

Article

Skin tears flap management in patient affected by dermatoporosis

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Abstract. *Background:* dermatoporosis is the chronic cutaneous atrophy syndrome that makes the skin particularly fragile and it is the main risk factor in the onset of skin tears (ST). The syndrome is still little known in our country and the particularity acquired by affected skin and its difficulty in regeneration is often ignored. STs, which are often underestimated both by the patient and by the medical staff, have, in the patient with dermatoporosis, an evolution towards the most difficult healing, in which the management of the free skin flap (FSF) is fundamental. If the management of the FSF is inadequate, the evolution of the injury goes towards chronicity, which leads to greater patient suffering and waste of resources.

Methods: following the spontaneous observation of the evolution of ST in patients with dermatoporosis from 2009 to date (about 150) it is clear that the preservation and the combination of vital FSF following an unsuitable cleansing or non-vital FSF, affects the reparative process even if you use appropriate advanced medications.

Based on the gained experience, a series of practical recommendations are defined in the immediate and late intervention for the management of the FSF on: the cleansing of the fund of the lesion and of the internal part of the vital flap, the preservation of the flap during cleansing, the primary dressing and the importance of maintaining the right humidity gradient in the interval between dressings.

Results: to illustrate this, it is brought into vision the evolution of ST in some clinical cases with FSF treated differently with the first two-hour to twenty-day intervention in patients with: age >80 years, Barthel Index from 0 to 100, dermatoporosis stage IIb-IV, ST type T2-T3, FSF >2 cm. Healing times from five to one hundred days, compared to the state and treatment of the flap.

Conclusions: the presence and condition of the flap directly affect the healing of ST, the phenomenon is to be explored through clinical studies. With adequate management of the FSF even in the patient with dermatoporosis the STs can reach healing within 15 days. The process lends itself to standardization for the resemblance of the patient's condition.

Keywords: Dermatoporosis; Skin Fragility; Atrophic Skin; Chronic Cutaneous Insufficiency; Skin Tears.

Dermatoporosis

Dermatoporosis is a relatively new term introduced in 2007 by Swiss dermatologists G. Kaya and JH. Saurat who have studied the pathogenesis more in depth, defining it as “a chronic cutaneous insufficiency/fragility syndrome”¹.

It is characterized by the structural impoverishment of the skin^{2,3} due to the alteration of the morpho-physiological mechanisms with consequent reduction of the quantity of collagen and hyaluronic acid^{4,5} and the deficit of the transmembrane glycoprotein CD441,^{4,6} normally present. This leads to its thinning with the alteration of viscoelasticity, making it particularly fragile and less resistant to the action of external mechanical forces.

For this reason, it can arise in the elderly subjects following the physiological process of aging (over 70) or be induced and/or accelerated by intrinsic or extrinsic factors.

Dermatoporosis manifests clinically with the presence of:

- atrophy and skin thinning;
- actinic purpura (of Bateman);
- starred scars^{1,2}.

Senile purpura is more common in women and it usually occurs on the extremities without the presence of coagulation disorders, with an average frequency of 10% in the 70-90-year-old population and in 90% of cases it is associated with multiple scars (**Photo 1**). Histologically it is due to the extravasation of the red blood cells in the dermis, then and once the purplish colour of the patches has vanished, a brownish pigmentation remains that corresponds to the dermal deposit of hemosiderin^{1,2}.

The stellate pseudo scars are scarred areas of star-shaped resulting from small traumas or spontaneous tearing of the dermis. Morphologically they may appear linear, stellate or like whitish plaques. At the histological level in the dermis there is a hypocellular and compact band of collagen and a reduction of elastic fibers. The epidermis, on the other hand, is atrophic^{1,2}.



Photo 1. – Clinical signs of dermatoporosis

It has been classified^{1,7} in:

- Primary dermatoporosis (due to the physiological aging process);
- Secondary iatrogenic dermatoporosis (when induced by topical/systemic administration of drugs such as corticosteroids and prolonged and incorrect exposure to UV rays).

