CONTACT POINTS

CONTACT DERMATITIS 2006: 55: 54–63 •

• CONTRIBUTIONS TO THIS SECTION WILL NOT UNDERGO PEER REVIEW, BUT WILL BE REVIEWED BY THE EDITOR •

Photoallergic contact cheilitis due to oxybenzone found in a lip cosmetic

Contact Dermatitis 2006: 55: 54

E. C. Veysey[1] and D. I. Orton[2]

[1]Department of Dermatology, Churchill Hospital, Old Road, Headington, Oxford, Oxon, and [2]Dermatology Department, Amersham Hospital, Whielden Street, Amersham, Buckinghamshire, Hp7 0jd, UK

Key words: benzophenone-3; cheilitis; oxybenzone; photoallergy

Case Report

A 79-year-old lady presented with a 2-month history of soreness and swelling of her upper and lower lips, associated with dryness, cracking and ulceration. Prior to this, she had been intermittently applying Vaseline, her own lipsticks and Blistex® (a brand of lip slave).

Patch tests were performed to the departmental standard series (based on the European standard series with a few additional allergens), a hand and face series, several mint products and 3 of her own lip cosmetics including Blistex®. Patch and photopatch tests were also performed to 2 sunscreens chemicals identified as constituents of the lip cosmetics (oxybenzone and ethylhexylmethoxycinnamate). The patch tests were entirely negative. The photopatch tests (UVA 5 J/cm^2) revealed photoallergy to oxybenzone (benzophenone-3) with no reaction at the non-irradiated site. The Blistex® contained oxybenzone, which she was advised to stop using, as well as avoiding all other cosmetics containing this UV filter. At follow-up, 4 months later, the cheilitis had resolved.

Discussion

Cheilitis is an inflammatory reaction of the lips, which can be caused by endogenous or exogenous factors. Exogenous factors include irritant and allergic contact dermatitis, the latter being far less common. A large variety of allergens have been identified; among them are ingredients of lip cosmetics, sunscreen agents, nail polishes, toothpastes and dentifrices (1, 2). To our knowledge, however, there are no previous case reports of photoallergy (in the absence of contact allergy) causing cheilitis.

Despite the increasing use of chemical UV filters in cosmetics and sunscreens, photoallergic reactions are rare. This was investigated in a large retrospective study of 2715 patients suspected of having a photodermatosis, who underwent photopatch tests between 1983 and 1998 (3). Photoallergic reactions were found in only 2.3% of patients, and the most common photoallergen was benzophenone-3 (oxybenzone). Of those with a photoallergy, just over 50% had an underlying photodermatosis, predominantly chronic actinic dermatitis or polymorphic light eruption. The authors comment that most of the common UV filter photoallergens have been removed from the market, except for benzophenone-3, which is currently the commonest contact photoallergen in widespread use. Overall, the authors concluded that photoallergy does not represent a common clinical problem.

The importance of contact allergy as a cause of cheilitis is well recognized, however, photoallergy is rarely considered, and phototests are not routinely performed. Consequently, we may be missing the diagnosis in a small but important group of patients. Our case illustrates that photoallergic contact cheilitis occurs and that photopatch testing may be an important investigation in these patients.

References

1. Ophaswongse S, Maibach H I. Allergic contact cheilitis. *Contact Dermatitis* 1995: 33: 365–370.
2. Strauss RM, Orton DI. Allergic contact cheilitis in the United Kingdom: a retrospective study. *Am J Contact Dermat* 2003: 14: 75–77.
3. Darvay A, White I R, Rycroft R J G et al. Photoallergic contact dermatitis is uncommon. *Br J Dermatol* 2001: 145: 597–601.

Address:
Dr Emma Veysey
Department of Dermatology
Churchill Hospital
Old Road
Headington
Oxford
Oxon
UK
Tel: +44 1494 734600
Fax: +44 1494 734662
e-mail: e_veysey@hotmail.com

Is contact allergy to glyceryl monothioglycolate still a problem in Germany?

Contact Dermatitis 2006: 55: 54–56

Wolfgang Uter[1], Johannes Geier[2], Holger Lessmann[2] and Axel Schnuch[2]

[1]Department of Medical Informatics, Biometry and Epidemiology, University of Erlangen-Nürnberg, Erlangen, and [2]Information Network of Departments of Dermatology, University of Göttingen, Göttingen, Germany

Key words: CAS 30618-84-9; contact allergy; glyceryl monothioglycolate

In the mid nineties, the major producers and importers withdrew 'acid'

permanent waving solutions containing glyceryl monothioglycolate (GMTG) from the German market. Subsequently, in December 1997, a 'technical rule for hazardous substances 540' was set in force in Germany, calling for the substitution of GMTG with other, less allergenic agents (1); current version (2). Action had been prompted by exceedingly high prevalences of contact allergy (CA) to GMTG in hairdressers especially, but not only, in Germany (3, 4) and, to a lesser extent, also in clients (5). The beneficial effect had soon become evident in terms of vastly declining prevalences of CA to GMTG until 1998 (3). However, question remains whether exposure has really ceased or whether GMTG is still an allergen of current relevance. Thus, the time series of CA to GMTG in hairdressers patch tested for occupational contact dermatitis (OCD), ranging from 1995 to 2005, stratified for age and based on the results collected by the IVDK (www.ivdk.org) was analysed.

Material

The CA research network IVDK has been described elsewhere (6). For the current analysis, all patients patch tested between 1995 and 2005, working as hairdressers and suffering from OCD or having worked as hairdressers and having suffered from OCD, were considered. This group comprised altogether 1414 cases, 1040 of these currently and 374 formerly working as hairdressers at the time of patch testing, with a proportion of 92.5% females. The yearly prevalence of CA to GMTG was calculated separately for three age strata: age up to 20, 21–32 and 33 and older, corresponding roughly to the tertiles of the age distribution. This data was subject to statistical testing for trend with the Cochrane Armitage test. For data management and analysis, the statistical program package SAS (version 9.1, SAS Institute, Cary, NC, USA) was used.

