

Do-it-yourself cement work: the main cause of severe irritant contact dermatitis requiring hospitalization

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Summary

Background. There are myriads of potentially irritant agents causing acute irritant contact dermatitis. In the large majority of cases, dermatitis is mild to moderate, and patients do not need hospitalization. However, some agents or special circumstances may cause severe dermatitis requiring more intensive therapy.

Objectives. The aim of this study was to evaluate causative agents of severe acute irritant contact dermatitis requiring hospitalization.

Methods. In this single-centre observational cohort study, we included 54 consecutive patients presenting with signs and symptoms of acute irritant contact dermatitis for which hospitalization was necessary. The severity of dermatitis was graded (grade I–IV) according to intensity, and details related to the skin irritation (irritant agent, area of exposure, time interval to onset of symptoms, and duration of hospitalization) were determined.

Results. All cases with severe ulcerative dermatitis (grade IV) were caused by wet cement, owing to prolonged skin contact. These cement burns are clearly associated with amateur work, younger age, male preponderance, and leg localization.

Conclusions. The study data provide clear-cut evidence that wet cement is a severely irritant substance that regularly causes the most severe form of acute irritant contact dermatitis. The main causative prerequisite for these cement burns is do-it-yourself work with poor protective measures.

Key words: cement burn; cement dermatitis; cement injury; chemical burn; contact dermatitis; necrosis; skin irritation; ulceration.

Irritant contact dermatitis in general is one of the most frequent skin diseases leading to outpatient dermatological consultation. The term irritant contact dermatitis includes several inflammatory reaction patterns, ranging from mild cumulative skin irritation to severe dermatitis after a single exposure to a caustic chemical (1). In

the clinical spectrum of acute irritant contact dermatitis, more severe cases with intensive pain, bullae formation or necrosis may need hospitalization. These patients generate rather high costs, both for the healthcare system and for the employers, owing to the patients' inability to work (2).

There are myriads of substances capable of causing acute irritant contact dermatitis, which is typically strictly confined to the exposed skin area (3, 4). In more severe forms of dermatitis, physical and/or chemical skin damage caused by the irritant substance is often intensified by individual risk factors, such as an impaired skin barrier resulting from atopy (5–7), alteration of the lipid barrier in older people, or a lack of skin protection (8). In principle, it is not possible to differentiate between

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very severe irritant contact dermatitis and a primary chemical burn. Both conditions usually present initially with erythema, oedema, and vesicles, which may progress to necrosis and ulceration 12–48 hr after contact with the irritant substance (8). Itching and burning up to apparent pain are often-noted subjective symptoms (8). It is possible to classify acute irritant contact dermatitis and chemical burns into four degrees of severity, whereby patients with dermatitis of grade III and IV severity may need hospitalization.

One important substance with a well-known high irritant potential to cause severe irritant contact dermatitis, in both the occupational and the do-it-yourself settings, is so-called ready-mixed cement (9–13). When mixed with water, these ready-mixed cement products release highly alkaline calcium hydroxide, which leads to an increase in the pH from 5 to 12 or 13 within minutes (10). This very alkaline milieu is capable of causing severe cell damage, including epidermo-dermal necrosis and ulceration, especially when it is intensified by mechanical abrasion, for example during work in a kneeling position (10).

The aim of this single-centre observational cohort study was to determine the main causes and clinical manifestations of acute severe irritant contact dermatitis requiring hospitalization.

Material and Methods

Patients

A total of 54 consecutive patients referred to our department for treatment of acute irritant contact dermatitis over a period of 7 years (2005–2011) were included in this retrospective single-centre observational cohort study. Individuals were eligible for inclusion when they fulfilled the following criteria: (i) a conclusive history and symptoms of acute irritant contact dermatitis; and (ii) inpatient treatment being necessary because of the severity of symptoms.

Patient history

Patient histories were obtained with a standardized questionnaire covering the episode of acute irritant contact dermatitis; additional information was retrieved from care-givers and general practitioners. The data collected included age and sex, information on the culprit agent, the severity and symptoms of acute irritant contact dermatitis (classification as described below), and duration of hospitalization. The time interval between chemical contacts and the onset of symptoms, and the localization of the dermatitis, were recorded.

