Dermatology Practical & Conceptual

Assessing Treatment Response to PD-1 Inhibitors in Cutaneous Squamous Cell Carcinoma: Real-World Challenges

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ABSTRACT

Introduction: Cutaneous squamous cell carcinoma (cSCC) is a common nonmelanoma skin cancer primarily driven by chronic sun exposure and advanced age. Standard treatments include wide local excision, Mohs surgery, and radiotherapy, with advanced cases often managed with chemotherapeutics. PD1 inhibitors like cemiplimab and pembrolizumab are FDA-approved treatments that have shown efficacy in locally advanced or metastatic cSCC not amenable to surgery or radiation.

Objectives: This study evaluated clinical responses to these inhibitors using RECIST 1.1 criteria through direct visual examination, addressing the applicability of the criteria in real-world clinical practice.

Methods: A cohort of 12 patients with cSCC treated at Duke University with cemiplimab or pembrolizumab was identified through the DeDUCE database using ICD-10 codes. Clinical responses were determined using RECIST 1.1 and modified WHO criteria and compared against clinical-based assessments.

Results: Results indicated varied responses: seven patients with progressive disease (PD), two with stable disease (SD), and three with complete response (CR). Challenges included distinguishing true progression from reactive conditions like erosive pustular dermatosis (EPD) and pseudoprogression as well as evaluating partial responses in ulcerated lesions, which are not defined within the criteria. Radiologic findings often required corroboration with clinical evaluations to avoid misclassification.

Conclusions: Accurate clinical determination of cSCC response to PD-1 inhibitors is complex, requiring multidisciplinary approaches and meticulous wound care. Incorporating these clinical nuances into revised response criteria would enhance treatment decision-making, balancing the risks of premature discontinuation against prolonged ineffective therapy.

Introduction

Cutaneous squamous cell carcinoma (cSCC) is a nonmelanoma skin cancer with risk factors including chronic sun exposure and advanced age [1,2]. Most cases of cSCC are treated with wide local excision, Mohs surgery, or radiotherapy, though advanced cases of cSCC are treated using a number of chemotherapeutic agents [2]. First-line treatment for locally advanced cSCC is surgical excision or radiotherapy, though in recent years, PD1 inhibitors like cemiplimab and pembrolizumab have been shown to improve outcomes in advanced cases of cSCC and have been employed as the first-line standard of care for patients with locally advanced or metastatic cSCC not amenable to surgery or radiation [1,3]. In 2018, cemiplimab became an FDA-approved treatment for patients with metastatic cSCC or locally advanced cSCC who are ineligible for surgical resection or radiation [4]. In 2020, pembrolizumab received FDA approval for the same indication.[5] Response rates to anti-PD-1 therapies have been documented up to 49%, with many studies documenting its efficacy and safety [6-8].

The Response Evaluation Criteria in Solid Tumors (RECIST) is employed in clinical trials to evaluate tumor response to treatment [9]. These criteria, initially published in 2000, define tumor response based on the size of measurable lesions and on the number of lesions, among other criteria. RECIST was most recently updated in 2009 as RECIST 1.1, a modified version of the original criteria; they include imaging studies, assessment of lymph nodes, and changes to the definitions of disease progression [7]. The RECIST 1.1 criteria are frequently used in clinical trials to evaluate progression of disease, and in the case of cSCC, the criteria can be used in conjunction with clinical response evaluated by digital medical photography to determine a composite clinical response [9].

Objectives

In reviewing the RECIST 1.1 criteria and the composite clinical response used in a phase 2 trial of cemiplimab, we posed the question: Can we determine clinical responses to PD1 inhibitor therapy for cSCC via a direct visual examination using the RECIST criteria for real-world use? We sought to answer this question with a review of the clinical course of a cohort of 12 patients with cSCC treated with cemiplimab or pembrolizumab identified at the Dermatology Department of the Duke University School of Medicine. Clinical photography from before and after anti-PD1 treatment is presented herein alongside the clinical course of patients, displaying the challenges of determining response to treatment and the potential for misalignment of clinical response determined by RECIST 1.1 as compared to real-world clinical response.