Results

Overall, 232 of the 1414 patients tested positive to GMTG, with 144+, 76++ and 12+++ reactions [altogether 16.4% positive; exact 95% confidence interval (CI) = 14.5–18.4], while few doubtful ($n = 39$) or irritant ($n = 5$) reactions were observed – all based on D3 readings. The time course of CA to GMTG in the three age strata of hairdressers suffering from OCD is shown in Fig. 1. It is evident that the proportion of sensitized was highest in the youngest age group at the beginning of the study period, while in 2004 and 2005, it has arrived at 0% ($n = 63$ tested; exact 95% CI = 0–4.6). The decrease in this age stratum, and in the 'medium' age stratum, was significant ($P < 0.0001$). In contrast, there was no time trend in the oldest age group. Investigating those patients with positive reactions in 2000 or later in the youngest age group further ($n = 14$), no conclusive pattern of geographical distribution possibly indicative of the sources of exposure can be observed. Although the six positive reactions in 49 young hairdressers patch tested which have been noted in Osnabrück (until 2003) seem to indicate a geographical cluster, the catchment area of this specialized department extends virtually all over Germany, including highly selected patients. The hand was the anatomical site affected most often both in GMTG positive (93.1%) and negative (85.9%) patients.

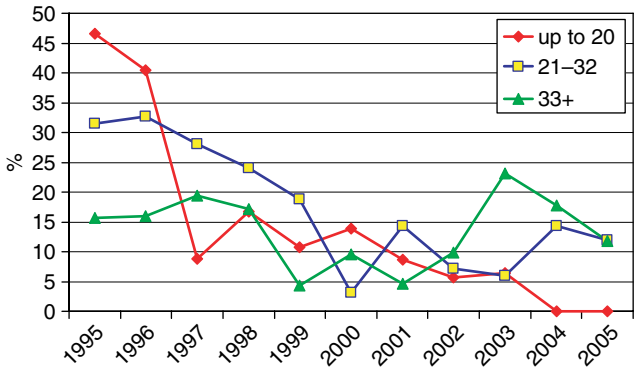

Fig. 1. Time course of the prevalence of sensitization to glyceryl monothioglycolate among hairdressers with occupational contact dermatitis, subdivide into three age strata.

Comment

Changes in the exposure to an allergen and a subsequently changing risk of CA to this allergen are best reflected in the CA prevalence observed in an age group in which the potential start of exposure is relatively recent. In such a subgroup, prevalence (and its changes) can be regarded as a relatively valid approximation of incidence (and its changes). In Germany, the training as hairdresser, with potential exposure to GMTG, may start at age 15. Hence, in hairdressers aged 20 when patch tested in 2004 or 2005, professional exposure will have commenced only since 1999 and 2000, respectively. The fact that no cases of CA to GMTG have been observed in the youngest age group in the 2 most recent years is reassuring – although it should be kept in mind that the relatively small size of this clinical sample does not preclude a certain, albeit low sensitization prevalence (cf. upper 95% confidence limit). Improved working hygiene could theoretically be an alternative, or additional, explanation for the observed trend. However, the use of protective gloves when applying 'acid' permanent waving solutions has shown no favourable trend (in Germany), according to available data (7). In conclusion, the beneficial effect of (largely) eliminating this compound can be well proven with the data presented. This might stimulate other national or EU-wide initiatives to firmly regulate GMTG, depending also on the burden of CA to GMTG in other countries (8, 9).

References

1. Anonymus. TRGS 540 'Sensibilisierende Stoffe'. *Bundesarbeitsblatt* 1997: (Dec) 58–63.
2. Anonymus. Technische Regel für Gefahrstoffe (TRGS) 540 – Sensibilisierende Stoffe. Ausgabe Februar 2000. *Bundesarbeitsblatt* 2000: (Feb) 73 ff.
3. Uter W, Geier J, Schnuch A. Downward trend of sensitization to glyceryl monothioglycolate in Germany. *Dermatology* 2000: 200: 132–133.

4. van der Walle H B, Brunsveld V M. Dermatitis in hairdressers. (I). The experience of the past 4 years. *Contact Dermatitis* 1994: 30: 217–221.
5. Schnuch A, Geier J. Glycerylmonothioglykolat-Sensibilisierung bei Friseurkunden. *Derm Beruf Umwelt* 1995: 43: 29.
6. Uter W, Schnuch A, Geier J, Frosch P J. The epidemiology of contact dermatitis. The information network of departments of dermatology (IVDK) in Germany. *Eur J Dermatol* 1998: 8: 36–40.
7. Schlesinger T, Revermann K, Schwanitz H J. Dermatosen bei Auszubildenden des Friseurhandwerks in Niedersachsen – Ein Vergleich zwischen 1989, 1994, und 1999. *Derm Beruf Umwelt* 2001: 49: 185–192.
8. Katugampola R P, Statham B N, English J S, et al. A multicentre review of the hairdressing allergens tested in the UK. *Contact Dermatitis* 2005: 53: 130–132.
9. Hasan T, Rantanen T, Alanko K, et al. Patch test reactions to cosmetic allergens in 1995–1997 and 2000–2002 in Finland – a multicentre study. *Contact Dermatitis* 2005: 53: 40–45.

Address:
Wolfgang Uter
Department of Medical Informatics, Biometry and Epidemiology
University of Erlangen/Nürnberg
Waldstr. 6
D-91054 Erlangen
Germany
Tel: +49 9131 8522720
Fax: +49 9131 8522721
e-mail: wolfgang.uter@rzmail.uni-erlangen.de

Contact dermatitis to α-lipoic acid in an anti-wrinkle cream

Contact Dermatitis 2006: 55: 56–57

A. Bergqvist-Karlsson[1], I. Thelin[2] and O. Bergendorff[2]

[1]Department of Dermatology, Akademiska sjukhuset, Uppsala, and [2]Department of Dermatology, Lund University, University Hospital, Lund, Sweden

Key words: allergic contact dermatitis; anti-ageing; cosmetics; α-lipoic acid; photo-ageing

Alpha-Lipoic acid (LA), also called 1,2-dithiolane-3-pentanoic acid and thioctic acid (Fig. 1) has been reported to have a protective effect in tissues under oxidative stress (1). In a recent study, LA was shown to improve photo-aged skin (2). In the US, the substance seems increasingly popular in health store products both for internal use and in anti-ageing products, and it has now its own homepage on the Internet (3)

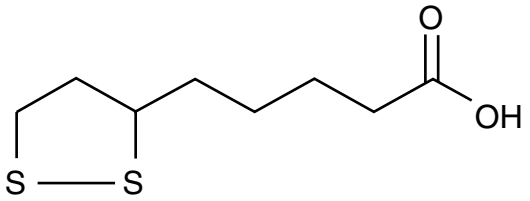

Fig. 1. Chemical structure of α-lipoic acid.

In Sweden, LA was launched in an anti-wrinkle cream ('cream A' in this report) in 2002.

