use of Rituximab, AntiTNFs and Tocilizumab has been described, presenting a good clinical response and with few adverse effects, as has been observed in our patients.

Disclosure of Interests: None declared

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AB0504

THE DIFFERENCES OF IMMUNE CHARACTERISTICS AND ITS RELATIONSHIP WITH DISEASE ACTIVITY IN LARGE VESSEL VASCULITIS PATIENTS

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Background: Large vessel vasculitis (LVV) is a rare refractory disease, giant cell arteritis (GCA) and Takayasu arteritis (TAK) constitute different types of primary LVV. Despite considerable discussion on the potential differences between GCA and TAK, the immunological difference and its relationship to disease activity remains unclear.

Objectives: To investigate the differences of peripheral blood immuno-phenotype between TAK and GCA, and to analyze the correlation between immune metabolism level and disease activity in TAK.

Methods: All patients with TAK and GCA who come to our department from January 2010 to January 2020 were included, all of them met the classification criteria of American college of rheumatology. General information, clinical manifestations, laboratory index, artery involvement and therapeutic schedule were collected. According to the National Institutes of Health criteria, there were 29 active and 23 inactive TAK patients.

Results:

[1] Baseline characteristics, treatment, involvement artery in LVV patients are summarized in Table 1.

Table 1. Clinical characteristics of patients with LVV

Variable	TAK, n=52	GCA, n=4
Age, X±S	29±13	64±7
Female, n(%)	45(86.6)	1(25)
Constitutional symptoms, n(%)	23 (43.4)	3(75)
Cephalic symptoms, n(%)	33 (62.3)	2 (50)
Peripheral limb ischemia, n(%)	22 (41.5)	-
Ocular signs, n(%)	3 (5.7)	2 (50)
Stroke, n(%)	2 (3.8)	-
GC, n(%)	40(76.9)	3(75)
GC dose >7.5mg/day, n(%)	36 (90)	2 (66.7)
Surgical intervention, n(%)	7(13.2)	-
Coronary artery, n(%)	3(5.7)	-
Common carotid artery, n(%)	31(58.5)	-
Subclavian artery, n(%)	23(43.4)	-
thoracic aorta, n(%)	6(11.3)	-
abdominal aorta, n(%)	13(24.5)	-

GC Glucocorticoids, bDMARDs biologic DMARDs

(2) Compared with GCA patients, TAK patients were younger (29±13 vs 64±7, P=0.015), with higher peripheral blood lymphocyte count of CD8+T cells and lower CD4/CD8 (Table 2).

Table 2. Comparison of peripheral immune cells, humoral immunity among TAK and GCA, active and inactive TAK

Variable	TAK, n=52	GCA, n=4	p value	inactive TAK, n=23	active TAK, n=29	p value
Neutrophils	5.5±2.3	5.8±1.1	0.827	4.4±1.6	6.3±2.5	0.009 [*]
Lymphocytes	2.4±0.8	1.0±0.6	0.010 [*]	2.3±0.7	2.4±0.9	0.901
Monocytes	0.5±0.2	0.7±0.1	0.218	0.5±0.2	0.6±0.2	0.431
CD3 ⁺ T	1959±490	1154±541	0.189	2195±92	1842±585	0.467
CD4 ⁺ T	1074±305	758±303	0.381	1250±198	987±333	0.376
CD8 ⁺ T	859±212	297±87	0.048 [*]	945±41	815±258	0.542
CD4 ⁺ CD8 ⁺	42±27	12±7	0.344	63±2	32±28	0.229
CD4-CD8-	90±75	91±63	0.999	97±74	87±68	0.914
CD19 ⁺ B	273±97	191±94	0.470	202±106	309±84	0.242
CD16 ⁺ CD56 ⁺	135±72	104±67	0.709	112±63	146±83	0.639
CD4/CD8	1.25±0.15	2.55±0.51	0.001 [*]	1.33±0.26	1.21±0.10	0.440
IgG	11.7±3.7	8.3±0.5	0.124	10.3±3.4	12.9±3.6	0.025 [*]
IgA	2.6±1.3	2.3±0.8	0.724	2.1±1.3	3.0±1.1	0.021 [*]
IgM	1.7±0.9	0.8±0.2	0.118	1.5±0.8	1.8±0.9	0.202
K light chain	9.5±3.9	6.6±0.3	0.222	8.1±3.3	10.8±4.0	0.033 [*]
λ light chain	5.2±1.9	3.9±0.3	0.260	4.7±1.9	5.8±1.9	0.086

