on C	Severe	N	N	No	Rubber Footwea	No	P	Feet	footwear site	Mild	ACD to Footwear	A	epoxy/MBT/thi uram/BR	2	2	N
75	Murugan	60	M	watchman	2 days	A	Severe	P	N	No	Plaster	HT/C	N	Hands/Wrist	Weeping dermatitis at plaster site	Severe	ACD to Plaster	P	colophony	3	3	N
76	murugan	35	M	painter	12 M	A on C	Severe	P	P	S/A	Paints	No	P	H&F/Legs	Pompholyx	Severe	ACD to Paints	P	K+ di chr/ Formaldehyde/ Epoxy resin/Colophon y	2	2	N
77	hudson	23	M	coolie	7 M	C	Severe	N	N	No	Oils/Greases	No	P	H&F/FA/Legs	H&f eczema pattern	Mild	ACD to Oils/Greases	A	Parabens/Form aldehyde	1	1	N
78	ravi	48	M	lawyer	10 M	C	Severe	N	N	No	Leather Footwea	M/H	P	Feet	feet dermatitis	Mild	ACD to Leather footwear	A	K+ di chr/Formaldeh yde	2	2	N
79	vijayakumar	18	M	student	8 M	C	Severe	N	N	No	Rubber Footwea	No	N	Feet	feet dermatitis	Mild	ACD to Rubber footwear	A	BR/THIU/PPD	2	2	N

Sl.no	Name	Age	Sex	Occupation	Duration	Onset	Pruritus	Oozing	Atopy	Seasonal variation	Contact alleged plants /cement /dye /nickel /etc	Asso medical disorders	Smoking/ alcohol	Areas involved	Pattern	Clinical Severity	Provisional diagnosis	Eosinophils	Patch test allergen	48 Hr reading	72 H	Adverse effect
80	esakiraja	22	M	student	2 M	C	Mild	N	N	No	Deodorant	No	N	T/Axillae	Chronic eczema/ back of neck & axillae	Mild	ACD to Deospray	P	FM/parabens/b alsum of peru	1	1	N
81	Madhialagan	34	M	Mechanic	2 M	C	Mild	N	N	No	Oils/Greases	No	N	H&F/FA/Legs	Lichenoid	Mild	ACD to Oils/Greases	A	Parabens	2	2	N
82	ramesh	25	M	mason	10 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	1	1	N
83	karthick	29	M	milkman	2 M	A on C	Severe	P	N	No	Leather Footwea	No	N	Feet	footwear site	Mild	ACD to Leather footwear	P	K+ di chr/Lanolin	2	2	N
84	maniraj	31	M	coolie	1 M	A	Severe	P	N	No	Leather Footwea	No	P	Feet	footwear site	Mild	ACD to Leather footwear	A	K+ di chr/Lanolin/Fo rmaldehyde	2	2	P
85	kalyani	44	F	agriculture	6 M	C	Mild	N	P	S	Plants	No	N	F/N/Flex	AD	Severe	ACD to parthenium	P	Parthenium	2	2	N
86	krishnan	41	M	mason	5 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	1	1	N
87	Murugan	23	M	mason	2 M	A on C	Severe	P	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	2	2	N
88	Sollamuthu	52	M	mason	5 M	A on C	Severe	P	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	1	1	N
89	Prabakaran	32	M	mason	4 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	2	2	N
90	pandi	40	M	mason	3 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	A	K+ di chr	2	2	N
91	velsamy	37	M	mason	7 M	C	Mild	N	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	1	1	N
92	shanmugathammal	35	F	mason	4 M	A on C	Mild	P	N	No	Cement	No	N	H&F	H&F	Mild	ACD to Cement	P	K+ di chr	2	2	N
93	mariappan	32	M	mechanic	3 M	A on C	Severe	P	N	No	Oils/Greases	No	N	H&F	H&F	Mild	ACD to Oils/Greases	A	Parabens/Form aldehyde	2	2	N
94	arul	27	M	mechanic	2 M	C	Mild	N	N	No	Oils/Greases	No	N	H&F	H&F	Mild	ACD to Oils/Greases	P	Parabens/Form aldehyde	2	2	N
95	sivan	31	M	coolie	3 M	C	Mild	N	N	No	Oils/Greases	No	N	H&F	H&F	Mild	ACD to Oils/Greases	A	Parabens/Form aldehyde	2	2	N
96	sivakami	29	F	farmer	6 M	C	Mild	N	N	S/A	Plants	No	N	F/N/V area/UB	ABCD	Mild	ACD to parthenium	A	Parthenium	1	1	N
97	rajavel	31	M	farmer	10 M	C	Mild	N	N	S/A	Plants	No	N	F/N/V area/UB	ABCD	Mild	ACD to parthenium	P	Parthenium	2	2	N
98	usha	23	F	student	3 M	C	Mild	N	N	No	Deodorant	No	N	T/Axillae	Chronic eczema/ back of neck & axillae	Mild	ACD to Deodorant	A	FM/balsum of peru	1	1	N
99	ravindran	25	M	student	4 days	A	Mild	P	N	No	Hair dye	No	N	Moustache	Acute eczema/ Moustache	Severe	ACD to Hairdye	A	No rxn	0	0	N
100	ramachandran	46	M	fisherman	2 days	A	Mild	P	N	No	Hair dye	No	N	Moustache	Acute eczema/ Moustache	Severe	ACD to Hairdye	A	PPD	1	1	N

KEY TO MASTER CHART

M	-	Male
F	-	Female
A	-	Acute
A on C	-	Acute on Chronic
C	-	Chronic
M	-	Months
Oozing, Atopy: P	-	present; N - absent
S	-	Summer exacerbation
S/A	-	Summer/autumn exacerbation

Associated Medical Disorders:

DM	-	Diabetes mellitus
HT	-	Hypertension
BA	-	Bronchial asthma
CAD	-	Coronary artery disease

Areas involved:

F	-	face
T	-	trunk
N	-	neck
UL/LL-		upper limb/lower limb
FA	-	forearm
UB	-	upper back
Flex	-	flexures
H&F	-	Hands and feet

Pattern:

ABCD- Air borne contact dermatitis

CAD - Chronic actinic dermatitis

Ex D - Exfoliative dermatitis

AD - Atopic dermatitis