Case Reports

Case no. 1

A 64-year-old woman was seen with a severe dermatitis on eyelids, trunk and arms. Two skin care products, cream B and lotion C, were suspected. She stopped using these, had topical steroid treatment and the dermatitis cleared. Epicutaneous tests were made 2 months later with cream B, lotion C, the standard series and an extended skin care/cosmetics ingredients series. Apart from a positive nickel reaction which could not explain the severe dermatitis, they were all negative.

3 months later, she returned with an infiltrated erythema all over face and neck which had started 6 hr after a single application of cream A. She now reported that she had in fact also been using it a few weeks before the earlier dermatitis episode but forgotten all about it. A patch test with cream A 2 months later gave an infiltrated and vesicular reaction. The manufacturer kindly provided all ingredients. Further testing with these in their respective concentrations gave a strongly positive reaction to one: LA 5% in pet. applied to the skin using Finn Chambers® mounted on Scanpor®. Testing with a serial dilution of this substance gave positive reactions down to 0.025%. 10 healthy middle-aged females were tested with LA at 5, 2.5 and 0.5% without any allergic or irritant reactions.

Case no. 2

A 54-year-old woman presented with a history of a severe facial and neck dermatitis for a couple of weeks, necessitating both external and internal corticosteroid treatment. She returned for epicutaneous testing manifesting a strong positive reaction only for cream A. She had used this for a few months before the reaction but stopped after her first visit to our clinic, and her skin was now completely healed. Further tests with the cream A components gave a positive reaction for 5% LA only. Retested with a LA dilution series, she was positive down to 0.025%.

Case no. 3

A 59-year-old woman with a history of nickel allergy started using cream A daily. After 6 weeks, an eczematous eruption developed in her face, on neck and hands. She was seen in an emergency department, given oral corticosteroids and hydrocortisone cream with some effect but continued with cream A daily for another month until seen by a dermatologist and recommended to stop. 6 weeks later when healed, she was tested with the standard series, cream A and some of the ingredients. Nickel, cream A as is and LA (5, 1 and 0.5%) were all positive.

Discussion

Allergic skin reactions after systemic administration of LA have been reported (4), and in a study of the effect of 5% LA on photo-ageing, transient local irritation was seen during the first weeks in a number of cases (2). In the adverse reaction reporting system of the Department of Cosmetics Section of the Swedish Medical Products Agency, 15 other reports on suspected adverse skin reactions to cream A have been submitted so far (M. Tammela, personal communication). To our knowledge, our 3 cases of contact allergy to LA seem to be the first ones reported.

Acknowledgement

Our sincere thanks to Ingalill Erikssohn, Department of Dermatology, University Hospital, Örebro for skilful help with preparation of the test substances of cases no. 1 and 3.

References

1. Panigrahi M, Sadguna Y, Shivakumar B R et al. α-lipoic acid protects against reperfusion injury following cerebral ischemia in rats. *Brain Res* 1996: 717: 184–188.
2. Beitner H. Randomized, placebo-controlled, double blind study on the clinical efficacy of a cream containing 5 % α-lipoic acid related to photoageing of facial skin. *Br J Dermatol* 2003: 149: 841–849.
3. Alpha-lipoic acid (homepage on internet). Copyright 2002–2003 by the Linus Pauling Institute, Oregon State University, Updated 23 July 2003; cited 18 August 2005 (available from http://lpi.oregonstate.edu/infocenter/othernuts/la/index.html).
4. Packer L, Witt E H, Tritschler H J. Alpha-lipoic acid as a biological antioxidant. *Free Radic Biol Med* 1995: 19: 227–250.

Address:
Ola Bergendorff
Department of Dermatology
University Hospital
SE-221 85 Lund
Sweden
Tel: + 46 46 173154
Fax: +46 46 172114
e-mail: ola.bergendorff@skane.se

The extent of black henna tattoo's complications are not restricted to PPD-sensitization

Contact Dermatitis 2006: 55: 57

Peter Jung, Gabriele Sesztak-Greinecker, Felix Wantke, Manfred Götz, Reinhart Jarisch and Wolfgang Hemmer

FAZ – Floridsdorf Allergy Center, Vienna, Austria

Key words: black henna tattoo; contact dermatitis; lawsone; PPD

Case Report

A 9-year old boy had a henna tattoo painted while on holiday in Italy. Two weeks later he experienced a painful, itchy blistering reaction at the application site, which disseminated to his arms, trunk and feet. Lesions healed, leaving a post-inflammatory hypopigmentation in the design of the original tattoo. 6 months later he had his hair dyed black. The next morning he developed papulovesicular eruptions at the hairline accompanied by pruritus. Patch testing revealed + + + positive reactions to paraphenylendiamine 0.2% (PPD), benzocaine 5% and isopropyl-diphenylenediamine 0.1% (IPPD) and a + + positive reaction to lawsone 10%.

Discussion

PPD is contained in both black henna tattoos and hair dye. We suppose that our patient had been actively sensitized to PPD in the black henna tattoo and subsequently developed contact allergic reactions due to PPD contained in the hair dye. In an analogous report (1), a patient had to be admitted to intensive care because of the severity of the contact allergic reactions due to PPD. Our patient also showed patch test reactions to lawsone 10%. Although PPD is the causative agent in the majority of cases of allergic contact dermatitis due to black henna tattoos (2), lawsone might have more clinical relevance as previously assumed in the context of black henna tattoos. We think that the acute allergic reaction caused by the much stronger, potent allergen PPD can facilitate concomitant hypersensitivity to lawsone, otherwise not provoking sensitization. Allergy to lawsone may lead to subsequent contact dermatitis due to lawsone-containing substances such as hair dyes and suntan creams (3). Therefore, it is important to patch test PPD and lawsone in patients with skin reactions following black henna tattoos to avoid future contact allergic reactions.

Furthermore, sensitization to PPD can provoke allergic contact dermatitis to cross-reactive, aromatic para-substituted compounds, such as benzocaine and IPPD (2, 4), both of which revealed patch test reactions in our patient. In this context, primary sensitization to benzocaine may lead to allergic reactions to PPD in henna tattoos as well (5).

The extent of potentially long-term implications on patients' health due to black henna tattoos are not restricted to PPD-hypersensitivity including active sensitization, long lasting post-inflammatory depigmentation and subsequent contact allergic reactions due to PPD-containing products, but also sensitization to simultaneously applied substances, for example lawsone, and sensitization to cross-reactive compounds, such as benzocaine and IPPD.

