Case Series

Use of antimicrobial Dialkyl Carbamoyl Chloride (DACC) surface dressings for the treatment of infected post-surgical complications in neonates with low risk of adverse reactions: case series in the AOU Meyer NICU

Citation: Nicolosi B., Parente E. "Use of antimicrobial Dialkyl Carbamoyl Chloride (DACC) surface dressings for the treatment of infected post-surgical complications in neonates with low risk of adverse reactions: case series in the AOU Meyer NICU" (2023) *infermieristica journal* 2(1): 39-45. DOI: 10.36253/if-2105

Received: April 8, 2023

Revised: April 12, 2023

Just accepted online: April 22, 2023

Published: April 30, 2023

Data Availability Statement: All relevant data are within the paper and its Supporting Information files.

Competing Interests: The Author(s) declare(s) no conflict of interest.

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From the 3rd trimester of pregnancy onwards, the skin presents a structure similar to the definitive one. The maturing process continues until birth and continues rapidly until 2-3 weeks later, unless the skin is exposed to a high-humidity environment or is covered by occlusive materials, which can slow down its maturation[1]. The stratum corneum reaches functional maturity around the 33rd gestational week[2].

The first skin cells are formed from the ectoderm, from which those of the nervous system also derive, in fact because of this relationship, the peripheral sensory function of the skin is understood[2].

The ectodermal cells reach the intermediate cell stage before finally maturing into keratinocytes. Around the 7th week a cell layer (periderm) is present, which disappears around the 24th week, with the formation of scales. The specialised skin cells, melanocytes, Merkel cells and Langerhans cells, also begin to differentiate early, and from the 24th to 25th week, they start their activity[2].

At the 24th week of gestation, newborns have a reduced stratum corneum. The skin is red, wrinkled, translucent, and apparently gelatinous. The subcutaneous tissue is absent, so the dermis lies directly in contact with the bone[3]. Consequently, removal of an adhesive dressing or patch, with removal of the skin, may result in loss of full thickness tissue.

Between the 26th and 29th weeks of gestation, fat begins to be deposited at the subcutaneous level and wrinkles tend to diminish. However, the barrier function of the skin remains poor; in fact, around the 26th week of gestation, up to 110 ml of water can be lost

in 24 hours[3].

The main function of the foetal sebaceous glands is the excretion of sebum, a complex mixture of relatively non-polar lipids. In infants, the sebaceous glands are well formed, hyperplastic and macroscopically visible on certain areas of the body, such as the nose[2]. The surge in their activity during the last trimester of pregnancy leads to the production of a dense, lipid-rich hydrophobic film called caseous varnish. This film is present in varying amounts on the infant's skin surface, both in utero, where it protects the skin from maceration by exposure to the amnion, and at birth. After birth, a proportion of caseous varnish is physiologically reabsorbed, while it is necessary to remove the remaining part with oily substances, otherwise the lipids could go rancid and cause damage to the skin. Its removal must be partial, as it plays an important role in preventing water loss, thermoregulation and innate immunity[2]. The latter is indispensable in protecting the infant from sudden exposure to micro-organisms, toxins, oxidative stress, fluctuating temperatures and humidity, and is made possible by its content of LL-37 peptide and lysozyme, i.e. two antimicrobial substances that act synergistically, lactoferrin, alpha-defensin and other antimicrobial peptides[2]. The caseous varnish, therefore, acts as a natural moisturiser and protects the outer layers of the skin to allow normal adaptation in the new aerobic environment[2]. After removal, the skin is reddened and slightly cyanotic at the extremities. Capillary dilatation is frequently present at the eyelids, forehead and nape of the neck, but these regress spontaneously.

The combination of an evolutionarily and functionally immature barrier, the absence or reduced production of caseous varnish, the large body surface area in relation to mass and a still-deficient immune system exposes newborns to a significant risk of infections and skin lesions, particularly those born with a gestational age of less than 33 weeks[2]. A further consideration resulting from the skin characteristics of the preterm infant is the increased risk of percutaneous toxicity from topical drugs, cleansers and even emollients[2].