In the normal aging process, all skin layers undergo structural and functional alterations⁸, and one third of people over 60 are affected by dermatoporosis⁹. More evident on the photo-exposed areas of

décolleté, forearms, back of hands, legs, the skin is subtle, translucent, discolored, dry and creased, with ample mobility due to the reduced adherence to the underlying tissues.

The first staging made by Kaya and Saurat in 2007¹ included four stages without subgroups. But it was revisited by the same authors in 2012² as follows:

- Stage I: it is the most frequent and its morphological markers are: cutaneous atrophy, senile purpura and stellate pseudo scars;
- Stage II:
 - a) localized skin tears <3 cm,
 - b) skin tears >3 cm;
- Stage III
 - a) superficial hematoma,
 - b) dissecting hematoma without skin necrosis;
- Stage IV: large areas of dissecting hematoma with cutaneous necrosis with potential lethal complications.

A targeted treatment of dermatoporosis has not yet been identified, but the positive results of research on the effect of topical application of products based on hyaluronic acid fragments^{2,4} and retinaldehyde² to improve tropism and reactivate the metabolism of hyaluronic acid, they will certainly be the starting point for the development of specific therapies.

Meanwhile, becoming aware of the transformations that the skin of the affected subjects undergoes, leads to greater awareness of the risk of easily injuring it.

With the deficit of hyaluronic acid at the level of the extracellular matrix, in which it represents one of the major components, it alters its function visco-elastic network, thus increasing the susceptibility of the skin to the action of external mechanical forces.

Capillary fragility due to morphological changes in the dermal-epidermal junction may result in subcutaneous bleeding also important as a result of minor trauma, followed by the formation of small hematomas or dissecting hematomas. The areas above and surrounding the hematoma for the created cleavage plane are exposed to tearing even for minimal shocks, or to necrosis if not promptly intervened in case of dissecting hematoma.

The presence of dermatoporosis is a real problem for those affected by the high risk of the onset of skin tears that are sometimes unavoidable, and only the therapeutic education of both the patient and the care giver can prevent most of it.

Skin tears (ST)

Skin tears are the lesions caused by the tearing of the skin with or without removal of the skin flap, because of the external action of mechanical forces (cut, impact, friction, tearing), in subjects with:

- fragile skin (in clinical state of advanced criticality, dermatoporosis);
- immature skin (newborns, premature infants);
- skin with decreased adherence to underlying tissues (due to partial or total cleavage, caused by hematoma and/or edema).

Defined and classified for the first time by Paine en Martin in 1990¹⁰ (revision in 1993¹¹), they have been subject of great international interest with consequent development of various international studies and guidelines¹²⁻¹⁵, especially in the USA, Canada and the United Kingdom. In 2011, following an international survey¹⁵ conducted on 1127 health professionals from 16 countries, it emerged that 81% of respondents were unaware or did not use any of the classification systems currently in use (**Table 1**):

- Payne-Martin Classification 1993¹¹;
- STAR Classification¹²;

- CAWC Best Practice¹⁴.

In addition, 89,5% were in favor of simplifying the methodology of documentation and evaluation of cutaneous lacerations¹⁶. In 2013, based on the review of the literature and the joint work of an international staff: "International Skin Tear Advisory Panel: A Tool Kit to Aid in the Prevention, Assessment and Treatment of Skin Tears Using a Simplified Classification System"¹⁷ was born, which proposes a simplified classification and has the aim of suggesting the tools for the prevention, identification and treatment of cutaneous lacerations (**Table 1**).

