Occupational skin diseases (Occupational dermatoses)

By

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The National Institute for occupational Safety and Health (NIOSH) has classified skin disease as one of the most pervasive problems facing workers in the United State. Since 1982, skin disease has been listed as one of the top ten work-related diseases based on potential for prevention, incidence and severity. Dermatologic diseases were shown to account for about 40% of all occupational diseases reported to the U.S. Department of labor.

As the largest organ in the body, the skin performs a variety of crucial homeostatic and protective functions. In constant contact with the external environment, the skin is particularly vulnerable to damage by physical and chemical agents of the workplace. The skin is a primary defender and good barrier protection (horny layer) represents only a fraction of the duties performed by the entire integumentary system. Pathologic responses of the skin can vary from excessive dryness and mild redness to more generalized exfoliate dermatitis that may be life threatening. In addition, neoplasms of the skin may result from primary skin exposure or through systemic absorption of carcinogen. Benign or malignant, such neoplastic events can have catastrophic consequences.

Occupational skin disease results primarily from four basic adverse stressors;

1. Mechanical (friction and pressure)
2. Chemical (organic and inorganic of various PHs)
3. physical (heat, cold, radiation)
4. Biologic (bacteria, fungi, viruses, parasites)

- **Contact Dermatitis**

Contact dermatitis is the single most prevalent occupational skin desease, accounting for ever 90% of the reported causes. Irritant and allergic contact dermatitis.

Diagnostic testing ---The patch test

Occupational contact dermatitis is frequently divided into two categories:

1. **Irritant contact dermatitis (ICD)**

   is a non-immunologic reaction that manifests as an inflammation of the skin caused by direct damage to the skin following exposure to a hazardous agent. The reaction is typically localized to the site of contact. Available data indicates that ICD represents approximately 80% of all cases of occupational contact dermatitis.

ICD may be caused by phototoxic responses (e.g., tar), acute exposures to highly irritating substances (e.g., acids, bases, oxiding/reducing agents), or chronic cumulative exposures to mild irritants (e.g., water, detergents, weak cleaning agents).

2. Allergic contact dermatitis (ACD)
is an inflammation of the skin caused by an immunologic reaction triggered by dermal contact to a skin allergen. For ACD to occur, a worker must be first sensitized to the allergen. Subsequent exposures of the skin to the allergenic agent may elicit an immunologic reaction resulting in inflammation of the skin. The reaction is not confined to the site of contact and may result in systemic responses.
ACD may be caused by industrial compounds (i.e. metals, epoxy and acrylic resins, rubber additives, chemical intermediates), agrochemicals (i.e. pesticides and fertilizers), and commercial chemicals.

Because the symptoms and presentation of ICD and ACD are so similar, it is extremely difficult to distinguish between the two forms of contact dermatitis without clinical testing (e.g. patch testing). The severity of contact dermatitis is highly variable and depends on many factors including:

1. Characteristics of the hazardous agent (irritant and/or allergen)
2. Concentration of the hazardous agent (irritant and/or allergen)
3. Duration and frequency of exposure to the hazardous agent (irritant and/or allergen)
4. Environmental factors (e.g., temperature, humidity)
5. Condition of the skin (e.g., healthy vs. damaged skin, dry vs. wet)

Prevention

1. Pre-employment medical examination
2. Mechanized
3. Protective measure (gloves, skin cream barrier)

Occupational skin diseases: Why, How and When?

Occupational Skin Disease

- A skin disease that is caused by physical, biological or chemical factor in work
- Also a worsening of pre-existing skin disease can be termed as occupational skin disease
- The start of occupational disease is considered to be the time a patient visited physician the first time

Occupational skin diseases - How common?

- In Finland approximately 1000 cases every year (pop. 5 Million).
- Approximately 20% of all occupational diseases
- Frequency is stable

Occupational skin diseases - what type?

- Most occupational skin diseases are contact dermatoses
- Allergic contact dermatitis
- Irritant contact dermatitis
- Contact urticaria
- Protein contact dermatitis
- Skin infections

OCCUPATIONAL SKIN INFECTIONS

- Scabies
- Fleas
- Paravaccinia
- Erysipeloides

OCCUPATIONAL SKIN CANCERS

- Basal cell carcinoma
- Spinous cell carcinoma
- Malignant melanoma

OCCUPATIONAL PIGMENT CHANGES

- Melanodermia
  - Increased pigmentation
- Leukodermia
  - Decreased pigmentation

OCCUPATIONAL ACNE

- Chloro-acne
- Oil acne
- Tar acne

CONTACT URTICARIA

- Immunologic contact urticaria
  - Caused by proteins that act as allergens
  - Proteins penetrate through skin and bind to IgE on the surface of mast cells
  - Binding causes histamine and other mediator release resulting in urticaria
  - Sometimes generalized reactions occur
  - Latex allergy

CONTACT URTICARIA

- Nonimmunologic contact urticaria
  - Caused by chemicals
  - Direct pharmacologic action on skin cells
  - No sensitization necessary
  - More common than suspected?

PROTEIN CONTACT DERMATITIS

- Repeated contact urticaria from protein allergens cause eczema (dermatitis)
- Kitchen work (repeated exposure to food allergens

PHOTOCONTACT DERMATITIS

- Toxic photocontact dermatitis (plants, psoralens)
- Allergic photocontact dermatitis (e.g., sunscreens)
- Permanent sensitization to light?

Irritant contact dermatitis

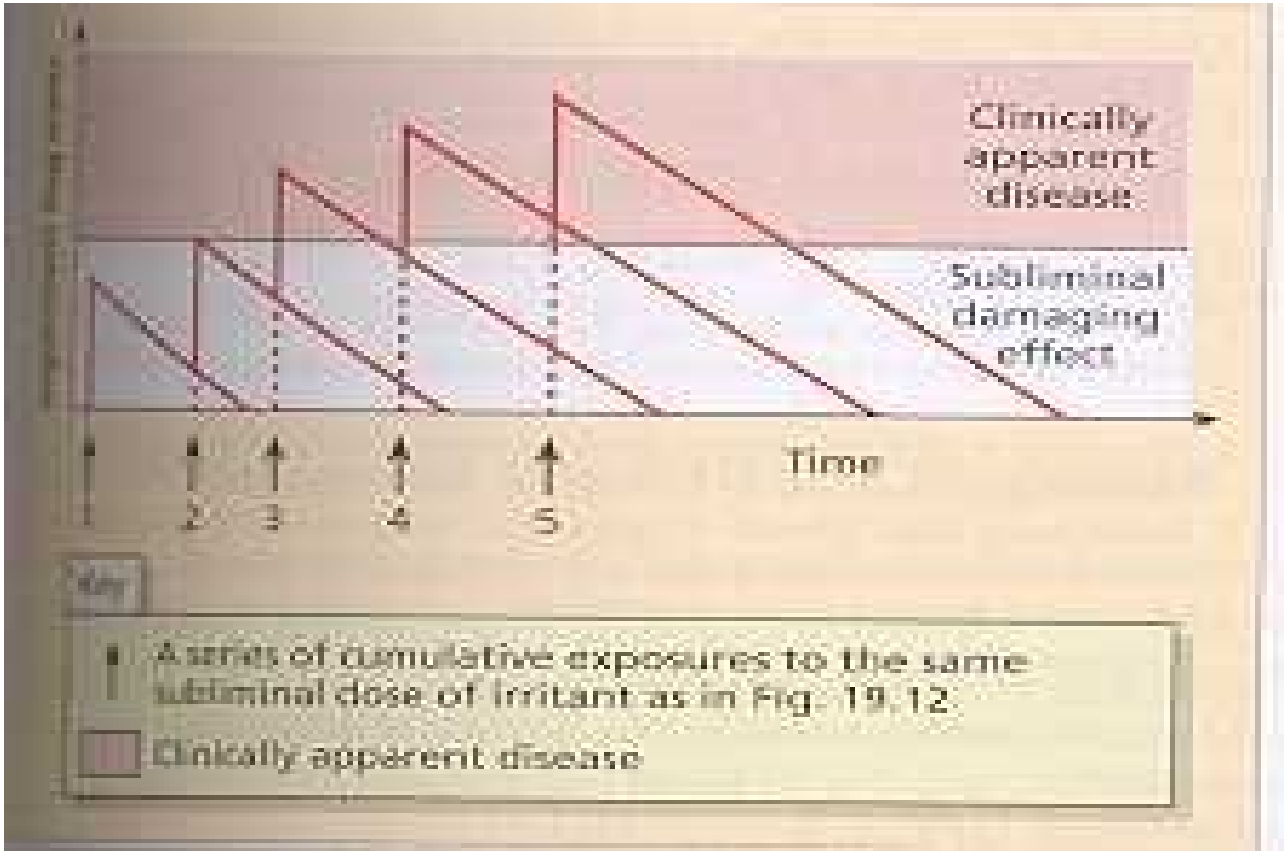
- Disease of the *stratum corneum*

- Solvents: Removal of lipids
- Acids, alkalics, salts: Destroy proteins
- Dust: Direct mechanical destruction