At 30 weeks, the subcutaneous tissue is evident and the stratum corneum has 2-3 thick cell layers, compared to 40 weeks, when it is 30 layers thick. Functional maturation of the skin occurs at 33 weeks, with the epidermis completely keratinised and the dermo-epidermal junction more resistant, although it remains fragile and easily damaged[3].

By the 36th week, the skin is structurally similar to that of the adult, although the epidermis and dermis are up to 60% thicker than the mature structure[3].

In a preterm infant, the epidermis is 55% thinner than in a full-term baby. The stratum corneum has the thickness of one cell, compared to 15 cells in the stratum corneum of a term baby. The immaturity of the stratum corneum of preterm infants leads to increased permeability, temperature changes, water loss, electrolyte imbalances and risk of infection. They also show an increase in skin cytokines compared to term infants, probably due to stress. In contrast, the stratum corneum of term infants has properties almost similar to those of adults. Preterm infants reach this level of maturation by 2-9 weeks of life[1]. In fact, there is evidence that, compared with gestational age, the skin develops rapidly within 2-3 weeks of birth and matures to the characteristics of that of the full-term infant[4].

Moisture in the environment and the maintenance of hydration at the stratum corneum are necessary for the normal desquamation process, as its interruption increases skin vulnerability, especially in preterm infants[5]. Physiologically, the epidermis and stratum corneum remain in equilibrium precisely because of the property of renewability, which reflects the distinct but closely related processes of hornification and desquamation[5]. Indeed, life in a dry environment requires protection from the constant dehydrating effects of exposure to air[5].

The formation of a barrier against trans-epidermal water loss is a direct function of gestational age: trans-epidermal water loss decreases dramatically with approaching term. This fundamental and vital postnatal function resides almost entirely in the 20 μm of the stratum corneum[5].

Another fundamental factor in the health of the skin of the preterm infant is the skin pH. The latter is essentially neutral, but rapidly develops an acid mantle through mechanisms distinct from bacterial colonisation: the development of skin acidity begins within the first 16 hours of life and is of particular importance, as it allows the inhibition of the proliferation of pathogenic bacteria, promoting instead colonisation by commensal micro-organisms[6]. The functions performed by the acid mantle, therefore, include antimicrobial defence and maintaining the integrity of the epidermal barrier[6].

Pre-term infants in particular, due to their inability to self-regulate temperature, display an additional aspect of skin vulnerability to the external environment. Central control of body temperature requires the activation of mechanisms, such as exocrine sweating and peripheral vasodilation/vasoconstriction, that are under the control of the autonomic nervous system. These functions are generally not adequately active at birth. This is

why newborns may have vasoconstriction of the extremities (acrocyanosis), large variations in red blood cell content (haematocrit) and blood volume. Peripheral cooling causes an increase in blood viscosity and a reduction in blood flow. After birth there is considerable reorganisation of the cutaneous vascular bed, with development of a sub-papillary plexus in the first 3 months of life[5].

Brown fat differentiates and deposits between the 26th and 29th week and skin folds slowly disappear, resulting in further difficulty in maintaining body temperature and acceptable glucose levels[3].

The anchoring structures of the epidermal cell (desmosomes, anchoring filaments, and haemidesmosomes) are smaller and less numerous.

All these differences result in a more fragile skin and greater skin permeability (increased trans-epidermal fluid loss, electrolyte imbalance, increased heat evaporation and increased absorption of locally applied products), especially in the preterm infant.

Wound healing in childhood occurs according to the four physiological phases: coagulation, inflammation, proliferation and maturation[7]. As has already been described, it is clear that there are intrinsic differences in the neonate, which influence wound healing and require special considerations, both for dressing choice and continuation of treatment[8].

Most of the evidence on the application of commercially available advanced dressings refers to efficacy compared with the adult population, so that in most cases, paediatric wound specialists have to adapt these products for paediatric use[9]. This inevitably also affects the availability of inadequately sized dressings for neonates especially[10].

The problem just analysed becomes even more impactful in the management of neonatal wounds when considering the formulation and contents of many advanced dressings. Indeed, these lack the evidence to guarantee their safe application in the neonatal, preterm and term population[9,10].

In the case of the treatment of infections, a further critical issue arises, since antibacterial substances may be responsible for allergic reactions, the emergence of resistance and toxicity.

