INFLUENZA INFECTION AS A TRIGGER FOR SYSTEMIC LUPUS ERYTHEMATOSUS FLARES RESULTING IN HOSPITALIZATION

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Background: Infections have been associated with a higher risk of systemic lupus erythematosus (SLE) flares, but the impact of influenza infection on SLE flares has not been evaluated.

Objectives: We evaluated the association between influenza infection and SLE flares resulting in hospitalization.

Methods: SLE flares resulting in hospitalization and influenza cases were ascertained from the Korean national healthcare insurance database (2014-2018). We used a self-reported case series design. In the interval analysis, the influenza infection incidence was defined as all other times during the observation period of each year. We estimated the incidence rates of SLE flares resulting in hospitalization during the risk interval and control interval and compared them using a Poisson regression model.

Results: We identified 1,624 influenza infections among the 1,455 patients with SLE. Among those, there were 98 flares in 79 patients with SLE. The incidence ratio (IR) for flares during the risk interval as compared with the control interval was 25.75 (95% confidence interval 17.63 – 37.59). This significantly increased the IRs for flares during the risk interval in both women (IR 27.86) and men (IR 23.90), all age groups (IR 17.09 – 37.65), and with and without immunosuppressant agent (IR 24.29 and 28.45, respectively), and with and without prior respiratory diseases (IR 21.86 and 28.82, respectively).

Conclusion: We found significant association between influenza infection and SLE flares resulting in hospitalization. Infection influenza has to be considered as a risk factor for flares in all SLE patients regardless of age, sex, medications, and comorbidities.

REFERENCES:

Disclosure of Interests: None declared

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CORRELATION OF PERIPHERAL CD4+GRANZB+CTLS WITH DISEASE SEVERITY IN PATIENTS WITH PRIMARY SJÖGREN’S SYNDROME

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Background: Sjögren’s syndrome (SS) is a chronic autoimmune disorder. The major histopathologic lesion of it is a focal lymphocytic infiltrate around ductal and acinar epithelial cells, which include a majority of CD4+ T. Several studies have shown that the epithelial cells in SS present diverse phenomena, such as MHC class II overexpression, CD4+T cells with cytotoxic activity (CD4 CTL) have been detected in various immune responses. They are characterized by their ability to secrete perforin and granzyme B to kill the target cells in an MHC class II-restricted fashion.

Objectives: So this study was to investigate the correlation of peripheral CD4+GRANZB+CTLS with disease severity and organ involvement in patients with primary Sjögren’s syndrome.

Methods: We recruited 116 pSS patients and 46 healthy controls using flow cytometry to examine proportion of CD4+GRANZB+CTLS in their peripheral blood, and immunofluorescence to test the expression of CD4+GRANZB+CTLS in labial gland. The correlations of CD4+GRANZB+CTLS and the relevant clinical data were analyzed.

Disclosure of Interests: None declared

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Table 1. Incidence ratios for SLE flares resulting in hospitalization after influenza infection

<table>
<thead>
<tr>
<th>Risk interval</th>
<th>Incidence ratio</th>
<th>95% CI</th>
</tr>
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<tbody>
<tr>
<td>During risk interval for 7 days / control interval</td>
<td>25.75</td>
<td>17.63 – 37.59</td>
</tr>
<tr>
<td>Days 1-3 / control interval</td>
<td>21.81</td>
<td>14.71 – 32.35</td>
</tr>
<tr>
<td>Days 4-7 / control interval</td>
<td>7.56</td>
<td>3.69 – 15.47</td>
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</tbody>
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SLE: systemic lupus erythematosus; CI, confidence interval

Disclosure of Interests: None declared

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APLS-ASSOCIATED RETINAL VASCULOPATHY AS A PRESENTATION OF THROMBOTIC MICROANGIOPATHY

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Background: Persistent antiphospholipid antibodies (aPL) positivity was a recognized risk factor for thrombotic events, obstetric morbidity and a variety of manifestations beyond thrombosis. The presence of some non-criteria manifestations including thrombocytopenia, hemolytic anemia, and APS nephropathy should prompt consideration for thrombotic microangiopathy (TMA). Patients with APS can also present with a variety of ocular and neuro-ophthalmic manifestations, such as retinal artery/vein occlusion, retinal arthritis, optic neurtis and ischemic optic neuropathy, with underlying mechanisms remained elusive. Retinal vasculopathy including retinal artery occlusion (RAO) or retinal vein occlusion (RVO) was recently found occurred more frequently in APS patients with thrombocytopenia, suggested other possible mechanisms besides thromboembolism.

Objectives: To explore risk factors and possible mechanisms of retinal vasculopathy among APS patients.

Methods: In this single-center case-control study among APS patients, we evaluated patients who fulfilled 2006 Sapporo APS Classification Criteria with or without retinal vasculopathy during 2018-2020 at Peking Union Medical College Hospital. Demographic data, aPL-related manifestations, cardiovascular risk factors and antibodies profile were compared and a logistical regression model was built. Hierarchical cluster analysis with the Euclidean distance and the Ward method was applied to identify clusters of variables.

Results: A total of 310 APS patients (67.4% female, mean age 38.1 years) were included, of whom 18 patients were diagnosed with retinal vasculopathy (9 with RVO and 9 with RAO). No significant differences was found among most demographic characteristics, clinical manifestations, or antibody profile. However, APS-related heart valve disease (OR 13.66, 95% confidence interval [CI] 4.55-40.98), APS nephropathy (OR 12.77, 95% CI 4.04-40.35), thrombocytopenia (OR 2.63, 95% CI 1.01-6.89) and low serum IgM (OR 3.67, 95% CI 1.30-10.40) were predictive of retinal vasculopathy (Figure 1 A). APS-related heart valve disease and nephropathy were also found statistical significant in multivariate logistical regression (Figure 1 B). They and other non-criteria manifestations were aggregated with retinal vasculopathy from cluster analysis of variables (Figure 1 C).

Conclusion: Patients with APS-related heart valve disease and nephropathy suffered a higher risk of retinal vasculopathy including RAO and RVO. The underlying mechanisms of aPLs-associated retinal vasculopathy may involve TMA, leading to a poor prognosis and therapeutic changes.

REFERENCES: