SARS-COV-2 Infection in Patients with Atopic Dermatitis: A Cross-Sectional Study

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Background:

SARS-COV-2 disproprotionately impacts certain populations with inflammatory conditions who have an elevated risk of respiratory comorbidities. In atopic dermatitis (AD), inflammatory cytokines, e.g. interleukin-13, can regulate SARS-COV-2 entry in airway epithelial cells by increasing transmembrane protease serine 2 and decreasing angiotensin-converting enzyme 2 expression.

Objective:

In this study, we aim to compare the rates of SARS-COV-2 infection, hospitalizations, and mortality among AD patients to controls without AD in a California-based population.

Methods:

A retrospective cross-sectional study was conducted using the University of California COVID Research Data Set (UC CORDS), a HIPAA secure medical records dataset for patients tested for SARS-COV-2 across UC medical centers. Information regarding SARS-COV-2 testing, patient demographics, hospitalization, and mortality were collected up to October 8, 2020. AD patients were diagnosed with "atopic dermatitis," "acute dermatitis," "atopic neurodermatitis," "nummular eczema," or "flexural eczema." Specific systemic treatment subgroups were identified (prednisone, methotrexate, cyclosporine, or dupilumab) for at least 30 days prior to SARS-COV-2 testing. Fisher Exact tests were used for statistical analysis when categories had less than five patients, while Chi-Squared tests were use for the rest.

Results:

A total of 269,299 patients were tested within the UC CORDS, with a 3.64% positive test rate (n=9808, 49% men, average age 42). Of these, 5,387 AD patients were tested for SARS-COV-2 and had a 2.95% (n=159, 47% men, average age 34) infection rate, which was lower than those without AD (n=5528, 40% men, average age 42 years) ($p<0.0063$). This observation was significant in women with AD compared to those without (2.70% vs 3.44%, $p=0.0220$), but not significant in men (3.29% vs 3.88%, $p=0.1433$).

In AD patients receving systemic mediations, SARS-COV-2 rates were not significantly different than those without: prednisone 2.6% (n=12; $p=0.6210$), methotrexate 2.7% (n=2), cyclosporine 0% (n=0), and dupilumab 0% (n=0). Hospitalizations within two weeks of SARS-COV-2 test (+/- 1 week) were assessed as a marker of infection severity. The hospitalization rate of SARS-COV-2 positive AD patients was 13.8% (n=22), which was not significantly different from those without AD at 19.3% (n=1858) ($p=0.9429$).

Lastly, the mortality rate of SARS-COV-2-positive patients in the UC CORDS was 2.1% (n=203, 62% men, average age 71), while that of SARS-COV-2-positive AD patients was 1.9% (n=3, 0% men, average age 82) and those on prednisone was 8.3% (n=1); yet, not significantly different from those without AD ($p=0.7502$, $p=1.0$, respectively).

Conclusion:

In this UC CORDS dataset, AD patients did not have increased risk for SARS-COV-2 infection, including AD patients on the immunomodulatory medications prednisone, methotrexate, cyclosporine, and dupilumab. This data do not account for the presence of respiratory comorbidities such as asthma. The overall lower AD age may account for the observed lack of significant difference. Limitations include use of tertiary center data, de-identified data with lack of clinical details, or follow-up. Future studies with identifiable datasets will help better assess this relationship. Understanding the mechanisms underlying SARS-COV-2 susceptibility are fundamental to develop better guidelines for populations at risk.