Table 1. Severity grading of acute irritant contact dermatitis

Grade I (mild)	Erythema
Grade II (moderate)	Erythema, oedema, small vesicles
Grade III (severe)	Larger vesicles: blisters, bullae
Grade IV (most severe)	Epidermo-dermal necrosis, ulceration

Grading of acute irritant contact dermatitis

The severity of acute irritant contact dermatitis was classified as mild, moderate, severe, or most severe, according to a modified version of the system used for thermal burns (i.e. first-degree, second-degree and third-degree burns) with respect to the leading cutaneous symptoms (Table 1).

Results

The mean age of the patients included was 54 years (range 12–96 years), and the sex ratio was balanced. Eleven patients (20.4%) presented with the most severe form of dermatitis, including necrosis and/or ulceration (Table 2). Reasonably, there was no case of a patient with grade I dermatitis requiring hospitalization. Most frequently, the irritant contact dermatitis affected the legs (20 patients, 37.0%), followed by one or both hands, respectively, and the trunk was affected in 9 patients (16.7%). The most prevalent agent causing an acute irritant contact dermatitis requiring hospitalization was wet cement (20.4%). Various topical medications (e.g. tar, dithranol, and antiseptics), particularly under occlusive bandaging, were responsible for 10 of the 54 hospitalizations (18.5%). Other causative substances determined were disinfectants in 8 patients (14.8%), biological fluids (e.g. urinary incontinence) in 7 patients (13.0%), and organic solvents in 6 patients (11.1%). The mean duration of hospitalization was 8.4 days, with a median of 8 days.

The mean age of patients with cement burns was 38 years (range 25–70 years), and 10 of the 11 patients were male. All 11 patients with cement burns developed grade IV dermatitis (Fig. 1), and the mean latency until clinical grade IV signs were present was 1.8 days, with a median of 2 days (Table 3). The first consultation with a dermatologist usually took place on day 2 or day 3, when grade IV symptoms had already developed. All 11 cases of cement burns were contracted during do-it-yourself work at home with ready-mixed wet cement products. In 10 cases, the legs were affected, whereas 1 patient presented with a cement burn on her arms. The mean duration of hospitalization caused by cement burns was 10.4 days, with a median of 11 days.

Table 2. Clinical data of patients with acute irritant contact dermatitis

Number of patients	54
Mean age in years (range)	54 (12–96)
Male/female ratio	27/27
Severity of acute irritant contact dermatitis, no. (%)	
Grade I	0 (0)
Grade II	30 (55.5)
Grade III	13 (24.1)
Grade IV	11 (20.4)
Localization, no. (%)	
Leg	20 (37.0)
Hand	9 (16.7)
Trunk	9 (16.7)
Head/neck	8 (14.8)
Arm	5 (9.3)
Foot	3 (5.6)
Suspected causative agent, no. (%)	
Cement	11 (20.4)
Topical medications	10 (18.5)
Disinfectants	8 (14.8)
Biological fluids	7 (13.0)
Solvents	6 (11.1)
Detergents	2 (3.7)
Foods	2 (3.7)
Plants	1 (1.9)
Unknown	7 (13.0)
Days of hospitalization	
Median	8
Mean	8.4
Minimum	1
Maximum	21

Discussion

In this single-centre observational cohort study, we investigated the main causes and specific circumstances of acute irritant contact dermatitis requiring inpatient treatment. Our data show that wet cement is an outstanding trigger substance that regularly causes the most severe form of irritant contact dermatitis. All 11 patients who presented with cement burns developed grade IV dermatitis, whereas no other irritant agents observed, such as topical medications, disinfectants, or biological fluids, were able to trigger such severe symptoms. Therefore, in our series, wet cement was the only substance possessing the potential to cause skin necrosis and, finally, ulceration.

Irritant contact dermatitis is a multifactorial skin inflammation, and its pathophysiology includes several contributory endogenous and exogenous factors, such as asteatosis, inappropriate application of care products, on the one hand, and extensive use of irritating substances on the other, as well as the intrinsic irritant potential of a specific substance (8). Spoo et al. reported in their review that cement burns appear as full-thickness burns



Fig. 1. (a) Typical ring-like pattern of cement-induced necrotic dermatitis. (b) Clinical picture after debridement of necrotic tissue. (c) Partially scarred healing.