Table 1. – Classification of the STs

Scale of Pain Martin ¹¹	STAR Scale ¹²	ISTAP 2013 ¹⁷
It considers the extent of tissue loss after the skin flap has been repositioned, it does not include the vital state of the flap.	It considers the tissue loss and the vitality of the cutaneous flap.	It considers the presence of free skin flap.
It consists of five levels: 1A. Epidermis and dermis are separated; the lesion is presented as an incision. 1B. Epidermis and dermis are separated; tissue loss is less than 1 millimeter. 2A. Epidermis and dermis are separated; tissue loss is 25% or less. 2B. Epidermis and dermis are separated; tissue loss is more than 25% 3. Complete tissue loss and absence of skin flap.	It is divided into: 1A. The margins can be realigned, and the skin flap is normochromic. 1B. Margins can be realigned but the flap is dark, pale, cyanotic or necrotic. 2A. Margins cannot be realigned, and the skin flap is normochromic. 2B. The margins cannot be realigned, and the skin flap is dark, pale, cyanotic or necrotic. 3. The flap is absent.	It is divided into: Type 1. No tissue loss. Type 2. Partial tissue loss. Type 3. Complete tissue loss.

The STs are defined as: "injuries caused by shear, friction and impact forces with consequent separation of the skin layers"¹⁸. They can be of partial thickness (separation of the epidermis from the dermis) or total (separation of the epidermis and dermis from the underlying structures).

The new simplified classification¹⁷ divides them into 3 types based on the extent of skin flap removal:

Type 1 - Without removal of the skin flap;

Type 2 - Partial removal of the skin flap;

Type 3 - Total removal of the skin flap.

The ISTAP simplified classification system was validated in Italian in 2018 by a group of nurses at the Niguarda¹⁹ Hospital in Milan, with a validity comparable to the validation in the original language. Their validated variant, not yet widespread in clinical practice, is as follows:

- Type1: Without loss of skin tissue.
Linear tear or skin flap that can be repositioned to cover the area of the wound;
- Type2: Partial loss of the skin flap.
Partial loss of the skin flap that cannot be repositioned in such a way as to cover the entire wound area.
- Type 3: Total loss of the skin flap.
Total loss of the skin flap that exposes the entire area of the wound¹⁹.

STs were estimated to be the most common acute lesions in adults¹⁷, with a prevalence equal to or greater than pressure lesions^{15,17}.

Risk factors are individual and/or related to assistance¹⁸⁻²⁰. Individual factors: age, general state, neurological and cognitive condition, skin condition (dryness, fragility, morpho-physiological alterations, dermatoporosis) degree of autonomy and mobility, multimorbidity, therapies in place, need to use devices and rigid and narrow devices (braces, busts, socks).

Factors related to the everyday environment and to the assistance: furniture (if not suitable with low or angular furniture, presence of carpets that can encourage slipping and falls, not suitable bed and hard and badly positioned aids), use of garments for transfer and bathroom, displacement and repositioning by third parties, management of drugs and ostomy. Having become aware of the extent of the phenomenon, from 2013 onwards, various clinical studies have been developed¹⁸⁻²¹, and with great sensitivity towards a problem so extensive, various guidelines have been developed on the management of this type of injury²²⁻²⁷.

Subjects and methods

Following the spontaneous observation of the evolution of ST in patients treated since 2009 to date (approximately 800), it has been observed that the main risk factor is the presence of dermatoporosis. It was also observed that most of the patients affected (about 95%) underestimate the risk, defining the consequent onset of skin lacerations as an evolution without remedy - a "condemnation", surrendering without even trying to change the usual attitudes or style of life. In all the observed cases, the request for wound care advice was received for: the chronicity of the lesion (70%), severe pain caused by the lesion (20%), strong bleeding (8%), awareness of the problem (2%).

At the care of patients with skin lesions, the patient is globally evaluated according to the Toven method²⁸ (with evaluation of: general state, autonomy, nutritional status, pain, risk) and an evaluation of the lesion, a method to which patients with STs have also been subjected to.

Following the targeted observation of the group of patients aged >80 years old and with dermatoporosis from stage II b - to IV² (about 150), the preservation and the combination of vital FSF without adequate cleansing or non-vital FSF have the same result: the repair process is impaired even if you use appropriate advanced dressings.

Following the gained experience in our practice in the local treatment of STs, special attention is given to the cleansing of the bottom and of the inner part of the flap (**Photo 2,3**) before repacking (**Photo 4**).

