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REVIEW ARTICLE

Corrective and Restorative Dermatology in Cancer Survivors: An Urgent Unmet Need!

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Abstract

The increasing efficacy of oncological therapies has led to a rapidly growing population of cancer survivors, shifting clinical attention from acute treatment-related toxicities to long-term and late sequelae. Among these, dermatological sequelae represent some of the most visible and persistent consequences of cancer treatments and may significantly impair quality of life. Persistent alopecia, nail disorders, pigmentary alterations, chronic radiation-induced skin changes, scarring, vascular abnormalities, mucosal alterations, and secondary skin cancers may develop or persist long after treatment completion, acting as constant reminders of the cancer experience. These conditions are not only cosmetic; they may be associated with functional limitations, psychological distress, and altered body image, particularly in patients treated during childhood or adolescence. While the role of supportive oncodermatology during active cancer treatment is well established, structured dermatological care in the survivorship phase remains insufficiently developed. This narrative review provides a comprehensive overview of the main long-lasting dermatological adverse events associated with chemotherapy, radiotherapy, targeted therapies, immunotherapy, endocrine treatments, and hematopoietic stem cell transplantation. In addition, available restorative and corrective dermatological interventions are discussed, with a focus on improving long-term outcomes and patient well-being. Recognizing and managing dermatological sequelae is an essential component of modern survivorship care, and dermatologists should play a central role in multidisciplinary follow-up programs dedicated to cancer survivors.

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Key Points

Advances in cancer therapies have resulted in a growing population of long-term cancer survivors with chronic dermatological sequelae, such as persistent alopecia, nail abnormalities, pigmentary disorders, telangiectasias, scarring, mucosal alterations, and chronic radiation-induced skin changes. Dermatological sequelae may cause functional impairment, psychological distress, altered body image, and reduced quality of life.

Childhood and adolescent cancer survivors are particularly vulnerable, due to the long-lasting impact on physical appearance and identity development.

Most supportive oncodermatology programs focus on acute toxicities, while post-treatment dermatological needs remain largely unmet. Dermatologists should play a key role in survivorship care, including surveillance for secondary skin cancers and delivery of restorative treatments.

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1 Introduction

The improvement in oncological therapies has led to a rapidly expanding population of cancer survivors, shifting clinical attention from acute toxicity toward long-term and late sequelae of treatment [1, 2]. Dermatological sequelae, including scars, persistent hair loss, nail disorders, pigmentary alterations, and chronic radiation-induced skin changes, represent some of the most visible, impactful and distressing long-term effects of cancer treatments [3]. These cutaneous manifestations are not merely cosmetic, as they may cause functional impairment and are strongly associated with psychological distress, reduced quality of life, and altered body image, particularly in survivors treated during childhood or adolescence [4]. In the survivorship setting, restorative oncodermatology may be broadly defined as the management of dermatological manifestations persisting for at least 6 months after completion of anticancer treatment and potentially requiring additional dermatological care. This concept includes both persistent toxicities with slow regression and *true* long-term sequelae that may remain stable over time (Table 1). In clinical practice, however, the boundary between these entities is often blurred, as some treatment-related manifestations (for example, pigmentary alterations or nail alterations) could persist for years before gradually improving.

The emerging field of supportive oncodermatology highlights the importance of early recognition and appropriate management of skin toxicities and their sequelae to improve long-term outcomes and therapeutic adherence [1]. While the relevance of supportive oncodermatology during active anticancer treatment is well demonstrated, its role in the post-treatment survivorship phase remains less clearly defined [5]. In this context, a comprehensive understanding of chronic dermatological sequelae and their management is essential to optimize survivorship care and to address the unmet needs of cancer survivors [3, 6].

Available evidence shows that dermatological sequelae persist in a substantial proportion of survivors, with up to 59% of adult survivors of childhood cancer reporting chronic skin-related problems and approximately 30% reporting visible scarring or disfigurement long after treatment completion [4, 7]. Despite this high prevalence, most supportive oncodermatology programs have been developed and evaluated primarily in the active treatment setting, where they have demonstrated significant improvements in quality of life and treatment adherence [5]. As an example, the implementation of a comprehensive cutaneous toxicity program for patients receiving epidermal growth factor receptor inhibitors (EGFRis) was associated with higher adherence to prophylactic guidelines, reduced need for rescue treatments, and fewer dose modifications, clearly demonstrating the benefit of integrated dermatological care during therapy [8]. However, these studies mainly address acute and treatment-related toxicities, and there is currently a paucity of data specifically evaluating the impact of structured supportive oncodermatology interventions on long-term dermatological sequelae and altered body image in cancer survivors after treatment completion [3, 4].

Despite the increasing number of cancer survivors, their long-term dermatological needs remain largely unmet, and dedicated survivorship-oriented dermatological care is still rarely implemented. There is an urgent need to provide appropriate care for these patients and to improve knowledge in this field, which is currently limited, with particular

Table 1 Expected reversibility of common dermatological manifestations in cancer survivors, distinguishing between persistent toxicities, defined as treatment-related conditions persisting ≥ 6 months after completion of anticancer therapy but potentially reversible, and *true* sequelae, which represent stable or irreversible alterations resulting from treatment-induced tissue damage

Dermatological manifestation	Expected reversibility	Time course	Classification
Persistent chemotherapy-induced alopecia	Often irreversible	> 6 months	Sequela
Endocrine-induced alopecia	Partially reversible	May improve after discontinuation	Persistent toxicity
Radiation-induced alopecia	Often irreversible	> 6 months	Sequela
Nail fragility	Partially reversible	Months with nail regrowth	Persistent toxicity
Chronic onycholysis	Variable	May persist months–years	Persistent toxicity/possible sequela
Post-inflammatory hyperpigmentation	Slowly reversible	Months–years	Persistent toxicity
Dermal hyperpigmentation	Often persistent	Years	Sequela
Hypopigmentation	Often permanent	Long term	Sequela
Radiation telangiectasias	Rare spontaneous regression	Months–years	Sequela
Radiation fibrosis	Irreversible	Progressive	Sequela
Chronic xerosis	Reversible	Improves after treatment	Persistent toxicity

For some conditions, particularly pigmentary and nail disorders, the long-term course may be variable

attention to childhood and adolescent cancer survivors. In this setting, dermatologists should play a key role both in the surveillance of secondary skin cancers and in the delivery of restorative dermatological therapies that help patients cope with life after cancer [3, 9].

This narrative review focuses on dermatological manifestations that may persist during survivorship and that can potentially benefit from corrective or restorative dermatological interventions.

2 Persistent and Long-Term Alterations in Hair and Nails

2.1 Persistent Chemotherapy-Induced Alopecia

Persistent (or permanent) chemotherapy-induced alopecia (pCIA) is defined as incomplete or absent hair regrowth persisting for more than 6 months after completion of cytotoxic chemotherapy and represents a late sequela of cancer treatment [10–12]. In contrast to classic chemotherapy-induced alopecia (CIA), which is usually reversible within 3–6 months after treatment discontinuation, pCIA reflects irreversible damage to the hair follicle stem cell compartment [11, 13, 14].

The clinical course of pCIA is typically preceded by an acute anagen effluvium occurring within the first weeks of chemotherapy, followed by delayed, incomplete, or absent hair regrowth [12, 15]. The reported incidence of pCIA varies widely in the literature, ranging from < 1% to approximately 40% depending on chemotherapy regimens, cumulative dose, treatment schedule, and study design [10, 11, 16–18]. Among chemotherapeutic agents, taxanes are most consistently associated with pCIA, with docetaxel showing a significantly higher risk compared with paclitaxel [13, 16, 19]. High-dose alkylating agents, particularly busulfan, cyclophosphamide, thiotepa, and melphalan, are also strongly implicated, especially in transplant settings or intensive chemotherapy protocols [4, 11, 13]. Additional agents including etoposide, carboplatin, and cisplatin have been associated with pCIA, particularly when used in combination regimens, highlighting the role of cumulative cytotoxic injury [11–13]. Weekly administration schedules, polychemotherapy, and higher cumulative doses further increase the risk of incomplete hair regrowth [10, 13, 14, 17].