References

1. Jasim Z F, Darling J R, Handley J M. Severe allergic contact dermatitis to paraphenylene diamine in hair dye following sensitization to black henna tattoos. *Contact Dermatitis* 2005: 52: 116–117.
2. Marcoux D, Couture-Trudel P M, Riboulet-Delmas G, Sasseville D. Sensitization to para-phenylenediamine from a streetside temporary tattoo. *Pediatr Dermatol* 2002: 19: 498–502.
3. Wantke F, Götz M, Jarisch R. Contact dermatitis due to henna, solvent red 1 and solvent red 3. *Contact Dermatitis* 1992: 27: 346–347.
4. Matulich J, Sullivan J. A temporary henna tattoo causing hair and clothing dye allergy. *Contact Dermatitis* 2005: 1: 33–36.
5. Arroyo M P. Black henna tattoo reaction in a person with sulfonamide and benzocaine drug allergies. *J Am Acad Dermatol* 2003: 48: 301–302.

Address:
Peter Jung
FAZ – Floridsdorf Allergy Centre
Franz Jonas Platz 8/6
A-1210 Vienna
Austria
Tel: +43 1 2702541
Fax: +43 1 270254278
e-mail: science@faz.at

A case of contact dermatitis from jojoba

Contact Dermatitis 2006: 55: 57–58

L. Di Berardino[1], F. Di Berardino[1], A. Castelli[1] and F. Della Torre[2]

[1]G. P. ASL Milan, and [2]Pneumologic Department, IN.R.C.A, Via Monteregio 13, 23880 Casatenovo (Lecco), Italy

Key words: contact dermatitis; delayed hypersensitivity; jojoba; patch tests

Jojoba (Simmondsia chinensis) is an arid perennial woody shrub grown in

several American and African countries. Jojoba seeds, which are rich in liquid wax, were used in folk medicine for diverse ailments. Now, they are frequently used in cosmetics, particularly body creams and hair care products (1–3).

We describe a case of delayed hypersensitivity reaction to Jojoba seed powder.

Case Report

A 43-year-old female patient, with a long personal history of inhalant allergy to grass pollen, mites, artemisia and wall pellitory, came to our observation with erythematous pomphoid lesions spread all over the body, particularly on the abdomen and forearms.

On the previous day, she had applied a cosmetic body cream.

Oral corticosteroid treatment induced a prompt remission of the lesions and complete recovery after approximately 1 week.

Skin patch tests were performed using all the components of the cosmetic cream, which were supplied by the manufacturer: nelumbo lucifera 15% (salt solution 0.9%), hibiscus 15% (salt solution 0.9%), camellia sinensis 15% (salt solution 0.9%), carbomer 1% (salt solution 0.9%), cetearyl 1% (vaseline), cyclomethicone 0.5% (vaseline), chlorphenesin 1% (salt solution 0.9%), parabeni mix 15% (vaseline), *S. chinensis* 15% (vaseline) and bambusa arundinacea 15% (vaseline).

All patch test results were negative except *S. chinensis* (jojoba) seed powder, which gave a positive response. Erythema, oedema and vesicles developed 72 hr after the application of the patch tests. The response was still evident 48 hr after removal of the specific patch. The same patch test gave a negative result in a healthy control subject.

4 months later the test was repeated, using only *S. chinensis* 15% (Vaseline), on the patient and 5 other healthy control subjects. The same positive response was observed in the patient, whereas there was no positive reaction in the control subjects.

Discussion

Clinical findings and positive patch test results to jojoba seed powder imply that the patient had developed a type-IV hypersensitivity to jojoba.

To the best of our knowledge, there is only one documented case of contact dermatitis from jojoba in international literature (2). On the contrary, it has been reported that liquid wax extracted from jojoba seeds demonstrates an anti-inflammatory activity in several experimental models (3).

As there is widespread use of products containing jojoba extracts, further studies are required to determine the factors provoking this rare sensitization.

References

1. Scott M J, Scott M J Jr. Jojoba oil. *J Am Acad Dermatol* 1982: 6: 545.
2. Wantke F, Hemmer W, Gotz M, Jarisch R. Contact dermatitis from jojoba oil and myristyl lactate/maleated soybean oil. *Contact Dermatitis* 1996: 34: 71–72.
3. Habashy R R, Abdel-Naim A B, Khalifa A E, Al-Azizi M M. Anti-inflammatory effects of jojoba liquid wax in experimental models. *Pharmacol Res* 2005: 51: 95–105.

Address:
Dr Luigi Di Berardino
Viale Piceno 1420123 Milano
Italy
e-mail: dblma@fastwebnet.it

Allergic contact dermatitis to rosin after a single accidental permanent marker skin contact

Contact Dermatitis 2006: 55: 58–59

E. Fesquet, B. Guillot and N. Raison-Peyron

Department of Dermatology and Allergology, CHU Montpellier, Saint Eloi Hospital, 80 Avenue Augustin Fliche, 34295 Montpellier cedex 5, France

Key words: adhesive tape; allergic contact dermatitis; permanent marker; primary sensitization; rosin

Case Report

A 33-year-old male virology research worker experienced in October 2005 an acute vesiculo-bullous dermatitis that appeared two hours after removing a medical adhesive tape ADHEROPLAST®, used for an orthopaedic surgical operation of his left shoulder. The lesions were localized on the areas covered by the adhesive tape: the left shoulder, the left flank and his lips (the nozzle of intubation area).

The acute allergic contact dermatitis improved after using a topical corticosteroid treatment, and left a slight postinflammatory hyperpigmentation.

The patient's medical history did not reveal any contact allergy to sticking plasters or surgical varnishes. He mentioned an allergic contact dermatitis of the axillary folds 10 years previously due to a deodorant ALASKA AXE, which was no more available commercially.

Nevertheless, he related he had developed, four months before, the same reaction on the dorsum of his left hand 1 day after an occupational accidental cutaneous contact with a black permanent marker PENTEL.

Patch testing with the European standard series (Trolab, Germany), fragrance series (Chemotechnique, Sweden) and oxidized (Trolab, Germany) and esterified rosin derivatives were performed, with the results summarized in Table 1.

Contact allergy to the patient's permanent marker was confirmed by patch testing to the ink 'as is': there were + at 2D and ++ at 3D.

We contacted the French manufacturer of the permanent marker who confirmed the presence of colophony (between 6% and 8%), only in this model.

The manufacturer of the surgical adhesive tapes ADHEROPLAST® also confirmed the presence of rosin in their tape.

The patient's reaction to sandalwood had probably a past relevance (past medical history of contact dermatitis to a deodorant).

Discussion

Rosin is used in some permanent markers as an ink thickener, to make sharp lines.

