INTERNATIONAL RESEARCH JOURNAL OF MULTIDISCIPLINARY STUDIES

Vol. 6, Issue 9, September, 2020 | ISSN (Online): 2454-8499 | Impact Factor: 1.8167 (GIF), 0.679(IIFS)

Contact vitiligo- A chemical induced vitiligo

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ABTRACT

Contact vitiligo is the loss of skin colour (whitening of skin) due to contact with chemicals. These chemicals destroy the skin pigment cells called as melanocytes.

A recent fascinating example of contact vitiligo is occurred in the summer of 2013 in Japan.

When the cosmetic company "Kanebo" developed a highly effective skin lighting cream and sold it to thousands of consumers. Unfortunately over 18,000 users developed contact vitiligo after using it, leading to recall the product. An active ingredient in the product is "Rhododendrol" also a phenol.

This type of chemicals appears to be anlog of the amino acid tyrosine that disrupt melanogenesis and result in autoimmunity and melanocytes destruction.

Keywords: Vitiligo, leukoderma, chemical, phenol, rhododendrol, Cellular stress, autoimmunity

Introduction

Contact vitiligo is an acquired leukoderma that occurs as a result of repeated topical or systematic exposure to a variety chemicals

This chemicals used to manufacturing of plastics, resins, synthetic rubber, paints, pretoleum products, deodorants, pesticides, disinfectants, germicide, photographic chemicals(1) and also presents in certain products of our daily use. Eg. Footwear, plastic watchstrap, purse, bindis.

Vitiligo is one of the few autoimmune diseases in which environmental factor are well-known, including the depigmenting effect of the chemicals monobenzyl ether of hydroquinone (MBEH) discovered by Oliver and colleagues in tanning factory but includes many others as well.(2)

This study will summarize the chemicals that have been clearly impacted as causing vitiligo, as well as mechanism, etiology, etiopathogenesis, clinical features, diagnosis and treatment.

Chemicals directly impacted in inducing contact vitiligo

Table no. 1 chemicals associated with contact vitiligo	
Systematic medication	Chloroquine, Fluphenazine(prolixin)
Cachol Derivatives	PTBC, p-Methycatechol, p-Isopropycatechol Pyrccatechol (1,2-benzenediol)
Phenol Derivative	Monobenzyl ether of HQ, HQ (p-hydroxyphenol) Monomethyl ether of HQ(p-ethoxyphenol) Monoethyl ether Of HQ(p-ethoxyphenol) PTBP, p-tert-Amlphenol, p-Phenylphenol, P-Octyphenol p-nonyphenol, Butylated hydroxytoluene p-Cresol-(4-methylphenol)
Sulfhydryls	B-Mecaptoethylamine hydrochloride Sulfanolic acid, Cystamine dihydrochloride
Optic preparation	Eserine (physostigmine), Thio-TEPA, Gunonitrofuracin
Miscellaneous	Mercurials, Cinnamic aldehyde, Arsenic, P-Phenylenediamine, Benzyl alcohol, Benzoyl peroxide, Coeticosteriods, Azelaic acid Phenyl glycidyl ether, Rhodamine B Carmusstine , Acrylates, Nickel, Fluorouracil Tretinoin, Dintrochlorobezene, Squaric acid dibutylester
Source- (3),(4),(5),(6),(7),(8)	

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A large number of chemicals inducing contact vitiligo have been reported in human and animal studies, as well as in experimental in vitro works.

Aromatic and aliphatic derivative of phenols and catechols are use in commercial products , such as varnishes, adhesives, pesticides, resins, industrial oil, disinfectants, rubber items and painting inks.