Clinically, pCIA presents as a diffuse reduction in hair density or as an androgenetic-like pattern, most commonly involving the frontal, parietal, and vertex scalp (Fig. 1a), frequently associated with alopecia of the eyelashes, eyebrows, underarm, and pubic hair [10, 12, 17]. In many patients, partial regrowth occurs after chemotherapy, but hair remains thin, fragile, and unable to reach its previous length, often not exceeding a few centimeters [13, 19]. Hair shafts may show altered texture, reduced diameter, and increased fragility, contributing to the perception of persistent thinning [10, 13, 15, 20]. Unlike scarring alopecia, scalp skin usually appears normal, with preserved follicular ostia and absence of erythema or scaling [11, 13].

Trichoscopy represents a fundamental non-invasive diagnostic tool and allows differentiation between reversible CIA, pCIA, and other alopecias [10, 15]. Typical trichoscopic findings include reduced hair shaft density, single-hair follicular units, variability in the diameter of the hair shaft, increased miniaturized hairs, multiple yellow dots, and persistence of short regrowing hairs beyond 6 months after chemotherapy (Fig. 1c) [11, 15, 17]. Diffuse yellow dots and persistent inflammatory signs have been identified as predictive markers of permanent alopecia [11].

Reflectance confocal microscopy further supports diagnosis by demonstrating chronic perifollicular inflammation and early fibrotic remodeling, suggesting irreversible follicular injury [11].

Histopathology consistently demonstrates a non-scarring alopecia with preserved follicular units, follicular miniaturization, increased telogen hairs, and absence of fibrosis [11, 13].

No therapy has been specifically approved for pCIA, and current treatment strategies are largely extrapolated from other non-scarring alopecias [10].

Topical minoxidil (dosage 2–5%) is frequently prescribed and may provide partial improvement in hair density and

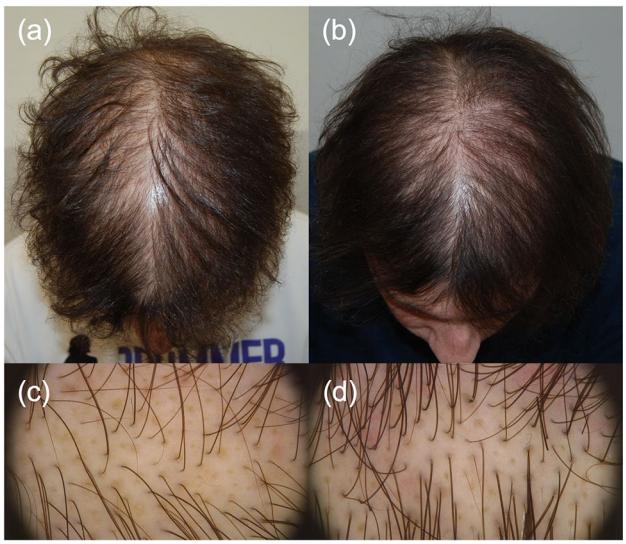

Fig. 1 Persistent chemotherapy-induced alopecia (pCIA). Clinical presentation with an androgenetic-like pattern, before (**a**) and after 1 year of oral minoxidil (**b**). Trichoscopy of pCIA shows reduced hair shaft density, single-hair follicular units, variability in the diameter of the hair shaft, increased miniaturized hairs, multiple yellow dots, and persistence of short regrowing hairs (**c**). Trichoscopic improvement after 1 year of oral minoxidil (**d**). Personal files

shaft thickness. It accelerates hair regrowth through the induction of angiogenesis, activation of prostaglandin-endoperoxide synthase 1, and prolongation of the anagen phase, with a consequent reduction in the telogen phase. For these reasons, it is recommended after the discontinuation of chemotherapy to enhance hair regrowth. Conversely, it is not recommended for the prevention of CIA, as it has shown clinical ineffectiveness and lacks a sound scientific rationale; indeed, by inducing peripheral arteriolar vasodilation, it may reduce blood flow velocity and increase the residence time of chemotherapeutic agents at the level of the hair follicle [10, 12, 19, 21]. The clinical efficacy of topical minoxidil in a preventive way remains to be demonstrated, although some preliminary data also supports this concept [21].

Recent observational evidence supports the use of low-dose oral minoxidil (0.5–5 mg/day), which has demonstrated significant increases in hair density and clinical improvement in cancer survivors with pCIA in retrospective series (Fig. 1b, d). Oral minoxidil appears to be well tolerated in oncological populations, with low discontinuation rates and no serious adverse events reported [22, 23]. In addition, spironolactone 100–200 mg/day may be added to achieve a better response [10, 24]. Available evidence does not demonstrate an increased risk of new primary breast or gynecological malignancies in women exposed to spironolactone [10, 25, 26]; however, these data are mainly derived from populations receiving lower doses and do not specifically address the risk of cancer recurrence, particularly in patients with a history of hormone receptor–positive malignancies. Therefore, in cancer survivors, the use of spironolactone should be preceded by an individualized risk–benefit assessment and discussed within a multidisciplinary oncology team.

Topical bimatoprost can increase length and thickness of chemotherapy-induced eyelashes hypotrichosis [27]. Platelet-rich plasma (PRP) therapy has emerged as a potential option, showing modest increases in hair density in randomized pilot studies involving patients with pCIA and endocrine-induced alopecia. However, the current evidence for PRP remains limited; efficacy may be confounded by diffusion effects and spontaneous stabilization of hair loss. Further controlled studies are needed before firm conclusions regarding its efficacy can be drawn [28]. Other therapeutic approaches, including antiandrogens and nutraceuticals, lack robust evidence and should be considered with caution [10].

Given the limited efficacy of available treatments, prevention remains the most effective strategy to reduce the burden of persistent alopecia [10, 29]. Scalp cooling is currently the only preventive intervention with strong evidence, significantly reducing the severity of CIA and potentially lowering the risk of pCIA, particularly in taxane-based regimens [10, 29, 30]. The main limitations of this approach include its effect being restricted to the period of drug infusion (including pre- and post-infusion phases) and its relatively high cost. In addition, several contraindications must be considered, including central nervous system tumors, hematological malignancies, tumors of the head and neck region, small cell lung cancer, and pediatric patients. It is also contraindicated in individuals receiving platinum-based chemotherapy and in patients with hypersensitivity or medical conditions that may be triggered or exacerbated by exposure to cold [31–34].

pCIA has a profound psychosocial impact, affecting body image, self-esteem, emotional well-being, and social functioning [19, 35, 36]. Cancer survivors with persistent hair loss report increased anxiety, emotional distress, and long-term impairment in quality of life [4, 19, 37]. For many patients, pCIA represents a visible and permanent reminder of cancer, so early counseling, preventive strategies, and long-term dermatological follow-up are mandatory [10, 35].

2.2 Persistent Endocrine-Induced Alopecia

Endocrine therapies may induce late-onset or persistent alopecia with a female pattern hair loss–like phenotype, independently of previous chemotherapy exposure, and it has been reported in approximately 15–25% of breast cancer survivors receiving aromatase inhibitors [19, 20, 38]. Alopecia is also more frequent and more severe when endocrine therapies are prescribed in combination with CDK4/6 inhibitors, both in the adjuvant or metastatic setting [39].

Clinically, patients typically present with progressive frontoparietal thinning and miniaturization of hair follicles, mimicking androgenetic alopecia (Fig. 2), and trichoscopy frequently reveals increased vellus hairs and variability in hair shaft diameter [38]. It has been associated with a significant negative impact on quality of life, particularly affecting emotional well-being and self-confidence, and may contribute to treatment discontinuation in a subset of patients [38].

Topical and oral minoxidil represent the first-line treatments and have shown moderate to significant improvement in the majority of treated patients [10, 38]. Also spironolactone prescription may be discussed, with a case-by-case approach [10, 24–26, 40]. Oral finasteride, bicalutamide, and dutasteride are contraindicated in patients with diagnosis of hormone-dependent breast cancer [10, 38].

2.3 Persistent Radiation-Induced Alopecia

Persistent radiation-induced alopecia (pRIA), also referred to as persistent radiation-induced scarring alopecia, is defined as incomplete hair regrowth 6 months after completion of radiotherapy and represents a dose-dependent late effect of ionizing radiation [12, 41]. The risk of developing persistent alopecia increases with higher cumulative scalp doses, and a threshold around 36 Gy has been

associated with a 50% probability of severe alopecia [4, 41]. Severe pRIA correlates not only with total dose but also with specific radiation techniques [41].

Radiotherapy causes direct damage to follicular stem cells in the bulge region and to the surrounding dermal microenvironment, leading to impaired follicular regeneration and progressive fibrosis [12, 42].

