

Poster presentation

CD39: a regulatory role in childhood arthritis

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Background

Human regulatory T cells (T reg) are classically defined as CD4⁺ CD25^{hi} Foxp3⁺. There is increasing evidence that T cells may be subject to regulation via the conversion of proinflammatory ATP to anti-inflammatory adenosine. Methotrexate, the standard therapy for children with JIA, is proposed to work via this mechanism. CD39 belongs to a family of ectoenzymes that convert ATP into an adenosine precursor. ATPase activity may therefore represent an important regulatory mechanism in JIA.

Materials and methods

Mononuclear cells from peripheral blood (PBMC) and synovial fluid (SFMC) of patients with JIA were analysed in parallel with PBMC from healthy controls. Samples were analysed by 5-colour flow cytometry for expression of Foxp3, CD25 and CD39. FACS sorted cells were assayed for ATPase activity.

Results

CD39 expression was demonstrated on a variety of mononuclear cells in both controls and JIA patients. Increased expression of CD39 was seen on SFMC. Mononuclear cells expressing CD39 showed rapid ATPase activity *in vitro*. We characterise a proportion of CD39⁺ T cells with a memory phenotype, express Foxp3 and have regulatory function.

Conclusion

ATPase activity may represent a novel mechanism by which regulation may occur in JIA. CD39⁺ CD4⁺ cells are enriched in the joint in JIA. CD4⁺ T cells which express both CD39 and Foxp3 may represent a population with

the capability to regulate via multiple mechanisms and therefore be more potent suppressors.