T helper (Th)17 radiation-induced hidradenitis suppurativa

To the Editor: We read with interest the case report describing the development of hidradenitis suppurativa (HS) lesions after radiation treatment in a patient with uterine adenocarcinoma.[1] The authors provided several thoughts on the possible mechanism behind radiation-induced HS. The purpose of this letter is to provide additional insight about the likely immunologic mechanisms behind radiation-induced HS. The reported case is an example of an abscopal-like effect[2] sharing adenocarcinoma antigens to antigens of apocrine glands of the groin. Abscopal effect is an immune-mediated phenomenon that has been heavily discussed in metastatic melanoma, whereby there is regression of nonirradiated metastatic lesions after irradiation of a distant melanoma tumor location.[3,4] Abscopal effects were also recently reported in advanced melanoma patients after immunotherapy, highlighting the therapeutic implications of this phenomenon.[5]

As the authors point out, apoptosis can be induced by irradiation.[1] It is likely that apoptotic uterine adenocarcinoma cells result in T-cell cross priming after radiation[6] and activate Th17 subset. A recent article points out that Th17 is an important subset in the immunopathogenesis of HS,[7,8] like it is in psoriasis.[9] This case and the article by Matusiak et al[7] make a strong case that HS, like psoriasis, is an autoimmune disease with Th17 and the attendant polymorphonuclear leukocytes involved. Oppman et al[10] identified the p19 third subunit of interleukin (IL)-12, and designated it as IL-23. This finding predicted the potential for treating psoriasis with anti—IL-12 ustekinumab and anti—IL-23 (guselkumab). This case of radiation-induced HS is likely to predict a positive outcome with clinical trials for HS with ustekinumab (IL-12 antagonist),[11] secukinumab (IL-17 antagonist),[12] and guselkumab (IL-23 antagonist).[13]

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Funding sources: None.

Conflicts of interest: None declared.

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https://doi.org/10.1016/j.jdcr.2017.12.005