Clinical presentation of pRIA usually is as a sharply demarcated alopecic patch corresponding to the irradiated field (Fig. 3a), although diffuse or mixed localized–diffuse patterns have also been described [12, 41]. It may initially appear as a non-scarring anagen effluvium, but when follicular injury exceeds a critical threshold, permanent follicular loss and scarring ensue [12, 41].

Trichoscopy of pRIA shows white patches, reduced hair shaft caliber, and decreased hair density (Fig. 3b) [41].

Histologically, pRIA shows a reduced number of terminal follicles, dermal fibrosis, and vascular damage, supporting an irreversible cicatricial process [12, 41].

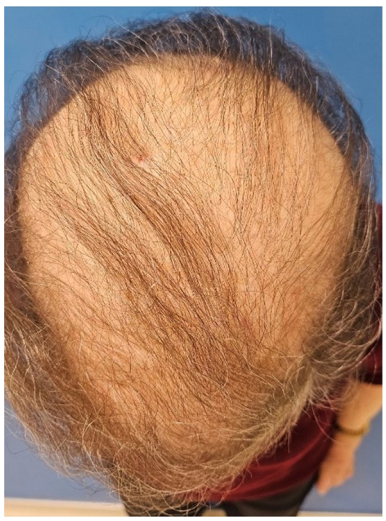

Fig. 2 Persistent endocrine therapy-induced alopecia due to long-term letrozole treatment. Personal files

From a therapeutic perspective, topical minoxidil, useful also for its antifibrotic power, may induce partial improvement by stimulating residual follicular activity, although responses are limited when scarring is established [41]. Surgical approaches including hair transplantation and plastic surgical reconstruction were reported to achieve clinical benefit in nonresponders, although evidence is mainly limited to small case series and expert experience [12, 41, 42]. Camouflage and prosthetic solutions are also important, including wigs, hair pieces, and scalp micropigmentation (tricopigmentation), especially when surgery is not feasible or the patient prefers non-surgical strategies [12, 43].

2.4 Scarring Alopecia

Scarring alopecia associated with targeted therapies is a rare but severe complication that usually develops secondary to inflammatory scalp disorders rather than as a direct toxic effect on the hair follicle [12, 43, 44]. Among targeted drugs, EGFRis are most frequently implicated through the induction of erosive pustular dermatosis of the scalp (EPDS) or folliculitis decalvans–like reactions [45, 46]. Pathogenetically, epidermal growth factor receptor (EGFR) blockade interferes with keratinocyte differentiation and follicular integrity, predisposing the scalp to abnormal keratinization, inflammation, and secondary infection that amplify follicular destruction [38, 46, 47].

EGFRi-induced EPDS typically presents with erosions, thick yellow-brown crusts, pustules, and progressive scalp atrophy, ultimately leading to scarring alopecia if not promptly treated [45, 48].

Early recognition of the inflammatory phase is crucial, because prompt treatment can arrest disease progression and limit cicatricial sequelae [45–47, 49–51]. Once scarring alopecia is established, therapeutic options are limited to stabilization and aesthetic restoration, including camouflage, scalp micropigmentation, or reconstructive surgery in selected stable cases [12, 43]. Current management

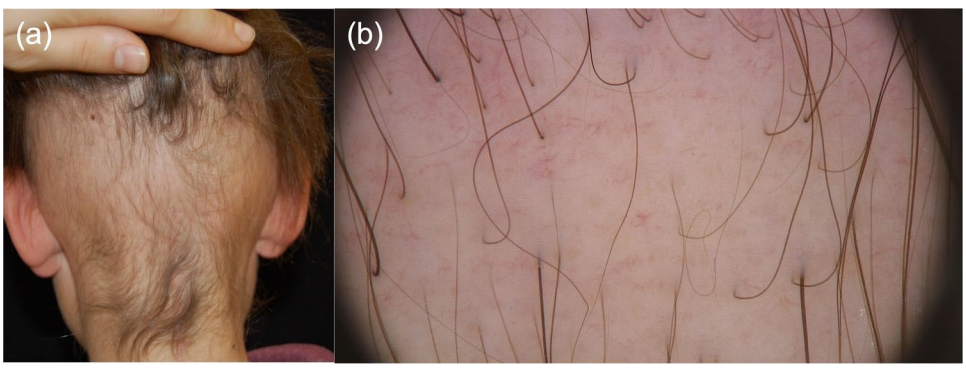

Fig. 3 Persistent radiation-induced alopecia (pRIA). Sharply demarcated alopecic patch corresponding to the irradiated field (**a**). Trichoscopy of pRIA shows white patches, reduced hair shaft caliber, and decreased hair density (**b**). Personal files

strategies are largely based on case reports and small case series and extrapolation from idiopathic cicatricial alopecias.

2.5 Hair Growth Disorders: Hirsutism and Hypertrichosis

With the increasing survival of patients with cancer, hair growth disorders such as hirsutism or hypertrichosis are being increasingly recognized as long-term dermatological sequelae of oncological therapies [12, 43, 52, 53]. Hirsutism is defined as androgen-dependent terminal hair growth in a male-pattern distribution in women, typical of patients under endocrine therapies through hormonal imbalance mechanisms [24, 38, 53], whereas hypertrichosis refers to androgen-independent excessive hair growth affecting non-sex-dependent areas of the body [54, 55]. Clinically, hirsutism associated with endocrine therapy typically presents as mild to moderate terminal hair growth in androgen-dependent areas, including the upper lip, chin, jawline, sideburns, and occasionally the lower abdomen, reflecting a male-pattern distribution, and develops gradually over months of treatment, often in parallel with scalp hair thinning or alopecia [12]. This condition is more frequently observed in postmenopausal women receiving aromatase inhibitors or selective estrogen receptor (ER) modulators, and is thought to result from estrogen deprivation with relative androgen predominance at the hair follicle level [38]. Although usually not associated with systemic hyperandrogenism, this form of hirsutism may be clinically distressing due to its facial localization and persistence during long-term endocrine therapy [24].

Facial hypertrichosis associated with EGFRis presents as excessive growth of pigmented vellus or terminal hairs, predominantly involving the malar areas, upper lip, and chin [56, 57]. Trichomegaly is characterized by abnormal elongation, thickening, increased curling, and pigmentation of eyelashes, often associated with targeted anticancer therapies, such as EGFRis, fibroblast growth factor receptor (FGFR) inhibitors and isocitrate dehydrogenase (IDH) inhibitors [45, 52, 56, 58–60]. In many cancer survivors, these hair changes may persist for months or years due to prolonged targeted therapy exposure or incomplete reversibility after treatment discontinuation [12, 43].

Although not life-threatening, hirsutism and hypertrichosis have a significant negative impact on body image, self-esteem, and social interactions, particularly in female patients [24, 43]. Facial hair overgrowth is often perceived as stigmatizing and can act as a persistent visual reminder in cancer survivors [12, 43]. Cosmetic approaches such as depilatory creams and laser hair removal are commonly recommended [12, 55, 56, 61]. Topical eflornithine cream has demonstrated efficacy and good tolerability in reducing facial hair growth, with a positive impact on patient-reported quality of life, although data specifically addressing cancer survivors remain limited [24, 62]. Spironolactone up to 200 mg/day for hirsutism could also be considered [3, 24].

2.6 Chronic Nail Changes

Chronic nail toxicities represent a relevant long-term sequela in cancer survivors because nail growth is slow and damage to the nail unit may persist for months after discontinuation of anticancer therapy [3, 63–65]. From a pathogenetic perspective, persistent nail abnormalities mainly result from direct cytotoxic damage to nail matrix keratinocytes and from inflammatory or vascular injury to the nail bed and periungual tissues [66–68]. Because the nail plate reflects past matrix injury, clinical manifestations may appear delayed and persist even when the causative drug has already been withdrawn (Fig. 4) [63, 69].