Methods

Patients were identified through DeDUCE, a database from Duke University used to identify patients through ICD-10 diagnosis codes and medications, among other criteria. The initial search included all patients with ICD-10 codes for cSCC who also received cemiplimab or pembrolizumab. This search identified 211 patients. We excluded patients with squamous cell carcinoma in situ, resulting in a modified cohort of 146 patients. Further exclusion of patients not treated with cemiplimab or pembrolizumab resulted in a cohort of 69 patients. We selected 35 patients with cSCC by further excluding patients with mucosal squamous cell carcinoma, including cancers of oropharynx, tonsils, and tongue. Finally, we selected patients whose clinical response was determined by clinical photography alone (radiological response minimally or not contributory for response assessment due to skin-only disease) resulted in our final cohort of 12 patients.

The evaluation of these 12 patients included medical record review for demographic data, including treatment course details, imaging review, clinical response assessments by size and clinical course post-treatment, and final outcome from treatment. Through comparison of their clinical images and radiological findings, the RECIST 1.1 criteria were used to determine each patient's response to treatment. The RECIST 1.1-based response was then compared to the patient's clinical course to understand how applicable the scale is to real-world clinical practice.

Overview of RECIST 1.1 Criteria

We utilized the clinical response modified WHO criteria as determined by photography in the context of skin lesions evaluated as follows: complete response of externally visible disease (vCR) is defined as the disappearance of all target

lesions, for at least four weeks, with histological confirmation, vs partial response of externally visible disease (vPR), achieved with at least 50% decrease in sum of diameters of target lesions, for at least four weeks. Progression of visible disease (vPD) is defined as at least 25% increase in the sum of diameters of target lesions, with an absolute increase of at least 5 mm, and stable externally visible disease (vSD) is defined as meeting neither vCR, vPR, nor vPD criteria [10].

The modified WHO criteria for clinical response are determined by the above criteria in conjunction with the presence or absence of new lesions. Any of the above categories with new lesions is considered clinical progression of disease (cPD), meaning progression in patients' overall disease. This is important to note, since many cSCC patients can develop new primaries. Similarly, vCR with no new lesion is determined to be clinical complete response (cCR), as is true for vPR to cPR and vSD to cSD. vPD with or without new lesions is considered cPD. However, for ulcerated cSCC lesion, there are alternate response criteria utilized. Complete response is defined as re-epithelialization of the entire target lesion, for at least four weeks. Partial response has no criteria and therefore not a response that can be achieved for ulcerated lesions. Stable disease is defined as meeting neither complete response nor progressive disease criteria. Progressive disease is defined as new ulceration of target lesion(s) separate from tissue biopsy or known trauma, without healing for at least two weeks.

In determining the composite response for visual assessment, the clinical response as detailed above is combined with the RECIST 1.1 evaluated response, based on radiology. For our cohort of patients, radiological evaluation was not contributory due to locally advanced disease with negative initial staging, allowing us to understand the clinical utility of the clinical visual assessment criteria.

Results

The demographics of the 12 patients and their primary tumor site are summarized in Table 1.

For each patient, the number of cycles of pembrolizumab or cemiplimab, prior treatment, clinical response, determination of response criteria, clinical course, and current state are detailed in Table 2.

Based on the RECIST 1.1 clinical and composite criteria, our cohort had 7/12 patients with progressive disease, 2/12 with stable disease, and 3/12 with complete response. Of the seven PD patients, two had <6 cycles of treatment with hyper-progressive disease (Figure 1), and 2/7 PD had pseudoprogression with eschar formation (Figure 2) and are still alive, demonstrating ultimate positive response. Finally, 1/7 PD patient was deemed progressive due to a new lesion, confirmed to be new primary near the locally advanced cSCC site; PD-1 inhibitor was discontinued, and the new nodule treated and resolved. However, there was progression of the original lesion.

Table 1. Demographics and primary tumor site.

Demographics	
Age (years)	
Mean (Range)	77.6 (63-93)
Sex	
Male	12
Female	0
Race/Ethnicity	
White	11
Declined/Not Reported	1
Primary Tumor Site	
Scalp	6
Chest	2
Face	2
Neck	1
Back	1

In the stable disease group, all were ulcerated lesions that could only achieve best clinical response of stable disease. Both patients were previously radiated lesions.

Of the three complete responders, two did not have any prior treatment before PD-1 inhibitor therapy, and the third patient had a small <3 cm tumor with resolution within 10 cycles.

Discussion

Our study highlights the nuances of RECIST 1.1 clinical response criteria determination and summarizes clinical pearls for improved clinical decision-making regarding treatment guidance.