^{*}P<0.05

(3) The levels of neutrophils, monocytes, IgG, IgA and K light chain active TAK were higher than inactive TAK, with lower lymphocyte level (Table 2). Further correlation analysis showed that the levels of neutrophils, IgG, IgA and K light chain were positively correlated with disease activity (r=0.434, P=0.005; r=0.380, P=0.019; r=0.452, P=0.004; r=0.380, P=0.022).

Conclusion: Compared with GCA patients, TAK patients had higher level of CD8⁺T lymphocytes, consistent with previous studies. In addition, TAK patients had persistent immune cell involvement, while humoral immunity is related to disease activity. Deeper studies may be required about the role of immune profile in large vessel vasculitis.

References:

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AB0505

SEASONAL INFLUENCE IN PMR: NOT ONLY SUMMER, BUT WINTER IS COMING TOO

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Background: The cause for polymyalgia rheumatica (PMR) is currently unknown. Disease onset may be triggered by a combination of genetic predisposition and environmental factors such as infection.¹ In different regions of Denmark a simultaneous peak incidence of giant cell arthritis and PMR occurred together with epidemics of *Mycoplasma pneumoniae*, *Chlamydia pneumoniae* and Parvovirus B19.¹ A seasonal epidemics pattern for PMR would be supporting evidence for an infectious cause.¹ However, the current evidence of seasonal effect on the occurrence and disease severity of PMR is limited and show conflicting results.^{2,3}

Objectives: To evaluate whether there is a seasonal effect on the risk of developing PMR in the Netherlands

Methods: We retrospectively collected data on patient-, disease-, and treatment characteristics from newly diagnosed PMR patients (clinical diagnosis) who visited our outpatient clinic during April 2012 and September 2017. Exclusion criteria was other concomitant inflammatory rheumatic disease. Based on the onset of PMR (start symptoms, not time of diagnosis) patients were grouped per month. Descriptive statistics were used [mean (SD), median (p25-p75) or n (%) as appropriate]. The Chi square goodness of fit test was used to determine whether the incidence of onset of symptoms was different between months of the year.

Results: In total 448 patients were included and 55 % were female and mean age was 66 years. Other baseline characteristics are described in table 1. The chi-square goodness of fit test to determine whether there was a peak incidence in months was p=0.06. As shown in figure 1 the incidence of onset PMR symptoms is higher in December-January, April through June with a peak in August. The April-June peaks coincides with incidence peaks of *Mycoplasma pneumoniae* infections and possibly Parvovirus B19 in spring and summer, the December-January peak coincides with Parvovirus B19 infections.^{4,5}

Table 1. Baseline characteristics (n=448)

Characteristic	
Female (%)	247 (55)
Age, years (SD)	66 (8.6)
PMR symptoms before diagnosis, weeks (IQR)	10 (6-16)
Neck pain (%)	205 (46)
Bilateral shoulder pain	412 (91)
/stiffness (%)	
Bilateral hip pain/stiffness (%)	380 (85)
Morning stiffness>45 min (%)	233 (52)
Peripheral arthritis (%)	35 (8)
Systemic symptoms* (%)	199 (44)
Elevated ESR mm/hour and / or CRP mg/l	309 (87)
ESR mm/hour (IQR)	37 (26-51)
CRP mg/l (IQR)	30 (15-54)

*Fever, night sweats, weight loss, anorexia

** ESR n= 428; CRP n=396

Conclusion: No definitive seasonal effect was found on risk of developing PMR, although a bimodal seasonal pattern compatible with the proposed respiratory infections is suggested.

References:

- [1] González-Gay MA et al. Polymyalgia rheumatica. *Lancet.* 2017 Oct Oct 7.