The interest of our observation is the probable primary sensitization to rosin by a single accidental skin contact with a permanent marker containing colophony. The patient had no previous history of contact allergy to rubber adhesives, surgical varnishes or any products containing rosin. After this episode, he developed a severe acute vesiculo-bullous dermatitis when his skin was in contact with surgical adhesive tapes containing rosin.

Table 1. Results of positive patch tests

	Day 2	Day 3
Rosin 20% pet.	++	+++
Abietic acid 10% pet.	+	++
Abitol 10% pet.	+	+++
Glyceryl rosinate (esterified rosin) 20% pet.	+/–	+
Sandalwood 20% pet.	++	++

Contact dermatitis to rosin from adhesive medical tapes and surgical varnishes is well known. Recently, we reported a primary sensitization to rosin from a surgical varnish (1).

Goossens et al. mentioned primary sensitization to esterified colophony derivatives (but not to natural colophony) from epilating products (2).

Allergic contact dermatitis to permanent markers has been rarely reported. In one case, the allergen was a Solvent Yellow 146 dye of a permanent marker used to trace the contours of irradiation field in a 62-year-old woman with an invasive-ductal carcinoma (3).

A contact allergy to a solution used to mark the sites of application of patch tests has also been reported (4). Negative results were obtained when patch testing was performed using the individual constituents of the marker solution (gentian violet, dihydroxyacetone and acetone). Chromatographic analysis of various combinations of these chemicals did not identify a chemical derivative which might have caused this reaction, suspecting a compound allergy.

Recently, Rademaker reported the case of a 13-year-old girl with a history of a recurring vulval dermatitis related to repeated contact with sanitary pad (5). Patch testing revealed a marked sensitivity (3+) to rosin. She denied previous reactions to adhesive tape/sticking plaster. During patch testing, she also developed reactions to a permanent waterproof marking pen used to mark the site of patch tests. The ink of this pen also contained colophony.

To our knowledge, no other case of allergic contact dermatitis to rosin from a permanent marker, especially with primary sensitization, has been reported.

References

1. Raison-Peyron N, Co Minh H B, Vidal-Mazuy A, Dereure O, Guillot B. Allergic contact dermatitis to a surgical varnish. *Allergy* 2005: 60: 1331.
2. Goossens A, Armingaud P, Avenel-Audran M et al. An epidemic of allergic contact dermatitis due to epilating products. *Contact Dermatitis* 2002: 47: 67–70.
3. Komericki P, Kern T, Aberer W, Kranke B. Contact dermatitis from Solvent Yellow 146 in a permanent marker. *Contact Dermatitis* 2001: 44: 256.
4. Cox N H, Moss C, Hannon M F. Compound allergy to a skin marker for patch testing: a chromatographic analysis. *Contact Dermatitis* 1989: 21: 12–15.
5. Rademaker M. Allergic contact dermatitis to a sanitary pad. *Australas J Dermatol* 2004: 45: 234–235.

Address:
N. Raison-Peyron
Department of Dermatology and Allergology
CHU Montpellier
Saint Eloi Hospital
80 Avenue Augustin Fliche
34295 Montpellier cedex 5
France
Tel: +33 467 336948
Fax: +33 467 336958
e-mail: n-raison@chu-montpellier.fr

Photoallergic contact dermatitis from dexketoprofen: study of 6 cases

Contact Dermatitis 2006: 55: 59–61

J. J. Goday-Bujan, J. Rodríguez-Lozano, M. C. Martínez-González and E. Fonseca

Department of Dermatology, Hospital Juan Canalejo, La Coruña, Spain

Key words: allergic contact dermatitis; cross-sensitivity; dexketoprofen; drugs; ketoprofen; non-steroidal anti-inflammatory drugs; photoallergic contact dermatitis; piketoprofen

Introduction

Since 1970, non-steroidal anti-inflammatory drugs (NSAIDs) have been commonly used due to their analgesic and antirheumatic properties. Topical formulations are available for some NSAIDs, and cases of contact or photocontact dermatitis have been described (1). Arylpropionic derivatives are the most frequent NSAIDs involved, and topical ketoprofen is the most important photosensitizing anti-inflammatory drug, the first photoallergy case being reported in Spain in 1985 (2, 3). Since 2002, isolated cases of photocontact dermatitis due to dexketoprofen, a new arylpropionic derivative with similar chemical structure to ketoprofen, have been reported (4–6). We report in this article 6 cases of photocontact dermatitis due to dexketoprofen.

Case Reports

In the last 4 years, 6 cases of photoallergic contact dermatitis from dexketoprofen after Enangel® application were seen in our department, of which the clinical and evolutive data are summarized in Table 1. In cases 4 and 6, a previous history of photocontact dermatitis to topical ketoprofen could be confirmed (Fig. 1). The delay between the application of dexketoprofen and the reaction was short, between 2 and 10 days. In case 2, photoallergy was caused by dexketoprofen application followed by UVA ray exposure, but in the remainder cases, a history of sun exposure after dexketoprofen application was confirmed. In all the cases, the reaction was restricted to the application areas, but a strong eczematous picture with bullous lesions was always observed.

Epicutaneous studies were performed with the topical product brought by the patient (Enangel®) as it is and with its components [dexketoprofen, trometamol, ethyl alcohol, carbomer, lavender (HBE-8028)] gently supplied by the manufacturer (Menarini Laboratory). We also tested the GEIDC standard series and our NSAIDs series in all cases (Table 2).

Patch and photopatch tests were performed following ICDRG criteria. Patch tests were carried out applying the allergens on a half of the back leaving them 2 days under occlusion; the readings were performed at 48 and 96 h. Photopatch tests were carried out applying the allergens on the other half of the back and taking the bandages off at 24 h and exposed to UVA rays at a dose of 5 J/cm^2. The first reading

Table 1. Clinical cases

Case number	Sex	Age (years)	Onset time (days)	Location
1	Male	23	<7	Knees and hands
2	Female	59	2 (UVA)	Right shoulder Right arm Right breast Right hand
3	Female	72	10	Legs
4	Male	34	2	Right wrist Back aspect of right hand
5	Male	68	10	Legs
6	Male	38	7	Left forearm Left elbow

Table 2. Non-steroidal anti-inflammatory drugs (NSAIDs) battery of our department

Allergen	Concentration and vehicle
Piketoprofen	2% pet.
Ketoprofen	1% pet.
Indomethacin	1% pet.
Indomethacin	5% pet.
Indomethacin	10% pet.
Acetaminophen	1% pet.
Acetaminophen	5% pet.
Acetaminophen	10% pet.
Salicylic acid	1% pet.
Salicylic acid	5% pet.
Benzydamine hydrochloride	3% pet.
Benzydamine hydrochloride	5% pet.
Bufexamac	5% pet.
Naproxen	5% pet.
Ibuprofen	5% pet.
Phenylbutazone	1% pet.
Fepradinol	1% pet.
Thiosalicylic acid	0.1% pet.
Diclofenac	1% pet.
Diclofenac	5% pet.
Diclofenac	10% pet.
Piroxicam	1% pet.
Sodium salicylate	0.1% pet.
Salicylamide	0.1% pet.
Methyl salicylate	2% o.o.
Oxifenylbutazone	10% pet.

o.o.: olive oil

was performed 24 h after UVA exposure and the second reading 48 h later. Controls were carried out in at least 8 healthy patients with negative results in all cases.

