

EPOXY RESIN AND ILLNESS IN THE WORKPLACE

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ABSTRACT

Epoxy Resin (ER) and Epoxy Resin Systems (ERS) were found to be the most common allergens in the workplace.¹ They have also been implicated in occupational asthma and cases of a reversible scleroderma-like presentation have also been described.² In addition to these, one of the main components of ER, bisphenol A (BPA), is currently very topical since it was found to cause endocrine disruption.³ ER and ERS are found across the spectrum of industry due to their wide range of uses. This article discusses contact dermatitis in a jewellery painter. The article outlines the nature of ER and its uses, the different types of contact dermatitis and their relation to ER and their management, the commonly affected industries and the association of ER with occupational diseases. The article highlights the need for the health care provider to suspect, diagnose and manage ER related disease as well as initiate changes in the workplace to improve health of all workers and to prevent loss of income to the company and worker because of time off from work due to ER related illnesses.

CASE

A 31 year old female presented with a 6 month history of a rash that started around her eyes and later extended to involve her neck and hands. The lesions were itchy blisters, which became weepy, painful when eroded, crusted, and left de-, hypo- and hyperpigmented patches upon healing. The itch was worse at work and was absent when she was on leave for a week, but came back when she returned to work. She had no history of atopy, eczema or other medical conditions.

She worked in the jewellery and silverware manufacturing industry where she created enamel jewellery using resins, hardeners, pigments and thinners. She had no skin problems at previous jobs which included plastic assembly, electrical assembly and clerical work.

Upon examination she had post inflammatory hyperpigmentation and hypopigmentation with patches of depigmentation. There was surrounding erythema with excoriations but no lichenification. This was noted on the face (periorbital, perioral and ears), the neck, upper back, forearms and hands. On the ear, crusts and peel were evident (Figure 1).

A diagnosis of contact dermatitis was made and because of the history and distribution, an airborne allergen exposure in the work environment was suspected. Due to the distribution and the remarkable pigmentary changes, a skin biopsy was taken from behind her ear to exclude discoid lupus erythematosus. The histology showed parakeratosis,

psoriasiform hyperplasia, necrotic keratinocytes, superficial to deep perivascular lymphocytes and eosinophils but no mucin, favouring contact dermatitis (Figure 2). Serum anti-nuclear antibodies were negative.

She was prescribed emollients as a soap substitute and moisturiser. Topical steroids and an evening dose of antihistamine were given therapeutically. She responded well



Figure 1a-1d: The patient's skin changes on presentation. (a) Significant excoriations on the upper back (b) Post inflammatory hypopigmentation with edge of hyperpigmentation and erythema. (c) Crust, scale and peeling skin on outer upper helix. (d) Erythema around eyes and on forehead with scattered vesico-pustules and post inflammatory pigmentary changes.

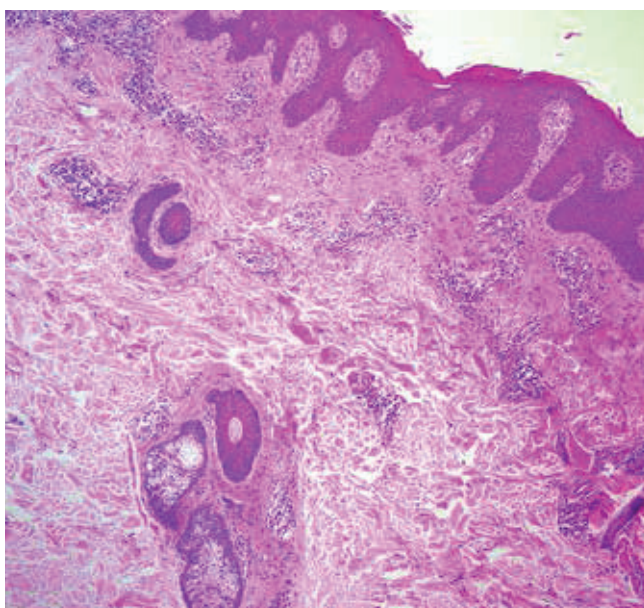


Figure 2: Histology of skin biopsy. The histology showed parakeratosis, psoriasiform hyperplasia, necrotic keratinocytes, superficial to deep perivascular lymphocytes and eosinophils but no mucin favouring contact dermatitis

to treatment with residual hyperpigmentation as well as patches of depigmentation.

