

Drug Associated Giant Cell Arteritis with Scalp Necrosis after Treatment with Pembrolizumab: A Case Report

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Research Article

Keywords: Giant cell arteritis, GCA, Check point inhibitor, Immunotherapy, Pembrolizumab, Scalp Necrosis

Posted Date: November 30th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-755713/v1>

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Version of Record: A version of this preprint was published at SN Comprehensive Clinical Medicine on January 12th, 2022. See the published version at <https://doi.org/10.1007/s42399-022-01120-5>.

Abstract

Giant cell arteritis (GCA) can be a rheumatologic complication of checkpoint inhibitor immunotherapy for the treatment of cancer. A rare and serious manifestation of GCA is scalp necrosis. We report a case of a patient who developed GCA and subsequent scalp necrosis after the initiation of immunotherapy with the checkpoint inhibitor pembrolizumab for the treatment of metastatic melanoma. Such advanced cases of GCA may be increasingly recognized in the immunotherapy era of oncology.

Presentation

A 90-year-old male with a history of hypertension was diagnosed with metastatic melanoma and treated with pembrolizumab intravenously every three weeks. Nine months after treatment with pembrolizumab, the patient developed jaw claudication, temporal headaches, and bilateral vision loss. Inflammation markers (erythrocyte sedimentation rate and C-reactive protein) were significantly elevated. Biopsies of bilateral temporal arteries demonstrated moderate to severe inflammatory infiltrates and focal mural calcifications consistent with giant cell arteritis (GCA). The patient was started on high-dose prednisone (60mg) with a taper, however, he was continued on treatment with pembrolizumab for metastatic melanoma as previously outlined.

Three months thereafter, the patient presented as a new patient to our clinic with recurrence of headache and jaw claudication. The physical exam at this point was notable for frontal-parietal scalp necrosis (Figure 1). Based on the timing and distribution of his scalp lesions, we made the diagnosis of scalp necrosis secondary to recurrent GCA as a likely result of an immune related adverse event from his pembrolizumab treatment. Given the severity of the presentation, we recommended holding pembrolizumab indefinitely. Our patient was resumed on high-dose prednisone (60mg) with taper and the addition of tocilizumab 162mg subcutaneously weekly as a disease modifying and steroid sparing agent. He had no further progression of scalp necrosis or recurrence of headaches thereafter. Unfortunately, his vision remained permanently impaired bilaterally.

Discussion

Scalp necrosis is a rare but serious complication of GCA. It signifies severely active disease [1]. In our case, this patient developed GCA and scalp necrosis as an immune-related adverse event (IRAE) after the use of pembrolizumab, a programmed death (PD)-1 inhibitor. IRAEs have been reported in 70% of patients treated with anti-PD1 immunotherapy [2]. By blocking the PD-1 pathway, checkpoint inhibitors increase T cell proliferation, increasing a patient's susceptibility to autoimmune diseases including GCA. Prophylactic treatment with immunosuppressive agents has not shown to prevent the incidence of IRAEs [3].

A few cases of GCA have been reported as IRAEs for immunopotentiating treatments such as nivolumab, ipilimumab, and pembrolizumab, but scalp necrosis has only been rarely cited as a complication of GCA

associated with immunotherapy. It is important to recognize this unusual cutaneous manifestation of GCA and a possible complication in patients undergoing cancer immunotherapy.

Declarations

Funding: None

Conflict of interest: None of the authors report any conflict of interest.

Ethics Approval: Not applicable for case report

Consent to participate: Informed consent was obtained from the patient for dissemination of the deidentified photographs and discussion of the case for publication and educational purposes.

Written consent for publication: Yes, written informed consent was obtained from the patient for this case report for publication

Availability of data and material: Not applicable

Code availability: Not applicable

Authors' contributions: All authors contributed equally to the intellectual analysis and drafting of this manuscript

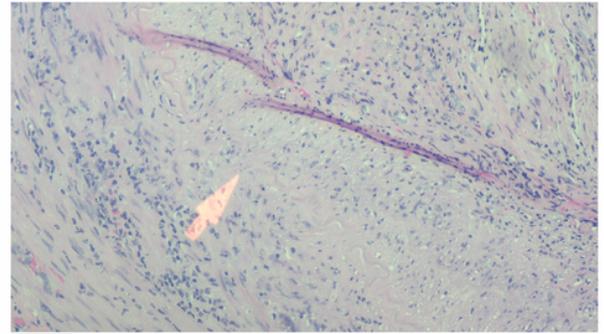
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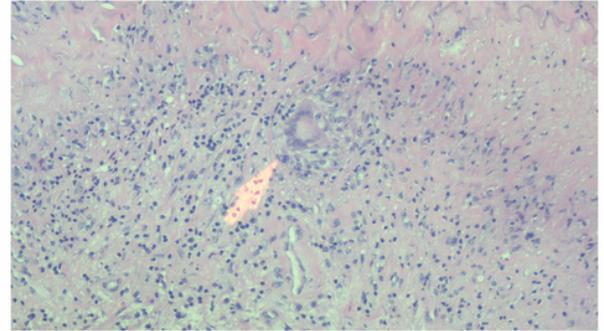
Figures



A



B-1



B-2

Figure 1: A- 10.0 x 8.0 cm right frontal-parietal lesion with central necrosis with smaller surrounding lesions; B- Abnormal temporal artery biopsy with features of severe intimal thickening with luminal compromise (1) and a media expanded by lymphoid infiltrate (2)

Figure 1

See image above for figure legend.

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