If the free skin flap is assessed as vital and the lesion has risen by no more than 36 hours, the cleansing is performed by moving the free flap (Photo 3) and cleaning with a non-injurious solution applied by spraying, before the wound bed with the removal of the clots and any debris

present, then cleansing the inside of the flap, always applying the spray solution and dabbing with gauze without exerting particular pressure.



Photo 2. – Type 1 ST, state of the injury upon taking charge.



Photo 3. – The cleansing of the free skin flap. State of the injury following cleansing.



Photo 4. - The combination of the FSE.

Following cleansing, the wound dressing is performed with advanced dressings (antimicrobials, haemostats or only absorbents) chosen with respect to the general condition of the

patient and the state of the lesion, with respect to the microenvironment and with the maintenance of the right humidity gradient in the interval between dressings.

To illustrate this, the evolution of STs is shown in some clinical cases with FSF treated differently with the first two-hour to twenty-day intervention in patients with: age > 80 years old, Barthel Index from 0 to 100, stage dermatoporosis IIb-IV, ST type T2-T3, FSF > 2 cm. Healing times range from five to one hundred days, compared to the status and treatment of the flap (Table 2).

Table 2. – Clinical cases of STs treated in patients with dermatoporosis.

CL C.	Sex	Age	Dermatoporosis stage	NRS Pain/Position	MNA	Hb (gr/dl) Albumin (gr/dl)	Barthel Index	ST Type	Cause	Size of the lesion	Free flap	Treatment before T0	Flap removal	Days ^h for T0*	Healing time
1	F	96	IV	10 leg	11,5	Hb - 10,0 Alb. - 3,0	40/100	T2	Fall Trauma	35x18	Necrosis	Stitches	Surgical Debridement	20 days	100 days** (120)^
2	F	96	IV	4 arm	11,5	Hb - 10,5 Alb. - 4,2	40/100	T2	Dressing Trauma	2,5x2	2,5	-	No	4 h	13 days (13)
3	F	82	IIIb	6 leg	18	Hb - 13,0 Alb. - 4,8	100/100	T2	Fall Trauma	10x7	3,5	Non determined advanced medications	Autolytic Debridement	12 h	13 days (25)
4	F	94	IIIa	6 leg	8	Hb - 9 Alb. - 2,6	0/100	T2	Trauma	8x4	4	-	Yes in the detersion	2 h	6 days
5	F	82	IIIa	7 leg	25	Hb - 12 Alb. - 3,0	100/100	T2	Trauma	6x3	3	Fat Gauze	Yes, voluntary after evaluation	4 days	22 days (26)
6	F	86	IIIa	8 arm	17	Hb - 10,0 Alb. - 3,2	100/100	T2	Trauma	12x8	9	Frontosan Idrofibre Ag gauze	Si. voluntary.	36 h	9 days
7	M	97	IIIa	4 hand	15	Hb - 9,0 Alb. - 2,6	20/100	T2	Home Trauma	6x3	3	-	No	3 h	5 days

* T0—moment of taking charge by Wound Care Specialist ; hours/days passed from the trauma ** days from T0 to healing ^ days from trauma to healing

Evaluations performed: nutritional (MNA, Hb, Albumin), autonomy (Barthel), pain (NRS), of the injury according to ISTAP 2013, of the free skin flap, of dermatoporosis according to Kaya-Saurat 2012 classification.