(grade IV) in up to 75% of the published cases (9). There may be several reasons why cement induces such severe forms of acute irritant contact dermatitis, which are commonly called cement burns. First, the admixed sand grains of the cement lead to small traumata of the skin, and thereby additionally impair the skin barrier (10). As a consequence, alkalis penetrate into deeper skin layers, dissolve proteins and collagen, dehydrate cells, and cause broad necrosis (10). Generally, alkali burns lead to liquefaction necrosis, which usually penetrates deeper than coagulation necrosis, owing to acid burns (11, 14). Therefore, cement burns are typically full-thickness burns with deep ulceration in most cases. An additional reason

Table 3. Clinical data of patients with cement burns

Number of patients	11
Mean age in years (range)	38 (25–70)
Male/female ratio	10/1
Severity of acute irritant contact dermatitis	
Grade I	0
Grade II	0
Grade III	0
Grade IV	11
Days until grade IV symptoms	
Median	2
Mean	1.8
Minimum	1
Maximum	3
Localization	
Leg	10
Hand	0
Trunk	0
Head/neck	0
Arm	1
Foot	0
Days of hospitalization	
Median	11
Mean	10.4
Minimum	7
Maximum	15

for the severity of cement burns is that contact with the alkaline wet cement does not lead to an immediate sensation of burning or stinging, with the consequence that patients often have had intense contact with the cement for several hours before the first bothersome symptoms occur (13).

Contact with wet cement usually occurs either in the occupational setting or during do-it-yourself work at home. There are several occupations with more or less intense exposure of the employees to wet cement, for example bricklayers, carpenters, cement masons, concrete finishers, concrete truck drivers, construction craft labourers, plasterers, and terrazzo workers (10). However, in the occupational setting, acute cement burns are relatively rare (10), and are estimated to occur in only 11–86 cases per 100 000 workers per year (15), probably because of effective education on and instruction in work protection measures, and surveillance of adherence to these. In contrast, do-it-yourself individuals handling wet cement are often not aware of the risks, and they frequently ignore manufacturers' warnings and instructions regarding skin protection. Measures such as wearing working clothes and safety shoes are often disregarded in the homework setting (13). A major review article analysing 51 cases of cement burns published over four decades reported that 40% of the cases were acquired during do-it-yourself work (9). In contrast, in

our study, all patients with cement burns were house owners and sustained their injuries during do-it-yourself work at home.

None of our patients was aware of the high irritant potential of wet cement, and in all 11 cases the cement burns were acquired during flooring of the garage. Ten of our patients were middle-aged males without expert knowledge of construction work, and in these cases the knees were affected, confirming the lower extremities as the predominant locality of cement burns (9). All of them worked while kneeling in the cement for a significant time span, leading to the typical clinical picture of a ring-like necrotic dermatitis on the ventral side of the knees, sparing the central patella region (Fig. 1). One patient was a woman who developed the cement burn on her arms while assisting her husband without wearing protective clothes. Although, in our study, 50% of the whole patient series who had been hospitalized for treatment of acute irritant contact dermatitis were female, there was only 1 woman presenting with a cement burn.

In our patient series, the first symptoms of cement burns were erythema and/or burning sensations, and appeared regularly within 4–8 hr after exposure. However, the first contact with a dermatologist did not occur until days 1–3, because the development of the severe full-thickness necrosis regularly takes >24 h, and patients initially have only minor symptoms. First aid procedures after skin contact with wet cement should include taking off the soaked clothes and washing away the remaining cement clumps (10). Neutralizing the skin pH with buffered phosphate solution or 1% acetic acid solution may be helpful, but carries a risk of further skin irritation (12). However, these immediate measures are usually not carried out, because patients simply do not realize that they have acquired severe skin damage. Treatment of already developed cement burns includes antiseptic and cooling topical treatment, as well as symptomatic analgesic treatment. Topical antibiotics should be avoided, because of the risk of generating resistant bacterial strains (11), and systemic antibiotics should only be administered if typical clinical signs of a skin infection occur. Topical corticosteroids may help to reduce the inflammation and thereby reduce tissue damage; however, in an experimental irritant contact dermatitis model, they did not show efficacy (16). Moreover, the antiproliferative properties of corticosteroids might lead to a further delay in the already long healing time over the course of several weeks (16, 17). Further controlled studies are required to prove a relevant therapeutic effect of corticosteroid creams beyond the effect of cooling and moistening. Surgery is necessary in approximately one-third of cases (8). When