Eschars, Erosive Pustular Dermatosis (EPD), and Pseudoprogression

Clinicians must be aware of impressive crust and eschar formation with PD-1 inhibition secondary to erosive pustular dermatosis (EPD), an inflammatory disorder that leads to development of extensive pustular lesions, erosions, and crusts, sometimes due to underlying cSCC.[11] Implementation of meticulous wound care and anti-inflammatory treatment is central to achieving clearance of wound bed before making a clinical decision of progression of cSCC disease. In our cases, anti-PD-1 therapy was discontinued for two patients with EPD; however, both patients ultimately did well and remain alive, suggesting an overall positive outcome.

Pseudoprogression anti-PD-1 therapy has been documented and defined in the literature as patients with radiographic

Table 2. Clinical course and outcomes.

Patient Characteristics	# of Cycles	Prior Treatment	Clinical Response	Determination of Response Criteria	Clinical Course	Current State (2/2024)	Clinical Pearls
Progressive Disease							
86M **AJCC T4a / BWH T3** **N0M0** **Clinical location:** Scalp	6	Surgery only with resection down to bone and integra graft placement → recurrence → PD1 INHIBITOR	Pre-treatment: 4 cm lesion Posttreatment: >8 cm lesion with eschar	**Clinical:** PD **Radiological:** SD **Combined:** PD **Treatment Decision: D/c** cemiplimab	2 cycles of intralesional 5-FU injections X10 sites + daily vinegar soaks and 10 days of 500 mg cephalexin BID Bx showed no evidence of SCC 3 months	**Alive,** no evidence of disease progression as of 11/2023	Scalp eschar secondary to erosive pustular dermatosis. Antiproliferative therapy and wound care following cemiplimab needed for clearance. Biopsy can be helpful in indeterminate cases
86 M **AJCC T4 / BWH T3, N0** **Clinical location:** Scalp	6	Tumor abutting parietal skull without erosive change, but possibility of microscopic erosion; Treated with radiation → recurrence →PD1 INHIBITOR		**Clinical:** PD **Radiology:** PD **Combined:** PD **Treatment Decision:** D/c pembrolizumab	5 cycles of intralesional fluorouracil; vinegar soaks and cephalexin to clear eschar	**Alive,** no recurrence of SCC as of 12/2023	Eschar, EPD not true SCC based on milder clinical course, important to clear and biopsy/ monitor base of lesion Radiology can be misleading due to subcutaneous edema on scans without visualization
88M **AJCC T4a / BWH T3, N0M0** **Clinical location:** Scalp	12	Mohs → progression → RT → progression with inner cortex involvement on CT head→ cemiplimab	Clinical evaluation challenging due radiation necrosis until new nodule	**Clinical:** PD (new lesion, original lesion SD) **Radiology:** SD **Combined:** PD **Treatment decision: D/c** cemiplimab	Surgery for new primary cSCC <2 cm on the scalp	**Alive,** disease progression post-discontinuation of PD1 inhibitor	Original large lesion responding to PD-1 inhibition. PD based on clinical response criteria due to development of a new cSCC.
83M **AJCC T3 / BWH T2a** **N2bMx** **Clinical location:** Left axilla	4	Cetuximab, excision with wound vac placement → Recurrence → Radiation → PD1 INHIBITOR	Rapid growth of initial lesions misdiagnosed as cysts (subcutaneous nodules).	**Clinical:** PD **Radiology:** PD **Combined:** PD **Treatment Decision:** D/c Pembrolizumab	Clinical course showed progressive disease managed by panitumumab → Carboplatin/ paclitaxel → RT → NED	**Alive,** no evidence of recurrent or metastatic disease	Rapidly growing SCC nodules pose a challenge due to lack of enough cycles to see immunotherapy response.

