Reply to “Pityriasis rosea during COVID-19 and its pathogenesis”

To the Editor: We thank Drs Drago and Ciccarese for their interest in our article and will share several thoughts and responses. Their first argument is that due to pityriasis rosea’s (PR) limited course, patients may be dissuaded from seeking dermatologic care, which is interestingly in contradiction to their findings of an increase in PR diagnoses relative to the total patient volume during the pandemic. This theory is certainly plausible that PR diagnoses or any non–life-threatening diagnoses would decrease due to shelter in place orders and behaviors, even with increased utilization of teledermatology. We, however, found otherwise by comparing the rates of PR to other inflammatory dermatoses, such as acne vulgaris. Using the TriNetX database, which encompassed over 212 million patients, as compared to the 8134 patients included between the 2 articles referenced. We demonstrated no significant reduction of selected control inflammatory dermatoses during the specific months of the pandemic and pre-pandemic, as compared to the number of PR cases recorded. It is important to note that the article supporting the claim that PR cases increased was derived from a single center review of PR diagnoses during and prior to the COVID-19 pandemic. Authors found a 3.8% decline in PR diagnoses from 2019 to 2020; however, this was interpreted as an increase in the context of an 80% decline in patient volume resulting from the pandemic. This approach is somewhat flawed, forgetting even a direct comparison to other inflammatory skin diseases was not conducted, as the proportion of one diagnosis as it relates to total patient volume in a single clinic will fluctuate.

Second, while there is agreement that the etiology of PR is viral in nature, Drago and Ciccarese argue PR uniquely results from HHV6/7 viral reactivation, rather than acute, initial infection. While we certainly agree that external forces such as COVID-19 infection or stress associated with the pandemic could result in HHV6/7 reactivation and in turn PR, the evidence is limited supporting this. The basis of this argument relies heavily on 1 article that examined 14 PR patients and found HHV6/7 DNA in 86% to 93% of lesional skin, 83% peripheral blood mononuclear cells, and 80% to 100% of saliva samples, suggesting a prior infection as salivary gland cells are a known reservoir for herpesviruses; the second study cited did not evaluate salivary samples limiting its utility.

Whether through reactivation, or active infection, our paper adds to the small but growing body of literature in support of a viral etiology for PR. That said, the significant reduction in PR cases during the COVID-19 lockdown as compared to other inflammatory skin diseases provides support for the role of initial infection in PR pathophysiology and certainly does not dismiss the role of reactivation. Arguably additional contributors to PR are likely out there, and we must admit that much about PR is still a mystery.

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Conflicts of interest

None disclosed.

REFERENCES


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