Moisture-associated skin damage and its management in neonatal and infant populations: a retrospective study in Italy

Objective: Moisture-associated skin damage (MASD) is a significant concern in the paediatric population, particularly among neonates, who exhibit the highest incidence due to their highly sensitive and fragile skin. The aim of the study was to identify the most effective treatment.

Method: A retrospective observational study was conducted at Meyer Children's Hospital IRCCS, Florence, Italy. Data from infants admitted to the neonatal intensive care unit of the hospital were collected from electronic health records. Demographic, clinical and nursing data were analysed to identify the key clinical features of MASD lesions, including their severity, healing time, and the relationship between MASD lesions and treatment approaches.

Results: The cohort comprised 102 infants (mean age 2.92 months). Incontinence-associated dermatitis (IAD) accounted for the majority (78.4%) of MASD lesions. Among patients with IAD, most exhibited persistent redness (26.3%) or skin loss (47.5%) without clinical signs of infection. In contrast, peristomal MASD cases constituted 21.6% of the total, with the majority classified according to the Study on Peristomal Skin Alterations (SACS) 2.0 as L2 erosive lesions (63.6%), predominantly occurring in quadrants 2, 3 or 5. A novel treatment (Vulnamin; Professional Dietetics S.p.A., Italy) was used to manage MASD lesions in 64.7% of patients. This innovative treatment significantly ($p<0.001$) reduced healing time by an average of 2.4 days and decreased the odds of relapse by 5.3 times compared with traditional treatments.

Conclusion: The findings of this study showed that use of effective MASD therapies can reduce healing time and relapse rate, leading to decreased nursing hours and associated costs.

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moisture-associated skin damage • neonatal intensive care unit • paediatric • retrospective study • wound • wound care • wound dressing • wound healing

Neonatal skin is a critical barrier, protecting newborns from the external environment. Although it shares similarities with adult skin, its fragility and immaturity make it uniquely distinct. Full-term neonatal skin consists of the primary layers of the epidermis and dermis; however, it features a reduced number of stratum corneum layers (2–3 compared to 20–30 layers in adults) and weaker cohesion between the epidermis and dermis. These characteristics make it particularly vulnerable to mechanical trauma, chemical irritation and infection.[1–3] In preterm neonates, this immaturity is even more pronounced, with a severely compromised skin barrier function that persists until around the 34th week of gestational age.[4]

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The skin barrier develops progressively after birth, with a gradual increase in the production of epidermal lipids and natural moisturising factor, which are essential elements for maintaining hydration and barrier function. Despite this maturation process, during the first months of life, the newborn is at high risk of transepidermal water loss (TEWL), leading to dehydration and increased skin permeability.[1,5] Due to these intrinsic fragility characteristics of neonatal skin, maintaining the skin microenvironment is essential both for preventing and treating skin injuries. The skin microclimate, which includes temperature, humidity and airflow near the skin surface, directly influences the barrier function. An inadequate microclimate can alter TEWL, increase the risk of irritation and impair wound healing.[6] Interventions, such as the use of advanced dressings, which promote a stable and controlled microclimate, are essential for improving clinical outcomes in neonates with skin wounds.[7]

The most common skin injuries in neonates include pressure injuries (PIs), medical device-related pressure injuries (MDRPIs) and moisture-associated skin damage (MASD). PIs result from prolonged immobilisation and compression of soft tissues, while MDRPIs are mainly caused by medical devices used in intensive care, which can exert prolonged localised pressure on the fragile skin of the newborn.[5,8]

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MASD incidence in the paediatric population is between 4–37%, with the highest incidence reported in neonates.[9] MASD refers to the skin damage caused by body fluids in direct contact with the skin and includes four prevalent forms:[10–12]

- Incontinence-associated dermatitis (IAD) caused by liquid stool or urine
- Intertriginous dermatitis resulting from perspiration
- Periwound moisture-associated dermatitis caused by wound exudate
- Peristomal moisture-associated dermatitis due to urinary or faecal effluent.[10]

