The clinicopathological characteristics of oral lichen planus and its relationship with dental materials

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The objective of this work was to carry out a clinicopathological study to ascertain whether clinical and histopathological differences existed between oral lichen planus OLP patients with and without metal restorations. The predominant clinical form in both groups was reticular white, with no statistically significant differences between the forms associated or not with metal. The histological variables showed no statistically significant differences between the groups.

Key words: dental patients; metals; oral lichen planus; oral lichenoid reactions.

The aetiology of oral lichen planus (OLP) is poorly understood, although genetic, infectious, pharmacological, immunological, neurological and psychological causes have all been proposed. Some authors consider a contact allergy to amalgam or other factors mentioned above to cause OLP, whereas others claim the existence of 2 separate diseases: oral lichenoid reactions (OLR) related to amalgam and OLP as an idiopathic disorder. OLP is thought to be a precancerous condition, whereas no malignant progression of OLR adjacent to dental amalgam has been shown (1–3). The role of dental amalgam restorations in the aetiology of oral lichen lesions remains controversial. Studies on the effect of eliminating amalgam in patients affected by OLR/OLP have also provided conflicting results (1, 4).

The objective of this work was to carry out a clinicopathological study to ascertain whether clinical and histopathological differences existed between OLP patients with and without metal restorations.

Patients and Methods
The study involved 50 patients with OLP, who were observed between 2001 and 2004 at the Oral Medicine Unit of the University of Murcia. The patients were diagnosed clinically and confirmed as having OLP by biopsy, according to the criteria of the WHO (5). We excluded patients taking drugs that might cause a lichenoid reaction and those with lesions of the skin or other locations other than the oral mucosa.

2 groups of 25 patients were established: group A (control) formed of 3 men and 22 women, mean age 52 years, including patients with OLP and without metal/amalgam; and group B formed of 7 men and 18 women, average age 51 years, and with metal/amalgam. According to the clinical appearance, the lesions were categorized as mainly white (reticular, papular and plaque like) or mainly red (atrophic and erosive), or both. In group B, the association of OLP with metals followed the criteria proposed by Thornhill et al. (1) (Table 1).

Results
The predominant clinical form in both groups was reticular white, with no statistically significant differences between the forms associated or not with metal. In group B, the most common association was type 2 (68% of cases) (Table 2). The histological variables showed no statistically significant differences between the groups (Table 3).

Table 1. Grading of strength of association between mucosal lesions and amalgam/metal

| Type I | No association, no lesions in direct contact with amalgam restorations |
| Type II | Slight association, <25% for contact of affected mucosa and amalgam |
| Type III | Strong association, >75% of affected mucosa in direct contact with amalgam restorations |

Table 2. Relationship between clinical form and association with dental amalgam

<table>
<thead>
<tr>
<th>Type of lesions</th>
<th>Degree of lesion–metal association</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Type 1</td>
</tr>
<tr>
<td>White oral lesions</td>
<td>2</td>
</tr>
<tr>
<td>Red oral lesions</td>
<td>0</td>
</tr>
<tr>
<td>White and red oral lesions</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
</tr>
</tbody>
</table>
The pathogenetic relationship between OLP and dental amalgam fillings is still a matter of controversy. Several studies suggest that such restorations may induce a lichenoid reaction in the oral mucosa in susceptible patients (4), and a high percentage of improvement of the lesions, following removal, although not all patients have shown the same response (1). In agreement with Dunsche et al. (3), we found no statistical differences in the clinical and histological parameters between OLP associated with amalgam and lichen planus lesions without metals, suggesting that the aetiology of such lesions should be investigated further.

References

Widespread contact dermatitis from tocopherol acetate
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Key words: allergic contact dermatitis; patch testing technique; tocopherol acetate; vitamin E.

Case Report
A 31-year-old, otherwise healthy, man presented with pruritic lesions on the face, penis, both upper extremities and hands. He had felt mild itching on his right wrist 3 days before his first visit. Having applied Mentholatum AD® (Rohto Pharmaceutical Co., Osaka, Japan), an over-the-counter antipruritic cream, which he had not used previously, the lesions had become more pruritic and increased in number. Physical examination showed lesions consisting of oedematous erythema and serous papules on the right wrist and dorsal aspect of the right hand. Erythematous lesions were seen on the face, left forearm and hand, and the penile shaft. We diagnosed contact dermatitis and autosensitive dermatitis on the basis of morphology and history.