Chronic onycholysis is the most characteristic long-lasting nail change associated with taxanes, particularly docetaxel and paclitaxel; however, despite its frequent occurrence in daily clinical practice, it remains poorly described and underreported in the literature and may persist as a long-term sequela after treatment cessation [63, 67, 70–73]. When taxane-induced onycholysis is severe, repeated nail bed exposure may lead to abnormal re-epithelialization and chronic nail dystrophy that does not fully resolve after chemotherapy discontinuation [63, 66, 67, 71]. Persistent onycholysis is also reported with pan-FGFR inhibitors [58]. In this context, management focuses on trimming detached nail plates, protecting the nail bed, and treating superinfections, especially those due to *Pseudomonas aeruginosa* colonization [63, 67, 70]. Preventive strategies include cryotherapy with frozen gloves during taxane infusion, which significantly reduces the incidence and severity of nail toxicity, and regular application of hydrating nail solutions [74, 75].

Persistent nail fragility is a frequent complaint after chemo and targeted therapy, and is characterized by thinning, splitting, lamellar onychoschizia, and reduced nail plate elasticity [76, 77]. This condition is commonly associated with taxanes, platinum drugs, anthracyclines, CDK4/6 inhibitors and tyrosine kinase inhibitors [63, 67–69, 78]. Long-term management includes avoidance of trauma, regular nail trimming, use of emollients on nail folds, and application of nail-strengthening lacquers such as hydroxypropyl chitosan formulations. Most recommendations are based on expert consensus and extrapolation from general dermatological practice [63, 76, 79, 80].

Chronic nail dystrophy may persist after repeated episodes of paronychia, particularly in patients treated with EGFRis or chemotherapy [79, 81, 82]. Persistent inflammation of the proximal nail fold can permanently impair matrix function, leading to ridging, thickening, and irregular

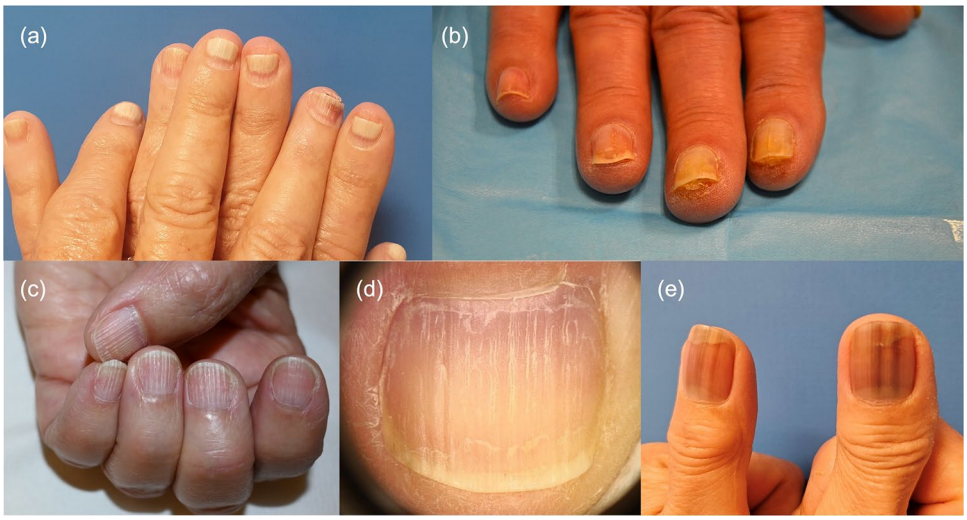

Fig. 4 Persistent onycholysis after taxanes (**a**, **b**). Persistent nail fragility (**c**, **d**). Persistent benign melanocyte activation of the nail matrix (longitudinal melanonychia) (**e**). Personal files

nail growth even after drug discontinuation [63, 65]. Early management of paronychia is therefore crucial to prevent long-term dystrophic sequelae [44, 81].

Nail involvement is also frequent in hematopoietic stem cell transplantation (HSCT) survivors, particularly in association with chronic graft-versus-host disease (cGVHD), and includes nail plate thinning, ridging, dorsal pterygium, and dystrophic changes, which may persist even after systemic disease stabilization [83, 84]. Management is mainly supportive, focusing on nail protection and treatment of associated cGVHD [84].

Chronic nail sequelae significantly impair quality of life in cancer survivors by causing cosmetic distress and anxiety related to incomplete recovery after cancer treatment [63, 64].

3 Persistent Pigmentary and Vascular Alterations

3.1 Persistent Hyperpigmentation

Persistent hyperpigmentation is a frequent survivorship sequela that may remain months to years after oncological treatments because pigment can be deposited in the dermis (melanin, melanophages, or other deposits) and is slowly cleared, particularly when inflammation and fibrosis coexist (Fig. 5) [3, 85]. However, the clinical course of treatment-related pigmentary alterations is variable. In some patients, gradual spontaneous fading may occur over months or years, whereas in others, dermal pigment deposition or associated fibrosis may lead to long-lasting or permanent discoloration.

In cancer survivors, long-lasting hyperpigmentation may follow radiotherapy, often developing after clinically evident radiation dermatitis and persisting with textural changes, atrophy, or fibrosis in the irradiated field [86–88].

Targeted therapies can also be associated with persistent or recurrent hyperpigmented patterns (often post-inflammatory or drug related), and pigmentary changes are described across multiple drug classes used for long periods in advanced disease (e.g., EGFRis), making persistence more relevant in survivors [60, 81, 89].

A paradigmatic chemotherapy-related example is bleomycin-induced flagellate pigmentation, which can leave prolonged hyperpigmented streaks even after discontinuation [90, 91]. Taxane regimens may contribute to pigmentary sequelae through inflammatory skin reactions and post-inflammatory hyperpigmentation. However, pigmentary sequelae are not limited to taxanes, as virtually any inflammatory or toxic cutaneous event occurring during chemotherapy may lead to persistent hyperpigmented lesions, due to chemotherapy-induced stimulation of melanogenesis, including toxic erythema of chemotherapy, repeated microtrauma, electrocardiographic electrode sites, phototoxic reactions, hand–foot syndrome, and "busulfan tan" [57, 71, 92–95].

The burden of persistent hyperpigmentation is not only cosmetic, because patient-perceived appearance changes can associate with negative symptoms and reduced quality of life, especially when lesions are visible [96, 97]. Hyperpigmentation induced by radiation therapy for breast cancer is considered as the most uncomfortable adverse event (before erythema), especially since it can persist for months or years after the end of treatment [98].

Management in long-term survivors is focused on strict photoprotection, trigger control (ongoing inflammation, friction, photosensitization), and evidence-based topical depigmenting agents (e.g., products containing hydroquinone 2–4%, retinoic acid, azelaic acid 5–10%, peeling agents, and corticosteroids), although evidence supporting their use specifically in cancer survivors is limited and largely extrapolated from studies in other pigmentary disorders [85, 99,

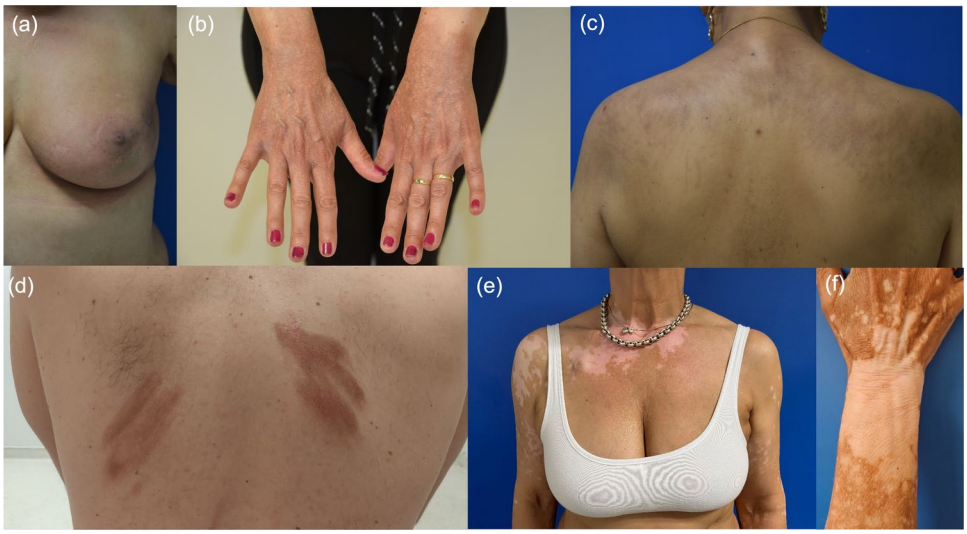

Fig. 5 Persistent pigmentary alterations. Hyperpigmentation secondary to breast radiotherapy (**a**), post-inflammatory hyperpigmentation following a toxic reaction due to chemotherapy (**b**), and bleomycin-induced flagellate hyperpigmentation (**c**, **d**). Vitiligo-like depigmentation associated with CDK4/6 inhibitors (**e**) and immunotherapy (**f**). Personal files

100]. For selected patients with persistent hyperpigmented lesions, laser therapy (e.g., Q-switched alexandrite, ruby, and Nd:YAG laser) can be considered [3, 101].

