Pediatric Rheumatology

BioMed Central

Oral presentation

Open Access

4.4 Different phenotype of anaemia in systemic juvenile idiopathic arthritis (s-JIA) compared to anaemia in other subtypes of JIA

CH Hinze, N Fall, MG Barnes, S Croswell, K Jennings, S Thornton, RA Colbert, DN Glass and AA Grom*

Address: Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA
* Corresponding author

from 15th Paediatric Rheumatology European Society (PreS) Congress
London, UK. 14–17 September 2008

Published: 15 September 2008

Pediatric Rheumatology 2008, **6**(Suppl 1):S8 doi:10.1186/1546-0096-6-S1-S8

This abstract is available from: http://www.ped-rheum.com/content/6/S1/S8

Background

In previous studies of PBMC gene expression in s-JIA we noted a strong erythropoiesis signature in patients with severe anaemia, correlating with an expansion of CD34+ progenitor cells [1]. Therefore, the origin of anaemia in s-JIA may be different from anaemia of chronic inflammation. We examined CD34+ progenitor cells and PBMC gene expression in patients with s-JIA and other types of JIA.

Methods

187 patients with JIA (21 s-JIA and 166 other subtypes) prior to DMARD treatment were studied. PBMCs were isolated using Ficoll gradient centrifugation and analyzed by flow cytometry after CD34 staining. Gene expression analysis was performed on Affymetrix HG U133 plus 2.0 arrays.

Results

Patients with s-JIA and anaemia had a significant elevation of CD34+ cells compared to patients with other JIA subtypes and anaemia (Table 1). Comparing PBMC gene expression in patients with s-JIA and anaemia and other JIA subtypes and anaemia revealed 671 differentially expressed probes (T-test). Unsupervised hierarchical clustering revealed homogeneous clustering of the s-JIA group. Strongly divergent gene clusters were identified with overexpression of erythropoiesis-related genes in patients with s-JIA.

Conclusion

Patients with s-JIA and anaemia have an increased output or survival of hematopoietic progenitor cells and a specific erythropoiesis signature. This contradicts the paradigm that anaemia in s-JIA is caused by decreased erythrocyte production due to chronic inflammation. In addition, we recently showed that many patients with s-JIA have fea-

Table 1:

Disease Category	N (flow data available)	Hb in g/dL	CD34+ (% of PBMC)
I. S-JIA (Hb < 11 g/dL)	18 (5)	9.4 ± 1.0*	0.20 ± 0.12*
II. Other JIA (Hb < 11 g/dL)	21 (8)	10.5 ± 0.4*	0.08 ± 0.03*
III. S-JIA (Hb > 11 g/dL)	3 (3)	11.9 ± 1.0*	0.09 ± 0.07*
IV. Other JIA (Hb > 11 g/dL)	145 (82)	12.7 ± 1.0*	0.08 ± 0.05*
P value (ANOVA)			1.65×10^{-6}

*Values are represented as mean ± standard deviation. Comparison of CD34+ count via T test: I vs. II: p = 0.008, I vs. III.: p = 0.19, I vs. IV: p = 0.000001, II vs. III.: p = 0.65, II vs. IV: p = 0.93, III vs. IV: 0.74).

tures of subclinical macrophage activation syndrome (MAS) [2]. We hypothesize that anaemia in s-JIA may be due to hemophagocytosis and subclinical MAS.

References

1. Fall N, Barnes M, Thornton S, Luyrink L, Olson J, Ilowite NT, Gottlieb BS, Griffin , Sherry DD, Thompson S, *et al.*: **Gene expression profiling of peripheral blood from patients with untreated new-onset systemic juvenile idiopathic arthritis reveals molecular heterogeneity that may predict macrophage activation syndrome.** *Arthritis Rheum* 2007, **56:**3793-3804.
2. Bleesing J, Prada A, Siegel DM, Villanueva J, Olson J, Ilowite NT, Brunner HI, Griffin T, Graham TB, Sherry DD, *et al.*: **The diagnostic significance of soluble CD163 and soluble interleukin-2 receptor alpha-chain in macrophage activation syndrome and untreated new-onset systemic juvenile idiopathic arthritis.** *Arthritis Rheum* 2007, **56:**965-971.