The results of patch and photopatch tests with the NSAIDs series and the components of Enangel® are summarized in Table 3. All patients showed positivity to dexketoprofen 1% and 2% in pet. in the photopatch test, except in case 5 (only to dexketoprofen 2% in pet.) (Fig. 2). In the first 3 cases, we could not do patch and photopatch testing with piketoprofen because the allergen was unavailable at that moment. In cases 4, 5 and 6, piketoprofen was tested at 1% and 2% in pet. with negative results.

Discussion

We present a series of 6 cases of photoallergic contact dermatitis from dexketoprofen studied in our department during the last 4 years. Dexketoprofen has become the most frequent NSAID cause of photocontact dermatitis, being in all cases photoallergic reactions caused by topical application and further sun exposure. Only three isolated cases have been previously reported, but one case may be a concomitant contact and photocontact reaction or a photoaggravated contact reaction based on epicutaneous study (++++ at D4 in patch test and ++++ in D4 in photopatch test) and not a pure photocontact dermatitis (6).

We emphasize that in all cases we found positivity to ketoprofen in the photopatch test. This could be explained by the chemical similarity between dexketoprofen and ketoprofen. In fact, therapeutic effect of ketoprofen (a racemic mixture of 2 enantiomers) is due to the (S)-(+)-enantiomer (dexketoprofen), while (R)-(–)-enantiomer is devoid of biological activity (7).

It is necessary to emphasize that in two cases there was a previous history of photoallergy caused by application of Fastum® gel that contains ketoprofen. In the remainder cases of our series, a previous sensitization to topical ketoprofen is possible because ketoprofen has been widely used in Spain during the last years. It is well known that photoallergy from ketoprofen is due to the benzophenone moiety of ketoprofen (8, 9), but not to the arylpropionic function, thus explaining the cross-reaction to fenofibrate (8) and benzophenone-containing sunscreens (9, 10). We believe that in the cases of photoallergy from dexketoprofen, fenofibrate, benzophenones or ketoprofen should be contraindicated.

We presented several cases of photoallergic contact dermatitis from dexketoprofen. Up to date, few cases have been published, one of them by our department (4–6). We thought dexketoprofen is an NSAID with high photosensitizing potential, similar to what is known from ketoprofen. In addition, dexketoprofen always cross-reacts to ketoprofen, but it does not always happen between dexketoprofen and piketoprofen (5). In fact, cross-reactions between ketoprofen and piketoprofen have been described in some cases (11), but not in others (12, 13). Although we could not carry out epicutaneous tests with piketoprofen in 3

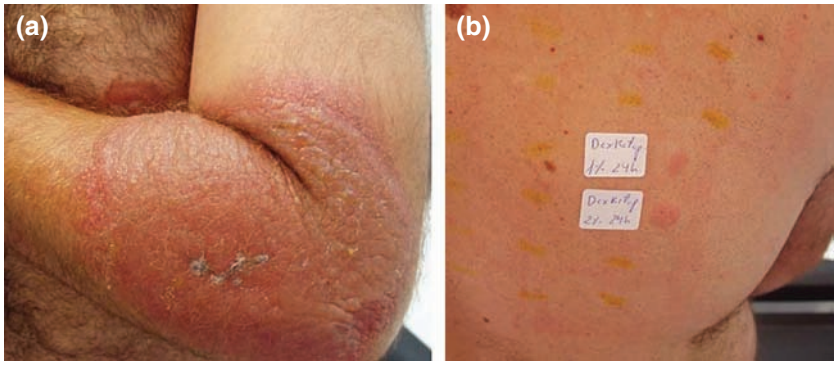

Fig. 1. (A) Intense eczema with bullous lesions on left arm in case 6. (B) Positive photopatch tests to dexketoprofen 1% and 2% pet. at 24 h.

Table 3. Positive epicutaneous tests

Case number	Allergens	PT		PPT	
		D2	D4	D1	D3
1	Dexketoprofen 1% pet.	–	–	+ + +	+ + +
	Dexketoprofen 2% pet.	–	–	+ + +	+ + +
	Ketoprofen 1% pet.	–	–	+ +	+ +
	Piketoprofen 1% pet.	NT	NT	NT	NT
	Piketoprofen 2% pet.	NT	NT	NT	NT
2	Dexketoprofen 1% pet.	–	–	+ +	+ +
	Dexketoprofen 2% pet.	–	–	+ +	+ +
	Ketoprofen 1% pet.	–	–	+ +	+ +
	Piketoprofen 1% pet.	NT	NT	NT	NT
	Piketoprofen 2% pet.	NT	NT	NT	NT
3	Dexketoprofen 1% pet.	–	–	–	+ +
	Dexketoprofen 2% pet.	–	–	–	+ +
	Ketoprofen 1% pet.	–	–	–	+ +
	Piketoprofen 1% pet.	NT	NT	NT	NT
	Piketoprofen 2% pet.	NT	NT	NT	NT
4	Dexketoprofen 1% pet.	–	–	+ + +	+ + +
	Dexketoprofen 2% pet.	–	–	+ + +	+ + +
	Ketoprofen 1% pet.	–	–	+ +	+ +
	Piketoprofen 1% pet.	–	–	–	–
	Piketoprofen 2% pet.	–	–	–	–
5	Dexketoprofen 1% pet.	–	–	–	–
	Dexketoprofen 2% pet.	–	–	+	+ +
	Ketoprofen 1% pet.	–	–	–	+ +
	Piketoprofen 1% pet.	–	–	–	–
	Piketoprofen 2% pet.	–	–	–	–
6	Dexketoprofen 1% pet.	–	–	+ +	+ +
	Dexketoprofen 2% pet.	–	–	+ +	+ +
	Ketoprofen 1% pet.	–	–	+ +	+ +
	Piketoprofen 1% pet.	–	–	–	–
	Piketoprofen 2% pet.	–	–	–	–

Other components of Enangel®: negative in all cases.
Other NSAIDS: negative in all cases.
PT: patch tests
PPT: photo patch tests

cases, in the other 3, the results remained negative (patch and photopatch tests), unlike two of the cases previously published (4, 6).