Table 3. – Photographic monitoring of some clinical cases evaluated in Table 2

CLINICAL CASE	1	3	4	7
Time 0 Upon taking charge	 20 days after the trauma, stitches has been applied to the patient	 12 days after trauma Maintained FSF not vital	 2 hours after trauma	 3 hours after trauma
Time in days of taking charge	 15 days	 10 days	 8 days – 2 dressings, 90% epitelized	 5 days – a dressing, epitelized at 80%, FSF completely taken
Time in days of taking charge	 90 days	 12 days	 13 days – at the removal of the third dressing	 Control at 15 days, not present retractable fabric
Treatment Executed after taking care of wound care	Negative pressure therapy Advanced antimicrobial dressings Medium moist	Advanced antimicrobial dressings Medium moist	Advanced antimicrobial dressings Medium moist Total treatment 3 dressings	Advanced antimicrobial dressings Medium moist Total treatment 2 dressings

Observations

- Skin tears (ST) in patients with dermatoporosis are more common on limbs, both in walking patients and in patients who need assistance in their movements. They are usually caused by minor trauma during dressing and assisted mobilization.
- STs in patients with dermatoporosis often have an evolution towards chronicity²⁹ for inadequate treatment of the lesion and free skin flap (FSF) due to:
 - poor knowledge of the syndrome;
 - underestimation of the trauma;
 - failure to evaluate the patient overall and the risk of infection;
 - failure to evaluate the FSF and the lesion;
 - lack of cleansing of the wound bottom and FSF;
 - no maintenance of the medium wet.
- In case of vital FSF, without adequate cleansing, the lesion has an evolution comparable to the lesion with non-vital flap.
- In situ maintenance of the non-vital flap slows the healing of the lesion and increases the risk of infection (see clinical case 4).
- The dressing in a dry environment, however, leads to the death of the flap (although vital at the time of dressing) even in the case of use of advanced dressings, or following proper cleansing. There is also the risk of involuntary removal of FSF as it adheres to the applied dressing.
- If soft and palpable subcutaneous hematoma (which exceeds the cutaneous level) has been present for more than 48 hours (**Photo 5**), the opening is almost inevitable, the skin

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tissue covering the hematoma is to be considered not vital. The phenomenon is to be explored through clinical studies.



Photo 5 - Presence of subcutaneous hematomas.

In these cases, it is advisable to empty the hematoma for the successful outcome of the recovery (Photo 6,7).



Photo 6. - Subcutaneous hematoma arising during the application of therapeutic stocking in a patient with dermatoporosis, without ongoing anticoagulant therapy.



Photo 7. - Depletion of the subcutaneous hematoma, ST healing.

- The flap is to be considered vital if:
 - the onset of the lesion dates to no more than 36 hours and in the applied dressings the humid environment has been maintained;
 - the color of the flap is the same as that of the surrounding skin;
 - the degree of hydration is like the surrounding skin. The dry flap, even if of the same color is to be considered not vital;
 - there was no palpable subcutaneous hematoma occurring at least 24 hours before the opening of the lesion.
- The application of any adhesive device to stabilize/fix the flap to the underlying layers has proved to be harmful, leading to the death of the flap following the stretching that occurs during movements.
- If the lesion is properly injected in the first few hours and proper cleansing of both the lesion and the internal part of the FSF is performed, even if the patient's age is advanced^{29,30} or the clinical conditions are not ideal, the flap takes root with complete healing times from 5 to 15 days if kept in the humid environment.
- The use of cytotoxic antiseptic solutions^{31,32} such as iodine povidone^{33,34,35}, hydrogen peroxide^{36,37} and mercurochrome^{31,32}, inevitably lead to cell death³⁶, FSF and exposed cellular layers, and they cause severe pains during application. Their use must be limited to the minimum necessary (serious trauma, damaged skin in a dirty environment), with the awareness that it will lengthen the healing time. The site of application of the antiseptics must be rigorously washed^{31,32} with physiological solution.

Conclusions

The presence and the condition of the flap directly affect the healing of STs³⁸, including the presence of subcutaneous hematoma. The phenomenon is to be explored through clinical studies. The use of cytotoxic solutions such as povidone iodine, hydrogen peroxide and mercurochrome compromise the vitality of the flap after their application.

With adequate management of the FSF even in the patient with dermatoporosis the STs can reach healing within 15 days. The process lends itself to standardization for the resemblance of the patient's condition.

