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Background: IgA vasculitis (IgAV) and IgA nephropathy (IgAN) are inflammatory conditions that share pathogenic and molecular mechanisms [1] and may represent different outcomes of a continuous spectrum of disease [2]. *Interleukin (IL)17A* has been identified as a common genetic risk locus for several immune-mediated diseases [3, 4].

Objectives: To determine whether IgAV and IgAN exhibit a different *IL17A* association pattern.

Methods: Five *IL17A* tag polymorphisms (rs4711998, rs8193036, rs3819024, rs2275913 and rs7747909) were genotyped in 388 Caucasian patients with IgAV, 99 patients with IgAN and 1,003 sex and ethnically matched healthy controls.

Results: No statistically significant differences between patients with IgAV and healthy controls and between patients with IgAN and healthy controls were observed when each *IL17A* genetic variant was analyzed independently (Table 1). Similarly, IgAV patients exhibited similar genotype and allele *IL17A* frequencies than those with IgAN (Table 1). Moreover, no genotype or allele differences between IgAV patients who developed nephritis and patients with IgAN were detected. Furthermore, haplotype frequencies were similar in patients with IgAV, IgAV and nephritis and those with IgAN.

Table 1. Genotype and allele frequencies of *IL17A* gene in patients with IgA vasculitis, patients with IgA nephropathy and healthy controls.

Polymorphism	Change	Data set	1/1	1/2	2/2	1	2
rs4711998	G/A	IgAV	53.4 (207)	38.9 (151)	7.7 (30)	72.8 (565)	27.2 (211)
		IgAN	49.0 (48)	42.9 (42)	8.2 (8)	70.4 (138)	29.6 (58)
		Controls	52.7 (529)	41.2 (413)	6.1 (61)	73.3 (1471)	26.7 (535)
		IgAV	57.0 (221)	38.4 (149)	4.6 (18)	76.2 (591)	23.8 (185)
rs8193036	T/C	IgAN	64.3 (63)	31.6 (31)	4.1 (4)	80.1 (157)	19.9 (39)
		Controls	60.3 (605)	35.2 (353)	4.5 (45)	77.9 (1563)	22.1 (443)
		IgAV	44.1 (171)	43.3 (168)	12.6 (49)	65.7 (510)	34.3 (266)
		IgAN	39.4 (39)	54.5 (54)	6.1 (6)	66.7 (132)	33.3 (66)
rs3819024	A/G	Controls	45.6 (457)	44.6 (447)	9.9 (99)	67.8 (1361)	32.2 (645)
		IgAV	44.6 (172)	43.3 (167)	12.2 (47)	66.2 (511)	33.8 (261)
		IgAN	39.8 (39)	53.1 (52)	7.1 (7)	66.3 (130)	33.7 (66)
		Controls	44.8 (449)	44.2 (443)	11.1 (111)	66.8 (1341)	33.2 (665)
rs2275913	G/A	IgAV	53.9 (209)	39.4 (153)	6.7 (26)	73.6 (571)	26.4 (205)
		IgAN	41.1 (39)	54.7 (52)	4.2 (4)	68.4 (130)	31.6 (60)
		Controls	53.0 (532)	39.4 (395)	7.6 (76)	72.7 (1459)	27.3 (547)
		IgAV	53.9 (209)	39.4 (153)	6.7 (26)	73.6 (571)	26.4 (205)

Conclusion: Our results revealed that IgAV and IgAN share a similar *IL17A* association pattern.

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AB0097

DIAGNOSTIC ACCURACY OF SERUM MARKERS IN LARGE VESSEL VASCULITIS AND CORRELATION WITH PET IMAGING

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Background: The onset of symptoms in large-vessel vasculitis (LVV) tends to be subacute, which often leads to a delay in diagnosis, during which time vascular disease may start and progress to become symptomatic. PET/CT has been recognised since the 2003s as a promising tool in evaluating of LVV. There is no gold standard diagnostic laboratory tests for this patient group, novel markers for active LVV is needed.

Objectives: to investigate the association between vascular inflammation, as detected by PET imaging and interleukin-6 (IL-6), pentraxin3 (PTX3), and B-cell-activating-factor (BAFF) in subjects with LVV.

Methods: The study included 67 patients patients with newly diagnosed GCA (n= 27) or TA (n=9) and healthy control (n= 31) who had been referred to the Rheumatology Unit at Gazi University, between December 2017 and August 2020. PET images obtained from an 29 patients (22 with GCA and 7 with TA) who had not received any corticosteroid treatment prior to PET imaging and blood sampling. IL-6, PTX3, and BAFF levels were determined quantitatively by enzyme-linked immunosorbent assay (ELISA) kits.

Results: 36 patients with LVV (20 females, 16 males; age 64.5±16.6) and 31 HC (14 females, 17 males; age 37.1±9.6) were analysed. Serum levels of IL-6, PTX3, BAFF, ESR and CRP are increased in patients with newly diagnosed LVV compared with those in control subjects. In a ROC analysis, serum IL-6 provided excellent discrimination of newly diagnosed LVV patients from HC, as indicated by AUCs>0.90. Serum BAFF also accurately distinguished newly diagnosed LVV patients from HC with AUCs>0.80. Serum PTX3 did not provide an AUC>0.80. In this study, we correlate vascular inflammation, as detected by PET imaging in newly diagnosed LVV patients, with the ESR, CRP, PTX3, IL-6 and BAFF. As a result, none of these markers has been associated with vascular inflammation as measured using PET.

Conclusion: In conclusion, our study shows that serum levels of PTX3, IL-6 and BAFF are increased in most LVV patients. The diagnostic value of BAFF and IL-6, both separately and in combination, should be further evaluated in larger cohorts of LVV patients, as well as in patients with infections or other inflammatory conditions. However, none of these markers has been associated with vascular inflammation as measured using PET.

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