Finally, we propose that for epicutaneous testing, dexketoprofen should be used at concentrations of 1% and 2% in pet.

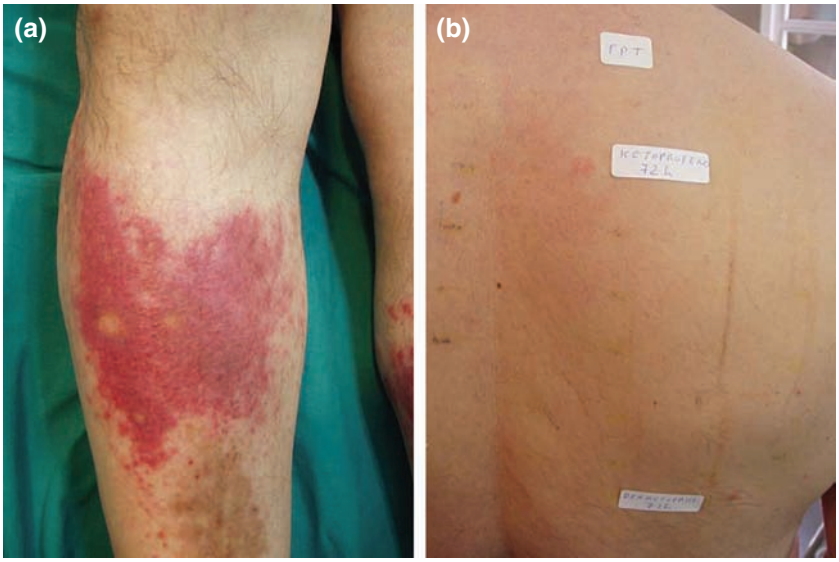

Fig. 2. (A) Acute eczematous reaction on legs in case 5. (B) Positive photopatch tests to dexketoprofen 2% pet. and ketoprofen 1% pet. at 72 h.

References

1. Ophoswongse S, Maibach H. Topical nonsteroidal anti-inflammatory drugs: allergic and photoallergic contact dermatitis and phototoxicity. *Contact Dermatitis* 1993: 29: 57–64.
2. Alomar A. Ketoprofen photodermatitis. *Contact Dermatitis* 1985: 12: 112–113.
3. Durieu C, Marguery MC, Gionardo-Labadie F, Joume Loche F, Bazex J. Allergies de contact photoaggravées et photoallergies de contact au kétoprofène: 19 cas. *Ann Dermatol Venereol* 2001: 128: 1020–1024.
4. Valenzuela N, Puig L, Barnadas M A, Alomar A. Photocontact dermatitis due to dexketoprofen. *Contact Dermatitis* 2002: 47: 237.
5. Cuerda E, Goday J J, Del Pozo J, García J, Peña C, Fonseca E. Photocontact dermatitis due to dexketoprofen. *Contact Dermatitis* 2003: 48: 283.
6. López R, Paniagua M J, Botey E, Gaig P, Rodríguez P, Richard C. Topical dexketoprofen as a cause of photocontact dermatitis. *J Investig Allergol Clin Immunol* 2004: 14: 247–249.
7. Barbanoj M J, Antonijoan R M, Gich I. Clinical pharmacokinetics of dexketoprofen. *Clin Pharmacokinet* 2001: 40: 245–262.
8. Le Coz C, Bottlaender A, Scrivener J. Photocontact dermatitis from ketoprofen and tiaprofenic acid: cross-reactivity study in 12 consecutive patients. *Contact Dermatitis* 1998: 38: 245–252.
9. Bosca F, Miranda M A. Photosensitizing drugs containing the benzophenone chromophore. *J Photochem Photobiol B* 1998: 43: 1–26.
10. Albés B, Marguery M C, Schwarze H P, Joumé F, Loche F, Bazex J. Prolonged photosensitivity following contact photoallergy to ketoprofen. *Dermatology* 2000: 201: 171–174.
11. García Bara M T, Matheu V, Pérez A, Díaz M P, Martínez M J, Zapatero L. Contact photodermatitis due to ketoprofen and piketoprofen. *Alergol Immunol Clin* 1999: 14: 148–150.
12. Goday J J, Oleaga J M, González M, Del Pozo J, Fonseca E. Photoallergic dermatitis from piketoprofen. *Contact Dermatitis* 2000: 43: 315.
13. Miralles J C, Negro J M, Sánchez-Gascón F, García M. Ketoprofen contact dermatitis with good piketoprofen tolerance. *Alergol Immunol Clin* 2001: 16: 105.

Address:
J. J. Goday-Bujan
Servicio de Dermatología
CHU Juan Canalejo – Abente y Lago Hospital
Paseo de Sir John Moore s/n
15006, La Coruña
Spain
Tel: +34 981 178000
Fax: +34 981 205375
e-mail: jgoday@inicia.es

Acute irritant contact dermatitis due to Juglans regia

Contact Dermatitis 2006: 55: 62–63

Iria Neri, Federica Bianchi, Federica Giacomini and Annalisa Patrizi

Department of Specialistic and Experimental Clinical Medicine, Division of Dermatology, University of Bologna, Bologna, Italy.

Juglone is the active ingredient of the green flesh of walnuts and is known to be a strong irritant. We report the first two paediatric cases of contact pigmentation and acute irritant contact dermatitis due to the juice of green walnut husks in two young nursery-school playmates.

Key words: childhood; contact dermatitis; juglone

Case Report

Due to its juglone content, the juice of fresh walnut husks of *Juglans regia* is a component of semipermanent vegetable hair dyes and so-called self-tanning products.

Juglone is the active ingredient of the green shells of walnuts and is known to be a strong irritant.

We report, to the best of our knowledge, the first 2 paediatric cases of contact pigmentation and acute irritant contact dermatitis due to the juice of green walnut husks in 2 young nursery-school playmates.

In September 2003, a 5-year-old boy was referred to us for evaluation of an itching, circumscribed, inflammatory patch on his right buttock, present for 5 days.

Physical examination showed 2 well circumscribed roundish erythematous raised patches of several cms in diameter, well defined and delineated on the right buttock. The child was otherwise in good health. Family and personal history was negative for atopy and ingestion of drugs. The child was no longer wearing a nappy. The mother reported that the lesion had appeared after the child had been playing with a schoolmate in the garden of his nursery school.