A work visit was scheduled to assess her occupational risks and exposures. She worked in a room with four other people doing the same job. They sat at a table and painted small amounts of a solution of epoxy resin, hardener and a pigment on to small metal shapes using a fine wooden stick. During this process minor contamination of the hands occurred and was immediately wiped off onto a cloth (Figure 3). The workers mixed their own solutions as needed and were responsible for cleaning their tools using lacquer thinners. The painted items were then batch dried in an oven in the same room. No formal extraction system was in place. She did not use personal protective equipment.

We collected samples of the possible offending agents for patch testing as well as the material safety data sheets. The epoxy resin used contained diglycidyl ethers of BPA (DGEBA).

A patch test using 45 commercially available allergens that commonly cause contact allergy was done initially. The patch

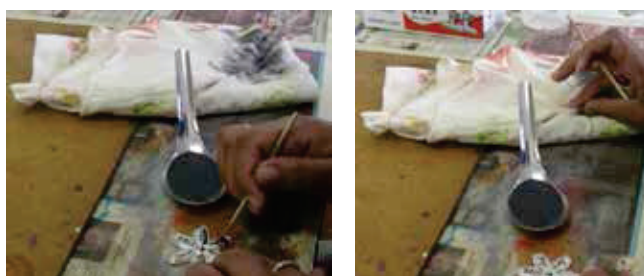


Figure 3a and 3b: (a) Workers paint epoxy resin on to small metal objects using a wooden stick. (b) Small amounts of resin that contaminate the fingers during painting are wiped off on a cloth

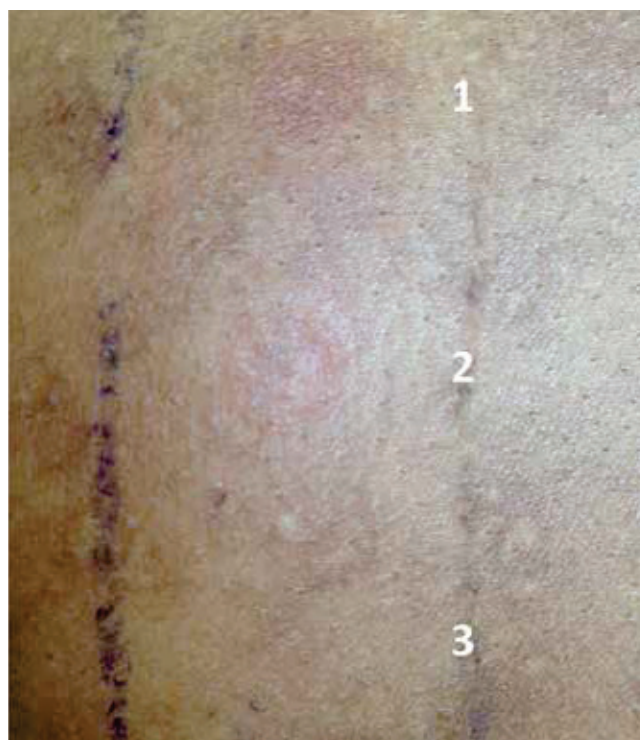


Figure 4: Own product patch test result. 1. Epoxy resin and hardener heat cured; 2. Lacquer thinner (cleaning solution) contaminated with epoxy resin monomer. (Organic solvent evaporated prior to testing); 3. Hand cream used by workers when hands feel dry

test was left *in situ* for 48 hours, and read at 72 hours. The Contact Dermatitis International Research Group Guidelines were followed for testing, reading and reaction interpretation. A doubtful positive ($\pm$) erythematous reaction to epoxy resin (1% Araldit® MY 740 from Ciba in petrolatum) and colophony (20% in petrolatum) was observed at 72 hours. An own product patch test was performed later using the workplace agents. These included resin and hardener; lacquer thinners contaminated with resin; various pigments used to colour the resin; a metal piece; and hand cream used by the workers. The epoxy resin and hardener were tested in the same concentrations as used in the workplace. The mixtures were dried on a small piece of filter paper, moistened with sterile water and then applied to her back in an aluminium Finn chamber. All the pigments used in the process were similarly tested, undiluted dried onto filter paper. She had a 1+ (indurated erythema) reaction to the epoxy/hardener combination, suggesting sensitisation to the particular epoxy resin used at work (Figure 4).

Due to social and personal problems, the patient left her work place and the skin is slowly regaining colour and she has not experienced any further problems (Figure 5).