Patch testing was performed with Mentholatum AD® cream and the standard series of the Japanese Society for Contact Dermatitis. The results were read on the International Contact Dermatitis Research Group scoring system 2 and 3 days after application. Positive reactions to Mentholatum AD® as is and 1.0% ammoniated mercury chloride were seen.

Further testing was performed with ingredients of Mentholatum AD® cream. The results are listed in Table 1. Positive reactions to tocopherol acetate and negative ones to other chemicals were observed.

Discussion
Topical vitamin E can produce allergic contact dermatitis and contact urticaria (1, 2). Among the reported cases of contact dermatitis from vitamin E, several developed widespread lesions despite local application

Table 1. Patch test results

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>Concentrations (pet.) (%)</th>
<th>Patch-test result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crotamiton</td>
<td>1%</td>
<td>–</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>1%, 0.5%</td>
<td>–</td>
</tr>
<tr>
<td>Glycyrhetic acid</td>
<td>0.2%, 0.1%</td>
<td>–</td>
</tr>
<tr>
<td>Tocopherol acetate</td>
<td>0.5%, 0.25%</td>
<td>+</td>
</tr>
</tbody>
</table>
(3, 4). Garcia-Bravo and Mozo (5) reported a case of generalized contact dermatitis diagnosed by a positive patch test reaction to 1% tocopherol acetate. Our case also developed eczematous lesions not only on the site of application but also on distant sites, and the patient was patch-test positive to tocopherol acetate 0.5% and 0.25% pet. Although tocopherol acetate 10% pet. is recommended as a patch-test material (6), a lower concentration might be sufficient in cases of contact dermatitis with widespread or generalized lesions.

Although our patient developed lesions after the first exposure to the OTC product, he had used other products containing tocopherol acetate previously because they are widely available (5, 7).

He also showed a positive reaction to 1.0% ammoniated mercuric chloride. Because the rash was not that of the typical baboon syndrome, and he had no overt history of contact with mercury, e.g. breaking a thermometer or applying Mercurochrome, mercury was not likely to have caused his condition. As this patch test was not repeated, we also cannot rule out excited skin syndrome as a cause (8).

Acknowledgements
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Discussion
N-(cyclohexylthio) phthalimide (CTP) was discovered in the 1960s and has been used commercially since the 1970s. It acts as a prevulcanization inhibitor for synthetic and natural rubber (1). It is particularly useful in the processing of solid rubber products to prevent the rapid onset of curing (2). There is a wide usage of CTP in the manufacture of large rubber articles such as tyres, tubes, hoses and belts (1). CTP has been incorporated in rubber series used for patch testing for many years. Rubber chemicals are common occupational sensitizers, but contact allergies to CTP have rarely been reported (2, 3, 4).

CTP is supplied for patch testing by Trolab®, Hermal, Reinbek, Germany, at a concentration of 1% pet. Kanerva et al. (2) reported that in their survey of 310 patients 30 had a doubtful or irritant reaction which might be explained by the fact that the currently used concentration of 1% is too high. More recently, Geier et al (4) published recommendations to change the concentration at which CTP should be patch tested from 1% to 0.5%. Hermal will provide CTP at a concentration of 0.5% on the international market from June 2004 (personal communication).

In our Department 321 patients have been tested to CTP since January 2002, and only 3 (0.9%), including the index case, reacted positively. We feel confident, therefore, that this patient had a true allergic contact reaction. The patient could not provide a material safety data sheet for the rubber tiles. Given the improvement of his hand dermatitis after avoidance of rubber material it is likely that CTP was relevant. The positive reaction to IPDI may
also be relevant because a glue used in playground fitting contains diphenylmethane diisocyanate and the 2 diisocyanates may cross-react.

