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Caustic Reaction Caused by Cement

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SUMMARY A case is reported of a patient who developed full thickness chemical burns of the skin after a prolonged contact while working with wet cement. The history, course of disease, and therapy are described. Cement is an alkaline substance (pH >12) leading to colliquative necrosis. Tissue damage is due to the exothermic reaction of calcium oxide and water forming calcium hydroxide. Patch test was performed to test sensitization to chromium, chromate and cobalt, the usual cement ingredients. In our opinion, such lesions may not be rare because cement is widely used in construction, but are rarely described or under-recognized.

KEY WORDS: cement burns; skin contact dermatitis; caustic reaction

INTRODUCTION

Daily dermatological practice indicates chronic cumulative irritant hand eczema or acute allergic contact dermatitis to predominate among construction workers in Croatia who handle alkaline materials such as mortar and cement. Contact allergic dermatitis is quite frequently diagnosed as an occupational disorder due to hypersensitivity to some allergenic component such as chromate and nickel ions. While the irritative and allergenic potential of cement is well recognized, it does not appear to hold for its caustic potential of causing third-degree chemical burns. Cement is a widely used mixture in construction. A corrosive alkali, calcium hydroxide, is liberated as water is added to the lime present in the cement mixture. Skin contact for prolonged periods produces deep chemical burns with thick eschar formation (1,2). Skin burns result from exposure to cement and its components. Of particular interest is the etiology of such burns. Wet cement is caustic (with a pH as high as 12.9) and can produce third-degree alkali burns after 2 hours of contact. Unlike professional cement workers, amateurs are usually not aware of any danger and may stand or kneel in cement for long periods (3). A general physician may recognize neither the seriousness of the injury in its early stages nor the significance of a history of prolonged contact with wet cement. All people working with cement should be warned about its dangers and advised to immediately wash and dry the skin if contact does occur. Do cement burns result from a high pH solution in contact with the skin under abrasive conditions for an undetermined period of time, or are they a consequence of hypersensitivity to chromates, or is there a role for calcium ion and ionic species with which the skin is in contact? Factors important in burn production appear to be alkalinity, duration of contact, and the abrasive nature of cement particles (2). The effect of cement dust upon living organisms consists in irritating, sensitizing and pneumoconiotic properties of its components (4). Cement burns present a potentially serious injury and were in the past considered a much more serious problem treated

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by plastic and reconstructive surgery (5). Patients were unaware of the potential of cement to cause burns, and had no precautionary warnings about cement (6).

Potentially very serious are injuries through explosion and contact with hot powder during manufacturing of cement (7). Hannuksela and Suhonen (8) had six patients who had been making a floor by levelling wet cement in a kneeling position. They all noted nearly identical curved ulcers on both sides of both patellae. General agreement is that direct skin contact, prolonged contact time, and pressure upon exposed areas or by persistent contact of cement with the skin by rubber boot polyethylene sheeting or rubber gloves are the conditions necessary for wet Portland cement to cause an acute ulcerative contact dermatitis (9). A case has been reported of a 63-year-old woman who sustained an acrylic cement burn of the sciatic nerve at hip replacement. She was treated by resection of the damaged segment and grafting (10). Xiao and Cai classify cement burns according to the cause or the environment in which the injury occurred into abrasion, heat and explosive type (11). Spoo and Elsner report on 51 cases encountered during four decades, with special reference to the common mechanisms of injury, localization of cement, exposure, preventive measures taken, and treatment (12). Cement burns are injuries that involve professionals at their workplace as well as amateurs during do-it-yourself work (12). According to Ricketts and Kiml, 51% of chemical burns occurred in domestic and 38% in industrial setting, most common being those caused by cement (25%), sulphuric acid (16%) and hydrofluoric acid (16%), mostly involving upper and lower extremities (13). Chemical injuries-burns to the eye account for a significant proportion of ocular trauma and alkalis were the most frequent causes (48% of cases) (14).