WHAT IS EPOXY RESIN?

Plastics are polymers, made from resins. The polymers consist of long chain-like molecules composed of simple repeating units. Plastics are synthetic polymers made up of low-, medium- and high-molecular weight resins.⁵

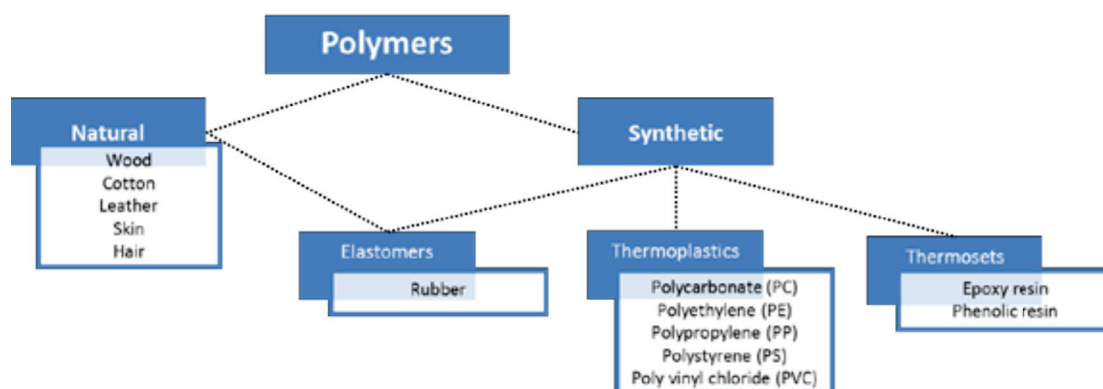
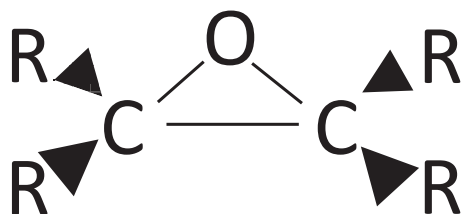


Figure 6: Classification of polymers

* Adapted from Amon, 2014

Synthetic polymers are divided into thermoplastic and thermoset types. Thermoplastic polymers are solid at room temperature, melted by heating for moulding purposes and can be recycled. Thermoset polymers are either solid or liquid at room temperature, are cured ('set/hardened') by heat, chemical reaction or suitable irradiation (e.g. ultraviolet light) and cannot be reshaped. ER is a thermoset plastic resin (Figure 6).^{5,6}

The term 'epoxy' in ER refers to a chemical structure known as an epoxide, which is a ring structure consisting of three components namely two carbon atoms and an oxygen atom which are bound to each other with a single covalent bond. The simplest epoxy molecule is ethylene oxide where two hydrogen molecules are bonded to the adjacent carbon molecules.



Epichlorohydrin is combined with (BPA) 2,2-bis(4-hydroxy-phenyl) propane to form DGEBA which makes up 75-90% of ERs.^{5,6} The molecular weight (MW) of the resin is determined by the ratio in which the two components are used. The higher the content of BPA in relation to epichlorohydrin, the higher the MW of the resulting ER. In terms of the physical characteristics, the higher the MW of the ER, the less volatile it becomes at room temperature.⁷ Higher MW also leads to a decrease in epoxide groups and the properties of the substance become more like that of a thermoplastic. A less common component of ER, bisphenol F may also undergo epoxidation like BPA, but compared to DGEBA, diglycidyl ether of bisphenol F (DGEBF) epoxy resins display lower viscosity and a higher epoxy content, which when cured leads to increased chemical resistance. The other components of ERS are listed in Table I.

PRODUCTION AND USES OF EPOXY RESIN

ER made its appearance in the late 19th century (first mentioned in 1891) and became commercially available in the mid-20th century. Commercially used ER was independently discovered by two scientists on different continents. The first was Dr Pierre Castan from Zurich, who developed it for use in dentures. His discovery was patented in 1938. On the other side of the pacific Dr Sylvan O'Greenlee, an American, made the same discovery whilst researching for suitable surface coatings. He patented it in 1943.⁸ Ever since its discovery, ER has gained such popularity due to its widespread uses that it is estimated that the yearly world production of ER today is more than half a million tons. Fifty-five percent is used in structural applications (boats, automobiles, aircraft, medical prostheses and sports equipment)⁹ and the rest in paints and coatings (paints and lacquers; laminates; electric insulation and encapsulation; floor covering; moulding; corrosion protection of metals; castings for metal tools; mending of cracks and fissures in concrete; and glues for metal, plastics, rubber, wood, glass, and ceramics).^{5,10-12} Other uses include electronic circuit boards, filling agents, printing inks, polyvinyl chloride products, and immersion oil for microscopes.^{5,10,12,13}