MASD is classified by the current version (11th revision) of the International Classification of Diseases (ICD)[13] under the term 'Irritant contact dermatitis due to friction, sweating or contact with body fluids' (EK02.2) and includes 'Intertriginous dermatitis due to friction, sweating or contact with body fluids' (EK02.20) as well as 'Irritant contact dermatitis due to saliva' (EK02.21), 'Irritant contact dermatitis due to incontinence' (EK02.22), 'Irritant contact dermatitis due to stoma [peristomal MASD (pMASD)] or fistula' (EK02.23), and 'Irritant contact dermatitis due to skin contact with prostheses or surgical appliances' (EK02.24). IAD is firmly established in paediatrics and is coded by the ICD as 'Primary irritant napkin dermatitis' (EH40.10).[14]

The prolonged exposure of the skin to moisture (e.g., urine, faeces, wound secretions or perspiration) damages the skin's outermost layer (epidermis) and results in mechanical and/or chemical irritation,[15] making the skin more susceptible to friction or shear damage[16] and pathogens.[17] However, skin exposure to moisture is not the sole cause of the inflammatory reaction—other pathophysiological factors are relevant for MASD promotion[10,18] and can be synergic.[19] Skin cleansing procedures and products, mechanical factors, indirect factors (e.g., care dependency, immobility, obesity, etc.) and comorbidities are also important contributing factors to the development of MASD.[15] The initial clinical manifestations of MASD include inflammatory erythema, eczema, maceration and pruritus.[20–22]

Currently, the management of MASD in paediatric patients includes emollients and zinc creams (mainly for IAD), absorptive barriers/dressings, hydrocolloid pastes or foam dressings, and topical antibiotics.[9,23–25] However, most of the products for MASD management are predominantly marketed to adult patients and have not been evaluated in the neonatal and paediatric population.[23] Even if the fundamentals of wound care in the paediatric population are similar to those applicable to adults, attention should be paid to some concerns typical of the paediatric population.[26] An impaired epidermal barrier and the immaturity of the immune system are critical for thermoregulation and risk for infection.[27] Moreover, the application of products and dressings can be challenging due to the site of application (e.g., frequent diaper changes) or to the hospitalisation conditions, especially for neonates housed in heated and humidified isolettes, at high risk of stress or chilling. Traditional application of emollients and topical antibiotics is not recommended due to the increased risk of sensitisation and development of resistance, as well as the associated bacterial and fungal overgrowth.[9] The treatment of MASD-related injuries, both for paediatric and adult populations, requires significant resources in terms of nursing hours and product costs, and is challenging for healthcare professionals.[28]

In this observational study, the aim was to identify the most effective MASD treatment by assessing its ability to reduce healing time and minimise relapses.

Method

Design, setting and participants

This retrospective observational study included all infants aged 0–364 days who were hospitalised for various reasons between January and December 2023 in the neonatal intensive care unit (NICU) of Meyer Children's Hospital IRCCS, Florence, Italy, and who experienced a MASD event.

All NICU electronic health records (EHRs) were examined for demographic, clinical and nursing data, including MASD diagnosis and therapy.

Study data

The following variables were collected and analysed:

- Demographic characteristics: age, sex, weight and gestational age
- Clinical data: MASD category according to the Ghent Global IAD Categorisation Tool (GLOBIAD)[29] and the Study on Peristomal Skin Alterations (SACS) 2.0[30] tools and its prevalent form, hospitalisation diagnosis, pharmacological treatments and nutrition (e.g., enteral)
- Nursing data: diaper (or other stool/urine collection system), assisted ventilation, therapy for MASD, healing time and relapses.

Statistical analysis

Data were analysed using both descriptive and inferential statistical methods. Descriptive statistics were used to summarise demographic, clinical and nursing data, with results reported as mean±standard deviation (SD) or median and interquartile range (IQR), depending on data distribution. Categorical variables were presented as frequencies and percentages. For inferential statistics, data were analysed using analysis of variance (ANOVA) or Student's t-test for continuous variables, and Chi-squared test for categorical variables. Odds ratios with 95% confidence intervals (CI) were used to compare probabilities of outcome occurrence between the two groups. To identify potential factors related to patients' demographics, which may affect the relapse rate, a logistic regression model was used.