CASE REPORT

M. P., a 25-year-old student, was admitted to the Outpatient Clinic of the University Department of Dermatology and Venereology, Zagreb University Hospital Center, for acute toxic, ulceronecrotic dermatitis of both shins, more pronounced on the right side (Fig. 1). The history and course of disease revealed that five days before, while laying foundation at his home, he had been kneeling in wet cement for 3 hours with only a sweatsuit on. At first he noted a burning sensation, then upon removing his clothes and socks he saw that his shins were erythematous. He kept on working for about 2 hours, mostly squatting. He washed the affected area thoroughly and vigorously with soap and water until they felt tender. He did not seek medical aid. His mother, a nurse, advised him to put on some ointment (silver sulfadiazine) and a solution for lavage. On the next day he became febrile, which persisted for 3 more days; he was taking systemic antibiotics. The pain increased and the inflamed area became worse. He decided to seek medical care, first at the Department of Surgery, then he was referred to our Department. Initial treatment included antiseptic baths and dressings in order to wash out and eliminate the layers of hardened cement and necrotic tissue. Upon their partial removal, we noticed persistent layers of hardened cement by 0.5 cm in width around the edges. On the day after admission he was referred to a surgeon for primary wound care and excision of necrotic areas. The wounds were debrided at our Department. We continued removing devitalized tissue with hydrogen peroxide, saline solution dressings, and corticoid and antibiotic ointments. The antibiotic cure was completed. Locally we also used vaseline gauze and hydrocolloid dressings. By day 7 of hospitalization the right shin area was covered with a tenacious black-green eschar, irregularly shaped, 27 x 9 cm. The patient was discharged on day 10.

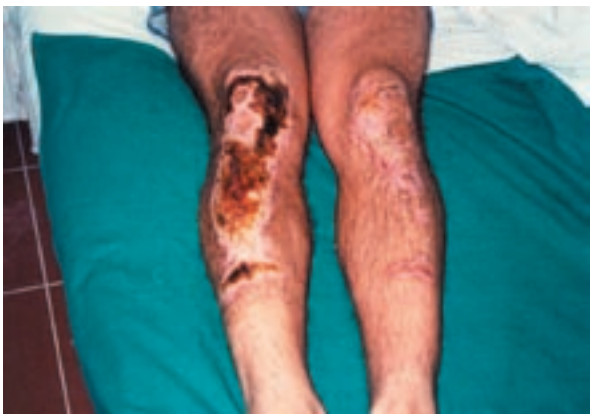

Figure 1. Thick tenacious eschar characteristic of caustic burn produced by wet cement.

RESULTS

All burns on the left shin re-epithelialized well. We gave the patient thorough instructions on how to continue home care with fibrinolytic ointments and antiseptic solutions. On control outpatient visit, no signs of secondary infection were observed. After one-month fibrinolytic therapy, the eschar had separated completely and the wound healed with an atrophic hyperpigmented scar. Four months af-

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ter the incident, there was a hypertrophic, linear scar on the medial side of the right knee, without limiting its function.

DISCUSSION

Chromates are common in our environment, for example, they may be found in degreasing solvents, detergents, paints, leathers, metalworking fluids, textile and printing inks, diesel engine, radiator fluids and wood preservatives. Some chromates are water-soluble and some are not. Water soluble chromates in cement and cement products dissolve in water used to mix cement, concrete, plaster, mortal or other products. It is obvious that our patient developed cement chemical burns of the full thickness of the skin on the right shin and superficially on the left shin, similar to those described in the literature (2,6,8,9). According to literature data, most authors agree that wet cement has a potential to damage the skin in three ways: 1) as an allergen to cause contact dermatitis due to hypersensitivity to hexavalent chromium in chromate compounds that are found in cement and are hard to treat. This is because once a person has been sensitized, any further skin exposure to chromates will bring the symptoms back; 2) as an abrasive due to the gritty nature of the course and fine aggregate within the wet cement (1-13). Wet cement and wet cement products can damage the skin surface because they are abrasive and highly alkaline; and 3) as an alkali it causes burns (2,14).

