massive intimal hyperplasia compared to TABs with mild intimal hyperplasia (p = 0.01).

Conclusion: Based on the expression of markers of inflammation and tissue remodeling, different subsets of infiltrating macrophages can be distinguished. Macrophages in vascular lesions of GCA patients display a distinct spatial distribution pattern within the inflamed vessel wall (Figure 1). Adventitial macrophages express highest level of pro-inflammatory cytokines indicating pro-inflammatory functions of these macrophages. Co-localization of CD206 and MMP-9 along media borders indicates a role for CD206+ macrophages in collagen digestion and angiogenesis. Additionally, the association of increased numbers of FR-β+ macrophages with extensive intimal hyperplasia suggests that FR-β+ macrophages promote myofibroblast proliferation.

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SAT0233 VALIDATION OF A NOVEL DISEASE CLASSIFICATION IN HACETTEPE TAKAYASU ARTERITIS COHORT

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Background: Takayasu Arteritis (TAK) is a rare idiopathic granulomatous large vessel vasculitis and it is a clinically heterogenous disease. Various classifications and subsets which based on distribution of arterial lesions were defined in the literature in order to exhibit and predict different disease courses.

Objectives: We aimed to validate a new disease subsets developed by Goel et al in our TAK series (1).

Methods: We retrospectively evaluated the medical records of 164 TAK patients followed at Department of Rheumatology and Department of Pediatric Rheumatology in Hacettepe University, Ankara, Turkey between August 2005 and October 2018. The pediatric-onset (< 18 years of age at disease diagnosis) and adult-onset patients fulfilled the Ankara 2008 and the American College of Rheumatology (ACR) 1990 criteria for TAK, respectively. All patients were assigned to three clusters (Cluster 1: Abdominal Predominant, Cluster 2: Aortic Arch Predominant, Cluster 3: Focal Disease) using a decision tree defined in the original study (1). Demographics, clinical and laboratory features, treatment regimens were compared between in three groups.

Results: There were forty-two (25.6%), sixty-three (38.4%) and fifty-nine (36%) patients in Cluster 1, 2 and 3, respectively. Demographic and clinical data was summarized in Table-1. Cluster 1 included approximately half of patients (45.8%) with pediatric-onset TAK (p= 0.048). Baseline acute phase reactants (Erithrocyte Sedimentation Rate:ESR and C-reactive protein:CRP) and age at disease diagnosis were slightly lower in Cluster 1, however none of them did not reach statistical significance (p= 0.94, p=0.99 and 0.56, respectively). In contrast, cerebrovascular accident rates were slightly higher in Cluster 2 (p=0.24). Although anti-TNF biological agents were more frequently used for treatment in Cluster 1 (p=0.004), cyclophosphamide and/or overall biological agents usage was similar in three groups (p=0.15).

Conclusion: Decision tree which defined by Goel et al. was applied to our single center pediatric and adult TAK cohort. In contrast to original validation cohorts, there was no difference among three clusters of our cohort except pediatric-onset disease and anti-TNF usage. Dissimilarities among the cohorts in terms of new classification system may be caused either number of patients or ethnic/regional differences.

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Table 1. Demographics and Clinical Features of TAK Disease Clusters

	Cluster 1 (n=42) (Abdominal Predominant)	Cluster 2 (Aortic Arch Predominant) (n=63)	Cluster 3 (Focal Disease) (n=59)	p
Female, n (%)	37 (88.1)	58 (90.1)	53 (89.8)	0.791
Age at TA diagnosis, years, Mean ± SD	29.3 ± 15.5	32.0 ± 13.7	32.6 ± 13.8	0.56
Pediatric-Onset (< 18 years of age at disease diagnosis)	11 (26.1)	7 (11.1)	6 (10.2)	**0.048**
Median disease duration, years (IQR)	4.5 (5.1)	5.2 (6.8)	4.9 (6.9)	0.83
Median ESR at TA diagnosis, mm/h (IQR)	48.5 (59)	53.0 (59.0)	62.0 (49.0)	0.94
Median CRP at TA diagnosis, mg/dL (IQR)	2.4 (10)	3.8 (9.0)	3.6 (8)	0.99
Hypertension (HT) , n (%)	18 (42.9)	17 (27)	15 (25.4)	0.13
Serebrovasculary Accident (SVA) , n (%)	3 (7.1)	11 (17.5)	6 (10.2)	0.24
Death, n (%)	3 (7.1)	2 (3.2)	2 (3.4)	0.56
Methotrexate, n (%)	22 (52.4)	38 (60.3)	34 (57.6)	0.72
Azathiopurine, n (%)	16 (38.1)	18 (28.6)	19 (32.2)	0.59
Leflunomide, n (%)	4 (9.5)	5 (7.9)	2 (3.4)	0.42
Cyclophosphamide, n (%)	19 (45.2)	27 (42.9)	18 (30.5)	0.24
Interleukin-6 blockage, n (%)	9 (21.4)	14 (22.2)	10 (16.9)	0.75
TNF-α inhibitors, n (%)	13 (31.0)	7 (11.1)	5 (8.5)	**0.004**
Cyclophosphamide and/or biologics, n (%)	27 (64.3)	37 (58.7)	27 (45.8)	0.15
Endovascular intervention*,n (%)	8 (19)	3 (4.8)	7 (11.9)	0.069
Vascular surgery*, n (%)	4 (9.5)	10 (15.9)	4 (6.8)	0.26

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SAT0234 RNA SEQUENCING IDENTIFIES AN IGA VASCULITIS ASSOCIATED SERUM MICRORNA SIGNATURE, DISCRIMINATING PATIENTS WITH IGA VASCULITIS FROM AGE- AND SEX-MATCHED HEALTHY SUBJECTS

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Background: Immunoglobulin A vasculitis (IgAV) is a small vessel vasculitis with IgA deposits in the vessel wall, manifesting with skin purpura, arthralgia/arthritis, gastrointestinal (GI) and renal complications. Altered serum miRNA signatures can reflect disease-specific pathology, which makes serum miRNAs promising disease biomarkers. We have recently reported that miRNA expression is changed in the affected skin of IgAV patients. The profile of serum miRNAs in IgAV, however, remains unknown.

Objectives: To determine the serum miRNA signatures in adult patients with IgAV as compared with healthy controls (HC) and explore *in silico* their gene targets and pathways.

Methods: Small RNA were isolated from sera of IgAV patients with high disease activity (sera collected at the time of diagnosis) and from the age- and sex-matched HC (n=6 each). Small RNA libraries were sequenced with Illumina technology and reads were mapped onto human genome (GRCh38). One IgAV sample was omitted from analysis due to too low number of recovered miRNA reads. Unsupervised clustering was based on the expression of 150 miRNAs with the highest variance across samples. The miRror platform was used to predict gene targets of differentially expressed (DE) miRNAs with log2 fold change $\geq |1|$ and $p_{adj} < 0.01$. The STRING protein networks platform was used for pathway enrichment analysis of these genes.

Results: Unsupervised clustering of serum miRNA reads clearly distinguished between IgAV patients and HC (Figure 1). Among 1918 annotated miRNA (miRBase), 446 miRNAs were detected in serum samples and 88 miRNAs were identified as DE between IgAV patients and HC (log2 fold change $\geq |1|$, $p_{adj} < 0.05$). Specifically, 28 miRNAs were elevated in the serum of IgAV patients (e.g. miRNA-22, -146a, -185, -320a/

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