This is an unusual case of an occupational contact allergy. CTP is widely used as a vulcanization retarder in various rubber products, and it may be that allergic reactions are more common than recorded to date.

**References**


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**Key words:** allergic contact dermatitis, black henna tattoos, para-phenylenediamine, patch testing technique

Allergic contact dermatitis from para-phenylenediamine (PPD) after skin painting with temporary black henna tattoos has been increasingly reported. When patch tested, such patients often develop extremely positive (++++) reactions, which may form bullae or erosions. Currently, the recommended concentration of PPD for patch testing is 1% in pet. We propose a new method of using PPD at lower concentrations to patch test patients who have previously been exposed to temporary black henna tattoos.

**Patients and Methods**

12 patients attending our Contact Dermatitis Clinic developed positive patch test reactions to PPD and had a previous history of exposure to temporary black henna tattoos between April 1997 and April 2004. Data recorded included age, sex, year and strength of patch test reaction to varying strengths of PPD used during patch testing. Application time in all but 1 case was 2 days. Recording of patch test reactions was in accordance with the standard criteria (1).

**Results**

Table 1 summarizes the findings. 11 of the 12 patients were female. The ages of these patients varied from 6 to 54 years though most were in their 20s. Half of the patients developed ++ or +++ reactions to PPD 1% pet. 1 patient developed a +++ reaction to PPD 0.3% pet., 4 patients developed + or ++ reactions to PPD 0.01% pet. and 1 patient developed a + reaction after PPD 1% pet. was applied for 20 min.

**Discussion**

Temporary black henna tattoos have become increasingly popular and are widely available at holiday resorts. This is reflected in the increasing frequency of cases presenting to our department. Pure natural henna (*Lawsonia inermis*) has very low allergenic potential but is often adulterated with varying concentrations of PPD to enhance the tattooing process. Brancaccio and colleagues (2) used high performance liquid chromatography to demonstrate that PPD was present in a black henna tattoo mix at a concentration of 15.7%. In a study by Kligman (3) all subjects exposed to such high concentration of PPD became sensitized. PPD at high concentrations is therefore a potent allergen and some individuals can become highly sensitized.

A previous study by our group (4) has demonstrated a dose–response relationship, where both concentration

<table>
<thead>
<tr>
<th>Henna tattoos</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>PPD 1% (20min)</th>
<th>PPD 0.01%</th>
<th>PPD 0.3%</th>
<th>PPD 1%</th>
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<td>2003</td>
<td>17</td>
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<td>++</td>
<td>+++</td>
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<tr>
<td>2</td>
<td>2002</td>
<td>39</td>
<td>Female</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>3</td>
<td>2004</td>
<td>6</td>
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<td>++</td>
<td>++</td>
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<td>++</td>
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<tr>
<td>4</td>
<td>2000</td>
<td>27</td>
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<td>++</td>
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<td>5</td>
<td>1999</td>
<td>21</td>
<td>Female</td>
<td>++</td>
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<tr>
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<td>1997</td>
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<td>++</td>
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<tr>
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<td>2002</td>
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<td>+</td>
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<td>+</td>
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<td>Female</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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and duration of skin exposure to PPD are important factors in the elicitation of contact dermatitis. Our current data show that patients with a history of using temporary black henna tattoos are often highly sensitized to PPD after exposure to such high concentrations and require only very low concentrations of PPD for elicitation of an allergic response. As the current practice of using PPD 1% pet. often results in unacceptably strong blistering reactions, we propose patch testing with PPD 0.01% pet., which can elicit a sufficiently positive reaction in a highly sensitized individual. If this is negative at the 1st reading, the concentration of PPD can then be stepped up to 0.1%, or even 1% to ensure that an allergic contact dermatitis from PPD is not missed.

References

Case Report
A 40-year-old man was referred for patch testing. He reported an 8-month history of a rash that began as blisters on his feet and then spread to involve his hands. He had been treated first with antibiotics and then with prednisolone. Further flares occurred, which required treatment with topical corticosteroids and narrow band UVB light, which improved his condition.