The majority of injuries with wet cement are located on the lower legs and knees, as in our patient (2,6,9). Wet cement has a pH of approximately 12.5. This is the result of the hydration of a number of oxides, mainly calcium oxide, to a supersaturated solution of their hydroxides. This is an exothermic reaction but the rise in temperature is slow and negligible. Such a highly alkaline solution is obviously very caustic and leads to protein disintegration, which permits further spread of the penetrating alkaline solutions. In this case the skin damage is not confined to the zone of action. Information on the harmful properties of cement in the occupational as well as in the domestic environment is needed. Controlling the hazard with cement is needed to reduce the risk of cement chromate dermatitis. The workplace should be safe and healthy (mortal, plaster, grout, concrete, etc.), it should not be wet, and any skin contact with wet cement or wet cement products should be limited (6,9). The addition of fresh ferrous sulfate lowers the amount of water-soluble chromate in cement. PVC gloves are recommended for handling wet cement or wet cement products. After handling cement products, the skin should always and immediately be washed with mild soap and clean water. A water-repellent barrier cream should be applied before putting on gloves. In case of dermatitis, anti-inflammatory therapy is needed. The following steps are emphasized in the treatment of cement burns: first, remove the contaminant with a cloth or similar material; dry in case of sodium or lime; wash with copious running water and soap; curette the burn areas to remove any contaminating chemical if the lesion is small (e.g., 2.5 cm in diameter). In other cases, excise the area as soon as possible, with grafting used as appropriate (19).

The doctors, especially casualty officers, should know the potential of progressive full thickness burns from wet cement. General public awareness should be increased (6).

CONCLUSION

Wet cement, although rarely, may cause third-degree chemical burns. In Croatia, cement is widely used in construction but cement induced injuries are rarely described or not well recognized. Cement burns should be treated surgically, with early excision of necrotic areas and skin grafting, especially if lesions are greater than 2.5 cm in diameter. The advantages of such a protocol are reduced duration of hospital stay, prevention of complications such as reactive inflammation to the eschar and secondary infections, and keloids. General public should be made more aware of the potential of injury from wet cement, especially among amateurs who should be informed on the respective first aid measures and the necessity to seek medical care. Medical staff, especially general practitioners, should be properly informed and educated in the early recognition, progressive nature and appropriate treatment of the lesions. Manufacturers should be persuaded to place directions, warnings and first aid information on all cement bags; also, mandatory preventive educational measures for professional cement workers such as the correct use of cement, appropriate protective garments, cleaning and drying of the skin after work, and skin protection are needed.

References

1. Lachapelle JM, Minne GJ. Burns related to the use of cement. Apropos of 4 cases. Ann Dermatol Venereol 1985;112:123-9.
2. Early SH, Simpson RL. Caustic burns from contact with wet cement. JAMA 1985;254:528-9.
3. Peters WJ. Alkali burns from wet cement. Can Med Assoc J 1984;130:902-4.
4. Maciejewska A, Bielichowska-Cybula G. Biological effect of cement dust. Med Pr 1991;42:281-9.
5. Feldberg L, Regan PJ, Roberts AH. Cement burns and their treatment. Burns 1992;18:51-3.
6. Boyce DE, Dickson WA. Wet cement: a poorly recognized cause of full-thickness skin burns. Injury 1993;24:615-7.
7. Morley SE, Humzah D, McGregor JC, Gilbert PM. Cement-related burns. Burns 1996;22:646-7.
8. Hannuksela M, Suhonen R, Karvonen J. Caustic ulcers caused by cement. Br J Dermatol 1976;95: 547-9.
9. Fisher AA. Cement injuries. Part II. Cement burns resulting in necrotic ulcers due to kneeling on wet cement. Cutis 1998;61:121.
10. Birch R, Wilkinson MC, Vijayan KP, Gschmeissner S. Cement burn of the sciatic nerve. J Bone Joint Surg [Br] 1992;74:731-3.
11. Xiao J, Cai BR. Classification and specificity of cement burns. Burns 1995;21:136-8.
12. Spoo J, Elsner P. Cement burns: a review 1960-2000. Contact Dermatitis 2001;45:68-71.
13. Ricketts S, Kimble FW. Chemical injuries: the Tasmanian Burns Unit experience. Aust N Z J Surg 2003;73:45-8.
14. Midelfart A, Hagen YC, Myhre GB. Chemical burns to the eye. Tidsskr Nor Laegeforen 2004;124:49-51.

For skin care, especially in winter time, use Nivea cream and oil.
From the Nivea collection of Zlatko Puntijar (1933)