which reported inflammatory alterations (high levels of CSF proteins in 2 subjects and oligoclonal bands in 4 patients).

Median Erythrocyte sedimentation rate (ESR) was 12 mm/h [IQR 18] while median C- Reactive protein was 0.34 mg/dL [IQR 0.32]. There was not complement deficiency in any patient. With regard to the tested auto-antibodies, 2 subjects showed also anti-La positivity. Out of the 4 patients tested for anti-AQP4 none was positive, even if 2 patients had NMOSD.

Conclusion: Anti-SSA positive may show a wide spectrum of neurological manifestations; CNS involvement was not associated to anti-AQP4 positivity in our cohort, even in patients with NMO/NMOSD. Further investigations are required to better disclose this association and to search for novel autoantibodies.

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FRI0259 SEROLOGICAL IMMUNE ABNORMALITIES ASSOCIATE WITH SPECIFIC PATHOLOGICAL ACTIVE LESIONS IN LUPUS NEPHRITIS

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Background: Serological immune abnormalities such as anti-double strand DNA (dsDNA) antibodies (Abs) and hypocomplementemia (HC) are characteristic of lupus nephritis (LN). International Society of Nephrology/Renal Pathology Society (ISN/RPS) Classification of LN defines pathological active lesions (ALs) as including endocapillary hypercellularity, karyorrhexis, fibrinoid necrosis, cellular/fibrocellular crescents, and wire-loop lesion/hyaline thrombi [Reference 1]. Few reports have focused on the clinicopathological impact of serological immune abnormalities on pathological ALs.

Objectives: To identify the clinicopathological association between serological immune abnormalities and pathological ALs in LN.

Methods: We enrolled 126 Japanese LN patients who were subjected to renal biopsy in 11 hospitals from 2000 to 2018. We determined various clinical parameters at the time of renal biopsy, including creatinine (Cr), estimated glomerular filtration rate (eGFR), total protein (TP), IgG, IgA, IgM, C3, C4, CH50, anti-nuclear antibodies (Abs), anti-double strand DNA (dsDNA) Abs, anti-Sm Abs, anti-RNP Abs in the sera, urinalysis findings, presence of comorbidities (antiphospholipid antibody syndrome, hypertension, hyperlipidemia, diabetes mellitus, and hyperuricemia), and use of any immunosuppressive medications before renal biopsy. Renal biopsy findings were classified by ISN/RPS Classification including ALs. Immune deposits were evaluated by immunofluorescence. Elevation of serum anti-dsDNA Abs level [dsDNA Abs (+)] was defined as >12 IU/mL. HC was defined by C3 <60 mg/dL. In patients of all pathological classes, we analyzed the association between serological abnormalities and ALs by univariate and multiple regression analyses.

Results: Of 126 patients (104 females; mean age 41.8 years), dsDNA Abs (+) and HC (+) were found in 83 (65.9%) and 80 (63.5%), respectively. There were no significant differences in renal function, comorbidities, immune deposits or immunosuppressive medications before renal biopsy between dsDNA Abs (+) and dsDNA Abs (-), or between HC (+) and HC (-) patients. In pathological study, endocapillary hypercellularity, karyorrhexis, fibrinoid necrosis, cellular or fibrocellular crescents, and wire-loop lesion/hyaline thrombi were found in 81 (64.3%), 33 (26.2%), 19 (15.1%), 51 (40.5%), and 41 (32.5%), respectively. dsDNA Abs (+) had a higher frequency of endocapillary hypercellularity, karyorrhexis, fibrinoid necrosis, and wire-loop lesion/hyaline thrombi than dsDNA Abs (-). HC (+) had a higher frequency of endocapillary hypercellularity, karyorrhexis, fibrinoid necrosis, cellular or fibrocellular crescents, and wire-loop lesion/hyaline thrombi than HC (-). On multiple regression analysis dsDNA Abs and C3 associated with fibrinoid necrosis and karyorrhexis, respectively ($\beta=0.25$, $p=0.049$; $\beta=-0.31$, $p=0.01$).

Conclusion: Serum anti-dsDNA Abs and HC were associated with fibrinoid necrosis and karyorrhexis, respectively. These results document that individual serological immune abnormalities associate with specific AL, suggesting that pathophysiological differences underlie each AL.

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Vasculitis

FRI0260

THE INPATIENT BURDEN OF TAKAYASU'S ARTERITIS IN THE UNITED STATES: A NATIONWIDE ANALYSIS

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Background: Takayasu's Arteritis (TAK) is a rare granulomatous inflammation of the large vessels. Its incidence has been reported to be 2-3 cases per million people in the United States. To our knowledge an inpatient nationwide study of TAK in the United States has not been conducted to date. The aim was to evaluate the demographic characteristics, reason for admission, procedures, mortality and resource utilization of patients with TAK using a national database.

Objectives: The primary goal was to estimate the national inpatient prevalence of admissions involving patients with TAK. Secondary outcomes were top reasons for admission, mortality, morbidity, resource utilization, length of hospital stay (LOS), and total hospitalization charges and costs, adjusted for inflation using the Consumer-Price Index.

Methods: All patients with a principal ICD9 diagnosis code for TAK were included using the NIS 2013 and 2014, the largest publicly available inpatient database in the US. Controls were 1:1 matched using a propensity score including age, gender, and Charlson Comorbidity Index (CCI). Multivariate logistic regression models were used to adjust for race, sex, patient zip code, income, CCI, hospital region, location, size and teaching status.

