

Permanent chemotherapy-induced alopecia presenting with erosive pustular dermatosis-like retention hyperkeratosis



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INTRODUCTION

Reports of permanent chemotherapy-induced alopecia (pCIA), defined as the absence or incomplete regrowth of hair for ≥ 6 months following completion of chemotherapy, are increasing. Most often associated with taxane-based chemotherapies and endocrine therapies, it affects 42% of breast cancer survivors.^{1,2} While most patients present with diffuse hair thinning, our patient presented with brown plaques in addition to hair loss.

CASE REPORT

A 70-year-old woman with a history of stage IIIB breast cancer presented with brown scaly plaques on the scalp and hair loss, associated with mild pruritus and no history of scalp trauma. The plaques appeared after she had developed chemotherapy-induced alopecia upon completion of breast cancer treatment with paclitaxel 1 year previously. Clinical exam revealed diffuse alopecia with large, thick, adherent plates of brown scale over the vertex and frontal aspect of the scalp (Fig 1). Retention of hair and smaller plaques were present on the occipital scalp. Gentle scale removal revealed a white-to-pink pustular-like base. Fungal culture was negative, and punch biopsy from the frontal aspect of the scalp showed retention hyperkeratosis. Improvement occurred following 4 weeks of treatment with Silvadene cream daily and a 14-day course of doxycycline 100 mg twice daily. Her maintenance regimen included daily compounded 6% salicylic acid with mineral oil, fluocinolone 0.01% topical oil,

Abbreviations used:

pCIA: permanent chemotherapy-induced alopecia
EPD: erosive pustular dermatosis

and alternating between ketoconazole 2% and T-Sal shampoos.

Five months after initial biopsy, the retention hyperkeratosis resolved; however, the patient expressed concern regarding lack of hair regrowth. The hair loss extended beyond the region of preceding erosive pustular dermatosis (EPD)-like retention hyperkeratosis, and she had decreased density of eyebrow and eyelash hair. The patient had no family history of alopecia but noted thin hair at baseline with a receding hairline that preceded chemotherapy. Dermoscopy demonstrated yellow globules, decreased follicular ostia, and mild scaling. Punch biopsy of posterior parietal scalp showed late-stage nonscarring alopecia with diminished follicular density, preserved sebaceous glands, and miniaturized follicles, most consistent with pCIA; there was no fibrosis or evidence of EPD (Fig 2). The patient's final diagnosis was EPD-like retention hyperkeratosis with pCIA. At last follow-up, the patient had been on minoxidil 5% foam twice daily for 8 months as well as spironolactone 50 mg twice daily for 5 months.

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Fig 1. Clinical exam illustrating diffuse hair loss and erosive pustular dermatosis-like eruption with thick plate-like scaling (*black circle*) at time of initial biopsy (**A**), after 1 month of treatment with keratolytic agents (**B**), and after 5 months of treatment (**C**).

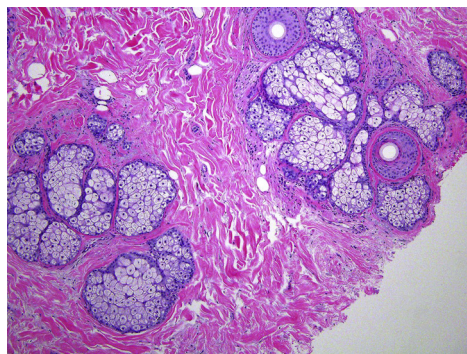


Fig 2. Horizontal sections at the level of the isthmus demonstrating diminished number of follicles with preserved sebaceous glands, miniaturized follicles, and no fibrosis in follicles on histopathology (Hematoxylin-eosin stain; original magnification: $\times 100$.)

DISCUSSION

Clinical features of pCIA typically include non-scarring alopecia with diffuse hair thinning, often with a pattern similar to androgenetic alopecia,^{1,3,4} and eyelash, eyebrow, axillary, and pubic hair loss.⁴ Histopathologic findings of pCIA include reduction in hair density, miniaturization, and end-stage fibrous tracts.^{5,6} To our knowledge, thick plate-like scaling with an erosive scalp has not been reported previously as an associated clinical finding of pCIA.^{1,2,4} The etiology of EPD and retention hyperkeratosis are not fully known and may be triggered by trauma. Chemotherapy could trigger EPD or retention hyperkeratosis, but this does not explain the patient's single episode, which usually follows a chronic recurrent course.

pCIA can be distressing, with negative impact on quality of life and self-esteem.^{4,7,8} Prevention with scalp cooling,⁹ prompt diagnosis, and early treatment may help reduce morbidity.⁴ There are

currently no guidelines for the treatment of pCIA, in part due to its incompletely understood pathogenesis. One hypothesis is that chemotherapies unmask underlying androgenetic alopecia in women,³ so this patient was treated with spironolactone, an androgen receptor antagonist. The patient was also treated with topical minoxidil 5%,¹⁰ a common treatment for CIA that has shown mixed results for pCIA. Oral minoxidil and platelet rich plasma may be considered as future treatments.¹¹

In conclusion, rare presentations of pCIA may include thick plate-like scaling resembling EPD. Repeat biopsy after clearance of lesions may allow for prompt diagnosis, and treatment for pCIA requires further research.

Conflicts of interest

None disclosed.

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