Frostbite complicating therapeutic surface cooling after heat stroke

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Dear Editor,

We report the case of an apparently healthy young woman found unconscious in a sauna suffering from heat stroke syndrome (GCS score 3/15; core temperature 41.9°C; BP 80/60, pulse 160/min). She was immediately treated with external surface cooling, initially using towels soaked with ice packs (~20 min) followed by cold wet blankets (~40 min), applied from the lower abdomen to the proximal half of the thighs. No accessory cooling method was used. She was also administered oxygen (FiO_2 100%) and a perfusion of 1,000 ml NaCl at ambient temperature. Within just about 1 h, the patient's core temperature was actively decreased from 41.9°C (tympanic, on-site) to 38°C (rectal, ER) with immediate effects on vital parameters. Nevertheless, the following day, the patient developed within the initial cooling zone progressive skin lesions, evolving from blisters to full-thickness skin and fat necrosis, which required skin grafting 43 days after the incident (Fig. 1a, b). The diagnosis was frostbite secondary to local cooling for heat stroke syndrome treatment.

Heat stroke syndrome is defined as core temperature exceeding 40°C associated with a change in mental status ranging from inappropriate behaviour or impaired judgment to delirium, epilepsy or coma [1–3]. In 20–65% of the cases, an acute circulatory failure (shock) is associated [1–3] as observed in our patient. Treatment consists mainly of therapeutic cooling and cardiovascular

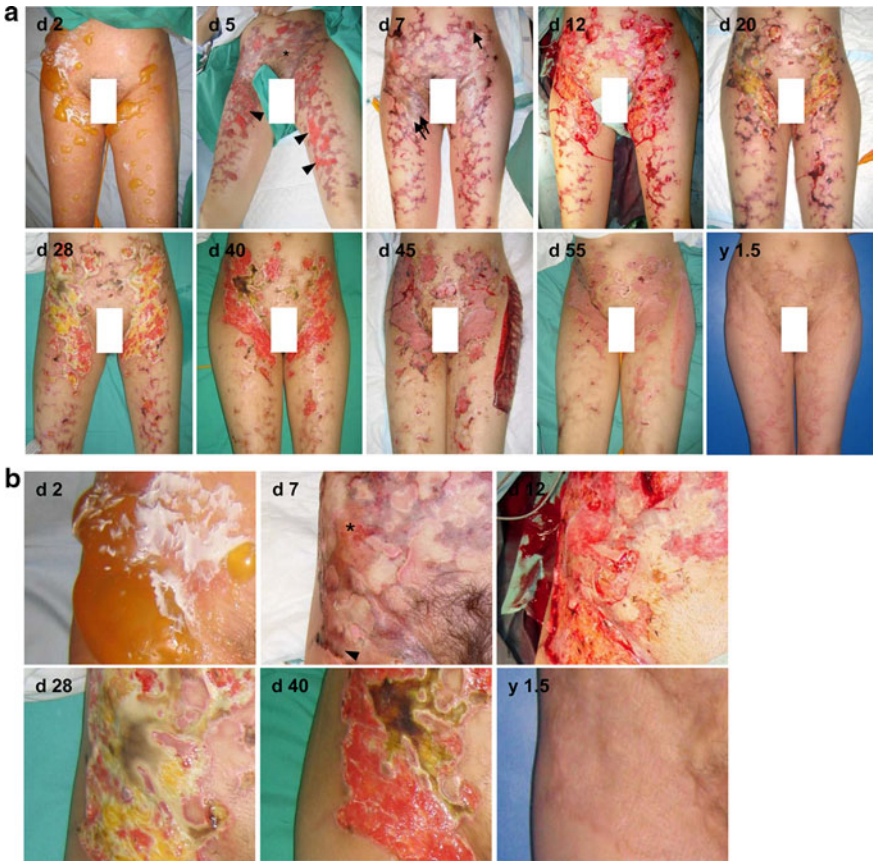

Fig. 1 **a** Blisters extending from the infra-umbilical region to the anterior surface of both thighs, including the inguinal folds, but preserving the pubic region, affecting ~10% of total body surface, the day after injury *(d2)*. Progressive evolution showed an increasing number of superficial (*asterisk*) and deep dermic lesions (*arrowhead*) at day 5 *(d5)*, which again turned into deep dermic (*arrow*) and full thickness lesions (*double arrow*) at day 7 *(d7)*. A first surgical debridement was performed at day 12 *(d12)*, followed by several other debridements due to progressive skin and fat necrosis *(d20)*. Well vascularised wounds with increasing amount of granulation tissue presented from day ~24 on *(d28)* towards day 40 *(d40)*, allowing skin grafting at day 43. First dressing after grafting at day 45 *(d45)* and day 55 *(d55)*, showing a full graft take. Absence of pathological scarring, including hypertrophic or unstable scars, 1.5 years after surgery *(y1.5)*. **b** Detail view of the right inguinal fold showing blisters at day 2 *(d2)* evolving to patchy lesions of superficial (*asterisk*) and deep dermic lesions (*arrowhead*) at day 7 *(d7)*. Following first debridement, underlying dermic and subcutaneous tissue was initially well vascularised *(d12)*. Despite additional debridements, progressive skin and fat necrosis was sparsely riddled with some islands of granulation tissue *(d28)*. Granulation tissue surrounding skin necrosis at day 40 *(d40)*. Fully healed split thickness skin grafts after 1.5 years *(y1.5)*

support. Bouchama et al. [1] reported that core temperature should be lowered below 39.4°C as quickly as possible, but neither the evidence of a specific endpoint temperature for safe cessation of cooling [2] nor the evidence supporting the optimal cooling method are well-established. Cooling may be induced by various procedures, including external surface cooling (ice packs, cold blankets or surface cooling devices), as well as intravascular cooling (ice-cold saline infusion or endovascular cooling catheter) [1]. Yet, although often safe and tolerable, external surface cooling may cause cutaneous vasoconstriction [1, 4], a risk factor that may lead to skin necrosis if used improperly, especially in case of shock. Interestingly, a few cases have been reported, essentially following soft tissue injury treatment [5], but no report following heatstroke cooling was found in the literature.

It may be assumed that the initial cooling by ice packs might have increased vasoconstriction of the cutaneous vessels related to the shock itself, resulting in a microcirculatory breakdown beyond ischemic tolerance and maintained by the application of a cold blanket, causing frostbite. Diagnosis was confirmed by immuno-histochemistry (anti-collagen type IV antibody).

External surface cooling, although recommended in case of heatstroke, must be considered as having a potential risk of tissue injury leaving important sequelae, especially in case of shock. Therefore, we recommend the following: (1) Infusion of cold fluids in all cases. If impossible, any external surface cooling method should be used. Once core temperature is <39°C, (2) direct skin contact with ice should be avoided and, (3) careful skin inspection and local warming undertaken if needed [massage, warm water spray (40°C) or hot air application (45°C)].

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