The prevention or treatment of infection in neonatal wounds thus becomes a significant challenge for all specialists in the field, especially when one considers that in children under 10 years of age, 67% of category 3 pressure ulcers are critically colonised or infected; of these, from a microbiological point of view, 92% present a polymicrobial profile[12]. The reasons for this

Tab. 1 - Infant and adult skin: similarities and differences[11]

Structural differences	Infant skin	Adult skin
Epidermis		
Corneocytes	Smaller	Larger
Granular cells	Smaller	Larger
Stratum corneum and epidermis	Thinner	Thicker
Microrelief lines	More dense	Less dense
Depth of surface glyphics	Similar to adult	—
Facultative pigmentation (melanin)	Less	More
Dermis		
Dermal papillae (density, size, and morphology)	More homogeneous	Less homogeneous
Distinct papillary-to-reticular dermis transition	Absent	Present
Compositional differences		
Epidermis		
Natural moisturizing factor concentration	Lower	Higher
pH	Higher (newborn only)	Lower
Sebum	Lower (7–12 month-old infant)	Higher
Stratum corneum water content	Higher	Lower
Dermis		
Collagen fiber density	Lower	Higher (young adult)
Functional differences		
Rate of water absorption	Higher	Lower
Rate of water desorption	Higher	Lower
Skin barrier function	Competent	Competent
Transepidermal water loss	Higher	Lower

greater susceptibility to infection are multifactorial, for example, in the infant skin barrier, compared to that of adults, there is a high percentage of immature neutrophils, which have a compromised adhesion and phagocytosis function and are consequently more prone to develop bacterial infections[13]. Overall, there are multiple causes of increased susceptibility to infection of skin lesions in newborns; these include skin thickness, sparse dermal capillary bundles, physiological oedema capable of interfering with the transport of oxygen on the skin surface, huge critical colonisation at skin level, systemic immune depletion, salivary, faecal urinary incontinence, the small size of various parts of the body facilitating self-on-self pressure ulcers as well as skin lacerations, patients with the greatest number of devices per body surface area available when admitted to critical intensive care areas[14].

Antimicrobial dressings with a dialkylcarbamoylchloride (DACC) surface

Advanced dressings that sequester microorganisms, i.e. those with a hydrophobic, bacterial-binding surface (DACC), reduce the risk of clinical infection, without exposure to potentially sensitising, toxic or resistance-developing substances[15]. This technology is characterised by a fabric support treated with a fatty acid derivative (DACC - Dialkylcarbamoylchloride) that makes it hydrophobic[14]. Applied directly to the wound bed, it captures bacteria and fungi through hydrophobic interaction. Pathogens are irreversibly bound and removed at each dressing change[14]. The high antibacterial activity of the DACC-coated dressing occurs due to its irreversible binding and inhibition of the growth of bound bacteria[15].

The main antimicrobial dressings contain agents such as silver, iodopovidone and biguanides, which may have bactericidal or bacteriostatic action depending on the concentration and type of molecule. Today consensus and major guidelines discourage the prolonged use of antimicrobial dressings, which certainly should be used, but at certain stages of treatment and under specific conditions[16]. Their inappropriate application may expose them to possible antibacterial resistance.

At this point, effective alternative methods for the management of infected wounds become necessary to address the emergence of antimicrobial resistance and thus limit its spread.

Alternatives indicated by a World Union of Wound Healing Societies document[17], suggest that to reduce the possibility of the development of resistance to antimicrobial agents, the use of products with physical and mechanical action to remove bacteria is indicated. Indeed, these dressings do not release any antimicrobial agents onto the wound bed, thus preventing the risk of bacterial resistance and allergies, and act by retaining and sequestering exudate and bacteria, which might otherwise return to the wound bed.

Bacterial uptake dressings, therefore, are bacteriostatic dressings and not bactericidal; this 'passive' control of the bacterial load prevents the breakdown of the bacterial cell wall and the consequent release of bacterial endotoxins, which worsen the inflammatory state and prevent wound healing[15].

