Third-Degree Chemical Burns from Chlorhexidine Local Antisepsis

Ezequiel Palmanovich MD, Yaron S. Brin MD, Lior Laver MD, Meir Nyska MD and Binyamin Kish MD

Department of Orthopedics, Meir Medical Center, Kfar Saba, affiliated with Sacker Faculty of Medicine, Tel Aviv University, Ramat Aviv, Israel

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BURNS cause coagulative necrosis of the epidermis and underlying tissues. The depth of the injury depends on the temperature to which the skin was exposed and the duration of exposure. Burns are classified into five different causal categories and depths of injury: a) flame, b) hot liquids (scald), c) contact with hot or cold objects, d) chemical exposure, and e) conduction of electricity. Chemicals cause injury by protein destruction, with denaturation, oxidation, formation of protein esters, or tissue desiccation.

Irritant contact dermatitis is the most common type of contact dermatitis. It is responsible for approximately 80% of cases of occupational contact dermatitis and is considered the most frequent cause of hand eczema [1]. Skin burn is the most severe complication of irritant contact dermatitis.

Chlorhexidine is an effective skin preparation agent for surgical interventions. Depending on the concentration, chlorhexidine has bacteriostatic, bactericidal, fungicidal, fungicidal and virucidal properties. However, it has been associated with a number of complications, such as anaphylaxis when used on mucous membranes and dermatitis in 7% of physicians surveyed [2].

In 1992, Watkins and Keogh [3] reported extensive chemical burns in neonates after chlorhexidine was used in an isopropyl solution. It was also associated with bullous corneal keratopathy in a woman following preoperative application to her face. Chemical burn was also reported in a 4 year old boy who underwent general anesthesia for elective orchidopexy; within a few hours, the child developed erythema and blistering around the upper buttocks and natal cleft region. The author recommended saline rinsing after antisepsis [4]. In 1995, contact dermatitis involving pruritic, burning or painful rashes caused by this antiseptic was reported [5]. No report of a deep burn was found in the literature.

In our service, preoperative antisepsis of the skin is performed with chlorhexidine soap (Septal Scrub®, chlorhexidine gluconate 4%, Teva-Israel), which is removed with chlorhexidine alcohol (alcohol chlorhexidine, 0.5% chlorhexidine gluconate in 70% isopropanol). Almost all surgeries involving the extremities in our service are performed with this technique. After this, a single-use tourniquet is applied (HemaClear™, self-contained, sterile, exsanguinating tourniquet, OHK Medical Devices, Israel) [Figure A].

PATIENT DESCRIPTION

A healthy 55 year old woman was admitted with a displaced ankle fracture to our department for operative treatment. The patient is a nurse in the emergency department in our hospital and is frequently exposed to chlorhexidine solution during the course of her work. No antecedents of drug, food or animal allergic reactions were reported by the patient. The surgery was performed using the chlorhexidine antiseptic technique. Approximately 48 to 72 hours after the operation, redness and pain in the posterior part of the knee was documented. After a week of complaints of mild pain, the patient returned to the outpatient clinic. On examination, a circular third-degree burn was discovered in the middle/distal third of the thigh and deep second and third-degree burns were observed in the posterior aspect of the same knee [Figures B & C]. No skin lesion was observed near the operative field. After consultation with a plastic surgeon, conservative treatment was recommended. During follow-up, no knee contracture was observed and the skin was regenerated with a mild scar [Figures D & E].

COMMENT

Irritant contact dermatitis is a localized inflammatory skin response to a wide range of chemical and physical agents. It results from the direct cytotoxic effect of irritants. The clinical manifestations of irritant contact dermatitis range from mild skin dryness and erythema to acute or chronic eczematous dermatitis and even skin necrosis (chemical burn). Developed in the mid-20th century, chlorhexidine has been used as a component of many products and routinely as a preoperative skin antiseptic. Its antimicrobial efficacy is increased when combined with alcohol. Preoperatively, the entire lower extremity is prepared for surgery with this solution. A HemaClear I tourni-
Two main scar zones
4 months after surgery

The tourniquet is routinely applied. In this case, the antiseptic solution pooled distal to the tourniquet and in the posterior aspect of the knee during surgery. It appears to be the cause of the deep second and third-degree chemical burns sustained in the area of the knee. However, the second region of burn was at the site of the pressure area of the tourniquet. The likelihood that the chlorhexidine accumulated under the tourniquet is small due to the localized high pressure of the HemaClear; however, it is possible that chlorhexidine accumulated just distal to the HemaClear tourniquet. Both burn sites were in the posterior area of the leg, indicating the site of accumulation by decubitus of the chlorhexidine.

Chlorhexidine solution can produce second and third-degree burns when used as a preoperative antiseptic solution. The surgeon must be alert and must carefully dry the area where a large quantity of chlorhexidine solution might accumulate.

Corresponding author: Dr. E. Palmanovitch
Dept. of Orthopedics, Meir Medical Center, Kfar Saba 44281, Israel
e-mail: ezepalm@gmail.com

References

Capsule

Cerebrospinal fluid CD19(+) B cell expansion in N-methyl-D-aspartate receptor encephalitis

There is increasing interest in the role of autoantibodies in acquired autoimmune central nervous system disorders. N-methyl-D-aspartate receptor (NMDAR) encephalitis is an autoimmune encephalitis defined by the presence of autoantibodies that bind to the NMDAR. Although there is evidence of NMDAR antibody pathogenicity, it is unclear which treatment results in the best outcome. Dale et al. measured the proportion of B cells in the cerebrospinal fluid of two children with NMDAR encephalitis (a 6 year old male and a 4 year old female), one in the acute phase and one in the relapsing phase. The proportion of CD19(+) B cells in both children was greater than 10%, significantly higher than seen in non-inflammatory neurological disorders (<1%). This finding supports the use of drugs, such as rituximab, that deplete B cells in severe or refractory cases of NMDAR encephalitis, and lends further support to the humoral autoimmune hypothesis in NMDAR encephalitis.

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