AN UNUSUAL CASE OF EXTENSIVE SELF-INFLICTED CEMENT BURN

Catalano F.,[1]* Mariano F.,[2] Maina G.,[3] Bianco C.,[3] Nuzzo J.,[4] Stella M.[4]

[1] Plastic Surgery Unit, Department of Surgical Specialties, Messina University Hospital, Messina, Italy
[2] Department of Medicine Area, Nephrology and Dialysis Unit, CTO Hospital, Turin, Italy
[3] Department of Traumatology, Orthopaedic and Occupational Medicine, University of Turin, Turin, Italy
[4] Department of Plastic Surgery, Burns Unit, CTO Hospital, Turin, Italy

SUMMARY. Cement is a fine powder used to bind sand and stones into a matrix of concrete, making up the world's most frequently used building material in the construction industry. First described by Ramazzini in his book "De Morbis Artificia Diatriba" in 1700, the effect of cement on the skin was presumed to be due to contact dermatitis. The first cement burns case was published by Rowe and Williams in 1963. Cement handling has been found to be responsible for many cases of occupational burns (generally full-thickness) usually affecting a limited TBSA, rarely greater than 5%, with localization especially in the lower limbs. We describe an unusual case of a self-inflicted cement burn involving 75% TBSA. A 28-yr-old building worker attempted suicide by jumping into a cement mixer in a truck. Upon arrival at our burn centre, clinical examination revealed extensive burn (75% TBSA - 40% full-thickness) involving face, back, abdomen, upper limbs and circumferentially lower limbs, sparing the hands and feet. The patient was sedated, mechanically ventilated, and subjected to escharotomy of the lower limbs in the emergency room. The following day, the deep burns in the lower limbs were excised down to the fascia and covered with meshed allografts. Owing to probable intestinal and skin absorption of cement, metal toxicity was suspected and dialysis and forced diuresis were therefore initiated on day 3. The patient's clinical conditions gradually worsened and he died on day 13 from the multi-organ failure syndrome.

Keywords: cement, burn, suicide

Introduction

Cement is a fine powder used to bind sand and stones into a matrix of concrete, making the world's most frequently used building material.[1] There are different types of modern cement, but Portland cement is the commonest used worldwide. Various standards, such as European Standard EN197-1,[2] describe the composition of a particular cement. The main components of cement are summarized in *Table I*.

Table I - Cement's main components

Cement	Mass %
Calcium oxide (CaO)	61-67
Silicon dioxide (SiO_2)	19-23
Aluminium oxide (Al_2O_3)	2.5-6
Ferric oxide (Fe_2O_3)	0-6
Sulphate	1.5-4.5

Cement has abrasive and hygroscopic properties and it can induce skin hydration, resulting in dry skin. The thermal damage of cement is due to an exothermic reaction[4] described in *Fig. 1*. Association with calcium oxide

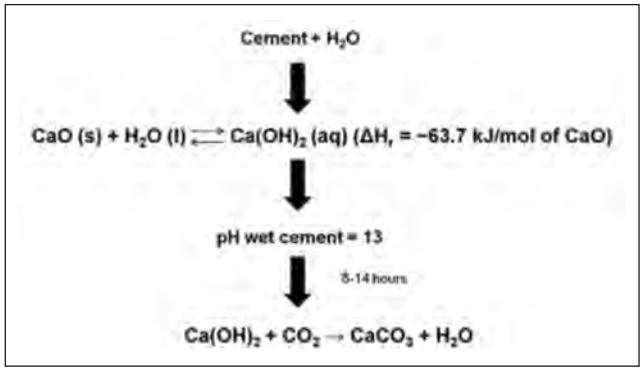

Fig. 1 - Exothermic reaction. CaO: calcium oxide; $Ca(OH)_2$: calcium hydroxide; ΔHr: released thermal energy; $CaCO_3$: calcium carbonate; H_2O: water; CO_2: carbon dioxide.

* Corresponding author: Dr Fabio Catalano, Via Marco Polo 425, 98125 Messina, Italy. Tel.: +39 3805175845; e-mail: fabiocat02@gmail.com

and water causes calcium hydroxide to be produced, its pH changes from 11 to 13, and it becomes more alkalotic for 8-14 h until calcium hydroxide is converted by air into an inert calcium carbonate. Alkaline substances cause tissue damage by dissolving proteins and collagen, dehydrating cells, and saponifying fat.