Irritant contact dermatitis (2)

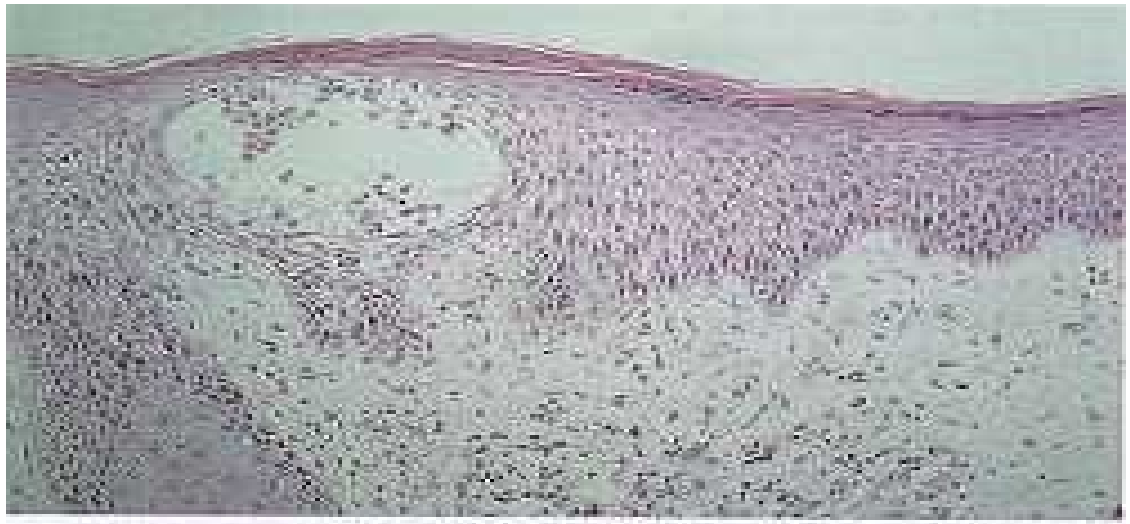
- Endogenous factors: Dryness vs wetness
- Sweating
- Age
- Atopic predisposition

Irritant contact dermatitis



Occcupational irritant contact dermatitis

- 35% Washing
- 10% Solvents
- 6% Plastics and adhesives
- 6% Foodstuff
- 5% Dirty, wet work
- 5% Mineral oils



(a)

Allergic contact dermatitis

- Caused by low-molecular weight haptens
- Hapten is “incomplete allergen”
- Binds to carrier protein for immunogenicity
- Low molecule weight enables penetration of hapten

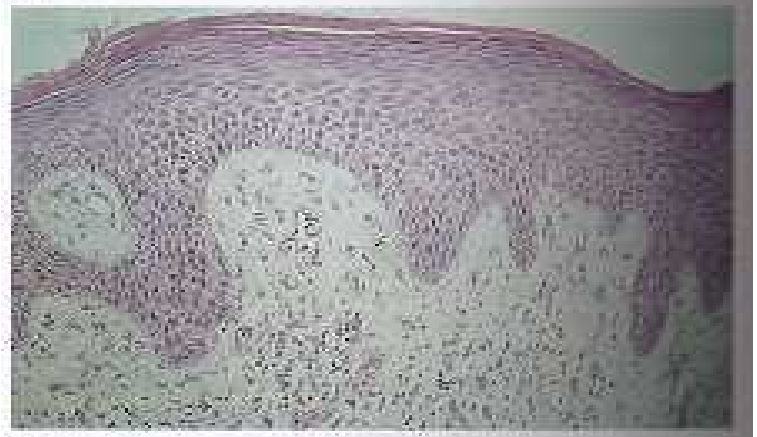
ALLERGIC CONTACT DERMATITIS - INDUCTION

- Induction (sensitization) occurs if hapten is allergenic and /or topical dosage is large enough
- Approximately 2 weeks later person is allergic to the same hapten chemical

ALLERGIC CONTACT DERMATITIS - ELICITATION

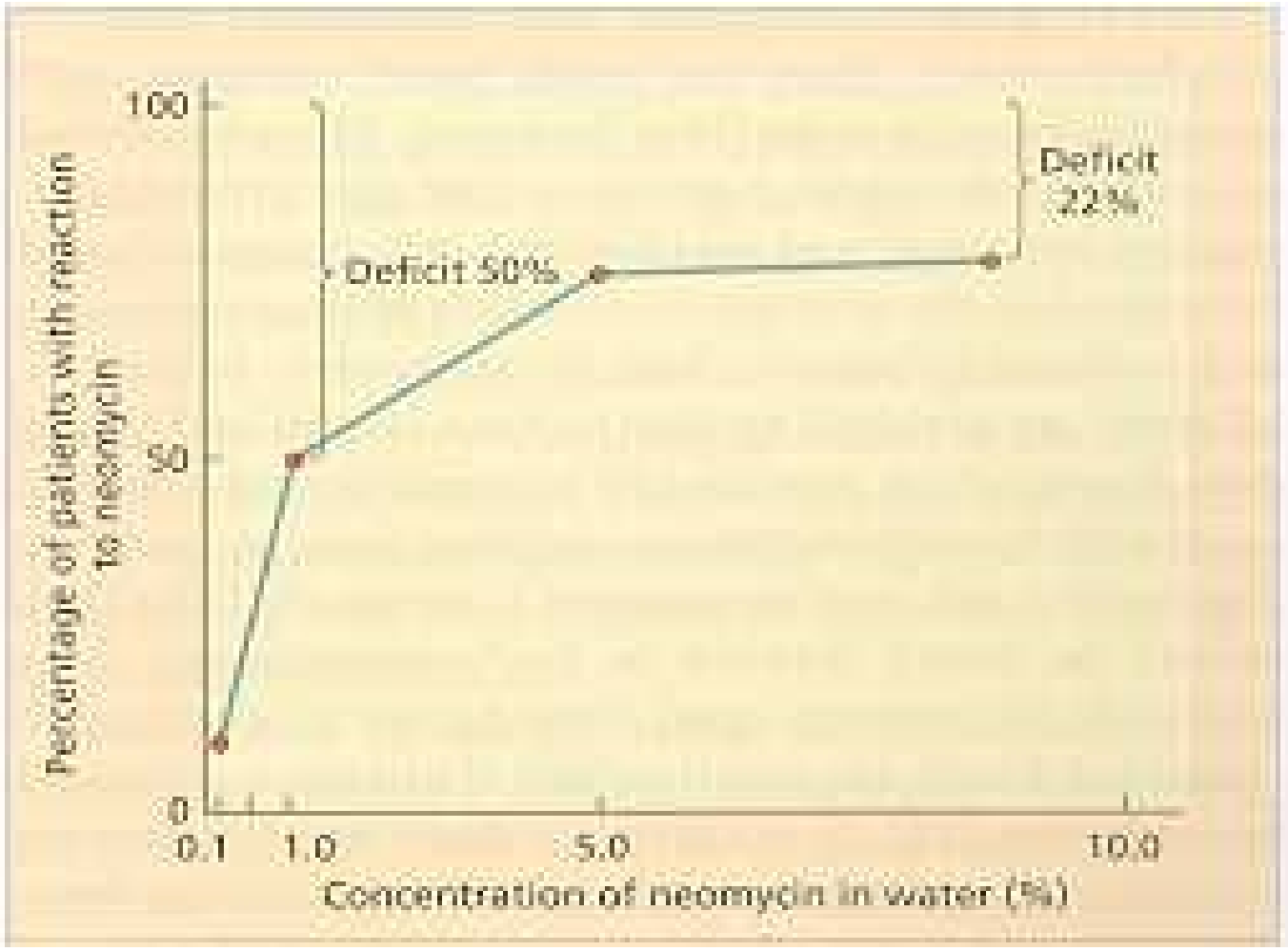
- Hapten penetrates through stratum corneum of a sensitized individual
- A classical Type IV reaction ensues in the form of eczema/dermatitis