The statistical testing was conducted at the two-sided α=0.05 level and the 95% CI was computed. Data analyses were performed using IBM SPSS Statistics Version 29 (IBM Corp., US).

Table 1. Hospitalisation diagnosis

Diagnosis	Total patients (n=102)		Patients treated with standard of care (n=36)		Patients treated with Vulnamin spray (n=66)	
	n	%	n	%	n	%
Respiratory tract infections	38	37.3	14	38.9	24	36.4
Recanalisation	30	29.4	11	30.6	19	28.8
Metabolic	8	7.8	2	5.6	6	9.1
Short bowel syndrome	6	5.9	4	11.1	2	3.0
Bowel obstruction	5	4.9	2	5.6	3	4.5
Intestinal atresia	5	4.9	2	5.6	3	4.5
Necrotising enterocolitis	3	2.9	1	2.8	2	3.0
Volvulus	2	2.0	0	0.0	2	3.0
Sepsis	1	1.0	0	0.0	1	1.5
Cerebral hypoxia	1	1.0	0	0.0	1	1.5
Cancer	1	1.0	0	0.0	1	1.5
Inguinal hernia	1	1.0	0	0.0	1	1.5
Peritonitis	1	1.0	0	0.0	1	1.5

Ethical considerations

Ethical approval for data collection was granted by the Pediatric Regional Ethical Committee of Tuscany, Italy (Prot. 168/2024, approved on 23 September 2024). Data were collected only for patients whose written informed consent had been obtained from either parents or a legal guardian. Parents or guardians were contacted by telephone to provide them with detailed information about the study, including its purpose and the use of anonymised clinical data documented in the patients' EHRs. If contact was unsuccessful after three attempts over a three-month period, the patient was excluded from the study. Upon successful contact, the informed consent form was sent via email to be completed and then returned to the investigators.

Results

Demographic characteristics of the sample

The screening process included 111 patients whose parents/legal guardians were contacted by the research team via telephone. Of these, nine patients were excluded due to unsuccessful contact attempts. The final sample consisted of 102 infants, of whom 53 (52.0%) were male, with a mean age of 1.77±1.25 months, and a mean birth weight of 3.96±1.22kg. The mean gestational age was 36.1±2.3 weeks, with 69 (67.6%) patients having a gestational age lower than the minimum normal pregnancy duration (<38 weeks). All of the patients were admitted to the NICU of the hospital for a variety of reasons (Table 1).

The demographic characteristics are reported in Table 2 for the total patient population, as well as for subgroups based on the most frequently received treatment for MASD lesions (i.e., standard of care (SoC; that is, treatments traditionally suggested as IAD prevention and treatment strategies[9,28]) and Vulnamin spray (Professional Dietetics S.p.A., Italy) treatment).

Clinical characteristics of the patient population

In the overall group, skin lesions categorised as MASD during hospitalisation were attributed to IAD in 80 (78.4%) cases and to pMASD in 22 (21.6%) cases. According to GLOBIAD scoring, among the patients with IAD, most presented persistent redness (26.3%) or skin loss (47.5%) without clinical signs of infection, while skin redness or loss with clinical signs of infection was observed in 26.3% of cases (Table 3). According to SACS 2.0 scoring, pMASD lesions were L1 erythematous lesions (36.3%) or L2 erosive lesions (63.7%) occurring in quadrants 2 and 3 or 5 (Table 3).

The pharmacological treatments consisted of antibiotics, vitamins, electrolytes, antireflux agents, cortisone and specific therapies (for example, for neurological or cardiological diseases), administered either alone or in combination to address the underlying medical condition. The predominant treatments were vitamins (91.1%), antibiotics (71.5%) and electrolytes (69.6%).

Most of the patients (48.0%) were on a combination of enteral, parenteral or suction nutrition, with enteral nutrition being the most common (61.7%), followed by suction (53.9%) and parenteral nutrition (45.1%), as shown in Table 3.