The therapeutic education of the patient with dermatoporosis for the awareness of the peculiar skin condition for the adaptation of his lifestyle, and to be able to apply an adequate medication³⁰ in case of appearance of the STs is to be considered the first essential intervention for the prevention of the onset of new lesions and of infection/chronicization of existing lesions.

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References

1. Kaya G, Saurat JH. *Dermatoporosis: a chronic cutaneous insufficiency/fragility syndrome. Clinicopathological features, mechanisms, prevention and potential treatments.* *Dermatology* 2007; 215(4):284-94.
2. Kaya G. *New therapeutic targets in dermatoporosis.* *J Nutr Health Aging* 2012;16(4):285-8.
3. Re Domínguez ML, Di Martino Ortiz B, Rodríguez Masi M, et al. *Dermatoporosis, an emerging disease: case report.* *Our Dermatol Online* 2016;7(2):191-194.
4. Barnes L, Ino F, Jaunin F et al. *Hyalurosomes Inhibition and Epidermal Atrophy.* *Journal of investigative Dermatology* 2013; 133:1017-1026.
5. Toma E. *La dermatoporosi: che cos'è e come riconoscerla - Il ruolo dell'infermiere nell'educazione del paziente.* *Infermiere Oggi* 2014; 24(4):20-22, ISSN 2037-4364.
6. Kaya G, Grand D, Hotz R, et al. *Upregulation of CD44 and hyaluronate synthases by topical retinoids in mouse skin.* *J Invest Dermatol* 2005;124(1):284-287.
7. Saurat JH, Mègeaude V, Georgescu V, et al. *A simple self-diagnosis tool to assess the prevalence of dermatoporosis in France.* *J Eur Acad Dermatol Venereol* 2017;31(8):1380-1386.
8. Farage MA, Miller KW, Berardesca E et al. *Clinical implications of aging skin: cutaneous disorders in the elderly.* *Am J Clin Dermatol* 2009;10:73-86.
9. Dyer MJ, Miller AR. *Chronic Skin Fragility of Aging: Current Concepts in the Pathogenesis, Recognition, and Management of Dermatoporosis.* *J Clin Aesthet Dermatol* 2018;11(1):13-18.
10. Payne R, Martin M. *The Epidemiology and management of skin tears in older adults.* *Ostomy Wound Manage* 1990;26(1):26-37.
11. Payne RL, Martin MC. *Defining and classifying skin tears: need for a common language.* *Ostomy Wound Manage* 1993;39(5):16-26.
12. Carville K, Lewin G, Newall N, et al. *STAR: a consensus for skin tear classification.* *Prim Intent* 2007;15(1):18-28.
13. Ratliff CR, Fletcher KR. *Skin tears: a review of the evidence to support prevention and treatment.* *Ostomy Wound Manage* 2007;53(3):32-42.
14. LeBlanc K, Christensen D, Orstead H, Keast D. *Best practice recommendations for the prevention and treatment of skin tears.* *Wound Care Canada* 2008;6(8):14-32.
15. LeBlanc K, Baranoski S. *Skin Tears: State of the Science: Consensus Statements for the Prevention, Prediction, Assessment, and Treatment of Skin Tears.* *Adv Skin Wound Care* 2011; 24(9Suppl):2-15.
16. LeBlanc K, Baranoski S, Holloway S, Langemo D, et al. *International Skin Tear Advisory Panel (ISTAP) – Validation of a New Classification System for Skin Tears.* *Adv Skin Wound Care* 2013;26(6): 263-265.