He reported wearing leather boat-shoes (made in China) intermittently over a 3-month period and had noticed dye leaking from the shoes onto his skin. Patch testing showed strong reactions to chromate (++++) and cobalt (++). The patient was diagnosed with allergic contact dermatitis (ACD) from chromate and advised to avoid chrome-tanned leather shoes.

He was reviewed 3 months later and reported that his foot dermatitis was much improved, but that he had developed dermatitis on his left anterior thigh, at the site of contact with his leather wallet. He was subsequently reviewed 3 months later, and reported that, after changing to a plastic wallet, the dermatitis on his thigh had completely resolved for 2 months, though it had recently flared again in the same localized area on his thigh, despite no known leather exposure.

Discussion
Chromate is a common allergen, and leather products are an important source of chromate exposure (1). A recent study by Moed et al. (2) investigating the phenomenon of local skin memory and flare-up reactions, reported that, after clinical recovery from an ACD reaction, CD4+CCR10+ memory T cells apparently persist locally. This may explain recurrent symptoms in a previously affected site, as in this case. Another possible cause for the flare of the rash may be oral ingestion of chromate, which has been previously reported (3, 4).

References
greenhouses. A few years later, she had had to discontinue wearing rubber gloves and boots, because of a burning sensation and eczema at contact sites.

Examination showed psoriatic plaques over the patient’s trunk and limbs. There was confluent erythema, hyperkeratosis with scaling and fissuring on the palms of the hands and lichenification on the dorsa. The distal forearms were also involved, with parakeratotic papules scattered along the borders of the dermatis. The patient underwent a standardized diagnostic procedure for farmers’ occupational diseases (1).

Biopsy from the involved forearm skin showed granulocytic infiltration of dermal papillae; there were foci of parakeratosis and spongiosis in the epidermis, the granular layer being preserved. This picture thus comprised features of both psoriasis and dermatitis. Prick and intracutaneous tests with environmental and occupational allergens were all negative.

Patch tests included European standard series (Chemotechnique, Malmö, Sweden), rubber series (Jaworski, Katowice, Poland) and pesticide series (Institute of Agricultural Medicine, Lublin, Poland). A positive reaction was recorded to thiuram mix 1% pet. on D3, D4 and D7. The test reaction corresponded with those chemicals are relevant sources of thiuram in agriculture and horticulture.

The present case, besides the rarity of the clinical picture, clearly shows that seed protectants and seeds treated with those chemicals are relevant sources of thiuram in agriculture and horticulture.

**References**


**Key words:** allergic contact dermatitis; benzyl alcohol; diclofenac; medicaments; polyethylene glycol monomethyl ether 350 (PEGMME 350); Solaraze® gel.

**Case Reports**

**Case no. 1**

A 65-year-old woman with disseminated superficial actinic porokeratosis developed contact allergy to stearyl alcohol in Efudix cream®. Thereafter, Solaraze® gel (Shire Pharmaceuticals, Basingstoke, UK) was prescribed, but she began to develop further contact dermatitis within a few weeks of starting to apply it. Patch tests were performed with the ingredients of Solaraze® gel, provided by the manufacturers, at the following concentrations: diclofenac 1% aq., benzyl alcohol 5% pet., polyethylene glycol monomethyl ether 350 (PEGMME 350) 1% and 5% aq., sodium hyaluronate 0.5%, 1% and 5% aq. and Solaraze® gel 5% and 10% pet. Positive reactions were seen to diclofenac 1% at D2 (+) and D4 (+++). Solaraze® gel 5% pet. at D4 (+) and Solaraze® gel 10% pet. at D2 (+) and D4 (+).

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Discussion

Solaraze® gel (diclofenac sodium), an NSAID licensed for the treatment of actinic keratoses, was introduced in the UK 3 years ago.

Diclofenac is a phenylacetic acid derivative and has previously been implicated in allergic contact sensitivity to treatments such as Solaraze® gel (1), eyedrops (2) and an anti-inflammatory gel (3).

PEGMME 350, an addition polymer of ethylene oxide and methyl alcohol, which is used to solubilize diclofenac, has previously been reported as an allergen in Solaraze® gel (4). In our case the skin reaction, occurring after a single application, suggests that a primary sensitization took place. Alternatively, it may represent a cross-reaction with the chemically similar polyethylene glycols, condensation polymers of ethylene oxide and water, which are commonly encountered in topical medicaments and are known to sensitize occasionally.