72M **AJCC T3 / BWH T2a, N0, M1** **Clinical location:** Scalp and right neck	7	Surgical resection with craniectomy and flap reconstruction; RT; TVEC clinical trial with progression→ PD1 INHIBITOR	New nodule on right supraclavicular areas	**Clinical:** PD **Radiology:** PD **Combined:** PD **Treatment Decision:** **D/c** Cemiplimab due to grade 3 colitis	Post-cemiplimab course complicated by infection in the right neck site post radiation	**Deceased**, cause of death: infection in the R neck site post-radiation	Clear indication of disease progression in skin can be a poor prognostic sign
64M **AJCC T4a / BWH T3** **Clinical location:** Mid chest	2	RT → recurrence with delayed diagnosis due chronic non healing wound→cemiplimab	Radiology scans showing improvement while skin ulceration was persistent which led diagnosis	**Clinical:** PD (not enough treatment) **Radiology:** SD/PD **Combined:** PD **Treatment decision:** **D/c** cemiplimab	Repeat radiation + docetaxel to painful chest wall lesion	**Alive**, clinically improved with RT	Too short PD1 inhibitor course, delay in diagnosis due to radiation therapy
78M **AJCC T4a / BWH T3, N1, M1** **Clinical location:** Scalp	20	Extensive Mohs on the scalp for poorly diff lesion → parotid mass 4 months post Mohs → excision and neck dissection → RT → recurrence with sagittal sinus invasion → Cemiplimab initiated	Immunocompromised patient, potentially progressive disease from 2018-2021 undetected	**Clinical:** PD **Radiology:** PD **Combined:** PD **Treatment decision:** Continued cemiplimab	Quality of life improved with cemiplimab	**Deceased** from progressive disease invading brain, no progression in the neck or metastases beyond parotid gland	Early diagnoses and treatment are helpful. Treatments can improve quality of life despite worsening disease. Clear lytic lesions on the bone
Stable Disease							
67M, **AJCC T3 / BWH T2a, N2, Mx** **Clinical location:** Scalp	41	Mohs, Surgical resection with graft placement, adjuvant RT→ recurrence within 4 months → PD1 INHIBITOR	Decreased nodularity during cemiplimab treatment	**Clinical:** SD **Radiology:** SD **Combined:** SD **Treatment Decision** Cemiplimab Completed	No post-cemiplimab interventions	**Alive**, stable small lung nodules, no metastatic disease in neck	Clinical response based on decreased nodularity of area, smaller areas of ulceration. Posttreatment image with erosions may be due to osteoma formation post-surgery and radiation

Table2 continues

Patient Characteristics	# of Cycles	Prior Treatment	Clinical Response	Determination of Response Criteria	Clinical Course	Current State (2/2024)	Clinical Pearls
85 M **AJCC T2 / BWH T2a, N0M0** **Clinical location:** Left preauricular	16	RT → recurrence 4 months post RT completion→ PD1 INHIBITOR	Pretreatment: ulcerated lesion Posttreatment: small ulcerations	**Clinical**: SD **Radiology:** CR **Combined:** PR **Treatment Decision:** D/c cemiplimab	No complications	**Alive,** skin cancer on left cheek healed, no evidence of metastatic disease	Small areas of ulceration post treatment count as stable disease Note: ulcerated lesions at baseline can achieve only CR or SD, no PR criteria
65M, **AJCC T3 / BWH T2b, N0M0** **Clinical location:** Chest	8	Incisional biopsy, no prior treatment → PD1 INHIBITOR	Care delayed due to COVID	**Clinical**: SD **Radiology**: CR **Combined:** CR **Treatment Decision:** D/c pembrolizumab	No complications posttreatment.	**Alive,** lesion resolved no metastatic disease	Clinical course demonstrated without upfront surgical or radiation treatment. Small areas of ulceration on the chest lead to stable skin disease. Note: ulcerated lesions at baseline can achieve only CR or SD, no PR criteria
Complete Response							
93M **AJCC T2 / BWH T2a** **Clinical location:** Right Temple	10	Mohs→ recurrence→ RT	2 cm tumor at baseline, no baseline imaging performed	**Clinical**: CR **Radiology**: N/A **Combined:** CR **Treatment decision: D/c** cemiplimab	Erosive pustular dermatosis during treatment	**Deceased,** heart failure	Early onset of PD1 inhibitor treatment at a smaller size despite patient's age, with good clinical outcome.
79M **AJCC T3 / BWH T2b** **Clinical location:** Left neck	63	Deemed not eligible for surgery or RT at baseline given facial nerve involvement	Facial nerve paralysis with a fast-growing tumor at baseline	**Clinical**: CR **Radiology**: PD **Combined:** PR **Treatment decision:** continued cemiplimab	Left neck subcutaneous nodule demonstrated on imaging	**Alive,** still on cemiplimab	Challenging as L neck nodule with slow growth in the setting of well tolerated PD1 inhibitor clinical course.

Abbreviations: RT: radiation therapy; CR: complete response; PR: partial response; SD: stable disease; PD: progressive disease; N/a: not available; BWH: Brigham and Women's Hospital Tumor Staging criteria; AJCC: American Joint Committee on Cancer.