17. LeBlanc K, Baranoski S, Holloway S, et al. International Skin Tear Advisory Panel: A Tool Kit to Aid in the Prevention, Assessment, and Treatment of Skin Tears Using a Simplified Classification System. *Adv Skin Wound Care* 2013;26(10).
18. Serra R, Ielapi N, Barbetta A, De Francid S. Skin tears and risk factors assessment: a systematic review on evidence-based medicine. *Int Wound J* 2018;15(1):38-42. Doi:10.1111/iwj.12815.
19. Bassola B, Cecci P, Lolli A, Lusignani M. Validazione in italiano del sistema ISTAP di classificazione degli strappi cutanei. Primo Congresso Nazionale della Federazione Nazionale degli Ordini della Professioni Infermieristiche 2018 Roma.
20. Bermark S, Wahlers B, Gerber AL, et al. Prevalence of skin tears in the extremities in inpatients at a hospital in Denmark . *Int Wound J* 2018. Doi: 10.1111/iwj.12847.
21. Brimelow RE, Wollun JA. The impact of care practice and healt dermographics on the prevalence of skin tears and pressure injuries in aged care. *J Clin Nurs* 2018; Feb 3. Doi : 10.1111/jocn.14287.
22. Koyano Y, Nakagami G, Iizaka S, et al. Skin property can predict the development of skin tears among elderly patients: a prospective cohort study. *Int Wound J* 2017;14(4):691–697.
23. Cooper P, Russell F, Stringfellow S. Managing the treatment of an older patient who has a skin tear. *Wound Essentials* 2006;1:119-20.
24. Holloway S, LeBlanc K. Dealing with Skin Tears. *Journal of Nursing in Practice* 2017;22:64-66.
25. LeBlanc K, Baranoski S. Skin Tears: Finally Recognized. *Adv Skin Wound Care* 2017;30(2):62-63.
26. Thompson-McHale S. Skin tears: assessment, prevention, classification and management. Clinical review. *Nursing Residential Care* 2013;15(11): 710-714.
27. LeBlanc K, Baranoski S, Christensen D, et al. The Art of Dressing Selection: A Consensus Statement on Skin Tears and Best Practice. *Adv Skin Wound Care*. 2016;29:32–46.
28. Toma E. La valutazione del paziente portatore I lesion cutanee. Fascicolo Toven wound care dedicato. Youcanprint 2017. ISBN 978-88-92675-88-9.
29. Vanzi V, Toma E. How to prevent and avoid common mistakes in skin tear management in the home setting. *Br J Community Nurs*. 2017 Sep 1; 22(Sup9):S14-S19. Doi: 10.12968/bjcn.2017.22.Sup9.S14.
30. Vanzi V, LeBlanc K. Skin tears in the aging population: Remember the 5 Ws. *EWMA Journal* 2018;19(1):15-20.
31. Müller G, Kramer A. Biocompatibility index of antiseptic agents by parallel assessment of antimicrobial activity and cellular cytotoxicity. *J Antimicrob Chemother* 2008 Jun;61(6):1281-7. Doi: 10.1093/jac/dkn125.
32. Kramer A, Dissemond J, Kim S, et al. Consensus on Wound Antisepsis: Update 2018. *Skin Pharmacol Physiol* 2018;31:28–58. Doi: 10.1159/000481545.
33. Atiyeh BS, Dibo SA, Hayek SN. Wound cleansing, topical antiseptics and wound healing. *Int Wound J* 2009;6:420–430.
34. Wilson JR, Mills JG, Prather ID, Dimitrijevic SD. A toxicity index of skin and wound cleansers used on in vitro fibroblasts and keratinocytes. *Adv Skin Wound Care*. 2005 Sep;18(7):373-8.
35. Kramer SA. Effect of povidone-iodine on wound healing: a review. *J Vasc Nurs*. 1999 Mar;17(1):17-23.

36. Quinn RH, Wedmore I, Johnson EL ,et al. Wilderness Medical Society Practice Guidelines for Basic Wound Management in the Austere Environment:2014 Update. *Wilderness & Environmental Medicine* 2014;25(3):295-310.
37. Yi Yang, Reid C, Mithun Nambiar , et al. Hydrogen peroxide in orthopaedic surgery – is it worth the risk?. *Acta Chirurgica Belgica* 2016; 116(4):247-250
Doi:org/10.1080/00015458.2016.1147235.
38. Toma E. La gestione del lembo cutaneo delle skin tears in paziente con dermatoporosi. *Atti del XIV Congresso Nazionale AIUC* 2017:111.