First described by Ramazzini in his book "De Morbis Artificia Diatriba" in 1700, the effect of cement on the skin was presumed to be due to contact dermatitis. The first cement burn case was published by Rowe and Williams in 1963.[5] In the most recent case series, the average TBSA% in cement burns is lower than 10%. We describe an unusual case of a self-inflicted cement burn involving 75% TBSA.

A 28-yr-old male patient employed as a building worker arrived at our burns centre about 24 h after a self-inflicted cement burn. The patient had jumped into the cement mixer of a truck, and had remained there for about three hours before the truck was unloaded and he was found. Clinical examination showed extensive burns (75% TBSA - 40% full-thickness) involving the face, back, abdomen, upper limbs, and circumferentially the lower limbs, but sparing the hands and feet (*Fig. 2*). The patient was hypothermic (34.6 °C) and an evident grey cement tattoo covered all the burned areas. An escharotomy of the lower limbs was performed in the emergency room on arrival, and the next day excision of the lower extremities was performed down to the fascia of the deep burns. The excised area was then covered with meshed allografts. The allograft take was 80% on day 5.

On day 1, abdomen radiography showed a radiopaque gastric image, indicating probable cement ingestion, while CT imaging revealed right renal effusion.

As metal toxicity was suspected as a result of intestinal and skin absorption of cement, it was decided to evaluate blood and urinary dosages of chromium (Cr) and aluminium (Al), which are the commonest toxic elements in cement. On admission, blood-Al was 478 μg/l and blood-Cr 316 μg/l; on day 3 blood-Al was 36 μg/l, urine-Al 80 μg/l, blood-Cr 206 μg/l, and urine-Cr 188 μg/l. Since hypotension and hypothermia were compensated on day 3, forced diuresis and dialysis were initiated. The post-dialysis values were: blood-Al: 31.2 μg/l, urine-Al: 60 μg/l, blood-Cr: 114.4 μg/l, urine-Cr: 62.9 μg/l. All these levels dropped further following subsequent dialysis.

Fig. 2 - Patient with extensive cement burn; lower limbs involved circumferentially, sparing the patient's feet.

Laboratory tests on admission showed a low white blood cell count of 1300/μl, increasing to a physiological range on day 4. Tests also revealed early impaired hepatic function; altered renal function was evident from day 10.

On day 9, blood culture was positive for *Aeromonas hydrophila* and *Acinetobacter baumannii*.

The patient died on day 13.

Discussion

As described by Xiao and Cai in 1995, cement burns can be divided into three types, depending on the mechanism of injury: abrasion, heat, and blast burn.[6] Abrasion is the most frequent mechanism, usually affecting a limited TBSA, with localization especially in the lower limbs.[7]

Blast burn injuries are caused by the accidental explosive discharge of cement powder from a kiln during manufacture.[8]

The thermal damage caused by cement may be explained by the exothermic reaction described in *Fig. 1*.

In the case reported here, we retrospectively assume that the patient's progressive multi-organ failure was likely to be the result of the cement burn, with toxicity and prolonged hypothermia. As an emergency measure, any soaked clothes must be removed, and the affected areas thoroughly irrigated to eliminate the toxic agent. Some authors[9] recommend the application of a buffered phosphate solution to limit the injury, but the rationale of this practice is debatable as the heat produced by the exothermic reaction of neutralization may increase the damage. The treatment of cement burns varies in the literature but is currently oriented towards early excision and grafting once the diagnosis of full-thickness burn has been made. In this patient, because of the extensive TBSA percentage and the lack of donor sites, an allograft was applied to the lower limbs as a first step in the surgical plan.

The systemic effects of this extensive burn were probably aggravated by prolonged hypothermia. Cardiovascular impairment (peripheral vasoconstriction, loss of plasma to extravascular compartments, cardiac output decrease, and "cold-induced" diuresis), marrow suppression and progressive marrow failure (leukocyte depletion evidenced by low white blood cell count may also be caused by the recruitment of white blood cells to the extensive zone of injury), liver function depression, gastric ulcers, impaired in-

testinal motility, and reduced total body metabolism are all documented[10] in association with hypothermia.