All IAD cases were associated with the use of diapers,

Table 2. Demographic and baseline characteristics

	Total patients (n=102)		Patients treated with standard of care (n=36)		Patients treated with Vulnamin spray (n=66)		p-value
	n	%	n	%	n	%	
Sex							
Male	53	52	17	47.2	36	54.5	0.388*
Female	49	48	19	52.8	30	45.5	
	Mean±SD	**Range**	**Mean±SD**	**Range**	**Mean±SD**	**Range**	
Age, years	1.77±1.25	0.2–6	1.84±1.36	0.4–6	1.73±1.19	0.2–6	0.679†
Weight, kg	3.96±1.22	1.98–8.97	4.14±1.21	2.30–8.30	3.86±1.22	1.98–8.97	0.280‡
GA, weeks	36.1±2.3	28–40	36.2±2.2	28–39	36.1±2.3	28–40	0.807†

*Chi-squared test; †Student's t-test; ‡Analysis of variance; GA—gestational age; SD—standard deviation

Table 3. Clinical data

	Total patients (n=102)		Patients treated with standard of care (n=36)		Patients treated with Vulnamin spray (n=66)	
	n	%	n	%	n	%
Moisture-associated skin damage						
IAD	80	78.4	27	75.0	53	80.3
pMASD	22	21.6	9	25.0	13	19.7
GLOBIAD score[28]						
1A	21	26.3	9	33.3	12	22.6
1B	7	8.8	3	11.1	4	7.5
2A	38	47.5	9	33.3	29	54.7
2B	14	17.5	6	22.2	8	15.1
SACS 2.0 score[29]						
T2/T3-L1	1	4.5	1	11.1	0	0.0
T2/T3-L2	4	18.2	1	11.1	3	23.1
T5-L1	7	31.8	3	33.3	4	30.8
T5-L2	10	45.5	4	44.4	6	46.2
Pharmacological treatments						
Antibiotic, vitamins, electrolytes	36	35.3	15	41.7	21	31.8
Antibiotic, vitamins	18	17.6	2	5.6	16	24.2
Antibiotic, vitamins, antireflux therapy, electrolytes	13	12.7	6	16.7	7	10.6
Vitamins, electrolytes	12	11.8	5	13.9	7	10.6
Vitamins, specific therapy	9	8.8	3	8.3	6	9.1
Vitamins, antireflux therapy, electrolytes	5	4.9	2	5.6	3	4.5
Antibiotic, electrolytes	5	4.9	2	5.6	3	4.5
Specific therapy	2	2.0	1	2.8	1	1.5
Specific therapy, cortisone	1	1.0	0	0.0	1	1.5
Antibiotic	1	1.0	0	0.0	1	1.5
Nutrition						
Enteral	25	24.5	7	19.4	18	27.3
Parenteral and suction	24	23.5	15	41.7	9	13.6
Enteral and suction	19	18.6	7	19.4	12	18.2
Parenteral and enteral	19	18.6	3	8.3	16	24.2
Suction	12	11.8	4	11.1	8	12.1
Parenteral	2	2.0	0	0.0	2	3.0
Parenteral and oral	1	1.0	0	0.0	1	1.5

GLOBIAD scoring: 1A Persistent redness without clinical signs of infection; 1B Persistent redness with clinical signs of infection; 2A Skin loss without clinical signs of infection; 2B Skin loss with clinical signs of infection

SACS 2.0 scoring: T is/are the quadrant(s) affected by the lesion; L1 Erythematous lesion (peristomal erythema without loss of substance); L2 Erosive lesion with loss of substance as far as and not beyond the basal membrane

IAD—incontinence-associated dermatitis; pMASD—peristomal moisture-associated skin damage

whereas other stool/urine collection systems were linked to pMASD cases.

Relationship between MASD lesions and their treatment

The study examined the treatments for both IAD and pMASD, and their relationship with the healing time (HT) and relapse rate (RR). The analysis identified two categories of treatments with significant differences in both HT and RR.

