Radiation therapy for B-cell cutaneous lymphoid hyperplasia

Robert B. Taylor · John A. Fortney · Ross B. Pollack
John S. Metcalf · Joseph M. Jenrette

Abstract Recent work has demonstrated that B-cell cutaneous lymphoid hyperplasia (BCCLH) lies in a spectrum of B-cell lymphoproliferative disorders that can progress to primary cutaneous B-cell lymphoma (CBCL). In light of this work, definitive therapy with methods such as radiotherapy is an important part of the treatment strategy. Few outcome data exist for patients with treatment-resistant BCCLH. We present a case study of a 63-year-old woman with BCCLH who failed immunomodulatory treatment but responded well to an aggressive course of radiotherapy. After 18 fractions of 6 MeV electron beam therapy with 200 cGy per fraction, the patient has been recurrence-free for 3 years. Acute toxicity was limited to Radiation Therapy Oncology Group grade II skin toxicity, which resolved within 1 month of treatment.

Key words Radiotherapy · Cutaneous lymphoid hyperplasia · Lymphocytoma cutis · Pseudolymphoma of Spiegler-Fendt

Introduction

B-cell cutaneous lymphoid hyperplasia (BCCLH) is a B-cell-rich, dense lymphoid infiltrate centered in the reticular dermis. It most often manifests clinically as a solitary nodule on the head, neck, extremities, breast, or genitalia with a doughy to firm consistency that can range from red-brown to violaceous in color. Other presentations include localized arrays of nodules, plaques, or papules and rarely generalized forms. Most cases are idiopathic, although some are the result of arthropod assault, infections, tattoos, acupuncture, trauma, gold jewelry, vaccinations, hyposensitivity injections, or medications.

Diagnosis is made through biopsy and the absence of systemic lymphoma B symptoms. The histology of BCCLH lesions typically reveals a wedge-shaped lymphoid infiltrate that spares the epidermis. A grenz zone of spared papillary dermis typically separates the involved dermis from the uninvolved epidermis.

BCCLH has been treated with immunomodulatory agents such as hydroxychloroquine, glucocorticoids, cryotherapy, antimalarials, cephalosporins, minocycline, laser therapy, photodynamic therapy, and radiation therapy. Successful radiation therapy for BCCLH has been described in the past, but there have been few recent examples of treatment doses and protocol. Given the need for definitive treatment of these lesions, we present our experience with one case to emphasize the current role for radiation therapy in BCCLH treatment.

Case report

A 63-year-old woman presented with a persistent pruritic, erythematous, indurated plaque on the right cheek measuring 7 × 6 cm (Fig. 1). She was free of systemic symptoms and a biopsy demonstrated
Topical nitrogen mustard therapy was attempted but was not tolerated due to an allergic response. EBT was reintroduced with a more aggressive treatment plan. A total of 18 fractions of 6 MeV EBT with 200 cGy were administered using a 0.5-cm bolus. The lesion completely resolved with the more aggressive course of radiation therapy, and the patient was lesion-free at the 3-year follow-up. The patient did experience Radiation Therapy Oncology Group (RTOG) grade II skin toxicity following the second round of radiotherapy, but it resolved by 1 month after treatment.

**Discussion**

BCCLH presents in a variety of ways, and this case demonstrates that treatment must be tailored to the lesion. Here, the first round of treatment followed a protocol similar to those discussed by Olson et al. and Gordon et al. with a total dose of 20 Gy. Although this dosage was sufficient to eradicate the lesions in some reports, it did not do so in our case, and more aggressive therapy of 36 Gy was required. The lesions successfully treated with ≤20 Gy in Olson et al.’s patients were of the nodular variety, whereas the lesion in this patient was plaque-like. It is possible that this difference is a factor that should be considered when deciding on a treatment protocol.

A defining feature of BCCLH is the polytypic expression of the immunoglobulin light chains. However, it has been recognized that 5%–30% of cutaneous lymphoid hyperplasia cases defined by clinicopathological and immunohistological criteria contain occult dominant immunoglobulin arrangements indicating a dominant B-cell clone. In light of these findings, BCCLH can be considered the benign end of a spectrum of B-cell lymphoproliferative disorders that include clonal BCCLH and primary cutaneous B-cell lymphoma. Thus, it is important to make all attempts to eradicate the disease and subsequently follow the patient at reasonable intervals. As demonstrated in this case, irradiation is an important treatment modality for BCCLH, especially for resistant BCCLH or in patients who do not tolerate other treatment modalities.

**References**