In the clinical setting, acute renal failure is observed in over 40% of patients with accidental hypothermia admitted to an intensive care unit.[11]

To our knowledge, no report has been published on acute cement toxicity, whereas the pathological effects of some metal constituents are well known. We chose to evaluate chromium and aluminium levels in view of the fact that severe acute hexavalent chromium poisoning (chromium VI) can lead to intravascular haemolysis, acute tubular necrosis, and acute renal failure.[12] It can also lead to liver toxicity with derangement of hepatocytes, necrosis, and lymphocytic and histocytic infiltration as well as an increase in Kupffer cells resulting in hepatic failure.[13] Gastrointestinal ulceration[14] and hypoxic changes in the myocardium have also been reported. Aluminium is well known for its neurotoxicity, described as the development of cerebral impairment, starting with speech disturbances, motor apraxia, and twitching, then slowly deteriorating into myoclonic jerks, seizures, and global dementia.[15] The most frequent cases in the literature are chronic intoxications, while acute poisoning is rare.[16] The lethal dosage of blood-Cr has been established as about 2-3 mg/l,[17] while no exact value exists in the literature for lethal blood-Al. Clinical data from case reports indicate levels of 359-1275 μg/l.[16]

Even though in the case presented here pre- and post-dialysis blood and urine concentrations of Al and Cr were lower than lethal levels, we believe that chromium (VI) absorption had a negative effect on early impaired hepatic function, the platelet count,[18] and progressive renal dysfunction.[19]

Renal impairment became evident in the final four days, when blood culture revealed *Aeromonas hydrophila* and *Acinetobacter baumannii* septicaemia. Septicaemia mainly develops in immune-compromised and trauma patients. In all cases reported in the literature, mortality was significantly associated with the severity of the underlying disease.[20,21]

In this case, the early low white blood cell count, probably due to the recruitment of white blood cells to the extensive zone of injury, associated with hypothermia-induced failure and bone marrow haematopoiesis, allowed aggressive bacterial colonization, which played a major role in the multi-organ failure.

Conclusion

To our knowledge, this is the first report of such an extensive burn due to cement. Our aim was to investigate the pathophysiological changes resulting from a lethal cement burn. From a retrospective analysis of the data, we think that the chemical burn associated with prolonged hypothermia sepsis and cement toxicity led to the patient's death, despite appropriate diagnostic and therapeutic planning.

RÉSUMÉ. Le ciment est une poudre fine utilisée pour lier le sable et les pierres dans une matrice de béton qui constitue le matériau de construction le plus utilisé au monde dans l'industrie de la construction. Décrit pour la première fois par Ramazzini dans son livre "De Morbis Artificia Diatriba" en 1700, l'effet du ciment sur la peau était attribué à une dermatite de contact. La première description d'un cas de brûlure due au ciment a été publiée par Rowe et Williams en 1963. La maniement du ciment a été jugé responsable de nombreux cas de brûlures professionnelles (généralement de toute épaisseur) qui intéressent en général une surface corporelle totale (SCT) limitée, rarement plus de 5%, avec une localisation surtout dans les membres inférieurs. Les Auteurs décrivent un cas peu commun de brûlure auto-infligée par ciment dans 75% de la SCT. Un ouvrier de 28 ans a tenté de se suicider en sautant dans la bétonnière d'un camion. À l'arrivée au centre des brûlés, l'examen clinique a révélé des brûlures dans 75% de la SCT (40% de toute épaisseur) qui intéressaient le visage, le dos, l'abdomen, et les membres supérieurs et inférieurs en circonférence, avec exclusion des mains et des pieds. Le patient a été mis sous sédation et ventilation mécanique et soumis à escarrotomie des membres inférieurs dans la salle d'urgence. Le lendemain, les brûlures profondes des membres inférieurs ont été excisées jusqu'au fascia et couvertes d'allogreffes en filet. L'absorption intestinale et cutanée du ciment a suggéré une toxicité métallique et pour cette raison la dialyse et la diurèse forcée ont été effectuées au troisième jour. Les conditions cliniques du patient se sont aggravées progressivement et il est mort le jour 13 à cause du syndrome de défaillance multi-organique.

Mots-clés: ciment, brûlure, suicide

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