3.2 Persistent Hypopigmentation

Persistent hypopigmentation in cancer survivors is most classically represented by vitiligo-like depigmentation induced by immune checkpoint inhibitors, which may remain long after treatment cessation and therefore functions as a chronic sequela rather than a transient manifestation [6, 89, 102–106]. In melanoma, vitiligo-like depigmentation has been associated with tumor response and outcomes, so, for survivors, the depigmentation can carry both positive prognostic meaning and long-term psychosocial burden [89, 102, 107].

Persistent hypopigmentation is also a recognized pigmentary sequela in cancer survivors treated with CDK4/6 inhibitors (mainly ribociclib), and multi-kinase inhibitors targeting c-KIT protein, particularly in patients exposed for long periods and in those who develop stable residual depigmentation after drug discontinuation [3, 89, 108].

Radiotherapy can also produce chronic pigmentary sequelae, including hypopigmented or depigmented patches within irradiated fields, especially when fibrosis, atrophy, or vascular alteration coexist [86, 87, 109].

The quality-of-life impact is substantial because hypopigmentation is highly visible and may affect identity and social interactions, and patient-perceived cosmetic changes can be linked to emotional distress, which is particularly relevant in long-term survivorship [96].

Treatment in survivors should be individualized and realistic, separating active inflammatory depigmentation (where topical corticosteroids or topical calcineurin inhibitors may help) from stable sequelae (where camouflage and acceptance-focused counseling may be more appropriate) [105, 110]. In this context, the use of systemic Janus kinase (JAK) inhibitors in cancer survivors requires particular caution, especially in patients previously treated with immune checkpoint inhibitors, given theoretical concerns regarding immunomodulation and malignancy risk [111]. By contrast, topical ruxolitinib results in minimal systemic exposure, and its oncological risk profile cannot be directly extrapolated from systemic data, and no specific warning signal has emerged regarding the occurrence of secondary skin neoplasms, including in higher-risk populations [112–114].

When repigmentation is feasible, light-based approaches (e.g., phototherapy and laser approaches used in pigment disorders) could be considered in expert centers, although supporting evidence in cancer survivors remains scarce [100, 101, 115].

3.3 Telangiectasias

Chronic telangiectasias are a well-recognized cutaneous sequela in cancer survivors and are most frequently associated with radiotherapy, particularly in patients treated for breast cancer and head and neck malignancies [86, 87]. These vascular alterations typically appear months to years after radiotherapy completion and persist as part of chronic radiation dermatitis (Fig. 6) [116]. Unlike transient vascular reactions observed during treatment, radiation-induced telangiectasias rarely regress spontaneously and therefore represent a stable late sequela in many survivors [86, 87]. Beyond radiotherapy, targeted therapies, especially multi-kinase inhibitors and EGFRis, antibody–drug conjugates such as T-DM1 and, to a lesser extent, chemotherapy agents, have been reported to induce or aggravate chronic vascular

and skin changes, including telangiectasias, particularly in patients receiving long-term treatment [60, 117, 118].

From a survivorship perspective, telangiectasias are clinically relevant because they are highly visible, often irreversible, and may represent a very impactful reminder of cancer treatment [3].

The treatment of chronic radiation-induced telangiectasias is mainly restorative, with vascular laser therapy, particularly pulsed dye laser, generally considered the preferred option [119]. Importantly, laser treatment of telangiectasias has been shown to significantly improve dermatology-specific quality of life, supporting active intervention even when lesions are not symptomatic [120].

3.4 Hot Flushes

Long-term hormonal deprivation (aromatase inhibitors, selective estrogen receptor modulators [SERMs] such as tamoxifen, gonadotropin-releasing hormone (GnRH) agonists for ovarian suppression, and androgen deprivation therapy in prostate cancer) can produce recurrent flushing that may persist for years because these treatments are often continued for long periods in survivors [24, 121]. Clinically, hot flashes are typically sudden waves of heat with facial, neck, and upper-chest erythema, sweating, and sometimes palpitations, nausea, or anxiety, and in men on androgen deprivation therapy, they may remain chronic and peak early after starting treatment [122]. In breast cancer survivorship, flushing is common during endocrine therapy, and trials reported hot flashes in approximately 30–50% of patients depending on the agent, supporting that this symptom can be very frequent and clinically relevant also beyond the acute setting [24]. It is also important to remember that in breast cancer patients (especially ER-positive disease), estrogen replacement is generally contraindicated [24]. In prostate cancer, hot flashes affect a large proportion of patients on androgen deprivation therapy (approximately 58–80%), and a meaningful subset describes them as the most important adverse quality-of-life effect, which is relevant in patients exposed for long time [24, 122].

Chronic vasomotor symptoms matter because severity correlates with worse perceived quality of life, sleep disturbance, pain, and psychological functioning, and they may even contribute to endocrine-therapy discontinuation in some patients, creating a survivorship management priority [24, 121].

For long-term management, a practical first step is to identify and reduce triggers (heat, alcohol, caffeine, smoking, stress) and use behavioral strategies (layers, breathable fabrics), with symptom diaries to monitor persistence and response over time [24]. When lifestyle measures are not sufficient in survivors, non-hormonal systemic options can be considered (e.g., selective serotonin reuptake inhibitors [SSRIs], serotonin-norepinephrine reuptake inhibitors [SNRIs], gabapentinoids, anticholinergics, alpha-adrenergic agonists, beta-blockers), and some regimens (venlafaxine, paroxetine, gabapentin, pregabalin) have been associated with approximately 55–60% reductions in hot flashes, while citalopram/fluoxetine may reduce by 50% of cases [24].

In parallel, repeated flushing episodes can overlap with or exacerbate rosacea, where survivors present persistent centrofacial erythema and flushing, possible telangiectasias, papules/pustules, burning/stinging, dryness, and ocular complaints, often triggered by heat, stress, spicy foods, hot drinks, or alcohol [123].

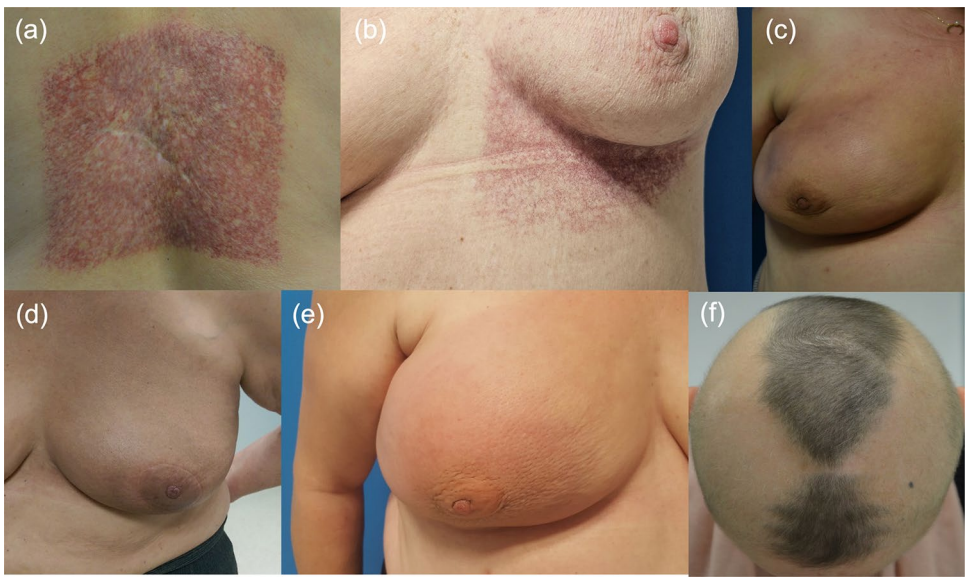

Fig. 6 Chronic radiation dermatitis. Persistent telangiectasia in the previously irradiated area (**a**, **b**). Pigmentation alteration and fibrosis (**c**, **d**, **e**), and alopecia (**f**). Personal files

4 Chronic Alterations in Skin Texture

4.1 Chronic Skin Texture Changes

Chronic skin texture changes represent a common and often underestimated sequela in cancer survivors and include atrophy, persistent roughness, and reduced skin elasticity [86, 87]. These alterations are frequently observed after radiotherapy and can persist indefinitely after treatment completion [116]. In addition to radiotherapy, targeted therapies, particularly EGFRis, are associated with chronic barrier dysfunction, xerosis, and long-lasting rough skin texture [60, 124]. Endocrine therapies and estrogen deprivation represent a highly relevant contributor to chronic skin thinning, persistent dryness, loss of elasticity, and impaired repair mechanisms, markedly amplifying texture-related complaints and accelerating cutaneous aging processes in long responders [125]. Patients treated with HSCT frequently develop chronic dermal or epidermal changes, largely driven by cGVHD, which represents one of the main causes of late morbidity after allogeneic transplantation. Cutaneous cGVHD may manifest with lichenoid, sclerodermoid, or poikilodermatous changes and is often associated with progressive fibrosis, skin atrophy, pigmentary alterations, and impaired barrier function (Fig. 7), leading to functional limitation and reduced quality of life [83, 126].