(a)



(b)

(c)


100
50
0
Percentage of patients with reaction to neomycin
Deficit 50%
Deficit 22%
0.1
1.0
5.0
10.0
Concentration of neomycin in water (%)

ALLERGIC CONTACT DERMATITIS TO RUBBER CHEMICALS

ALLERGIC CONTACT DERMATITIS TO RUBBER IN SOCKS

ALLERGIC CONTACT DERMATITIS TO PRESERVATIVE IN OINTMENT (KATHON CG)

ALLERGIC CONTACT DERMATITIS TO CHROMIUM IN CEMENT

ALLERGIC CONTACT DERMATITIS TO PERFUME IN SHAMPOO

ALLERGIC CONTACT DERMATITIS TO NICKEL SULPHATE - FACIAL CONTACT THROUGH FINGERS

ALLERGIC CONTACT DERMATITIS TO NICKEL PRESENT IN KEYS HELD IN POCKET OF TROUSERS

POMPHYLOX

FUNGAL INFECTION (TRICHOPHYTON RUBRUM)

ERTYHEMA AB IGNE

ATOPIC DERMATITIS

DIAGNOSIS OF OCCUPATIONAL SKIN DISEASE

- Patient history: Does skin disease relate to work?
- Exposure: Are there causative agents (allergens, irritants) in the work-place?
- Clinical symptoms: Are they in accordance to clinical disease?

CLINICAL FEATURES OF OCCUPATIONAL SKIN DISEASE

- When did disease start?
- In which skin area was the first symptom?
- What is work technique?
- Free time, other works
- Cleaning measures
- Protection
- Vacation, holidays

CLINICAL FEATURES OF CONTACT DERMATITIS

- Skin disease starts on the area of contact
- Dorsal aspects of hands and fingers, volar aspects of arms
- Redness, edema -> blisters, ulcerations
- Itch, pain, heat, stinging
- Contact dermatitis heals after exposure is discontinued

CLINICAL FEATURES OF CONTACT URTICARIA

- Hives (edema) appear on sites of contact within minutes
- The hives disappear within 1-4 hours
- Mild: Only itching
- Severe: Systemic symptoms (anaphylaxis)

DIAGNOSTIC TESTS

- PATCH TESTS
- PRICK TESTS
- SCRATCH TEST
- OPEN TEST
- USAGE TEST

PATCH TESTS

- Diagnosis of allergic contact dermatitis
- Hapten (~0.001-20%) in vehicle
- Finn Chamber
- 48 h apllication on back
- Reading at 2 days, 4-5 days (7-9 days)
- ?+ (erythema), + (erythema, edema), ++ (+vesiculation), +++ (+ bulla), IR (irritation)

PRICK TESTS

- A drop of allergen placed on top of skin
- Skin broken with lancet
- Positive control histamine
- Negative control vehicle
- Positive reaction: at least 3 mm **and** histamine size
- Overall negative: Antihistamine
- Overall positive: Dermografismus

Occupational Skin Diseases

Introduction

- The second cause of occupational diseases (23-25% of all occ.diseases)
- A skin disease that is caused by physical, biological or chemical factor in work
- Also a worsening of pre-existing skin disease can be termed as occupational skin disease (Psoriasis , Acne)

Absenteeism & Cost

- 4 million working days are lost due to occupational skin disease and the UK Health & Safety Executive has calculated an associated cost to British industry of £200 million per year.

Work place agents that induced skin disorder

- **Chemicals**

Acids
Alkalis
Solvents
Oils
Detergents
Resins
Plastics
Metals
Petroleum product
Plant & wood

- **Physicals**

Temperature
Ionizing radiation
Non ionizing radiation

- **Biologic**

Viruses (orf-wart-herpes)
Bacteria(anthrax-erisopeloid)
Fungi(candida-dermatophyte)
Parasites(scabies-(schistosomiasis)

- **Mechanicals**

Pressure
Friction
Vibration

Important causal agents of occupational skin illness

- **Machinery**
  - Cutting oils
  - Solvents
- **Metal products**
  - Solvents
  - Metallic salts
- **Electronic equipment**
  - Solvents
  - Plastic & resin
- **Food products**
  - Fruits & vegetables
  - Soap & detergents
- **Agriculture**
  - Chemicals
  - Fruits & nuts
- **Health services**
  - Soap & detergents
  - Infectious agents

Diagnosis Of Occupational Skin Diseases

- History : present illness, occ.information, personal history
- P/E
- Diagnostic techniques
- Supplemental information

Questions

- When did disease start?
- In which skin area was the first symptom?
- What is work technique?
- Free time, other works
- Cleaning measures
- Protection
- Vacation, holidays

Classification of skin diseases

- Occupational dermatitis
- Occupational photosensitivity reactions
- Occupational phototoxicity reaction
- Occupational skin cancers
- Occupational contact urticaria
- Occupational acne
- Occupational skin infections
- Occupational pigmentary disorders
- Miscellaneous

Case 1

Erythema , dryness and itching on the right hand of a printer man

What is your diagnosis ?

Contact Dermatitis

- Occupational dermatitis is an inflammation of the skin causing itching, pain, redness, swelling and small blisters.

- Contact dermatitis is an eczematous eruption caused by external agents, which can be broadly divided into:

  - Irritant substances that have a direct toxic effect on the skin (irritant contact dermatitis, ICD)

  - Allergic chemicals where immune delayed hypersensitivity reactions occur (allergic contact dermatitis, ACD).

Prognosis Of Occupational Dermatitis After Treatment

- 25% complete recovery
- 25% refractory
- 50% remitting / relapsing

How exposure can occur

Direct handling

Contaminated surfaces

Splash ing

Immersion

Deposition

Classification of ICD

- Acute
- Chronic

Clinical Features Of Contact Dermatitis

- Location
  - Skin disease **starts** on the area of contact.
    - Dorsal aspects of hands and finger
    - Volar aspects of arms
    - Interdigital webs
    - Medial aspect of thighs
    - Dorsal aspects of feet
  - May in face (forehead, eyelids, ears, neck) and arms due to airborne irritant dusts and volatile irritant chemicals

Acute Irritant Contact Dermatitis

- Commonly seen in occupational accidents
- Irritant reaction reaches its peak quickly, within minutes to hours after exposure
- Symptoms include stinging, burning, and soreness
- Physical signs include erythema, edema, bullae, and possibly necrosis
- Lesions restricted to the area where the irritant or toxicant damaged the tissue
- Sharply demarcated borders and asymmetry pointing to an exogenous cause
- Most frequent irritants are acids and alkaline solutions


DOIA
(c) University Erlangen,
Department of Dermatology

Chronic (cumulative) ICD

- Repetitive exposure to weaker irritants

  -Wet : detergents, organic solvents, soaps, weak acids, and alkalis

  -Dry : low humidity air, heat ,dusts , and powders

- Disease of the *stratum corneum*

Occupational at high risk

- **Cleaner**
- **Housekeeping**
- **Construction**
- **Food service**
- **Medical dental**
- **Engineer**
- **Hairdresser**
- **Mechanic**
- **Printer**
- **Butcher**
- **Agricultural/Gardening**
- **Machinist**

Clinical Features Of Contact Dermatitis

- Sign and symptoms
  - Cumulative (exposure to weak irritants)
    - Delayed pain and burning
    - Vesicles and little pruritus
    - Lichenifications, fissures