On the suspicion of an irritant contact dermatitis without a recognized cause, a zinc oxide paste was prescribed to improve the symptoms.

A week later, the lesion had become red-brown in colour and flatter than at the previous examination (Fig. 1).

The mother referred that during a conversation with the teacher, she learned that the child had played with another child bouncing with his buttocks on fresh, green walnut husks, that had fallen to the ground in the garden.

A diagnosis of contact pigmentation and acute irritant contact dermatitis due to the juice of green walnut husks was made, to the great surprise of the mother.

A few days later, physical examination showed a combustiform appearance followed by lamellar desquamation on the child's right buttock (Fig. 2). On this occasion, the family was accompanied by the playmate who presented similar findings on the buttocks.

Physical examination of this 4-year-old boy showed the presence of 3 similar but less pronounced erythematous, oedematous, geometrically circular lesions on the left buttock. The friend was probably less lively than the first patient, and had jumped more gently on the green walnut husks.

Discussion

Juglans regia, the common walnut, is a large tree that commonly grows in Italy. The active principle of the whole Walnut tree, as well as of the nuts, is Juglone. This substance is known to cause irritant reactions as well as skin hyperpigmentation (1) as in our cases. However, allergic contact dermatitis is a very rare event (1).

The mechanism of skin pigmentation does not involve the melanocytes but occurs through the rise of $C = N$ chromophore groups with a pigmenting action ranging from red to brown. In fact, the $C = O$ groups of juglone have a high affinity for the NH_2 groups of keratin, giving rise to consequently $C = N$ chromophore groups that absorb the violet colour and reflect the yellow and red. The same mechanism explains the hyperpigmentation due to henna (2). Both agents belong to the family of naphthoquinones.

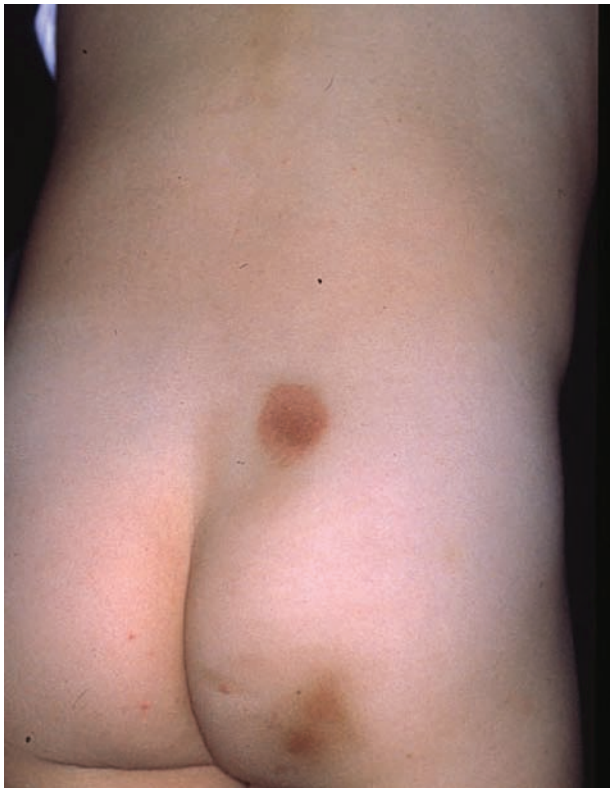

Fig. 1. The circumscribed roundish red-brown patches well defined on the right buttock of the child.

This report is not under simultaneous consideration by any other publication.

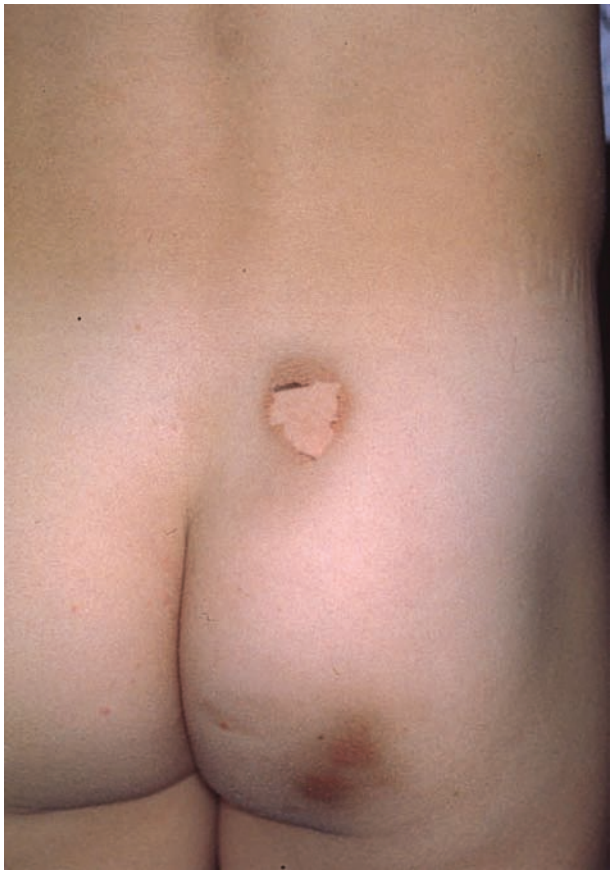

Fig. 2. Same patient with lesions in late phase with lamellar desquamation on the buttock.

The acute irritant and hyperpigmented contact dermatitis due to juglone has been mainly observed during the early autumn in agricultural workers and housewives who remove the green husk of walnuts (2). Recently a case of contact pigmentation and acute contact dermatitis of the hands due to walnut juice has been reported in a woman who shelled several kilos of fresh walnuts (2).

Our patients represent the first paediatric case reports of this condition. In our cases, the diagnosis was delayed by the unusual contact with the walnut husks in a covered area of the body, that is, the buttocks.

When a clinical picture of circumscribed hyperpigmented and combustiform lesions appears suddenly on the skin, possible contact with strong irritant agents, like juglone, should be taken into consideration.

References

1. Siegel J M. Dermatitis due to black walnut juice. *AMA Arch Derm Syphilol* 1954: 70: 511–513.
2. Bonamonte D, Foti C, Angelini G. Hyperpigmentation and contact dermatitis due to Juglans regia. *Contact Dermatitis* 2001: 44: 101–102.

Address:
Dott.ssa Iria Neri
Clinica Dermatologica Via Massarenti 1
40138 Bologna
Italy
Tel: +39 51 6363475/6364841
Fax: +39 51 347847
e-mail: iria.neri@tin.it