A SoC approach and/or therapy with a class IIb medical device Vulnamin spray (National Classification of Medical Devices classification M040499) were used. Vulnamin spray therapy (VSP) was used in 64.7% of the patients, while the remaining 35.3% were treated with creams and lotions (SoC), which included formulations with a skin barrier protective effect (58.3%) and other properties (41.7%), as shown in Table 4. VSP was used to treat 66.2% of the patients with IAD and 59.1% of the patients with pMASD, while the remaining patients were treated with SoC.

Table 4. Standard of care

Treatment	n	%	Mechanism of action
Cholestyramine	3	8.3	Chelation
10% zinc oxide	2	5.6	Skin barrier
30% zinc oxide	2	5.6	Skin barrier
Eosin	4	11.1	Astringent
0.2% sodium hyaluronate	3	8.3	Skin barrier
Eosin and 10% zinc oxide	2	5.6	Skin barrier
Antimycotic	7	19.4	Antifungal
Honey	6	16.7	Skin barrier
Liquid skin protective emulsion	7	19.4	Skin barrier

In the entire cohort, healing time was significantly shorter (–2.4 days) in VSP-treated patients (4.2±1.3 days) compared with SoC-treated patients (6.6±1.9 days), with a statistically significant (p<0.001) difference (Fig 1a, Table 5). Similar results were observed in the cohort of patients with a gestational age <38 weeks (Fig 1b, Table 5), where healing time was 4.2±1.3 days for VSP-treated patients and 6.3±1.9 days for SoC-treated patients, with a statistically significant mean difference of 2.1 days (p<0.001). All VSP-treated patients were discharged from the hospital with MASD lesions fully resolved, while five SoC-treated patients (including three with a gestational age of <38 weeks) were discharged with unresolved MASD lesions. The healing time for IAD-related skin lesions (Fig 1c, Table 5) was 4.2±1.3 days in VSP-treated patients and 6.6±2.0 days in SoC-treated patients (p<0.001). Similarly, for pMASD lesions (Fig 1d, Table 5), the healing time was 4.5±1.4 days in VSP-treated patients and 6.4±1.7 days in SoC-treated patients (p<0.01). The healing time was not influenced (p>0.05, generalised linear model) by the patients' weight in any of the cohorts.

In the entire cohort, the relapse rate (Tables 5, 6) was significantly lower (p<0.01) in the VSP-treated cohort (12.1%) compared with the SoC-treated cohort (42.4%). Logistic regression analysis showed that the odds of relapse for SoC-treated patients were 5.3-times higher (95% CI: 1.94, 14.69) than for VSP-treated patients (p<0.01). This corresponds to an 81.3% lower risk of relapse (95% CI: 0.07, 0.52) in VSP-treated patients compared with SoC-treated patients.

In the cohort of patients with a gestational age <38 weeks, the relapse rate was 13.3% in the VSP-treated patients and 50.0% in the SoC-treated patients. In this cohort, the odds of relapse for SoC-treated patients were 6.5-times higher (95% CI: 1.96, 21.56) than for the VSP-treated patients (p<0.01), corresponding to an 84.6% lower risk of relapse (95% CI: 0.05, 0.51) in VSP-treated patients compared with SoC-treated patients.

In patients with IAD, the relapse rate was significantly lower (p<0.01) in VSP-treated patients (9.4%) compared with SoC-treated patients (41.7%). The odds of relapse for SoC-treated patients were 6.9 times higher (95% CI: 2.01, 23.40) than for VSP-treated patients (p<0.01), corresponding to an 85.4% lower risk of relapse (95% CI: 0.04, 0.50) in VSP-treated patients.

In patients with pMASD, the relapse rate was 23.1% in VSP-treated patients and 44.4% in SoC-treated patients. The odds of relapse for SoC-treated patients were 2.7 times higher (95% CI: 0.42, 16.83) than for VSP-treated patients (p>0.05), corresponding to a 62.5% lower risk of relapse (95% CI: 0.06, 2.37) in VSP-treated patients compared with SoC-treated patients.