In survivorship care, management of chronic texture changes is mainly supportive and preventive, focusing on long-term use of emollients, barrier-repair products, and avoidance of irritants to improve comfort and skin function [24]. For selected patients with markedly altered texture, procedural approaches, such as reconstructive surgery procedures, dermal fillers, and laser treatments could be considered to improve skin pliability and appearance, although supporting evidence remains limited and largely derived from small studies or expert opinion [3]. Topical agents such as retinoids can be cautiously used in stable survivors to improve roughness and epidermal renewal, provided tolerability is adequate in previously irradiated or sensitized skin [127].

Chronic skin texture changes have a documented negative impact on long-term quality of life and body image, reinforcing the importance of proactive management of these sequelae within structured oncological survivorship programs [128].

4.2 Scars and Fibrotic Lesions

Long-term scars in cancer survivors derive mainly from oncological surgery and from radiotherapy [86]. Chronic scars may cause pain, pruritus, burning, hypoesthesia, and dysesthesia, with limitation of range of motion and daily activities [115, 129, 130]. Radiotherapy-related sequelae can also include chronic radiation dermatitis and radiation fibrosis, which are clinically relevant in survivorship because they develop or persist beyond the acute phase and can worsen with time [115, 131]. Hypertrophic scars and keloids represent a burdensome subset because they can be symptomatic, raised, itchy, painful, and sometimes restrictive, and they may persist for years after cancer surgery or trauma from repeated procedures [132, 133].

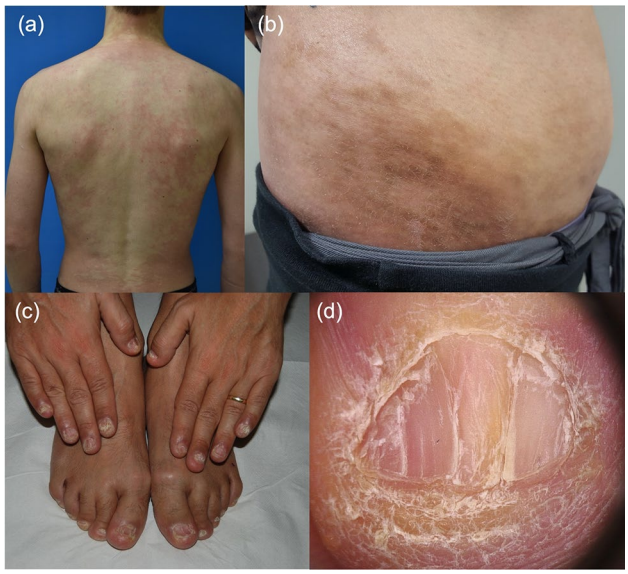

Fig. 7 Sclerotic skin changes associated with chronic graft-versus-host disease (cGVHD) involving the trunk in a childhood cancer survivor (**a**) and an adult (**b**). Lichenoid cGVHD affecting the nails (**c**, **d**). Personal files

The psychological impact is often heavy, because scars can affect identity, body image, sexuality, and relationships, and can induce self-consciousness and avoidance behaviors in survivorship [4, 86, 134, 135].

In particular, validated patient-reported outcome tools (e.g., FACE-Q) used in patients treated for facial skin cancers showed that appearance-related psychosocial distress is present especially early but may persist as self-consciousness in a subset, supporting targeted supportive interventions [136–138].

For long-term management, first-line survivorship measures are silicone gel or sheets, massage, and photoprotection to optimize maturation [139]. When scars remain symptomatic or cosmetically distressing, options include intralesional corticosteroids, plastic surgical revision for selected mature scars, and lasers, tailored to scar type [3, 132, 139]. For hypertrophic and restrictive scars, fractional ablative laser can improve texture and function, and laser-assisted delivery of topical corticosteroids (e.g., triamcinolone acetonide) has been described as a multimodal approach to improve difficult scars [132, 133].

For radiation-related fibrosis, survivorship-oriented guidance supports a multidisciplinary approach (skin care, rehabilitation, physiotherapy, symptom control). Specific systemic options, such as pentoxifylline 400–800 mg/day and/or vitamin E 1000 IU/day administered for at least 6 months, have been proposed for the management of radiation-induced fibrosis in late effects; however, their clinical effectiveness remains controversial [131, 140, 141].

Because chronic radiation dermatitis and fibrosis are common but heterogeneous, consensus-based survivorship recommendations emphasize structured assessment and longitudinal follow-up to select the right combination of topical care, procedural interventions, and rehabilitation strategies [3, 115, 142]. Finally, survivorship care should not underestimate scars as only aesthetic concern, because appearance-related ones and chronic symptoms can reduce social functioning and overall quality of life, and addressing scars can be part of normalization after cancer [7, 137].

Figure 8 illustrates a simplified decision-making algorithm for the potential use of laser therapies in restorative dermatology, summarizing the main laser approaches described in the literature for selected clinical indications. These proposals are derived from available literature, mostly limited to small studies and case series, and should therefore be interpreted as exploratory approaches rather than guideline-based recommendations.

5 Mucosal Sequelae

Oral sequelae are frequent but underestimated complications of cancer treatments, affecting both adult and childhood survivors and significantly impairing quality of life [143–146]. Xerostomia and salivary gland dysfunction are common long-term effects, particularly after head and neck radiation therapy, chemotherapy, endocrine, targeted and immune therapies, or HSCT (Fig. 9) [83, 143, 144, 146–149]. Dry mouth has been reported in up to 67% of head and neck cancer survivors [148], and increases the risk of dental decay, periodontal disease and osteonecrosis of the jaw [144, 147]. Salivary alterations also lead to mucosal sensitivity, infections, taste disorders, sleep disturbances, and difficulties in chewing, swallowing, and speaking [143, 144]. Persistent taste changes occur in 11–60% of patients depending on cancer type and treatment [150].

Preventive strategies for dry mouth and taste changes rely on baseline oral assessment before cancer therapy and implementation of oral hygiene protocols. Management is mainly symptomatic and multidisciplinary, including saliva substitutes or stimulants for xerostomia and dietary counseling for taste disorders [150–152].

Oral mucosal pigmentations are frequently associated with chemotherapy or targeted therapies and, although benign, may cause patient anxiety and require follow-up [152]. Patient education and integration of oral medicine into survivorship care pathways are essential to optimize outcomes and quality of life [153].

