

Abstract N°: ID-193

Topic: Adverse drug reactions, TEN

Ocrelizumab-Induced Atopic Dermatitis-like Drug Eruption: A Case Report

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Introduction

Ocrelizumab, a humanized anti-CD20 monoclonal antibody, is used for the management of relapsing-remitting multiple sclerosis (RRMS) and primary progressive multiple sclerosis (PPMS). While psoriasiform dermatitis, thought to result from B-cell depletion and subsequent T-cell dysregulation induced by ocrelizumab, has been documented in the literature, the emergence of atopic dermatitis-like eruptions has not been previously reported. Herein, we describe a case of atopic dermatitis-like eruption associated with ocrelizumab administration.

Materials and Methods

A 67-year-old male patient presented to the neurology clinic with a one-year history of progressive gait disturbance. Neurological examination revealed bilateral clonus and mild gait ataxia. Cranial contrast-enhanced magnetic resonance imaging demonstrated multiple demyelinating plaques. Cerebrospinal fluid (CSF) electrophoresis revealed type 2 oligoclonal bands. The patient was diagnosed with primary progressive multiple sclerosis and initiated on ocrelizumab therapy. The patient had no history of taking any medications other than ocrelizumab. Six and a half months following treatment initiation and four days after administration of the third ocrelizumab infusion, the patient developed pruritic, erythematous, ill-defined eczematous papules and plaques in the antecubital and popliteal fossae. The patient was referred to the dermatology department, where a skin punch biopsy was performed.

Results

Skin biopsy revealed epidermal spongiosis and acanthosis with superficial pustule formation, accompanied by a mixed inflammatory infiltrate in the superficial dermis characterized by a dense perivascular eosinophilic infiltrate with scattered neutrophils. The histopathological findings were consistent with drug-induced eruption. The patient was prescribed emollients, topical methylprednisolone aceponate, and oral bilastine. At the two-week follow-up, complete resolution of the cutaneous lesions was observed. Given the mild severity of the eruption, discontinuation of ocrelizumab therapy was not deemed necessary.

Conclusions

The existing literature has documented various cutaneous adverse events associated with ocrelizumab therapy, predominantly psoriasiform eruptions, as well as palmoplantar pustulosis, cutaneous vasculitis, nummular dermatitis, alopecia areata, and pyoderma gangrenosum. To the best of our knowledge, this represents the first documented case of atopic dermatitis-like eruption associated with ocrelizumab administration. The pathophysiological mechanism may involve B-cell depletion induced by ocrelizumab, resulting in subsequent immune dysregulation. Although further investigation is warranted to elucidate this association, clinicians should maintain heightened vigilance for cutaneous

manifestations when administering novel biological agents.