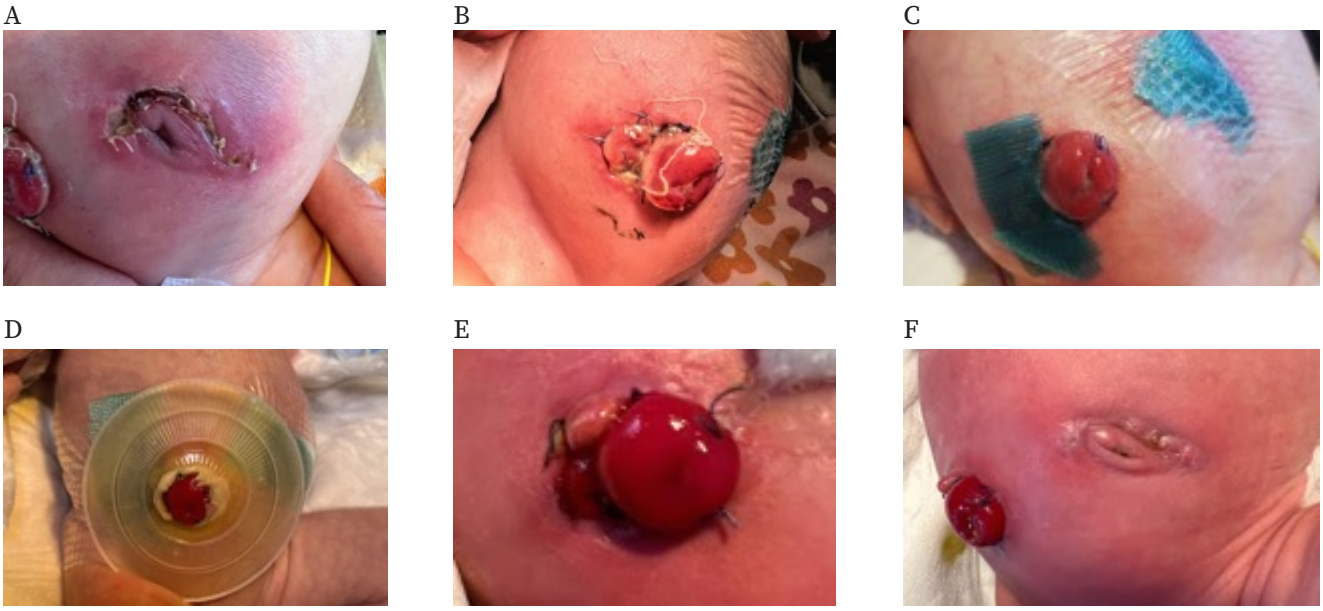
Thanks to its purely physical principle, it also does not release any potentially harmful antimicrobial agents, which could create a risk of bacterial resistance and allergies[18].

Series of cases

Figures 1- below show cases of complex neonatal wounds treated with bacterial-capturing DACC dressings with a hydrophobic, bacteria-binding surface.