HEALTH EFFECTS OF EPOXY RESIN

Contact dermatitis is the most common illness reported to be due to ER.¹⁰ Contact dermatitis can be subdivided into irritant contact dermatitis (ICD), allergic contact dermatitis (ACD), contact urticaria and protein contact dermatitis.¹⁴ All types of contact dermatitis, except for protein contact dermatitis, can be induced by ER. The features of each of these are summarised in Table II as related to ER components. ACD is the

TABLE I: COMMON ER ADDITIVES

ADDITIVES TO ER

Colourants
Fillers
Tar
Ultraviolet light absorbers
Solvents
Reinforcing agents
Flame retardants
Plasticisers

Adapted from Cao, 2009⁶¹

TABLE II: TYPES OF CONTACT DERMATITIS AND ERS IMPLICATED SUBSTANCES

	IRRITANT CONTACT	ALLERGIC CONTACT	CONTACT URTICARIA
SUBSTANCE	Irritant (hand soap, lacquer thinners)*	Allergen (DGEBA-containing resin, pigments, metals)*	Allergen (DGEBA-containing resins)*
TRIGGER	Any irritant.	Sensitised substance.	Sensitised substance.
MECHANISM	Irritation and damage to skin barrier or directly skin toxicity.	Type IV delayed hypersensitivity.	Non immunologic or Type 1 IgE-mediated hypersensitivity.
TIME OF ONSET	Immediate to days.	12 to 48 hours once sensitised.	Onset <1 hour once sensitised. Resolves <24 hours.
PREVALENCE	++	++	+
MORPHOLOGY	Eczema Acute: red, vesicular/papules, weeping, crusting, and peel. 	Eczema: In the case of ER, allergic is more common than irritant. Acute: red, vesicular/papules, weeping, crusting, and peel. 	Urticaria Transient migratory red papules/plaques with wheals. 
	Chronic: scale and lichenification. 	Chronic: scale and lichenification. 	
LOCATION	Limited to exposed site.	Limited to exposure sites but can become generalised.	Localised or generalised.
SYSTEMIC INVOLVEMENT	No	No	Possible
EXPOSURE	No previous contact needed.	Previous contact sensitisation.	Previous contact sensitisation.
DOSE	Dose dependant or cumulative.	Any dose once sensitised. Higher doses needed for sensitisation.	Any dose once sensitised. Higher doses increase risk for sensitisation.
SUBSEQUENT EXPOSURE	Not always clinically reactive; Does not always react (sub-irritant dose); Skin adaptation (hardening).	Always react unless develop tolerance.	Always react unless develop tolerance.
PATCH TEST	-	+	-
PRICK TEST	-	-	+

*used by our index patient

most common dermatosis associated with ER and accounts for up to 95% of ER induced contact dermatitis. ICD accounts for only 4% while 1% of patients will present with contact urticaria.¹⁵ ACD typically presents as acute (red, blistering, weepy and/or oedematous) or chronic (lichenified and scaly) plaques. This usually starts on the hands and then spreads to involve the arms, face and neck. Involvement of distal sites is the result of indirect contact with ER via touch, but exposed sites can also be affected via volatile vapours of resins or additives.¹⁰

Contact dermatitis due to resins is mostly due to contact with epoxy (93%)¹⁵ versus other resins or additives.¹⁶⁻¹⁸ Table III lists all resin systems implicated in contact dermatitis. DGEBA is not only the most common compound in ER, but is also the most common cause of ER contact dermatitis (60-80%).^{6,15,19-23} DGEBA is an oligomer of 340 molecular weight.⁵ Table IV lists other ER ingredients less commonly responsible for sensitisation and allergic skin reactions. The low molecular weight ERS are associated with a higher chance of sensitisation^{17,24} and vice versa. The most common sources for low molecular weight