Results: Of a total of 2,840 hospital admissions for patients with TAK, 2,680 were propensity-matched to admissions without TAK and included in the study. Mean age was 49 years, 81% were female. The inpatient prevalence of TAK was 4.6/100,000 admissions. After adjusting for confounders, patients with TAK had propensity-matched mortality odds of 1.44 ($p=0.43$) when compared to patients without TAK. The top reasons for admission were large vessel vasculitis, sepsis secondary to pneumonia and urinary tract infection, carotid artery stenosis/occlusion, and arthralgias. For secondary outcomes, patients with TAK displayed increased odds of stroke, aortic aneurysm, peripheral vascular disease and aortic valvulopathy when compared to patients without TAK. When CCI was evaluated it was noted that all the patients had an index lower than 4 and the great majority lower than 2 which could be related to the young age of this population. Despite the low CCI, these patients had 7.35 days of in-hospital stay which represented 2.1 days more when compared to non-TAK patients. For resource utilization, patients with TAK displayed higher odds of peripheral vascular intervention and arteriography when compared to patients without TAK. Patients with TAK displayed significantly higher adjusted total hospital costs, charges and LOS compared to patients without TAK.

Conclusion: The inpatient prevalence was higher than suspected and could be related to the severity of the disease and complications associated with TAK. TAK was associated with an increased risk of stroke, aortic aneurysm, peripheral vascular disease and vascular interventions, but no increase mortality in this group of patients. The costs associated with hospitalization were substantial.

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FRI0261

RISK FACTORS FOR TREATMENT FAILURE IN PATIENTS WITH GIANT CELL ARTERITIS TREATED WITH TOCILIZUMAB PLUS PREDNISONE VERSUS PREDNISONE ALONE

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Background: Risk factors for treatment failure in patients with giant cell arteritis (GCA) are poorly understood.

Objectives: To identify predictors of treatment failure in GCA patients receiving tocilizumab (TCZ) or placebo (PBO) in combination with prednisone in a randomized controlled trial.¹

Methods: Two hundred fifty GCA patients received weekly or every-other-week TCZ plus a 26-week prednisone taper (TCZ+pred) or PBO plus a 26- or 52-week prednisone taper (PBO+pred). Patients who achieved and maintained clinical remission (CR) from week 12 to week 52 while adhering to the protocol prednisone taper were classified as responders. CR, adjudicated by investigators, was defined as the absence of disease flare (GCA signs or symptoms, and/or ESR elevation attributable to GCA that required further treatment [eg, rescue prednisone]) regardless of CRP level. Treatment failure was defined as failure to achieve CR by week 12 or occurrence of flare between weeks 12 and 52. Both TCZ groups and both PBO groups were combined for this analysis. Potential predictors investigated included baseline demographics, disease- and treatment-related factors, and health-related quality of life (HRQOL) measures. Univariate and multivariate analyses were performed.

Results: Overall, 45% (113/250) of patients were responders: 27% (27/101) in the PBO+pred groups and 58% (86/149) in the TCZ+pred groups. In contrast, 44% (111/250) of patients experienced treatment failure: 66% (67/101) in the PBO+pred group and 30% (44/149) in the TCZ+pred group. The other 10% (26/250) of patients were nonresponders for reasons other than treatment failure: 7 in the PBO+pred group and 19 in the TCZ+pred group. In univariate analysis, female sex and lower baseline SF-36 Physical Component Summary (PCS), Mental Component Summary, and FACIT-Fatigue scores were associated with treatment failure among PBO+pred-treated patients, whereas higher patient global assessment of disease activity scores and lower SF-36 PCS, FACIT-Fatigue, and EQ-5D scores were associated with treatment failure among TCZ+pred-treated patients (Figure 1). Among TCZ+pred-treated patients, no treatment response difference according to sex was observed. Age, previous relapse, starting prednisone dose, and GCA clinical features (cranial or polymyalgia rheumatica symptoms) were not associated with treatment failure in either group based on univariate analysis. Multivariate logistic regression demonstrated that PBO+pred treatment, female sex, worse FACIT-Fatigue scores at baseline, and historical CRP >2.5 mg/dL all independently increased the risk for treatment failure (Figure 2).

Conclusion: Female GCA patients responded particularly poorly if treated with prednisone alone according to univariate analysis. Female sex, impaired HRQOL at baseline, history of elevated CRP, and treatment with prednisone alone are independent risk factors for treatment failure in GCA. These factors may be considered when determining which treatment would be best for a particular patient.

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Figure 1. Univariate analysis of treatment failure

BL, baseline; CRP, C-reactive protein; EQ-5D, Euro-Qol 5D; ESR, erythrocyte sedimentation rate; GCA, giant cell arteritis; MCS, Mental Component Summary; PBO, placebo; PCS, Physical Component Summary; PMR, polymyalgia rheumatica; Pred, prednisone; PIGA, patient global assessment; SF-36, Short Form 36; TCZ, tocilizumab; VAS, visual analog scale.
14 patients in the PBO+prednisone group and 23 patients in the TCZ+prednisone group were nonresponders for reasons other than treatment failure. Percentages are based on number of responders/treatment failures (n).

*p<0.05 treatment failures vs responders (based on χ^2 test for categorical variables and t test for continuous variables).

BL, baseline; PBO, placebo; PMR, polymyalgia rheumatica; TCZ, tocilizumab.
Odds ratio >1 indicates the first group has higher odds of treatment failure than its comparator based on a multiple logistic regression model including all variables selected in univariate analysis prior to multivariate analysis.

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