and relapse rate (p=0.137) were not significant between the different treatment groups.

Abstract FRI0490 – Table 1. Risk factors affecting survival according to Cox regression analysis

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Hazard ratio</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 75 years old</td>
<td>2.35</td>
<td>(1.1–5.03)</td>
<td>0.027</td>
</tr>
<tr>
<td>BVAS &gt; 20</td>
<td>2.08</td>
<td>(0.99–4.32)</td>
<td>0.05</td>
</tr>
<tr>
<td>Creatinine &gt; 1.5 mg/dL</td>
<td>1.18</td>
<td>(0.58–2.38)</td>
<td>0.654</td>
</tr>
<tr>
<td>Early severe infection</td>
<td>3.23</td>
<td>(1.63–6.48)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

* Birmingham Vasculitis Activity Score (BVAS) Version 3

Abstract FRI0490 – Table 2. Risk factors affecting early severe infection in non-severe AAV patients

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Hazard ratio</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 75 years old</td>
<td>2.1</td>
<td>(0.85–5.32)</td>
<td>0.105</td>
</tr>
<tr>
<td>Conventional treatment</td>
<td>3.0</td>
<td>(1.11–7.89)</td>
<td>0.030</td>
</tr>
<tr>
<td>Lung involvement</td>
<td>1.0</td>
<td>(0.43–2.48)</td>
<td>0.806</td>
</tr>
<tr>
<td>Creatinine &gt; 1.5 mg/dL</td>
<td>1.0</td>
<td>(0.41–2.33)</td>
<td>0.963</td>
</tr>
</tbody>
</table>

Conclusions: Early severe infection is an independent predictor of death in patients with AAV, and conventional treatment has a potential risk of death due to severe infection. This study supports the current EULAR recommendation that several treatment strategies are recommended according to the disease severity of vasculitis. AAV patients who receive conventional treatment should be carefully monitored to reduce the occurrence of severe infection, especially in early phase of treatment.

REFERENCES:

Disclosure of Interest: None declared

FRI0491

SMOKING AS A RISK FACTOR FOR GIANT CELL ARTERITIS: A SYSTEMATIC REVIEW AND META-ANALYSIS

D. Brennan1, P. Ungprasert2, K.J. Warrington2, M.J. Koster2, 1Internal Medicine; 2Rheumatology, Mayo Clinic, Rochester, USA

Background: Tobacco smoking is a well-established risk factor for the development of several autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus. A similar association between smoking and giant cell arteritis (GCA) has been suspected but remains unclear due to limited study size and conflicting epidemiologic data.

Objectives: To conduct a systematic review and meta-analysis to further investigate the association between smoking and the development of GCA.

Methods: Two investigators (D.B. and M.K.) independently searched published studies indexed in MEDLINE and EMBASE from inception to February 2017 using the terms “giant cell arteritis,” “temporal arteritis,” “cranial arteritis,” and “Horton disease.” Recent conference abstracts available online were also reviewed. The following inclusion criteria were used: 1) original observational study comparing patients with GCA to healthy controls; 2) inclusion of smoking history; 3) provision of absolute numbers and/or statistical comparisons with 95% confidence intervals. Study eligibility was independently determined by the two investigators, with disagreements reviewed by a third investigator (P.U.) and resolved by consensus. RevMan 5.3 software was used for the data analysis. Point estimates and standard errors were extracted from individual studies and were combined by the generic inverse variance method of DerSimonian and Laird. Given the high likelihood of between-study variance, we used a random-effect model rather than a fixed-effect model. Statistical heterogeneity was assessed using the Cochran’s Q test.

Results: The initial search yielded 3312 articles. Of these, thirteen studies (8 prospective and 5 retrospective case-control studies) with unique cohorts were identified and included in the primary analysis (ever vs. never smoking history). Patients in the GCA cohort were more likely to have a history of smoking with an odds ratio of 1.19 (95% CI, 1.01–1.39) [figure 1A]. Considerable heterogeneity was present (I²=85%). Five of these studies included information on current smoking status. One additional study, which only reported current smoking status, was also included. The GCA cohort showed an association with current tobacco use with an odds ratio of 1.18 (95% CI, 1.01–1.38) [figure 1B].

Conclusions: Our study demonstrated a statistically significant increased risk of GCA among smokers compared to non-smokers.

Disclosure of Interest: None declared

FRI0492

CLINICAL CHARACTERISTICS OF PARENCHYMAL NEURO-BEHÇET’S DISEASE: A RETROSPECTIVE ANALYSIS

D. Yan1, J. Liu1, D. Wu1, L. Peng1, Z. Liu2, W. Zheng1. 1Peking Union Medical College Hospital, Beijing; 2The Second Affiliated Hospital of Soochow University, Soochow; 3The Second Affiliated Hospital of Soochow University, Soochow, China

Background: Neurological involvement is one of the most serious complications in Behcet’s disease (BD).

Objectives: To investigate the clinical characteristics of parenchymal neuro-Behcet’s Disease (pNBD).

Methods: We retrospectively reviewed all the medical records of BD patients who were admitted to our institute between 2000 and 2016. The diagnosis of NBD was based on the 2014 International Consensus criteria for NBD. Eighty-four BD patients without neurological involvement were randomly matched by sex and age as control.

Results: 42 patients (25 male and 17 female) with pNBD accounted for 4.2% of the 1009 hospitalised BD patients during that period. The mean age at BD onset and at neurological onset was (30.1±11.1) years old and (35.3±12.1) years old, respectively. The majority of patients developed neurological symptoms after other initial systemic symptoms of BD in a median period of 2 months (range from 0–49). Neurological onset was concurrent with the onset of BD in 6 cases (14.3%). The most frequent location was brainstem (23/42, 54.8%). Spinal cord involvement was presented in five cases, in which four with cervical cord involved. 13 cases suffered from multiple lesions. Pyramidal (21/42, 50.0%) and headache (14/42, 33.3%) were the most common manifestations of pNBD. Lumbar puncture was performed in 40 patients, in which 80% (32/40) of patients had normal pressure and 55% (22/40) had elevated protein levels (0.51±0.24) g/L). Compared with the controls, the prevalence of ocular involvement (uveitis, retinal vasculitis, scleritis) was significantly higher in pNBD (35.7%) (p=0.041, OR=2.36, 95% CI=1.03–5.44) (table 1). Cranial MRI in 32 patients showed the lesions were mainly in the midline structures, including brainstem (22/42, 52.4%), periventricular (13/42, 31.0%), centrum semiovale (8/42, 19.0%). Typically, the lesions were hyperintense in T2. All pNBD patients received corticosteroids (≥ 1 mg/kg/d) and 23 patients (54.8%) received the pulse dose (1 g/d). Cyclophosphamide was the most commonly used immunosuppressant (39/42) and 10 cases took more than two immunosuppressants (including methylotrexate and azathioprine). Biological agents were administrated in six refractory pNBD patients, including Infliximab in 4 cases, Tocilizumab in 1 case, and Interferon-α2a in 1 case. Intrathecal injection of dexamethasone 10 mg and methotrexate 10 mg was given to 28 patients. With a median follow-up of 28 months (4 to 156 months), 22 patients (52.4%) achieved clinical improvements, while 10 patients (23.8%) relapsed and 4 patients died (the mortality was 9.5%). Six patients lost to follow up.