MBEH, PTBP, PTBC, PPD,Rhododendrol are most common causal molecule in this chemicals groups

MBEH-

In 1939, Oliver, Shwartz, and Warren reported a case series of workers in a leather manufacturing company who developed patchy depigmentation on their hands and arms. In fact, 50% of the workers in this factory and others who wore a particular brand of gloves developed depigmentation on skin that contacted the gloves, and several of them also had similar lesions on remote areas that did not contact the gloves. The ingredients used in manufacturing the gloves were obtained by the medical team, and each systematically applied to the workers through patch testing. Only patches containing the antioxidant MBEH induced an inflammatory response, which was then followed by depigmentation. This chemical ingredient was removed from the gloves, and workers subsequently repigmented[9]

PTBP- Para-tertiary Butylphenol

Exposure to PTBP is widespread in the industry of synthetic leather, plastic, adhesives and germicide detergents

Sources of Exposure to PTBP	
Latex glues	Duplicating paper
Insecticide	Painting ink
Rubber antioxidants	Detergents
	Source-[10]

A case series reported depigmentation of the hands and forearms in 12 hospital worker, caused by phenolic germicide detergents [10]

A case series reported depigmentation of the of the hands and forearms in 99 automobile factory worker, caused by Para-tertiary Butylphenol[11]

A case series reported the characteristics of 100 consecutive patients who presented with depigmentation under their bindi, a decorative item worn on the forehead of many Indian women, often using an adhesive resin. [12]

PTBC- Para- Tertiary Butycatechol

A case reported depigmentation of hands in 75 tappet assembly workers due to exposure of PTBC, present in an assembly oil[12]

PPD- Para-phenylenediamine

A case reported depigmentation of forehead due to use of hair dye which contain PPD[13]

Rhododendrol.

In the summer of 2013 when Kanebo, a Japanese company, was forced to recall a new skin lighting cream after over 18,000 users developed vitiligo the active ingredient is rhododentrol.

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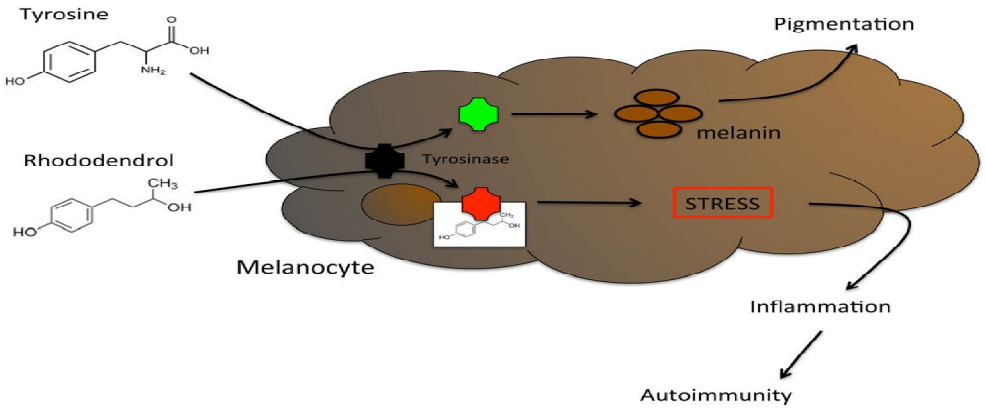
Etiopathogenesis



Tyrosine is processed into melanin by the enzyme tyrosinase. Chemicals that act as tyrosine analogs interact with tyrosinase (or other melanin-producing enzymes), disrupt melanin production, and induce the cellular stress response, which leads to inflammation and autoimmune destruction of melanocytes.

Mechanism -

Depigmentation from chemicals may be associated with an initial reaction consistent with allergic contact dermatitis, and so some have dismissed chemical-induced depigmentation as a nonspecific post-inflammatory change or koebner response to inflammation. However many cases occur in the absence of any apparent dermatitis, and most who experience contact dermatitis to these agents do not develop depigmentation. In fact, they typically develop post-inflammatory hyperpigmentation[(14)]

Clinical feature

Table no.3- Clinical feature

Age of onset	Usually Adulthood
Confetti macules	Present
Macules color	White to off-white
Lesion distribution	Mainly hands, face, arms, remote macules
Lesion margin	Sharp but ill defined

Diagnosis

To diagnose contact vitiligo

A detailed history is especially important in isolated cases.