TABLE III: SYNTHETIC PLASTIC RESINS AND CD**SYNTHETIC PLASTIC RESINS KNOWN
TO CAUSE ACD AND ICD**

Epoxy
(Meth)acrylic
Polyurethane
Phenol-formaldehyde
Polyester
Amino resin
 Melamine-formaldehyde
 Urea-formaldehyde
Polyvinyl
Polystyrene
Others
 Polyolefin
 Polyamide
 Polycarbonate

Adapted from Cao, 2009⁶¹

ER exposure include paints, surface coating and adhesives.¹⁵ Higher MW ERS can cause sensitisation, but this will be in the setting of already irritated or damaged skin, commonly due to substances like solvents or hardeners.¹⁵ Sensitisation occurs within the first three months of initial contact with ER.¹⁹ Properly cured ER does not cause sensitisation, but under certain conditions up to 25% of ER remains uncured, especially if cured at room temperature.⁵

The bulk of health problems related to ER occur in its manufacture and use prior to curing.¹⁰ Handling the final cured product is problematic if it is machined or heated. In the occupational environment low MW ERS components causing health problems include monomers from uncured or incompletely cured ER resins, hardeners, reactive diluents (RD), degradation products, and other additives listed in Table IV.^{5,10} RDs are low MW ERS which have a low viscosity at room temperature and are added to ERS to reduce the viscosity of other resins. Non occupational exposure to ER causing health problems have been described, mostly due to hobby glues or residues of unhardened resin on coated objects (e.g. knee patches, bottle caps, film cassettes, metal packages etc.).^{25,26}

Hardeners (mostly aliphatic polyamines, such as ethylenediamine (EDA)) and reactive solvents (mostly phenylglycidyl ether) account for about 10-15% of ER associated contact dermatitis.^{6,19,27} In a recent large cohort described in Finland, 142 of 1844 cases of occupational skin conditions were reported to be due to ER.¹⁵

High prevalence industries for ERS related ACD are the marble industry (45%)²⁸ and construction industry (18%).²⁹ High prevalence industries for ERS sensitisation are aircraft manufacture (56%),³⁰ factory workers manufacturing plastic skis (27%),³¹ paint manufacturing (21%)¹⁶ and construction (20%).²⁹ Sensitisation to DGEBA based ER can induce both type 1 and type 4 allergic reactions.³² Not all studies have included hardeners and RDs together with ER in the patch test battery and therefore the actual number of people sensitised to ERS is likely to be higher. Table V lists occupations

TABLE IV: EPOXY RESIN TYPES**TYPES OF EPOXY RESINS**

Diglycidyl ether of the bisphenol A (DGEBA)*
Diglycidyl ether of the bisphenol F (DGEBF)
Brominated
Cycloaliphatic
Heterocyclic dimethyl hydantoin
Phenol novolak

**used by our index patient*

Adapted from Cao, 2009⁶¹

with frequent exposures to ER compounds.

While DGEBA is the most common component causing skin problems in ERS, hardeners come in second place, non DGEBA resins third and RDs in fourth place.¹⁵ There is also significant cross reactivity between DGEBA and DGEBF due to their structural similarity³³ as well as between RDs.¹⁵ Additional substances added to ERS include accelerators with hexavalent chromate which are well recognised causes of ACD.^{22,34}

When confronted with work related allergens, an individual can be sensitised via the cutaneous or the pulmonary route. It has also been shown in animal studies that cutaneous sensitisation can lead to an asthma-like response when the offending agent is inhaled thereafter.³⁵⁻³⁹ The most common occupational contact allergens and their relation to occupational asthma are shown in Table VI as per the Asthma in the Workplace and the Health and Safety Executive Asthmagen Documents. The association of ERS induced contact dermatitis with asthma is well established and several common hardeners are implicated, but the link with ER monomer has only been described in a few case studies.⁴⁰⁻⁴² In practice when an individual is working with ER, they are likely to be exposed to multiple components in the ERS, many of which have been shown to induce asthma.