Finally, vulvovaginal atrophy (with loss of elasticity), insufficient lubrification, dyspareunia, and genitourinary syndrome are frequently reported in female cancer survivors, particularly after chemotherapy, pelvic radiotherapy, and prolonged exposure to endocrine therapies [121]. Non-hormonal local therapies such as vaginal moisturizers and lubricants represent the first-line supportive approach in many cancer survivors. In this context, topical formulations containing hyaluronic acid have shown potential benefits in improving vaginal dryness, inflammation, and dyspareunia in women receiving radiotherapy for gynecological cancers,

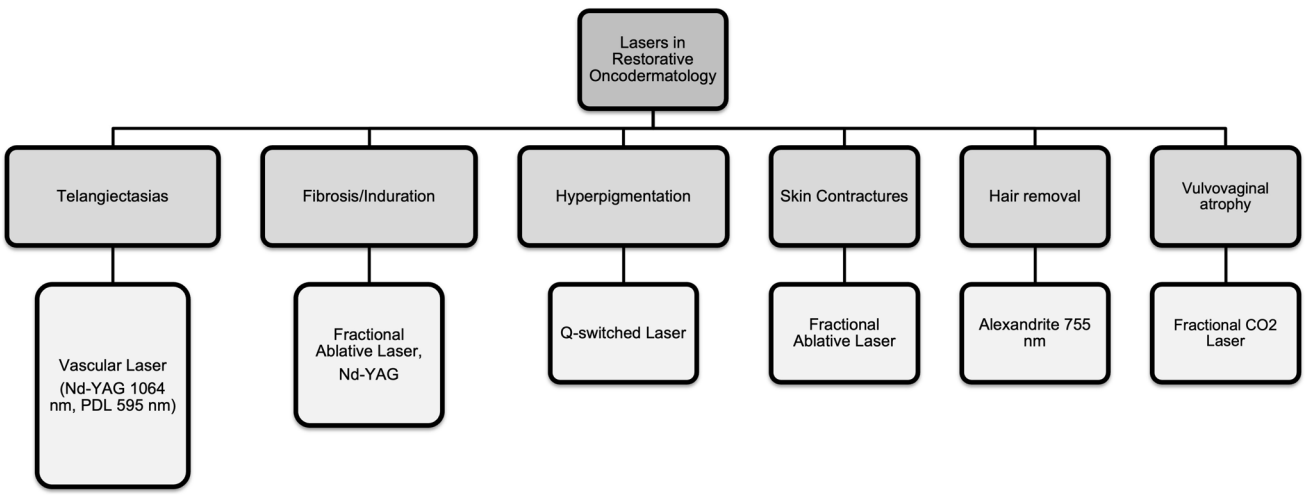

Fig. 8 Proposal for decision algorithm for laser treatment in restorative dermatology, with the most suitable specific laser technologies

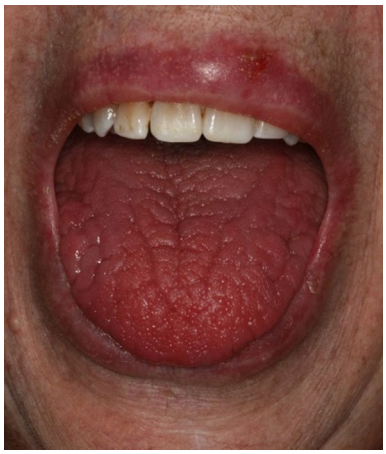

Fig. 9 Persistent grade 2 xerostomia following anti-programmed cell death protein 1 (anti-PD-1) therapy. Personal files

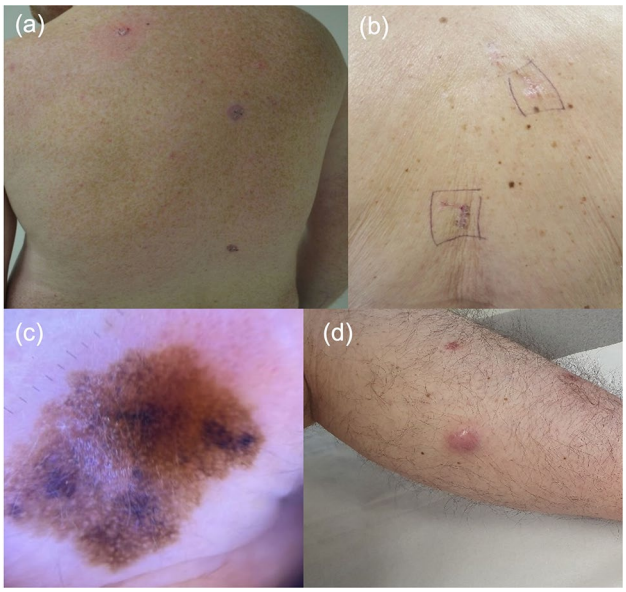

Fig. 10 Second cutaneous malignancies in long-term cancer survivors: basal cell carcinoma (**a**, **b**), melanoma (**c**), and Merkel cell carcinoma (**d**). Personal files

likely due to their hydrating and tissue-repair properties; however, available data are heterogeneous and mainly derived from small clinical trials or observational studies, supporting moderate-to-limited evidence for their use in this setting [154, 155]. Laser therapies have also been proposed as non-hormonal options. Fractional CO_2 laser therapy has been investigated to improve vaginal elasticity, lubrication, and sexual function, with preliminary prospective and observational studies reporting symptomatic improvement in patients with genitourinary syndrome of menopause; nevertheless, current evidence remains limited and largely based on small or uncontrolled studies, and further randomized trials are required to confirm its long-term safety and efficacy [156–158]. Effective collaboration with specialized gynecological teams is absolutely essential in this context.

6 Secondary Skin Cancers

The development of secondary cutaneous malignancies (Fig. 10) is one of the most important long-term complications in cancer survivors and is strongly related to previous exposure to chemotherapy, radiotherapy, targeted therapies, and immunomodulating agents [9]. This risk is particularly relevant in childhood cancer survivors, who have a longer life expectancy and therefore a prolonged latency period during which therapy-related carcinogenic effects could emerge [159].

Radiotherapy represents one of the strongest factors of second cancer risk, with a clear dose–response relationship and higher susceptibility when exposure occurs at younger ages [87, 160]. In childhood cancer survivors, radiation-induced secondary tumors often arise within or adjacent to the irradiated field and mainly include non-melanoma skin cancers [161]. Cutaneous malignancies are among the most frequent therapy-related second cancers, particularly basal cell carcinoma and squamous cell carcinoma, which may develop after a long latency period, often exceeding 10–15 years and sometimes occurring decades after radiotherapy or combined modality treatment [86, 160, 161]. Post-radiotherapy angiosarcoma is another rare late complication arising in previously irradiated areas, particularly in breast cancer patients, where lymphoedema represents the main etiological factor [162].

Chemotherapeutic and immunosuppressive agents (e.g., hydroxyurea, alkylating agents, fludarabine, and methotrexate) have also been associated with long-term oncogenic risk [163–167].

Immunosuppressive states due to the use of long-term targeted therapies, including JAK inhibitors such as ruxolitinib, have also been associated with an increased risk of second primary malignancies [9, 168–170]. Importantly, most data suggesting an increased risk of non-melanoma skin cancer derive from patients receiving systemic ruxolitinib for myeloproliferative neoplasms [168–170], a population characterized by advanced age, cumulative prior therapies, and disease-related immune dysregulation, all of which may independently contribute to carcinogenesis. Therefore, a direct causal relationship with the drug itself cannot always be clearly distinguished from underlying disease-related risk factors [168, 170].

Given the prolonged latency and the cumulative risk [9, 87, 160], long-term dermatological surveillance is essential in cancer survivors. Periodic full-body skin examinations should be recommended, particularly in patients previously

exposed to radiotherapy (with careful inspection of irradiated fields), those treated during childhood or adolescence [161], and individuals receiving prolonged immunosuppressive therapies [166, 167]. Surveillance should begin during survivorship follow-up and continue lifelong in high-risk populations, according to a frequency determined by the specialist.

7 Sequelae in Childhood Cancer Survivors

In a cohort of adult survivors of childhood cancer, dermatological conditions were reported by nearly 60% of patients [7].

Biologically, anticancer therapies administered during periods of active growth may alter stem cell reserve and tissue homeostasis, leading to reduced regenerative capacity and progressive organ vulnerability across the life course, thereby contributing to the high cumulative prevalence of chronic conditions documented in long-term survivors [171].

In pediatric patients, alopecia induced by chemotherapy, radiotherapy, or HSCT is usually transient; however, pCIA has been reported in approximately 12–15% of childhood cancer survivors, especially following conditioning treatments with thiotepa and busulfan [4, 37, 172]. In children, pCIA most frequently presents as diffuse thinning rather than patterned hair loss and may be under-recognized due to limited long-term dermatological follow-up [4, 37, 172]. Childhood cancer survivors with pCIA experience significant impairment in quality of life, social integration, and emotional well-being, with psychosocial consequences persisting into adulthood [4, 37]. The treatment of this condition in children and adolescents is similar to that in adults [10, 173].