Table 5. Healing time and odds of relapse

Cohort	n	Healing time VSP versus SoC, days	Relapse rate, %	Odds ratio
Entire cohort	99	4.2 versus 6.6 (–2.4)	<81.3	5.3 times higher*
GA <38 weeks	67	4.2 versus 6.3 (–2.1)	<84.6	6.5 times higher*
IAD	80	4.2 versus 6.6 (–2.4)	<85.4	6.9 times higher*
pMASD	22	4.5 versus 6.4 (–1.9)	<62.5	2.7 times higher*

*Higher in SoC group compared to VSP group; GA—gestational age; IAD—incontinence-associated dermatitis; pMASD—peristomal moisture-associated skin damage; SoC—standard of care; VSP—Vulnamin spray therapy

Discussion

The study aimed to analyse the demographic characteristics, clinical data and nursing information of NICU-transferred infants to identify the key clinical features of MASD lesions. Additionally, the most common types of MASD lesions, their severity, healing time, the relationship between MASD lesions and their treatment, as well as the relapse rate and associated risk were assessed.

The findings revealed a higher incidence of IAD lesions (78.4%) compared with pMASD lesions (21.6%). Most patients with IAD showed redness or skin lesions without clinical signs of infection, whereas pMASD lesions were characterised by erythematous or erosive lesions occurring in no more than two quadrants. IAD occurrence was associated with diaper use.

In the patient cohort, two primary treatments for MASD lesions were identified. The first treatment, SoC, involved creams and lotions with skin barrier protective effects and/or additional properties, such as antimycotic or sequestrant actions. Zinc oxide acts as a barrier that protects the skin by reducing TEWL and preventing irritation, thereby maintaining a stable microenvironment.[6,22] Similarly, acrylate-based skin protectants form a breathable film that reduces friction and regulates moisture levels, helping to maintain a stable microclimate.[31] Hydrocolloid pastes, by retaining moisture, promote autolytic debridement and help regulate temperature, essential factors for optimal wound healing.[32] Additionally, topical antibiotics support a healthy microenvironment by controlling bacterial colonisation, reducing the risk of infection, and ensuring better wound healing conditions. All these dressings promote an occlusive mechanical function.[9,23–25]

Products that form an occlusive mechanical barrier, such as films, dressings and other occlusive materials,

Table 6. Relapse rate and odds ratio

		Patients treated with standard of care	Patients treated with Vulnamin spray	Total	Odds ratio	95% CI
Entire cohort Chi-squared test p-value=0.001	Yes, n (%)	8 (12.1)	14 (42.4)	22 (22.2)	5.3	1.94–14.69
	No, n (%)	58 (87.9)	19 (57.6)	77 (77.8)		
GA < 38 weeks Chi-squared test p-value=0.001	Yes, n (%)	6 (13.3)	11 (50.0)	17 (25.4)	6.5	1.96–21.56
	No, n (%)	39 (86.7)	11 (50.0)	50 (74.6)		
IAD cohort Chi-squared test p-value=0.001	Yes, n (%)	5 (9.4)	10 (41.7)	15 (19.5)	6.9	2.01–23.40
	No, n (%)	48 (90.6)	14 (58.3)	62 (80.5)		
pMASD cohort Chi-squared test p-value=0.290	Yes, n (%)	3 (23.1)	4 (44.4)	7 (31.8)	2.7	0.42–16.83
	No, n (%)	10 (76.9)	5 (55.6)	15 (68.2)		

CI—confidence interval; GA—gestational age; IAD—incontinence-associated dermatitis; pMASD—peristomal moisture-associated skin damage

influence the skin microclimate by directly affecting the hydration level of the stratum corneum and the skin temperature.[6] The presence of occlusive materials prevents the dissipation of heat and moisture through conduction and convection, leading to heat accumulation and increased stratum corneum hydration. This increased hydration can reduce the skin's mechanical resistance, raise the coefficient of friction, and consequently increase the risk of PIs. Moreover, more occlusive materials tend to elevate skin pH and promote bacterial proliferation, thereby aggravating the risk of skin damage.[6]