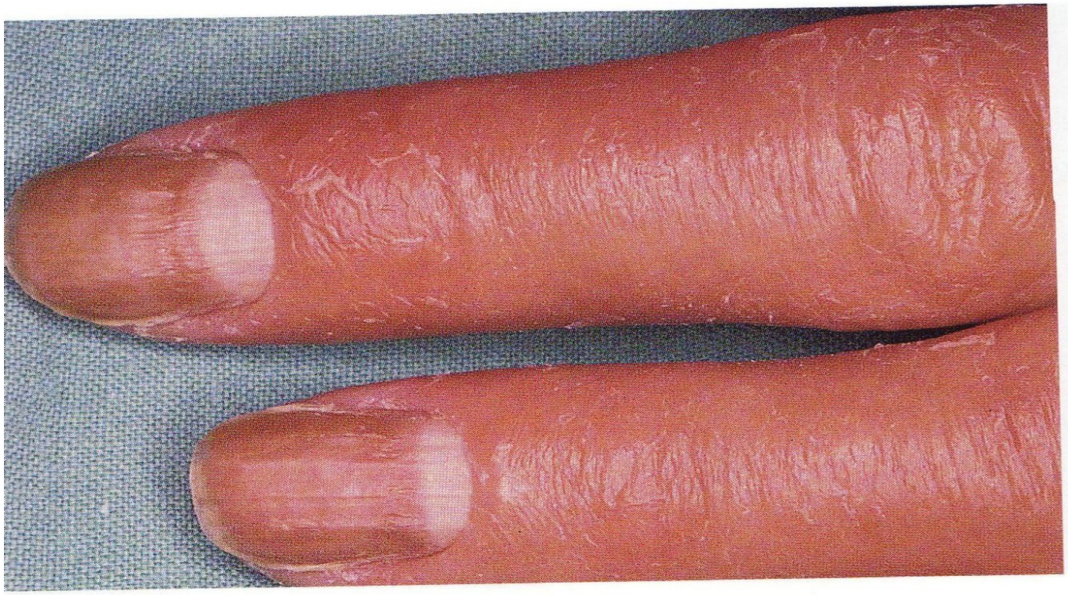
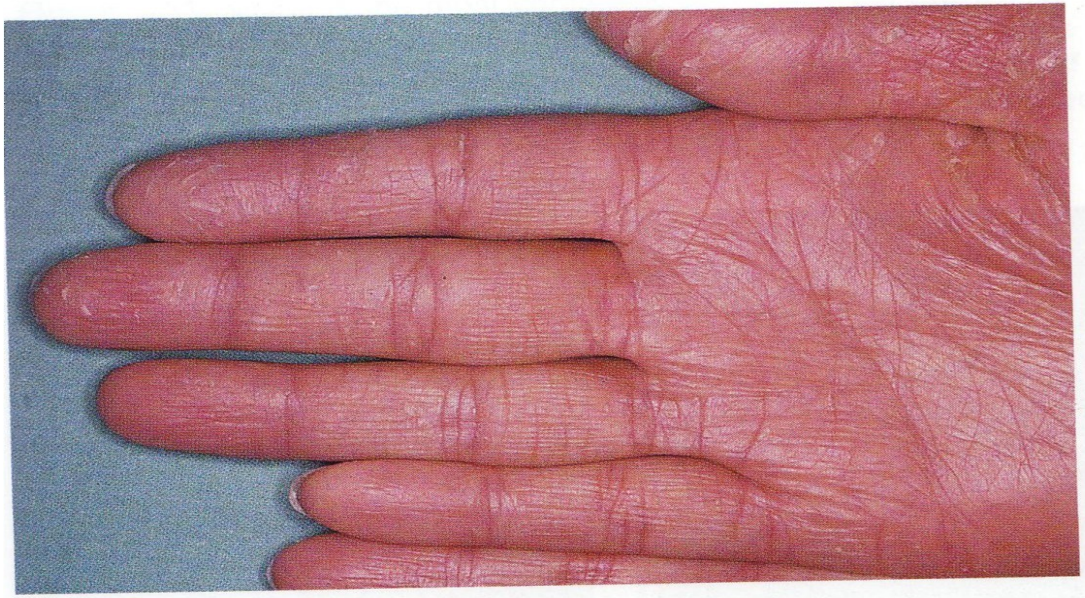


Figure 7.27 This intensive care unit nurse had irritant contact dermatitis. Patch tests were negative, and repeated washing of the hands with disinfectant products was believed to be the source of this dermatitis. Irritant contact dermatitis is more common than allergic contact dermatitis in most studies (Camarasa and Conde-Salazar, 1987; Meding and Swanbeck, 1990).



(**a**)

Figure 7.28 It is not unusual for a worker to suspect the chemicals that they handle in their work to be responsible for their dermatitis. (**a**) A laboratory technician worked with the materials seen in (**b**), and believed these to be the cause of the eruption. The correct diagnosis was irritant contact dermatitis, and patch tests were negative (Camarasa and Conde-Salazar, 1987).

Late fissuring in irritant contact dermatitis

1. Elimination or substitution of hazardous substances, e.g. chromate.
2. Technical control measures, e.g. damp dusts, ventilation extraction.
3. Personal protection, e.g. gloves, barrier creams, emollients, personal hygiene.
4. Identification of susceptible individuals, e.g. pre-employment health questionnaires, patch tests.
5. Education/training and health surveillance, e.g. induction, surveillance programmes.

Figure 3. Occupational contact dermatitis: strategies for prevention.

Case 2

A builder man presented with erythema , scaling and pruritus on his hands

What is your diagnosis ?

Allergic Contact Dermatitis

- Caused by low-molecular weight haptens
- Hapten is “incomplete allergen”
- Binds to carrier protein for immunogenicity
- Low molecule weight enables penetration of hapten

- Hapten penetrates through stratum corneum of a sensitized individual
- A classical Type IV reaction

Allergic contact dermatitis

- **The most common causes of an occupational allergic contact dermatitis are:**

1- rubber (23.4% of cases)
2- nickel (18.2% 0f cases)
3- epoxy and other resins (15.6%)
4- aromatic amines (8.6%)
5- chromate (8.1%)
6- fragrances
7- cosmetics (8.0%)
8- preservatives (7.3%)

- **Patch testing with the suspect material will confirm the correct diagnosis**
- **Typically there is an eczematous patch test reaction**

Clinical Features (Acute Form)

