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"Pembrolizumab-Induced Grover's Disease in a Patient with Merkel Cell Carcinoma"

Introduction: Immune checkpoint inhibitors (ICIs) have transformed treatment of various cancers including Merkel cell carcinoma (MCC) but are associated with immune-related adverse events (irAEs). Dermatologic irAEs occur in 30-40% of patients on PD-1 inhibitors such as pembrolizumab and in up to 50% of patients on ipilimumab. Patients commonly present with maculopapular, lichenoid, psoriasiform, or eczematous eruptions. Grover's disease (GD), a transient acantholytic dermatosis, is rare but increasingly reported in oncologic patients, with only limited cases linked to pembrolizumab. The true incidence of GD is difficult to determine, as it can be misdiagnosed as folliculitis, miliaria, or drug eruption.

Case Presentation: A male patient with MCC of the left nasal ala underwent Mohs surgery in March 2024, followed by adjuvant radiation. After detection of metastases, pembrolizumab was started and remission achieved. Months later, the patient developed a pruritic papular eruption on the chest and trunk that progressed to the arms and back despite topical and systemic corticosteroids, emollients, wet dressings, and antihistamines. Biopsies revealed acantholytic dyskeratosis, suprabasal clefts, and superficial perivascular lymphocytic infiltrates consistent with GD. Pembrolizumab was discontinued, after which the eruption stabilized. MCC remained in remission.

Discussion: Our patient's clinical and histologic features paralleled classic GD and align with prior reports of ICI-associated cases. GD in oncologic patients may be triggered by malignancy itself or therapy, with immunotherapy the most frequently described. The underlying pathogenesis of GD remains unclear. Proposed triggers include sweat duct obstruction, xerosis, and heat stress. ICI-associated GD is thought to reflect immune dysregulation. Studies support multiple mechanisms, including CD4+/CD8+ infiltrates in ipilimumab-induced GD, Th1/Th17 skewing with PD-1 blockade, and epitope spreading in cases of concomitant bullous pemphigoid and GD under nivolumab. Collectively, these findings suggest that ICI-induced GD may represent a direct result of the drug's intended immune stimulation misdirected at the skin. Recognition requires biopsy, since misclassification as nonspecific drug rash is common without histology. Management is supportive, with topical or systemic corticosteroids often effective, though ICI discontinuation is sometimes necessary.

Conclusion: This case highlights pembrolizumab-induced GD in a patient with MCC, contributing to the sparse literature on this association. Awareness of GD as a rare but treatable cutaneous irAE is critical as use of ICIs continues to expand.