Benzy alcohol is an uncommon allergen despite being widely used as a preservative and fragrance; it also has local anaesthetic properties. Allergic contact sensitivity has previously been reported in fragrance allergy (5) and contact sensitivity to numerous medicaments. Cross-sensitization to benzyl alcohol has been described in patients who have been sensitized to Myroxylon pereirae resin (balsam of Peru); however, there was no such additional reaction in our patient. To our knowledge, this is the first report of allergic contact dermatitis from benzyl alcohol in Solaraze® gel.

This case-series highlights the importance of patch testing with the individual constituents of a putative allergenic product.

Acknowledgements

We thank Shire Pharmaceuticals for their cooperation in providing ingredients of Solaraze® gel and Dr Ian Howe for his helpful comments.

References


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Case Report

A 53-year-old woman, without past medical history, was referred with dermatitis that had begun after she returned from holiday in Tahiti. Because of widespread insect bites, she had applied several topical remedies including 2 tamanu oil samples, bought in a supermarket and over the counter in a pharmacy. The dermatitis consisted of acute eczema of her upper and lower limbs and face, which cleared after discontinuation of topical drugs and administration of potent topical corticosteroids.

Key words: CAS [241148-25-4]; cosmetics; coumarins; EINECS [310-127-6]; patch testing; photodermatitis; plants.

Discussion

Tamanu oil is known as Calophyllum inophyllum (INCI Europe) and Calophyllum tacamahaca (INCI USA). It has identification numbers CAS [241148-25-4] and EINECS [310-127-6] (1, 2). The tree Calophyllum tacamahaca L. (known as tamanu or atti), of the Guttiferae family, is indigenous to South-east Asia, currently growing in Asia and the South Pacific. It is profuse in Polynesia, growing up to 25–30 meters high.

Tamanu oil is extracted from seeds (almonds) or raw fruits, filtered and stabilized with tocopherol (vitamin E). In the cosmetics industry, it is used as a skin-conditioning agent, particularly as an emollient (1, 2). It is more widely employed in traditional medicine, both in Tahiti and in China, for various disorders like cuts, scrapes, burns (from boiling water, sun, or X-rays), insect bites and stings, abrasions, acne, scars, psoriasis, diabetic sores, anal fissures, dry skin, blisters, eczema, herpes sores and to reduce foot and body odour. It is applied to the neck to relieve sore throat and is massaged into the skin to relieve muscular, neuralgic, shingles, leprosy neuritis, rheumatism and sciatica pains. Polynesian women utilize it for promoting healthy, clear, blemish-free skin and to prevent diaper rash and skin eruptions (3–5).
Tamanu oil mainly contains lipids: neutral lipids (92%) like palmitic, stearic, oleic and linoleic acids, glycolipids (6.4%) and phospholipids (1.6%). It also contains specific molecules such as inocalophyllins (6), some of them having anti-inflammatory, antibacterial and antiviral properties. For example, 4-phenylcoumarins like calo-coumarin-A have an inhibitory effect on the growth of Epstein–Barr virus and can be regarded as cancer chemopreventive agents (7). Others coumarins like inophyllins have been considered as inhibitors of HIV-1 reverse transcriptase (8).

This report seems to be the first of allergic contact dermatitis from tamanu oil. A slight degree of photo-worsening can be proposed as the patch tests were more intense after UVA irradiation. The exact allergen(s) remains to be identified but seems to be a genuine component of tamanu oil and not the tocopherol generally added as an anti-oxidant.

References


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Contact dermatitis from the staples of neuroreflexotherapy

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Case Report

We describe a 52-year-old female patient, with a personal history of metal intolerance and mechanical low back pain, who was seen in our Department of Dermatology because of dermatitis on the lower back.

In the last year, she had undergone 2 interventions of neuroreflexotherapy (NRT). After the 1st such intervention, surgical implants were kept on the skin surface of the lower back and gluteal area for 3 months, during which she did not develop any complication. The staples were removed for 1 month, but reimplemented during a 2nd such intervention. A few days later, pruritic erythematous lesions developed around the surgical staples, after which the staples were removed (Fig. 1).