Cases of a scleroderma-like presentation have been reported in association with ER contact. It seems to have an acute onset and has a fairly good prognosis since the condition is reversible after exposure is stopped. It also does not entail systemic involvement.² This relationship is controversial. In a systematic review conducted in 2013 looking at the association of systemic sclerosis with occupational exposures, ER exposure, as a cause for scleroderma, was found to have been proven statistically in only one study, whereas the other three quoted studies did not report a statistical relation between systemic sclerosis and ER exposure.⁴³

BPA is one of the major components of ER. Other than in ER, BPA is also often used in polycarbonate plastics and is commonly found in the manufacturing of everyday consumer goods such as food containers, baby bottles, beverage and food can linings, and also in certain industrial articles such as water pipes. BPA is produced in

TABLE V: OCCUPATIONAL EXPOSURES TO ERS**OCCUPATIONS WITH FREQUENT EXPOSURES TO ERS COMPOUNDS**

Adhesive and glue manufacturing;
 Aircraft manufacturing;
 Appliance finishing;
 Assembling;
 Automobile manufacturing;
 Boat manufacturing;
 Can coating;
 Cement work;
 Ceramics;
 Construction and building;
 Electric insulation;
 Electronics;
 Floor covering;
 Furniture building;
 Lacquering;
 Laminating;
 Laboratory work with microscope immersion oil;
 Machining;
 Marble work;
 Metal casting;
 Other plastic material (e.g. polyvinyl chloride) Manufacturing;
 Paint, lacquer, and varnish manufacturing;
 Painting;
 Printing ink manufacturing;
 Sculpting;
 Sporting equipment manufacturing (e.g. tennis rackets, skis);
 Soldering;
 Terrazzo work;
 Tile setting;
 Tunnel work;
 Varnishing;
 Wood processing.

Adapted from Cao, 2009⁶¹

excess of 3 million metric tons per year⁴⁴ and is regularly encountered in everyday life so that up to 90% of people living in the USA will have detectable amounts of BPA in their urine. Because of incomplete polymerisation, BPA can contaminate food and drink, especially under hot, basic or acidic conditions.⁴⁵⁻⁴⁷ BPA has been the subject of intense research due to the 2008 findings of the National Toxicology Program's (NTP) Centre for Evaluation of Risk to Human Reproduction (CERHR) that BPA poses 'some' health concern.⁴⁸ BPA enters the body via inhalational, oral or transdermal routes. BPA has been found to be an endocrine disrupting compound (EDC)⁴⁹ that was promoted as a xenoestrogen⁵⁰ for medical use when its estrogenic properties were first discovered. In addition to binding oestrogen receptors and exhibiting oestrogen activity, BPA also binds androgen receptors where it has an anti-androgenic effect as well as binding thyroid receptors where it has an anti-thyroid hormone effect. Clinical conditions resulting from this endocrine disruption include female and male infertility, precocious puberty, hormone dependent tumours such as breast and prostate cancer and polycystic ovary syndrome (PCOS) as well as causing brain development abnormalities.⁵¹ Exposure to BPA can be accurately monitored in the urine and in the future this will likely be used to monitor bioaccumulation to assess health risk.⁵²

WHEN EPOXY RESIN DISEASE IS SUSPECTED

When one is faced with a probable reaction to ER, the

TABLE VI: WORK PLACE EXPOSURES AND ASTHMA

SUBSTANCE	ASTHMA IN THE WORK-PLACE	HSE ASTHMAGEN
1. Components of epoxy resin systems	Yes	Yes
2. Thiuram	Not listed	Not listed
3. Carba mix	Not listed	Not listed
4. Nickel sulfate	Yes	Yes
5. Cobalt chloride	Yes	Yes
6. Potassium dichromate	Yes	Yes
7. Glyceryl thioglycolate	Not listed	Not listed
8. P-phenylenediamine (PPD)	Yes	Watch list
9. Formaldehyde	Yes	No
10. Glutaraldehyde	Yes	Yes

Adapted from Arrandale, 2012¹

diagnostic approach relies on a thorough history, as well as a physical examination (focussing on skin and lungs), before confirmation with a patch test in cases of skin reactions. The patch test needs to include LMW ER with high amounts of DGEBA oligomer MW 340 (in 1% petrolatum), to confirm sensitivity and ACD. This was present in our commercial patch test material and the weakly positive reaction observed was the first clue to a possible cause for her dermatitis. It may be necessary to use a patch test with the specific resin that the patient uses, obtained either from the workplace or the manufacturer. If these are all negative, testing hardeners and RDs can be considered, always keeping appropriate dilutions, solvents and pH in mind for all patch testing related to own substance testing including ERS.

In the event of an 'unexpected' positive to epoxy on patch test, one may be dealing with the "epoxy-by-proxy" phenomenon in which case it is useful to enquire about occupations of close contacts.⁵³ The contact may also be from a hidden source and often careful detective work will reveal the answer.