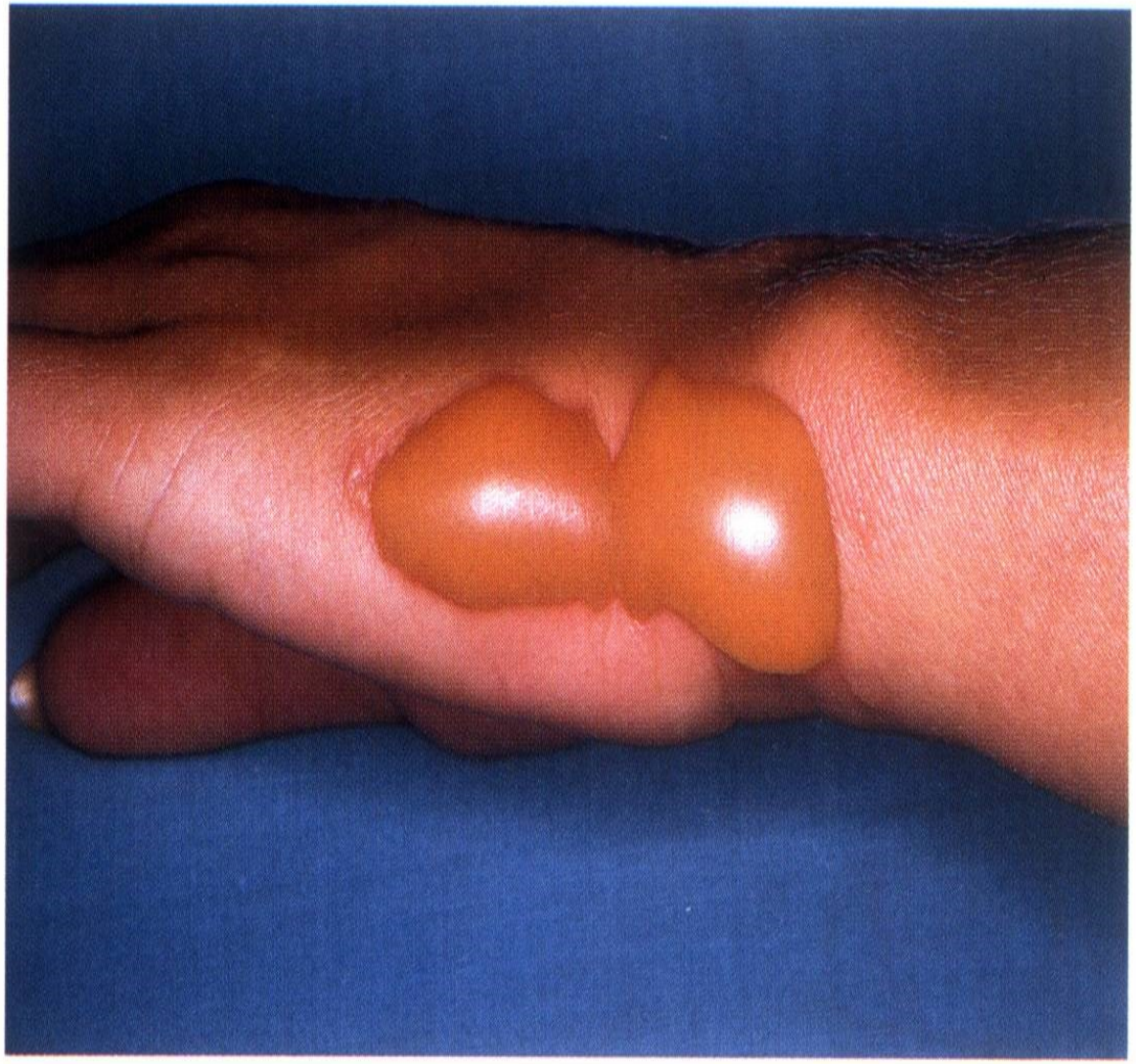
- Rash appears in areas exposed to the sensitizing agent, usually asymmetric or unilat.
- Sensitizing agent on the hands or clothes is often transferred to other body parts.
- The rash is characterized by erythema, vesicles and sever edema.
- Pruritus is the overriding symp.

Clinical Features (Chronic Form)

- Thickened , fissured, lichenified skin with scaling
- The most common sites:
  - Dorsal aspect of hands
  - Eyelids
  - periorbital

222 Bullous allergic contact dermatitis from *Rhus* species in a North American nursery worker.

Figure 1.3 *Allergic:* This is a classic example of allergic contact dermatitis, showing typical clinical lesions, with vesicles, blisters and exudation.



(a)

Figure 7.51 Acute dermatitis developed in this woman after she made leather handbags glued together with a *p-tert*-butylphenol–formaldehyde resin type of product. The dermatitis is seen in (**a**). The glue is seen in (**b**). Patch test to the *p-tert*-butylphenol–formaldehyde resin in the standard screening series was negative, but, as noted in the introduction to this section, the specific resin may be required to confirm the allergen.

Figure 4.66 The tips of the fingers can be the exclusive site of allergic contact dermatitis, as seen in this dentist because of the nature of his exposure to acrylates. Florists may develop a nearly identical fingertip eruption from *Alstroemeria* plants (van Ketel et al, 1975). Working with tulip bulbs can likewise cause a fingertip eruption, and both garlic and onion can also produce this picture (Sinha et al, 1977). (See also Figures 7.92–7.95.)

Figure 4.99 The rubber insole of the black rubber boots worn by a construction worker was responsible for this plantar dermatitis. The allergens proved to be the antioxidants added to the insole: *N*-isopropyl-*N*′-phenyl-*p*-phenylene-diamine (IPPD) and cyclohexyl-phenyl-paraphenylenediamine (CPPD).

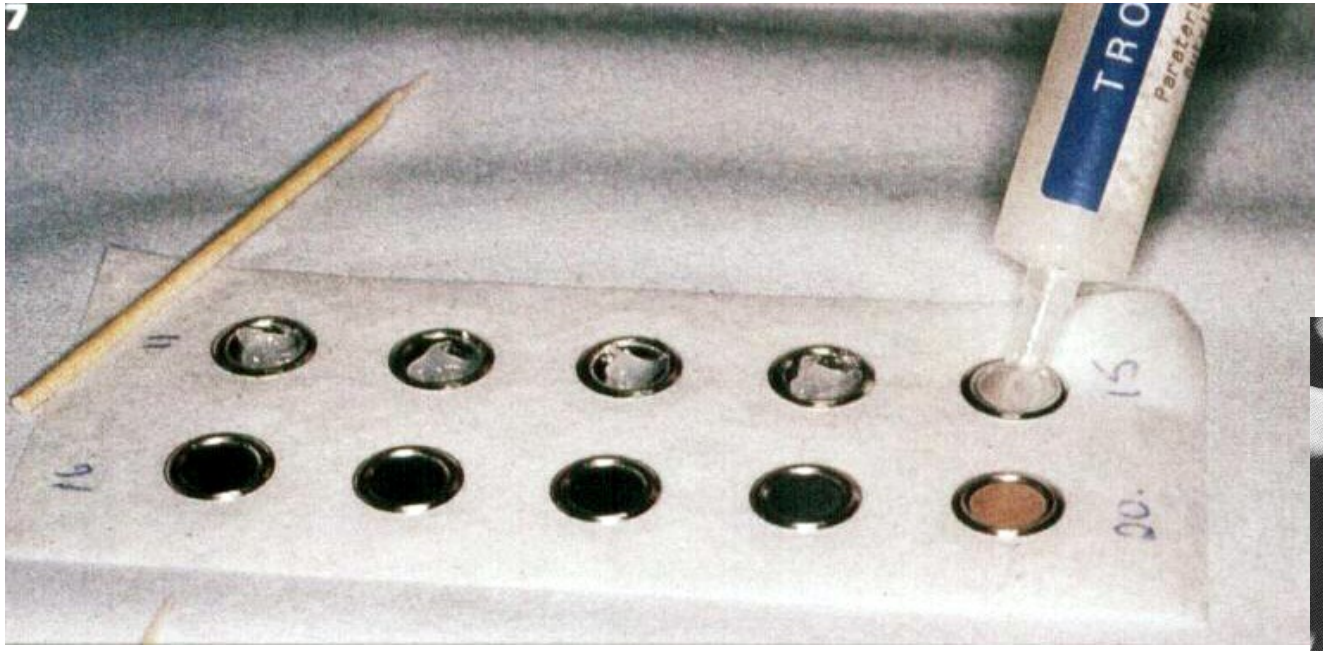
Figure 4.100 This chronic dermatitis on the dorsum of the foot was caused by chromate contained in the leather of Spanish-manufactured footware.

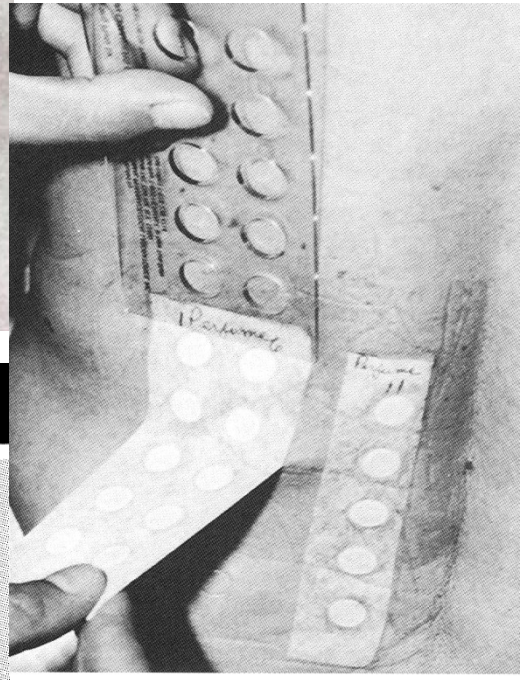
Chronic OACD in an employee with exposure to cable filler gel confirmed with patch test