Beyond alopecia, childhood cancer survivors frequently experience a broad spectrum of chronic dermatological sequelae, including permanent scarring, pigmentary alterations, and skin fibrosis [4, 7, 174]. These visible and often permanent cutaneous sequelae have a strong impact on body image development, particularly when cancer treatment occurs during critical periods of childhood and adolescence, a phase in which physical appearance plays a central role in identity formation and social integration [175]. Long-term cohort data from the Childhood Cancer Survivor Study demonstrate that adult survivors report significantly lower mental health–related quality of life compared with the general population [176]. Similarly, large survivorship analyses have shown increased global distress and depressive symptoms associated with impaired social functioning in survivors of childhood malignancies [177]. Importantly, visible treatment-related sequelae such as scarring, disfigurement, and persistent hair loss may negatively influence body image development, which in turn mediates psychological distress and post-traumatic stress symptoms in adult survivors [171, 178].

In pediatric and adolescent survivors, cumulative exposure to multiple therapeutic modalities and individual genetic susceptibility further amplify the lifetime risk of second cancers [4, 7, 179, 180]. Longitudinal data from the Childhood Cancer Survivor Study demonstrate that late mortality and treatment-related deaths, including those due to subsequent neoplasms, continue to increase decades after diagnosis [180]. These considerations underline the importance of lifelong surveillance programs, including dermatological screening, particularly for patients treated during childhood [9, 181, 182].

8 Vitamin and Micronutrients Deficiencies

Vitamin and micronutrients deficiencies represent a common and clinically relevant long-term sequela in cancer survivors and arise from multiple interacting mechanisms, including chemotherapy- and radiotherapy-induced malabsorption, chronic gastrointestinal toxicity, pancreatic insufficiency, reduced dietary intake, and long-lasting metabolic and inflammatory alterations [183, 184]. Food insecurity and persistent nutritional challenges have also been described in adolescent and young adult cancer patients, further contributing to inadequate micronutrient intake years after treatment completion [185].

Deficiencies of vitamin D, vitamin B12, folate, iron, and fat-soluble vitamins are frequently reported, particularly in survivors of gastrointestinal, pancreatic, testicular, and pediatric cancers, and may contribute to chronic fatigue, osteoporosis, neuropathy, cognitive impairment, and impaired immune function [186–188]. From a dermatological perspective, micronutrient deficiencies may also manifest with characteristic skin, hair, and nail findings that can serve as early clinical clues in cancer survivors, especially in those with malabsorption or restrictive dietary intake. Reported manifestations include xerosis and follicular hyperkeratosis (including phrynoderma), angular cheilitis and glossitis associated with B-vitamin deficiencies, pellagra-like photosensitive dermatitis in niacin deficiency, and perifollicular petechiae in vitamin C deficiency [189, 190]. Hair and nail changes may also occur, including diffuse alopecia associated with iron or zinc deficiency, "corkscrew hairs" in vitamin C deficiency, and nail fragility or koilonychia in iron deficiency [189, 190].

In pediatric cancer survivors, nutritional deficiencies may persist into adulthood and interact with other late effects, amplifying the burden of chronic health conditions [188, 191]. Therefore, dermatologists involved in survivorship care should consider nutritional deficiencies in the differential diagnosis of unexplained mucocutaneous findings and

Table 2 Proposed algorithm for dermatological survivorship assessment in cancer survivors

Cancer survivor referred for dermatological assessment

Step 1. Treatment history and risk stratification

- Prior anticancer treatments: surgery, chemotherapy, radiotherapy, targeted therapy, immunotherapy, endocrine therapy, HSCT, cGVHD history, etc.
- Timing from treatment completion
- Current cancer status and ongoing therapies
- Risk factors for secondary skin cancers
- Relevant comorbidities and nutritional risk

Step 2. Identify the main dermatological survivorship issue(s)

- Hair disorders: persistent alopecia, hypertrichosis, hirsutism, scarring alopecia
- Nail disorders: persistent onycholysis, fragility, chronic paronychia, nail dystrophies
- Pigmentary sequelae: hyperpigmentation, hypopigmentation
- Vascular and texture changes: telangiectasias, fibrosis, chronic radiation dermatitis, xerosis, atrophy
- Scars and functional impairment
- Mucosal sequelae: xerostomia, oral sensitivity, vulvovaginal atrophy
- Suspicious lesions or secondary skin cancers
- Possible nutritional deficiency signs

Step 3. Assess clinical priority

- Urgent/high priority: suspicious skin cancer, rapidly progressive lesion, severe fibrosis, contracture severe pain, infection, bleeding, ulceration, cGVHD
- Non-urgent but with negative impact on patients quality of life or social interaction: alopecia, facial pigmentary changes, visible scars, telangiectasias, chronic nail alterations, mucosal symptoms

Step 4. Dermatological evaluation

- Full dermatological examination: skin, hair, nail, and mucosal
- Dermoscopy for suspicious lesions
- Trichoscopy and/or onychoscopy when indicated
- Biopsy, imaging, laboratory tests when indicated
- Evaluate functional impairment, symptoms, psychosocial distress, body image impact, quality-of-life burden

Step 5. Management

A Surveillance and prevention
- Skin cancer screening
- Photoprotection
- Education on self-examination
- Long-term follow-up scheduling

B Supportive medical treatment
- Skin, hair, nails, mucosal sequelae
- Nutritional supplementation if indicated

C Restorative and procedural treatment
- Laser therapy
- Scar management
- Fillers, peeling if appropriate
- Reconstructive or surgical referral

D Multidisciplinary referral
- Dermatologist
- Oncologist
- Radiotherapist
- Oral medicine specialist
- Gynecologist
- Plastic surgeon
- Psychologist
- Physiatrist, physiotherapist
- Nutritionist
- Specialized nurse

Step 6. Long-term follow-up

- Monitor improvement, stability, or progression of sequelae
- Reassess quality-of-life and body image burden
- Adapt treatment according to survivorship phase
- Continue surveillance for secondary skin cancers and late toxicities

cGVHD chronic graft-versus-host disease, *HSCT* hematopoietic stem cell transplantation

collaborate with oncologists and primary care providers in targeted laboratory evaluation. In survivors at higher risk of malabsorption, such as those who underwent gastrointestinal surgery or experience chronic gastrointestinal toxicity, periodic monitoring of key micronutrients (particularly vitamin B12, iron, and vitamin D) may be appropriate according to survivorship recommendations [191]. Long-term survivorship care should therefore include structured nutritional surveillance and individualized supplementation strategies, preferably guided by laboratory evaluation, since evidence supporting routine supplementation in cancer survivors remains limited and largely observational. Avoid indiscriminate

vitamin use, as excessive or inappropriate supplementation has been associated with potential adverse oncological outcomes [192, 193].

9 Conclusion

Dermatological sequelae represent a significant burden in cancer survivors, persisting long after the end of oncological treatments [3]. Persistent alopecia, nail alterations, pigmentary disorders, vascular changes, scars, and secondary skin cancers can substantially impair quality of life and contribute to long-lasting psychological distress [4]. These sequelae are particularly impactful in childhood cancer survivors, who face prolonged exposure to the physical and emotional consequences of therapy-related skin damage over their lifetime [37, 159, 172]. A multidisciplinary survivorship approach, integrating dermatology in the oncological follow-up, is crucial to improve functional outcomes, aesthetic concerns, and overall well-being [3]. Table 2 summarizes a practical algorithm for dermatological survivorship assessment in cancer survivors. Finally, this must necessarily involve the creation of appropriate dermatological consultations that are entirely dedicated to the dermatological care of cancer survivors.

Future prospective studies and controlled trials are needed to better define the efficacy and safety of restorative dermatological interventions in cancer survivors, as current recommendations are often based on limited or heterogeneous evidence.

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Declarations

Conflict of Interest Luca Rapparini, Timila Assia Touhouche, Davide Fattore, Azael Freites-Martinez, Pauline Hirtz, Zoe Apalla, Emmanuelle Vigarios, Delphine Maret, Michela Starace, and Vincent Sibaud declare that they have no conflicts of interest that might be relevant to the contents of this article.

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent for Publication The patients, or patient's parents/guardians, in this article have given written informed consent for the publication of their photos, according to the Declaration of Helsinki principles.

Availability of Data and Material No datasets were generated or analyzed during the current study. A review of the literature was conducted using previously published data.

Code Availability Not applicable.

Authors' Contributions All authors contributed to the conception and drafting of the manuscript and provided clinical images. All authors critically revised the manuscript for important intellectual content and approved the final version of the manuscript.

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