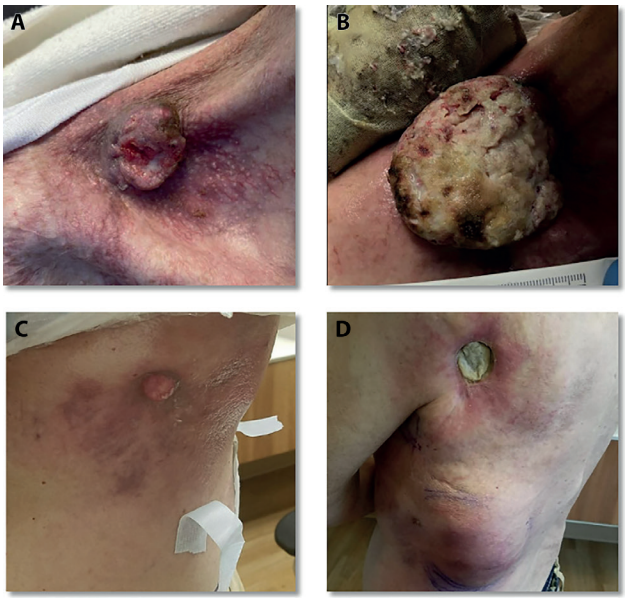

Figure 1. Highlighted cases of hyperprogression. (A, C) Pretreatment. (B, D) Posttreatment.

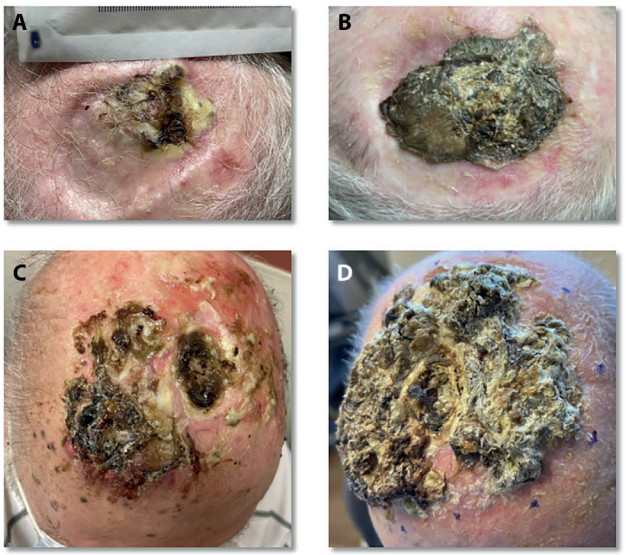

Figure 2. Highlighted cases of erosive pustular dermatosis and pseudoprogression. (A, C) Pretreatment, (B, D) posttreatment.

progressive disease who continue treatment and have subsequent complete or partial response [12,13]. In the context of cSCC, this has been described as radiographic progression of inflammation, edema, and necrosis. On examination, pseudoprogression has been described as "tumor flare," presenting with initial tumor growth [13]. The clinical challenge of distinguishing between EPD and tumor flare may result in misclassification as progressive disease. Further clinical evaluation for symptoms of true progression, such as a biopsy and close follow-up, are necessary to distinguish between progressive disease and pseudoprogression such as EPD. Though continued treatment in the case of tumor flare may lead to eventual response, it is not necessary to continue treatment for those with CR and EPD.

Cases that are highlighted with true progression showed nodules under the skin (Figure 1). However, one case of progression was due to a new nodule near the primary site, confirmed as new primary. Given the RECIST 1.1 criteria, treatment with PD-1 inhibitor was stopped, leading ultimately to progression of disease. It is now known that PD-1 inhibitors can lead to atypical squamous proliferations in the skin, a treatable condition, and it is critically important to distinguish these lesions from new cSCCs, as described in the clinical RECIST criteria, to prevent premature discontinuation of PD-1 inhibitor treatment [14].

Partial Response in Ulcerated Lesions

In considering our patients' response to anti-PD1 therapy using both clinical response and RECIST 1.1 criteria, several complexities in evaluation of both clinical and radiological response emerged. Firstly, the inability to characterize partial treatment response in cSCC with baseline ulcerations and ongoing erosions. In our study, most patients had ulceration of lesions at baseline due to prior radiation therapy, and healing over time showed significant reduction in the size of ulceration. As there are no criteria for partial response in ulcerated lesions, the evaluation of a patient with a partially healed wound is stable disease. Clinicians, including dermatologists, oncologists, and wound care specialists, are needed to evaluate the true cause of the ulcerations, for example, poor vasculature, or previously irradiated site vs active tumor bed. Additionally, prior extensive surgery down to the bone or radiation can lead to osteoma formation and continued ulceration of the wound (Figures 3–4). Proper characterization of erosions can prevent unnecessarily long treatment duration as well as premature cessation of treatment.