Diagnosis

- Complete history
  - Occupational
  - Non-occupational
- Physical examination
- Patch test

Patch test

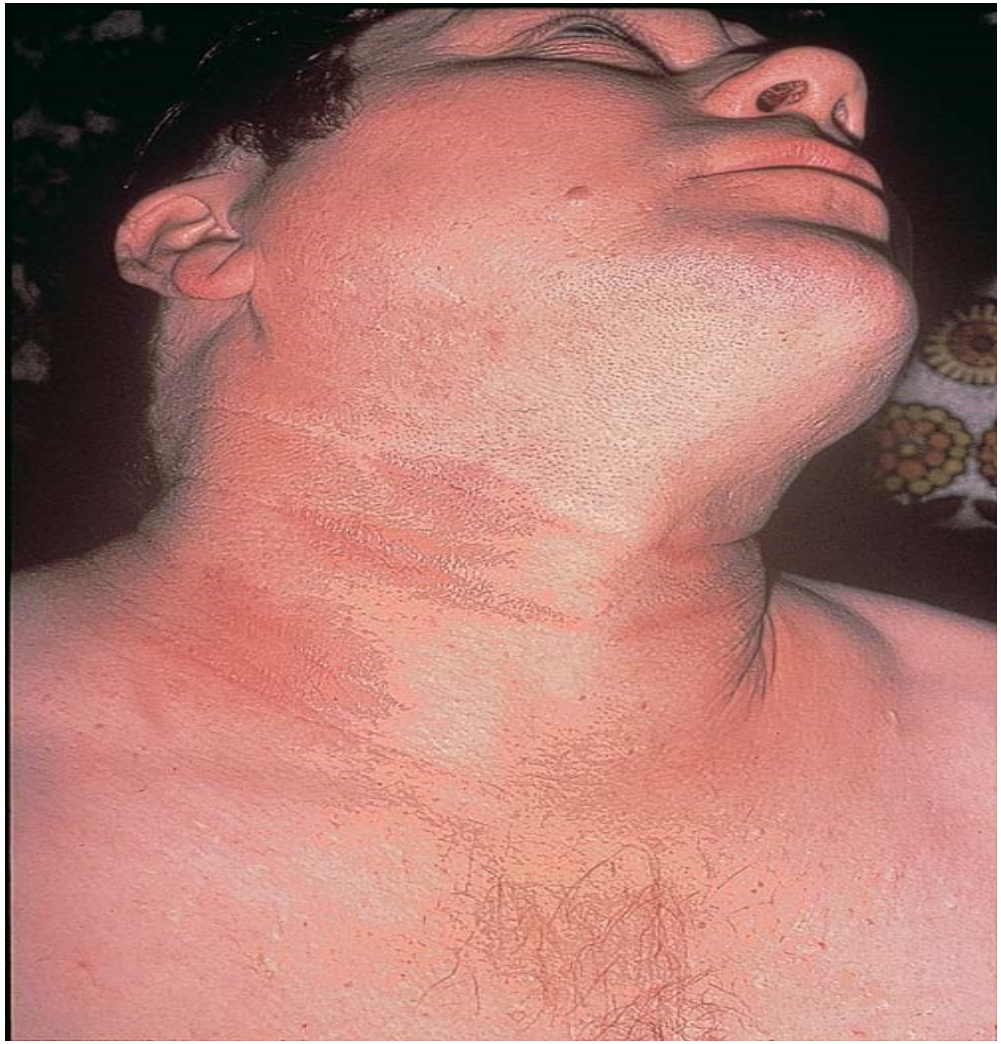




Patch Test Reading: Morphology Codes*

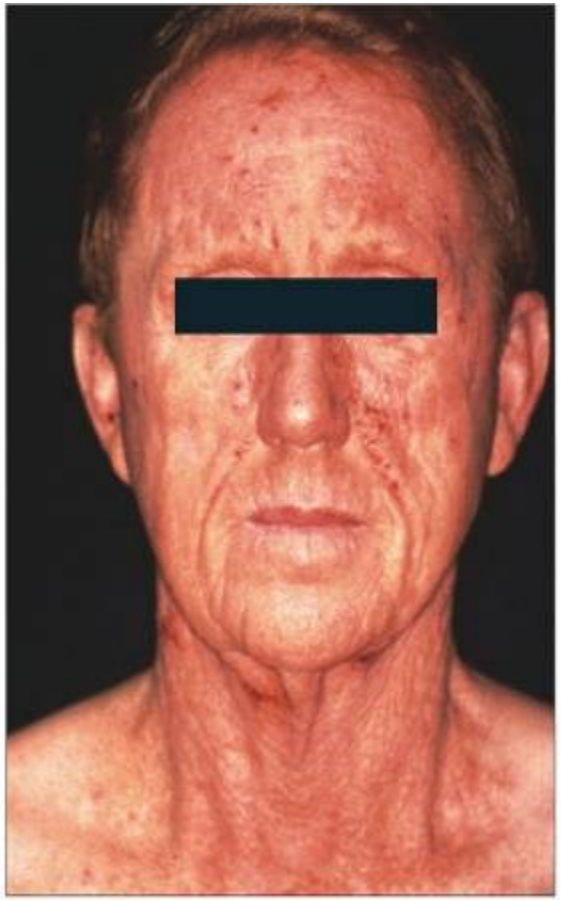
+/−	=	Macular erythema only
+	=	Weak (nonvesicular) reaction: erythema, infiltration, possibly papules
++	=	Strong (edematous or vesicular) reaction
+++	=	Extreme (spreading, bullous or ulcerative) reaction
IRR	=	Irritant morphologic appearance
−	=	Negative reaction
NT	=	Not tested

Airborne contact dermatitis



Airborne Irritant Contact Dermatitis

- Develops on irritant-exposed skin of the face and periorbital regions
- Often simulates photoallergic reactions
- Involvement of the upper eyelids, philtrum, and submental regions help to differentiate from photoallergic reaction



Case 3

A worker with itchy papules on his forearm

What is your diagnosis?

DX : Fiber glass dermatitis (kind of CD)

- Mechanism of skin injury is via direct penetration
- Pruritus and tingling are the usual initial symptoms
- Subsequently erythematous papules develop (often with follicular accentuation) on exposed areas when there is airborne exposure or on the forearms when there is contamination of a work surface
- Paronychia is common and airborne exposure may also cause burning eyes, sore throat and cough

Diagnosis :

- Clinical findings
- Confirmed by finding the fibers on tape stripping of affected skin or examining skin scrapings in KOH 20%.

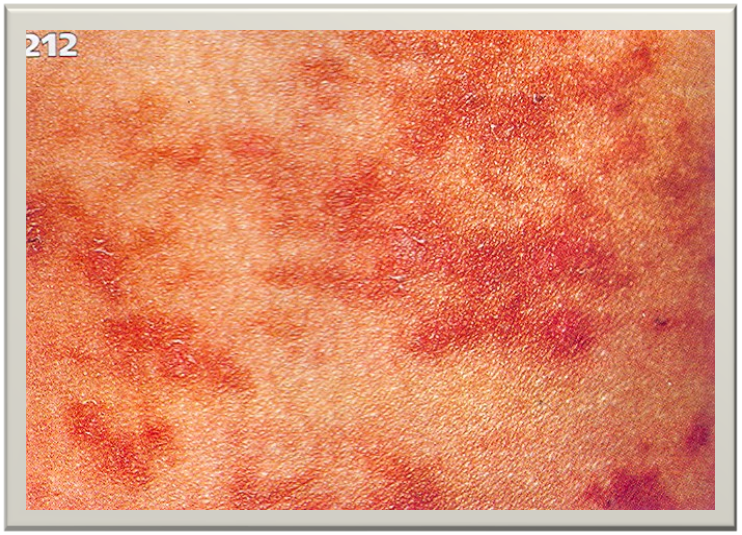
Course:

- Resolves rapidly after cessation of exposure.
- In most individuals, hardening occurs and symptoms resolve over a few weeks despite of continued exposure.

Case 4

Erythematous bullous reaction on the elbow of a gardener and hyperpigmentation after resolving this eruption

What is your diagnosis?