The second treatment, VSP, used a class IIb medical device. The mechanical action of VSP is supported by the hydrating and film-forming properties of medium molecular weight (>300kDa) sodium hyaluronate (HA), which is stabilised and protected against hyaluronidase degradation by a mixture of charged amino acids (glycine, alanine, valine and proline).[33] Additionally, the humectant, buffering and pH-balancing characteristics of amino acids help maintain a favourable microenvironment in the skin lesion, promoting tissue re-epithelialisation.[34] The mechanism behind the product efficacy can be related to its unique combination of hyaluronic acid and amino acids.[33]

The healing time for SoC-treated patients was approximately seven days, whereas VSP-treated patients had a healing time that was two days shorter, with no differences observed between IAD and pMASD lesions. The relapse rate in SoC-treated patients was 40–50%, whereas a significantly lower rate of approximately 10% was observed in VSP-treated patients, corresponding to an 80% lower risk of relapse. These results allow us to deduce the protective and preventive action of the HA and four amino acid combination against MASD.

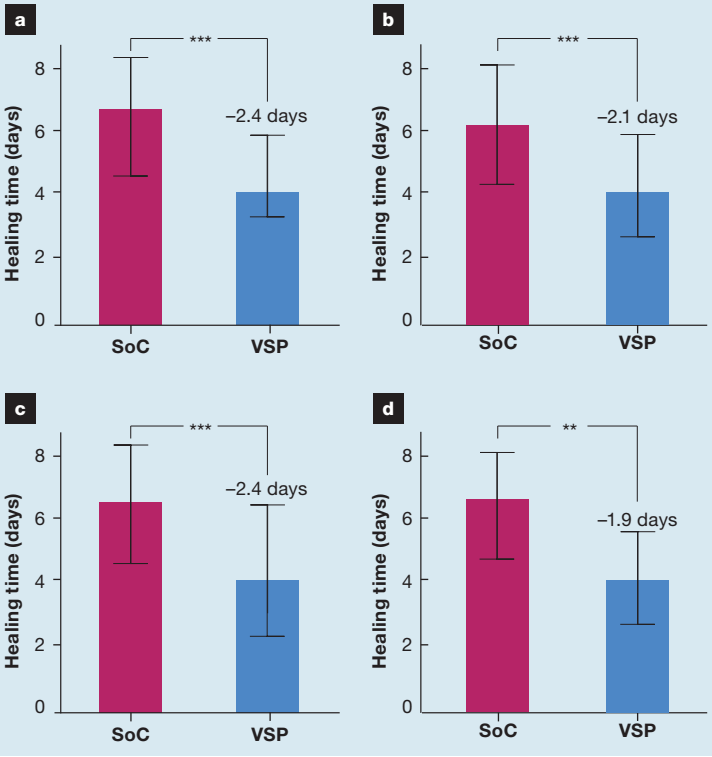
Limitations

To the best of the authors' knowledge, this is the first study to report on the demographics, clinical characteristics and nursing data of MASD lesions, in a small cohort of patients hospitalised in the NICU at Meyer Children's Hospital IRCCS in Italy. The study's relatively small sample size, including the sample size of VSP and SoC groups, is a limitation and will be addressed in future research.

Conclusion

The present study demonstrates a strong association between the treatment of MASD lesions, and their healing time and relapse rate in NICU patients. These findings highlight the need for developing strategies for

Fig 1. Healing time in the Vulnamin spray treatment group (VSP) compared to the standard of care group (SoC). Entire cohort **(a)**; patients with gestational age <38 weeks **(b)**; incontinence-associated dermatitis cohort **(c)**; peristomal moisture-associated skin damage cohort **(d).** **p<0.01; ***p<0.001



Reflective questions

- What strategies can be implemented to effectively protect the skin barrier function in preterm infants?
- What are the advantages and limitations of traditional standard of care therapies?
- How can the choice of treatment influence hospital stay duration and the quality of care perceived by parents or caregivers?

MASD lesion treatment beyond the traditional emollients and zinc creams, hydrocolloid or foam dressing, and topical antibiotics. The technological innovation of VSP as a therapeutic procedure represents a significant advancement in MASD management, effectively reducing healing time, recurrence rates and costs,[35] thus improving clinical outcomes in neonates. **JWC**

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