necroses develop, debridement of the dead cell masses is recommended to help fibroblasts and keratinocytes start dermal and epithelial regeneration. Extended ulcerations can be covered by skin grafts; this might reduce the risk of intense scarring with consecutive contractures.

Conclusions

To reduce the number of cement burn cases in the do-it-yourself setting, there are two important preventive

approaches. First, there has to be better education on the hazards of wet cement and on measures for adequate skin protection in building supply and do-it-yourself stores, where individuals usually acquire their construction material. Second, there is a need for more prominent warning notices on packages of construction material, including clear and coherent instructions how to proceed in the case of accidental contact with wet cement even before overt clinical symptoms develop.

References

- 1 Levin C Y, Maibach H I. Irritant contact dermatitis: is there an immunologic component? *Int Immunopharmacol* 2002; **2**: 183–189.
- 2 Fluhr J W, Darlenski R, Angelova-Fischer I, Tsankow N, Basketter D. Skin irritation and sensitization: mechanisms and new approaches for risk management. 1. Skin irritation. *Skin Pharmacol Physiol* 2008; **21**: 124–135.
- 3 Basketter D A, Chamberlain M, Griffiths H A, Rowson M, Whittle E, York M. The classification of skin irritants by human patch test. *Food Chem Toxicol* 1997; **35**: 845–852.
- 4 Slodownik D, Lee A, Nixon R. Irritant contact dermatitis: a review. *Australas J Dermatol* 2008; **49**: 1–9.
- 5 Meding B, Swanbeck G. Predictive factors for hand eczema. *Contact Dermatitis* 1990; **23**: 154–161.
- 6 Nicholson P J. Occupational contact dermatitis: known knowns and known unknowns. *Clin Dermatol* 2011; **29**: 325–330.
- 7 Avnstorp C. Risk factors for cement eczema. *Contact Dermatitis* 1991; **25**: 81–88.
- 8 Seyfarth F, Schliemann S, Antonov D, Elsner P. Dry skin, barrier function, and irritant contact dermatitis in the elderly. *Clin Dermatol* 2011; **29**: 31–36.
- 9 Spoo J, Elsner P. Cement burns: a review 1960–2000. *Contact Dermatitis* 2001; **45**: 68–71.
- 10 Chung J Y, Kowal-Wern A, Latenser B A, Lewis R W II. Cement-related injuries: review of a series, the National Burn Repository, and the prevailing literature. *J Burn Care Res* 2007; **28**: 827–834.
- 11 Sherman S C, Larkin K. Cement burns. *J Emerg Med* 2005; **29**: 97–99.
- 12 Mehta R K, Handfield-Jones S, Bracegirdle J, Hall P N. Cement dermatitis and chemical burns. *Clin Exp Dermatol* 2002; **27**: 347–348.
- 13 Keles A, Aygencel G, Kahveci O, Bildik F, Demircan A. Contact with wet cement: report of a case. *Contact Dermatitis* 2008; **58**: 173–174.
- 14 Falker G. Kolliquationsnekrose nach Kontakt mit Zementestrich. *Akt Dermatol* 1985; **11**: 101–104.
- 15 Nicholson P J, Llewellyn D (eds) *Occupational Contact Dermatitis & Urticaria*, London, British Occupational Health Research Foundation, 2010.
- 16 Levin C, Zhai H, Bashir S, Chew A L, Anigbogu A, Stern R, Maibach H. Efficacy of corticosteroids in acute experimental irritant contact dermatitis? *Skin Res Technol* 2001; **7**: 214–218.
- 17 Lee J Y, Effendy I, Maibach H I. Acute irritant contact dermatitis: recovery time in man. *Contact Dermatitis* 1997; **36**: 285–290.