The principles of management of contact dermatitis due to ER are similar to standard management of all forms of CD. Even though early job transfer generally leads to a good clinical outcome⁵⁴ due to decreased aggravation of the dermatitis as well as decreased sensitisation to other



Figure 5a and 5b: The patient's skin changes on leaving work. (a) Mild erythema and resolving post inflammatory pigmentary changes with excoriation periauricularly and involving the ear helices. (b) Leucoderma of dorsum of hands showing re-pigmentation

substances like hardeners and RDs,²⁹ our experience it that this step often leads to a decrease in salary or a dismissal and is not a good option. Many patients are unwilling or unable to change their work and are therefore left with continued exposure to ER and active dermatitis, despite measures to try and reduce contact with ER and medical treatments such as super potent topical steroids.¹⁵

For our patient and co-workers it was necessary to prevent hand contact with the ER. A system had been developed by the workers over time in which spills or skin contamination were wiped clean on a cloth during the day. The use of alternate cleaning tools other than fingers was suggested for all workers. The cleaning of ER and pigment contaminated tools should be done without hand contact with the cleaning solvent and dissolved contaminating ER monomers. The installation of an extraction system over the curing oven was recommended. The patient was advised on alternate ER exposures in activities of daily living and in the work environment.

When choosing personal protective equipment (PPE) for ER exposed workers, the glove of choice would be either heavy duty vinyl or multi-layered folio-type material, as plastic and rubber gloves are penetrated by ER. These are often very clumsy to work with and would have hindered the fine motor skills needed for the patient's work. The best alternative was to use gloves for splash protection only, which should be discarded immediately on contamination. There is a paucity of studies on the effectiveness of barrier creams in the prevention of contact dermatitis to ER. At best these act as emollients and if given as barrier creams create unsubstantiated expectations in workers. Important preventative measures are the regular maintenance and cleaning of contaminated equipment and workspaces without skin contact. The use of alternate systems other than ER needs to be considered. This includes the use of higher MW epoxy resins due to their lower potential to sensitise. If at all possible, LMWs of 340 and 624 are best avoided.^{17,55} When using hardeners they should be aliphatic polyamine-free and RDs should be of high-molecular weight (>1000).⁵⁵ In our case, the safety data sheets demonstrated that the epoxy resin contained BPA and that the hardener was a cycloaliphatic polyamine. It is essential to properly train and educate workers exposed, although failure to disclose ER as an ingredient remains a challenge in many products.^{56,57} Pigments that have been implicated in contact dermatitis include phthalic anhydride in ER,⁵⁸ cupric-phthalocyanine complexes⁵⁹ and organic pigment (Irgalite Orange F2G)⁶⁰ in plastic gloves.

Our index case demonstrated the most common occupational clinical picture due to ER, namely allergic contact dermatitis. This assessment is supported by her history of being in direct contact with the uncured substance at work and the fact that the ER to which she was exposed contained DGEBA. DGEBA is the commonest ER allergen which is of low MW and thus

more likely to sensitise on exposure. Additionally when she was away from work her skin improved and upon re-exposure it worsened. The clinical picture was that of exposure contact dermatitis, possibly due to vapour exposure because ventilation was minimal. Own product patch testing confirmed a reaction to the ER used. In terms of BPA toxicity, she did not display any features of endocrine disruption most likely due to the limited exposure. She did not have any features of asthma.

She responded to medical treatment but decided to leave the job. On follow up, the dyspigmentation of her skin was noted to be slowly improving. She has been informed about avoiding future contact with ER and was advised on the other health risks possibly implicated in ER exposure, although these are highly unlikely to be associated with her limited exposure at work.

CONCLUSION

ER induced occupational illness is common relative to other exposures, especially when it comes to skin disease. ER is used as part of ERS which contains many other potential hazardous substances and it is used over a wide spectrum of industry where contact with paint, surface coating and adhesives is common. Sensitisation to ERS can lead to an individual being cross sensitised to other ERS or to develop ERS induced asthma. The attending physician must be alert to the fact that in the right setting new onset dermatitis or asthma may be occupational in nature and that ER must be interrogated as a possible cause when taking a history and must be confirmed by patch test. The physician must also make sure that measures are put into place to eliminate the need for further exposure to ER, in order to improve the skin condition and prevent sensitisation to other substances or the development of occupational asthma. Suspected cases of occupational ER related disease should ideally be referred to a specialist occupational health facility.

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DECLARATION OF CONFLICT OF INTEREST

The author declares no conflict of interest.

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