Interpretation of Radiological Findings

Patients 1 and 2 underscore the challenges posed by radiological findings, which may be misleading, necessitating a comprehensive clinical evaluation alongside imaging. Both patients had progressive disease by clinical size criteria (consistent with pseudoprogression due to EPD clinically) and showed progressive disease by radiology; as such, the decision was made to stop treatment with immunotherapy. It should be noted that radiological progression can be misleading due to subcutaneous edema and inflammation on the scans, and a complete clinical picture is required to determine the outcome status.

Conclusion

We conclude that clinical determination of response to immunotherapy in cSCC can be challenging due to multiple prior cSCC treatments, including extensive surgery and radiation,

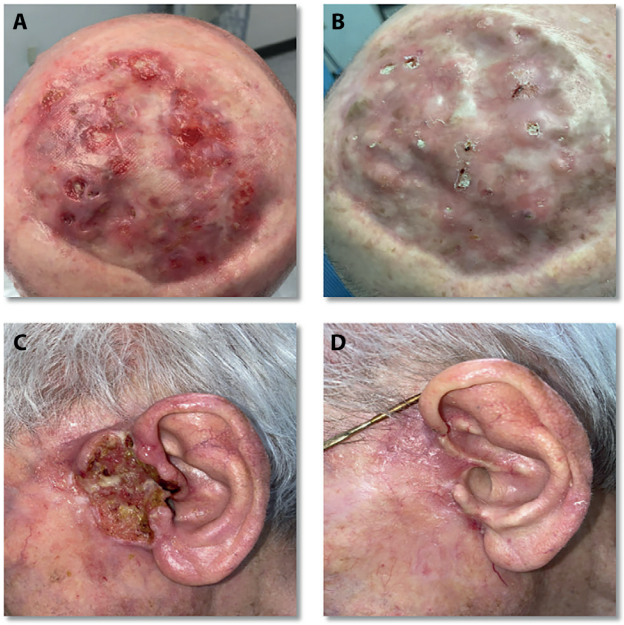

Figure 3. Highlighted cases of stable disease of ulcerated lesions, with osteoma formation. (A, C) Pretreatment, (B, D) posttreatment.

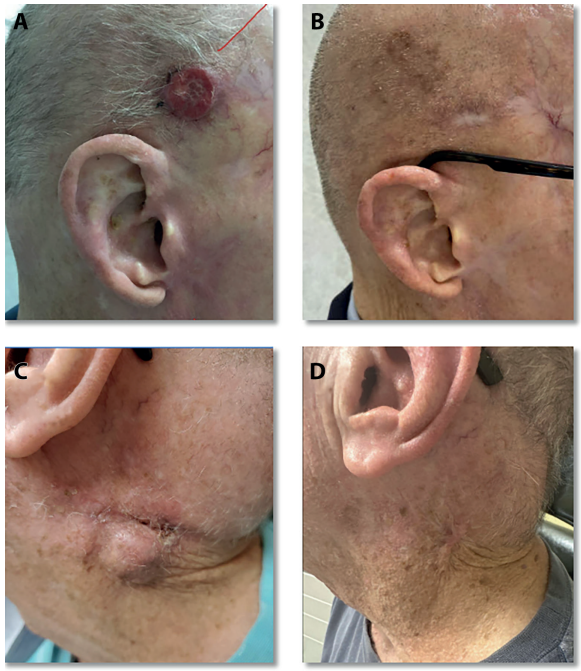

Figure 4. Complete responses. (A, C) Pretreatment, (B, D) posttreatment.

due to inflammation caused by these agents as well as to the limitations of radiological testing. Ulcerated lesions at baseline pose a significant challenge, and an active multidisciplinary approach is needed to determine the cause of ongoing erosions. When treatment continuation decisions are at stake, biopsy sampling can be very helpful to determine response status. Anti-inflammatory and wound care treatments to clear eschars and crust are necessary for accurate clinical determination of tumor response. Finally, new lesions resembling cSCC on PD-1 inhibitor therapy must be evaluated to determine recurrence/progression of locally advanced lesion vs new primary cSCC vs atypical squamous proliferation to determine an appropriate course of action. Revised criteria taking these factors into account would allow physicians to make more informed decisions about treatment, avoiding premature treatment cessation or prolonged ineffective treatment.

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