Contact photodermatitis

- Some chemicals may cause CD only in the presence of light
- Sunlight or artificial light sources that emit specific wavelengths
- 2 categories:

  -phototoxic

  -photoallergic

Phototoxic

- Coal-tar derivative
- Dyes (Eosin)
- Drug

  -phenothiazines

  -sulfonamides
- Plants&derivative

  -lemon

photoallergic

- Antifungal agents
- Fragrances
- Halogenated salicylanilide
- Phenothiazines
- Sunscreens
- Whiteners

Where involved ?

- Exposed areas: face, ant. V of the neck, back of the hand, uncovered sites on the arm&leg
- Hairy areas, upper eyelids, and below the chin may be spared

85 After making a large batch of celery soup this patient used a sunbed producing phototoxic blisters 48 hours later on the backs of her hands.

Case 5

Erythematous patches with tingling on the fingers of a health worker

What is your diagnosis ?

Occupational Causes

- Latex allergy (m/c)
- Formaldehyde
- Food industry
  - Plants
  - Vegetables
  - Animal products
- Pharmaceutical industry
  - Streptomycin

DX : Contact urticaria (due to latex)

- Pruritus and wheal-and-flare reaction
- Develops within a few to 60 minutes of exposure and resolves within 24 hours
- It is the protein content of latex rubber that is responsible for the associated contact urticaria
- In suspected cases of latex-induced contact urticaria, the specific IgE test may be negative, requiring prick testing with a commercial latex extract
- Symptoms include the development of pruritus and localized urticaria soon after donning latex gloves

Management of contact urticaria

1- Avoidance :

occupational hygiene

use of personal protective equipment (In the case of latex, the use of powder-free gloves containing low levels of protein should reduce the development of latex hypersensitivity in the future by reducing the level of exposure

change of occupation

2- Systemic antihistamines & epinephrine depending on the severity of the attack.

3- latex desensitization without significant risk of systemic adverse reactions in the future

Occupational Skin Cancers

- The second m/c form of occupational skin diseases
- About 17% of all cases of occupational skin diseases

Occupational Skin Cancer

- Ultraviolet light
- Poly cyclic aromatic hydrocarbones
- Arsenic
- Ionizing radiation
- Trauma

Table 1. Occupations at risk for occupational skin cancer

Causative agent	Occupation
Arsenic	Manufacture of insecticide or herbicide
	Agricultural exposure to pesticide
	Smelting of copper, lead, zinc
	Mining of arsenic
Polycyclic hydrocarbons	Distillation of coal tar
	Manufacture of coal gas
	Working with shale oil, creosote, asphalt and chimney soot
Ultraviolet irradiation	Outdoor work, e.g. agriculture, driving, fishing and construction
	Welding
	Laser exposure
	Certain printing processes
Ionizing radiation	Nuclear plant operations
	Diagnostic X-ray work
	Uranium mining
Burn	Welding

Neoplasm
Poly Aromatic Hydrocarbons

- Dimethylbenzantheracene , Benzyprine
- After latent intervals of 6-20 years :

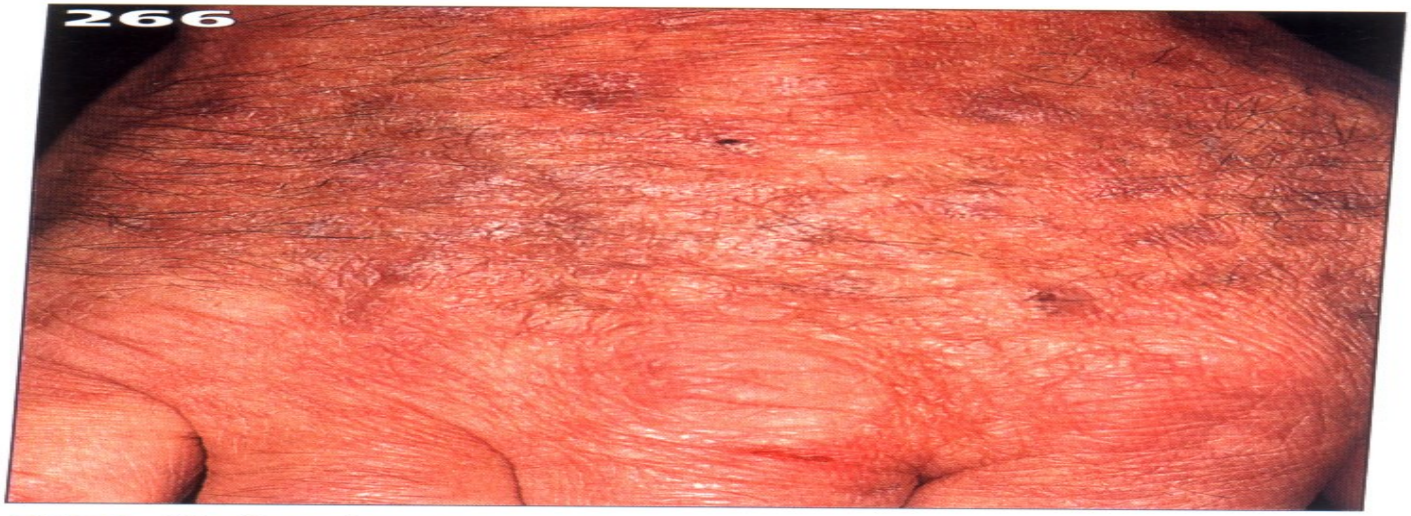
  keratotic papillomas (tar warts) in face ,forearms ,hands ,ankles ,dorsal feet ,scrotum
- Co factors: UV ,trauma

Neoplasm
Arsenic

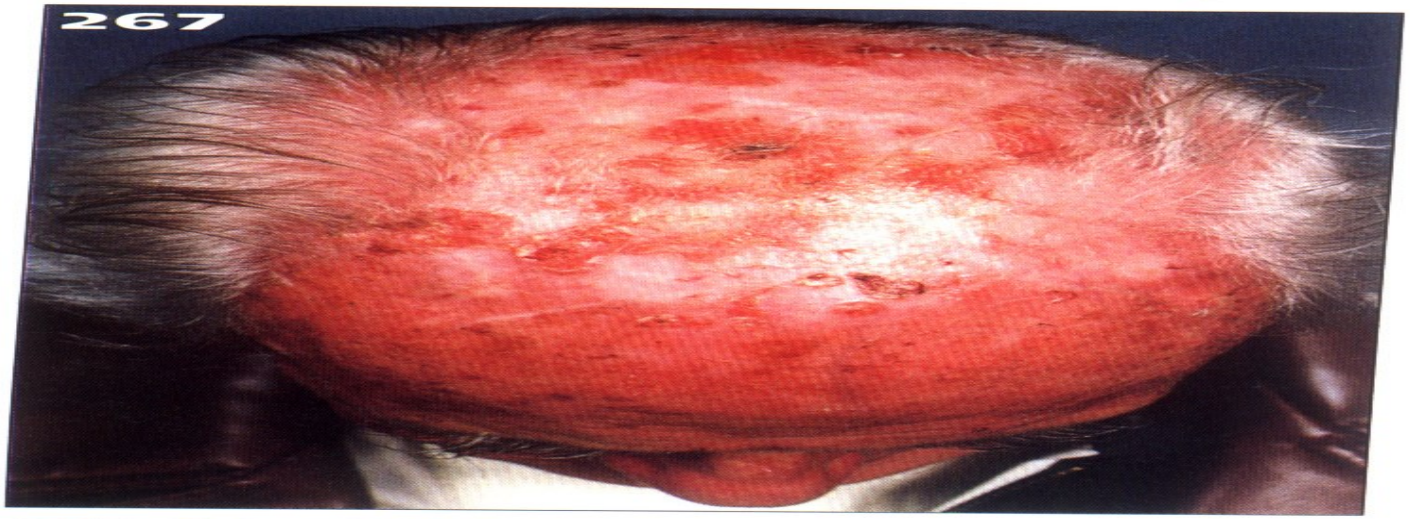
- Chronic exposure: (water, fowlers solution ,inorganic arsenic)

  punctuate ,keratotic papules (arsenic keratosis) ,on palms and soles

- No exposed skin surfaces ,intra epidermal SCC (Bowens disease)