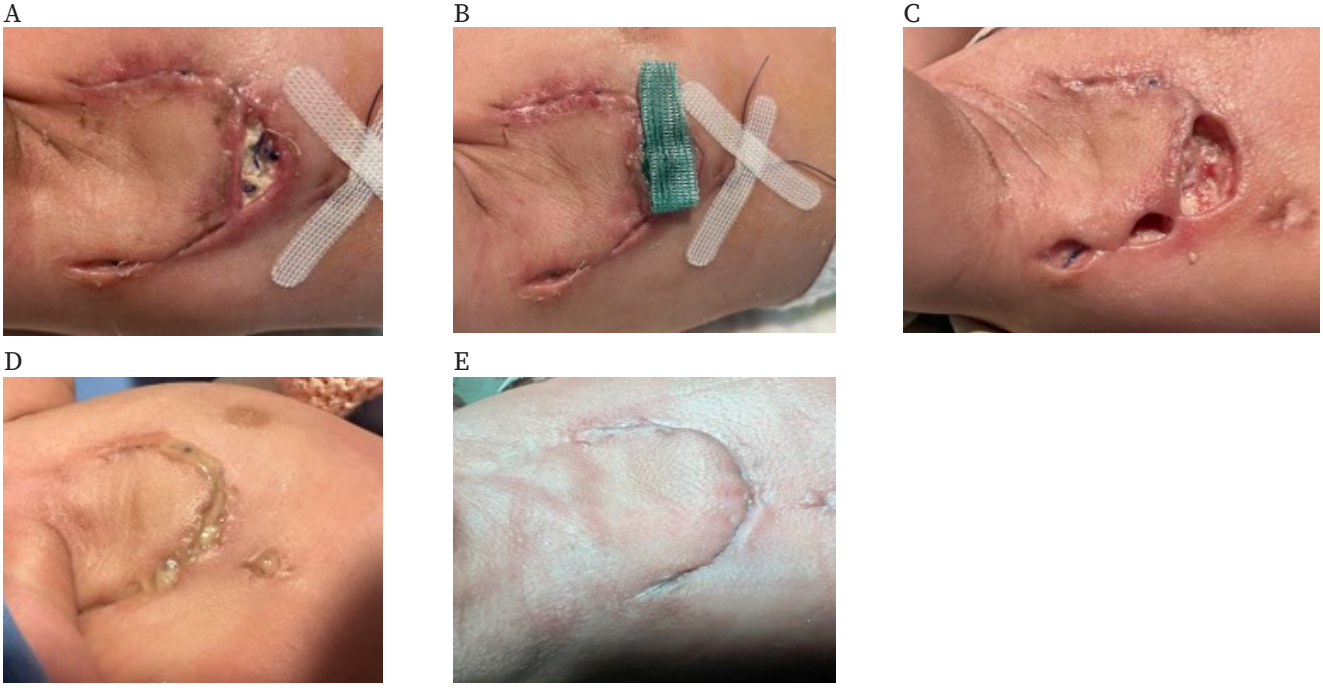
A) Complicated surgical outcome, with topical signs of infection, in 38 week-old infant with NEC. B) Peristomal lesion with dehiscence and signs of local infection. C) Bacterial gel dressing replaced every 48 hours for 10 days. D) Ostomy appliance with underlying dressing. E, F) Resolution of infection and dehiscence, optimal healing.

Figure 1: case of post-operative complication from ostomy placement.



A) Complicated surgical outcome, with topical signs of infection, in 40 week-old infant with tetralogy of Fallot and oesophageal atresia. B) Bacterial gel dressing replaced every 48 hours for 5 days. C) Fundus cleansed and ready for decisive treatment. D) Application of platelet-rich plasma. E) Resolution of dehiscence and optimal healing.

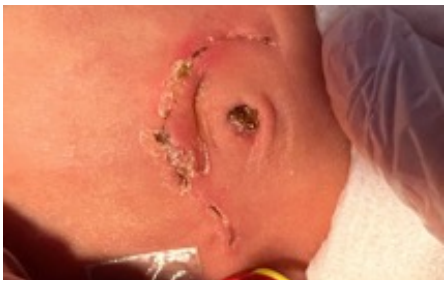
Figure 2: case of post-operative complication of oesophageal atresia surgery



Notes: A) Complicated surgical outcome, with topical signs of infection, in 37 week-old infant with malrotation. B) Bacterial gel dressing replaced every 48 hours for 6 days. C) Resolution of infection and optimal healing.

Figure 3: A case of post-operative complication of intestinal recanalization surgery.

A



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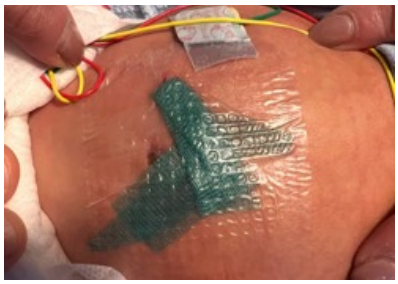

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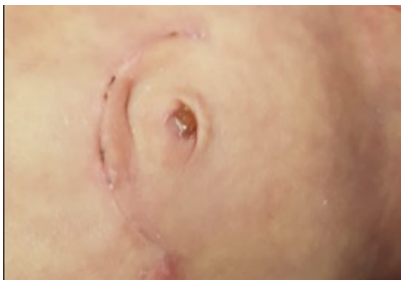

Conclusions

It may be concluded that the bacterial uptake dressing coated with DACC plays an important role in controlling the bacterial load of a wound, reducing the overall demand for antibiotics and without the use of antimicrobial substances. These characteristics do not lead to any known contraindications for use. Applications can be repeated without time limit and can be used on any type of patient, including newborns.

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