Patch testing was performed with the European standard series and a metals series (Chemotechnique, Malmö, Sweden). After 2 and 4 days, a positive (++) reaction to nickel was observed.

The material of the surgical staples was stainless steel AISI 316, composed of chromium (18%), nickel (14%), molybdenum (3%), manganese (2%), silica (1%), phosphorus (0.045%), carbon (0.03%), sulfur (0.02%) and iron (the rest).

Discussion

NRT is a relative new technique, indicated in the treatment of chronic non-specific low backpain, which has been developed by the Kovacs Foundation (Spain). It consists of the stimulation of cutaneous nerve fibers related to the nerves involved in pain, inflammation and muscle contracture. Surgical staples are implanted on the skin surface of the back over certain trigger points and kept in place for approximately 3 months. The trigger points are hypersensitive points, from which local or irradiated pain originates, and which are found by palpation and identified by each patient. The trigger points are situated in certain dermatomes, which depend on the clinically implicated metameres in each case, as well as in specific areas of the ear.

In the ears, little metallic staples are implanted with about 2-mm penetration into the skin. On the back, special surgical staples are implanted without pain and with adequate depth into the skin, in order to obtain a normal life for several weeks.

The surgical implantation of staples consisted of 5 segments at the same level; 1 central superior segment, which rested above the skin; 2 lateral segments of 2.5 mm each, which penetrated the skin and formed an angle of 90 degrees with the central segment; and 2 central inferior segments of 2 mm each, which were opposed to each other under the skin and which formed a

Fig. 1. Staples implantation of surface.
symmetric angle of 90 degrees with the lateral segments.

Nickel is the most common cause of allergic contact dermatitis and its prevalence continues to increase (1). Whereas in the past this was mainly occupational, the increasing use of nickel in jewellery and consumer products extended the problem among women and the fashion of piercing also in men.

Steel is principally composed of iron and chromium to which other substances, mainly other metals, are added to modify the physical and chemical properties. The majority of modern surgical material is made of stainless steel with a high resistance to corrosion. The name stainless implies less oxide. However, although it oxidizes less, it does oxidize. The safety of the surgical steel implants is proportional to its resistance to corrosion, which depends largely on its composition and chemical structure. For example, whereas molybdenum increases the resistance of steel to corrosion, sulfur reduces it (2). In industry, several techniques are employed to increase the resistance of steel products, such as the polishing of its surface and the addition of a fine external layer of ferrochromate-oxide. As long as the external layer remains intact, the scarce cations that may be released are iron and chromium and not the metallic ions of the internal layers such as nickel.

It is well known that nickel-plated items may readily release nickel onto the skin, which may cause sensitization to nickel or allergic contact dermatitis in previously sensitized individuals. Such nickel-plated items may release around 100 μg/cm²/week of nickel (2). However, the nickel-releasing capacity of steel is still debated. There are many types of stainless steels, some of which may release sufficient nickel to elicit allergic contact dermatitis, especially those that contain a large amount of sulfur (1–3). Some authors divide the stainless steels into high- and low-sulfur stainless steels according to the capacity to elicit allergic contact dermatitis from nickel. An example of a high-sulfur stainless steel is AISI 303, which may release 1.5 μg/cm²/week and elicit allergic contact dermatitis from nickel (2).

Some low-sulfur stainless steels, such as AISI 304, AISI 430 and AISI 316L, release less than 0.03 μg/cm²/week of nickel in acid medium and theoretically do not elicit allergic contact dermatitis (2, 3). Steel items are considered safe when they release less than 0.5 μg/cm²/week of nickel, although it is known that some nickel-sensitive individuals may react to objects that release less nickel (4, 5).

We describe herein a patient who developed sensitization and allergic contact dermatitis from nickel, which was released by a surgical implant made of steel AISI 316L, which confirms that low-sulfur stainless steel, with a low rate of nickel release to the outside medium, is not as safe as it is thought to be.

References

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