266 Solar keratoses.



267 Solar keratoses.

264 Tar keratoses.

265 Tar keratoses.

259 Pigmented tar keratoses.

260 Squamous cell carcinoma on the back of the hand of a soldier who had observed nuclear weapons tests.

261 Multiple BCC following arsenic ingestion.

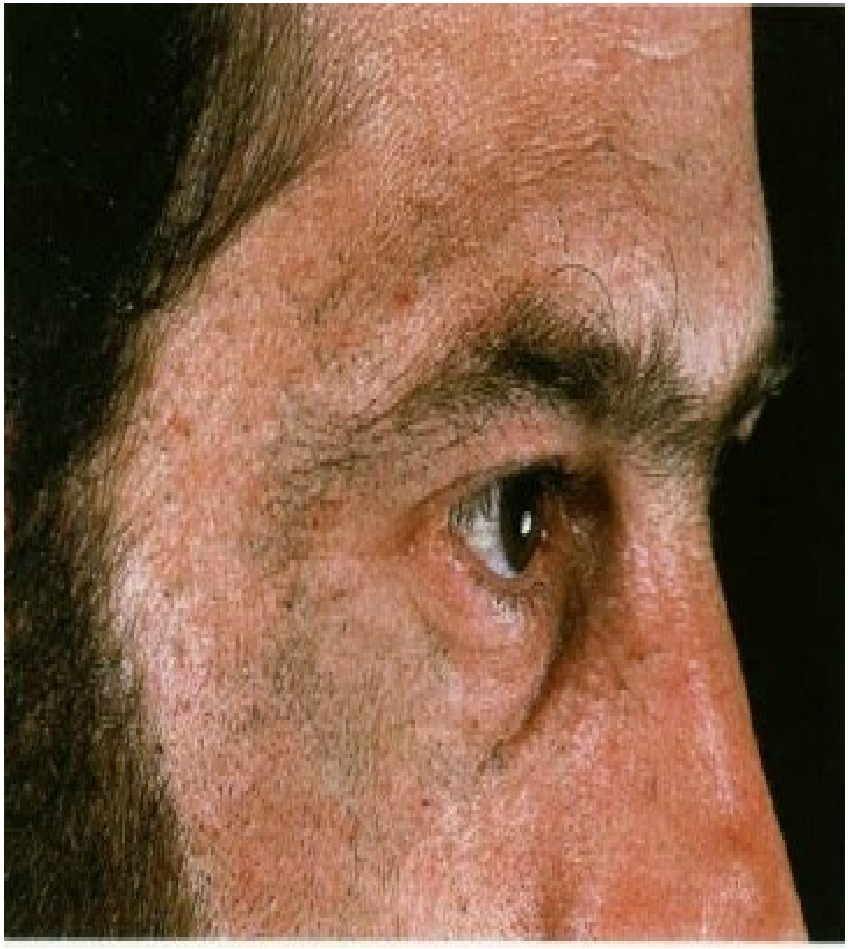
268 Pigmented BCC.

269 Ulcerating nodular cystic BCC.

270 Morphoeic BCC on the nose.

271 Superficial BCC on the shoulder of a professional yachtsman.

Case 7



A worker man presented with numerous comedons on his face.

What is your diagnosis ?

Case 8

A worker man with numerous retroauricular comedones and cysts

What is your diagnosis?

Environmental Acne

- Preexisting acne vulgaris may be aggravated by various occupational stress

1-Tropical acne: acne prone individuals employed in tropical climates

2-Acne mechanica: tight fitting work clothing ,pressure from seat belt

Oil Acne

- Lubricating petroleum greases ,oils ,and pitch fumes may cause follicular plugging and postular folliculitis and is seen not infrequently in machinists and automotive mechanics.
- Mechanism : stimulation of follicular keratinization followed by ductal occlusion

Chloracne

- Caused by polychlorinated or poly brominated aromatic hydrocarbons (halogen acne)
- Mechanism: induction of metaplasia ,keratin filled cysts
- Noninflammatory comedones and cysts in malar crescents and posterior auricular folds
- Poly Chlorinated Biphenyl (PCB)

Age
Differential Features of Acne

• Oil acne	Any age
• Acne vulgaris	Peak incidence, ages 11-20
• Chloracne	Any age

Distribution
Differential Features of Acne

- Oil acne — Exposed area
- Acne vulgaris — Face ,Neck ,Chest
  - Chloracne — Face, Especially Malar Crescent & Auricular Creases, Axillae, Groin, Nose Spared

Associated Conditions
Differential Features of Acne

- Oil acne — None
- AcneVulgaris — None
- Chloracne — Xerosis, Conjunctivitis, Actinic Elastosis, Pheripheral Neuritis, Liver Abnormalities

239 Oil folliculitis with follicular plugging and inflammatory papules (courtesy of the National Institute for Occupational Safety and Health, Cincinnati, OH).

Pigmentary disorders

Pigmentary disorders

- Melanosis

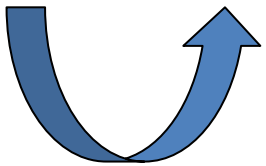
  -repeated trauma, friction, chemical & thermal burns, UV

  -coal tar, pitch, asphalt, creosote

- Leukoderma

  -hydroquinone, phenol

  -hand & forearms spread



(a)

(b)

Figure 4.101 (**a**) The cause of the loss of pigment on the dorsum of the foot was an adhesive that contained *p-tert*-butylphenol–formaldehyde resin. The depigmentation was not post-inflammatory. This type of depigmentation does not require antecedent dermatitis, and is seen in industrial exposures to this same class of compounds (Rietschel and Fowler, 1995, p.770). (**b**) An example of post-inflammatory depigmentation.

SEQUELAE OF CONTACT DERMATITIS

Figure 1.14 This 31-year-old woman with post-inflammatory hyperpigmentation had an antecedent nickel dermatitis due to a jeans button.

Figure 1.15
Hyperpigmentation may be the sequela of many forms of dermatitis; in this example the initial eruption was stasis dermatitis.

Figure 1.16 This is a 76-year-old man with post-inflammatory hyperpigmentation following mechanical dermatitis from his belt.

Physical cause of occupational skin disorders

- Mechanical trauma:callus,corn, lichenfication
- Permanent callus leading to early retirement
- Callus with painful fissure become infected
- Prevention : not necessarilly

heat

- Burn,miliaria, intertrigo
- Burn: after burn hypopigmentation susceptible actinic damage
- Hyperpigmentation and scar are disfiguring
- Miliria: sweat retention
- 3 Types: -
- M.crystalina: upper epidermis
- M.rubra: lower epidermis
- M.perfounda: upper dermis

- DX : clinical picture, Hx of excessive heat exposure
- Prevention: avoiding of exposure, hexachlorophen soap, frequent clothing changes


heat rash

continue

- Intertrigo:macerated, erythematus lesion in body fold
- Result excessive sweating in obese worker
- Common site is interdigital space between third and fourth finger
- Bacterial and fungal infection is common

cold

- Frosbite,chilblain
- Frosbite: progressive vasoconstriction cause impairment circulation
- Clinical symptom in mild form: redness, transient anesthesia, superficial bullae →Initial redness replace by white waxy appearance → blistering & later necrosis

- Long-term effects: Raynaud-like change paresthesia, hyperhydrosis
- Scc develop in old scar
- Rewarming, analgesic, surgical debridement
- Prevention: protective clothing, educating.

cold

- Chilblain:mild form of cold injury
- Reddish,blue,swollen,boggy discoloration with bulla and ulceration
- Finger,toe,heel,nose,ear are effected
- Genetic is important back ground
- Treatment : symptomatically

Vibration syndrome

- Vibration tool in cold weather produce vasocostriction of digital arteries.(30-300)
- pallor, cyanosis, erythem of finger named raynaud phenomen
- Papular name : dead or white finger.

- Tingling, numbness, blanching of the tip of finger occurred
- Asymmetry is diagnostic
- Prevention: design of tools, insulation, protection of hands from cold weather.


With thanks