No helpful laboratory test are available to conform the diagnosis expect " patch testing"

Imbalance of some blood parameters such as liver and thyroid enzyme levels

To detect the presence of chemicals inducing contact vitiligo, gas chromatography, thin- layer chromatography, infrared spectrometry, high pressure liquid chromatography and paper chromatography may be required.

Treatment

In the workplace, it is important to prevent and minimize exposure to known depigmenting agents.

Psoralen –UV-therapy use for contact vitiligo[(15)]

Spontaneous repigmentation has been reported in some cases after avoidance of causing agents.[(16)(17)]

Discussion

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Contact vitiligo is a depigmentation of skin induced by exposure to the variety of chemicals. Most cases results from skin contact but ingestion or inhalation can also be inducing factors. Contact vitiligo is reported as a disease with an occupational industrial origin, and with involvement of the hands and forearms, contact vitiligo can be induced by a large number of non occupational consumer products and involve all body areas.

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Fig no. 1-

Depigmentation of the feet in the distribution of sandal straps.

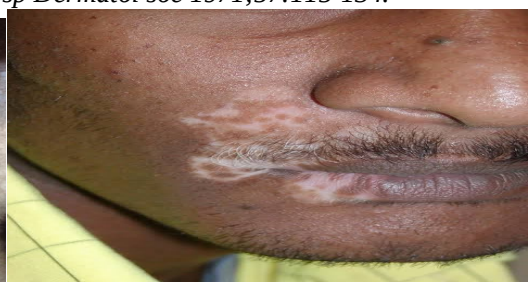

Fig no. 2

Man with depigmentation on the face after treating his goatee with a comb-in dye.

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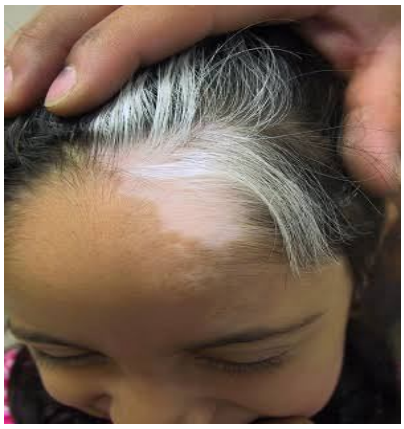

Fig no. 3
Depigmentation of the scalp occur due to hair dye.

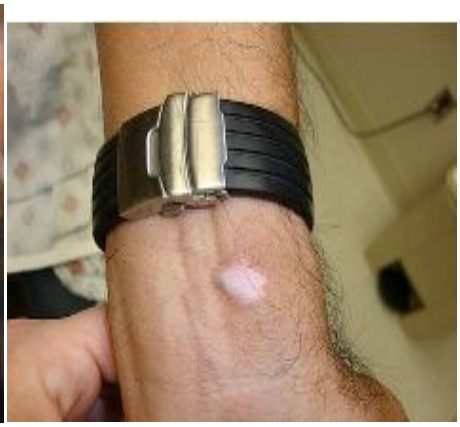

Fig no. 4
Depigmentation of the forearms due to watch straps.

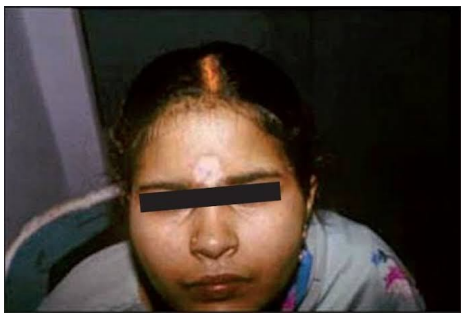

Fig no. 5
Depigmentation occur due to adhesive using in bindi contact with forehead.