New player in the endometriosis pathogenesis: Immunoregulatory Treg

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Endometriosis is one of the most common gynecological diseases in women. Is associated with immune system impairments and associated with local and systemic immune changes. Regulatory T (Treg) cells have been implicated in the switch from predominantly inflammatory to anti-inflammatory responses. The natural Treg cells, phenotypically defined as CD4+CD25+ cells that express the transcription factor FoxP3 but not the ? chain of the interleukin (IL)-7 receptor (CD127), display several additional regulatory molecules. However, some authors describe that Treg cells have immunoregulatory capacity if they express CD39 as a marker. CD39 is a surface molecule and an ectoenzyme that in cooperation with CD73 have the potential of T cell inhibition. The objective of this study was to identify the immunoregulatory capacity of Treg in patients with deep endometriosis. We analyzed peripheral blood from 24 women with deep endometriosis and 11 healthy subjects. Flow cytometric assay were performed. Inclusion criteria for both groups were women between 18 and 45 years old, regular menstrual cycles, without the using hormonal treatment for at least 3 months and without associated diseases. No differences in the natural Tregs or conventional T cells were observed between deep endometriosis and healthy subjects. However the deep endometriosis patients showed a lower percentage in the PBMC compare with healthy subject, p=0.04. Conventional T cells have only a low basal expression of CD39. The CD39 mediates suppressive activity of human Treg cells, probably through the generation of adenosine. Other soluble factors, such as IL-10 and TGF-beta are also released by activated Treg cells and predominantly mediate suppression in vivo. In the CD39/CD73 ectonucleotidase cascade, ATP is the crucial substrate for adenosine production, and therefore access of Treg cells to ATP at inflamed tissue sites is mandatory. These Treg cells (CD39+CD73+), together with other tissue-resident cells, might produce sufficient adenosine to prevent exacerbated tissue damage during ongoing immune reactions. In conclusion, if the deep endometriosis patients has lower in these cells in the periphery, its important investigate in the lesion (tissue) and these data reveal new player in the endometriosis pathogenesis and could be a tissue biomarker in this disease.

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Auteurs :
Références : , , ,

Auteurs

Karina Carvalho 1, Isabella Deho 1, Ana Eduarda Carvalho 1, Luiz Rizzo 2, Sergio Podgaec 2, Mauricio Abrao 3,

1. Research Institute, Hospital Israelita Albert Einstein, São Paulo, BRAZIL
2. Research Institute, Hospital Israelita Albert Einstein, Research Institute, BRAZIL
3. Obstetrics and Gynecology, Faculdade de Medicina Universidade de São Paulo, São Paulo, BRAZIL

Auteurs (raw format)

Carvalho Karina - email : karina.ladislau@einstein.br Etablissement : Hospital Israelita Albert Einstein Service : Research Institute Ville : São Paulo Pays : BRAZIL Présentateur : Oui
Deho Isabella - email : isazdeho@gmail.com Etablissement : Hospital Israelita Albert Einstein Service : Research Institute Ville : São Paulo Pays : BRAZIL Présentateur : Non
Carvalho Ana Eduarda - email : anaeduardac@gmail.com Etablissement : Hospital Israelita Albert Einstein Service : Research Institute Ville : São Paulo Pays : BRAZIL Présentateur : Non
Rizzo Luiz - email : lvrizzo@einstein.br Etablissement : Hospital Israelita Albert Einstein Service : Research Institute Ville : Research Institute Pays : BRAZIL Présentateur : Non
Podgaec Sergio - email : sergiopodgaec@me.com Etablissement : Hospital Israelita Albert Einstein Service : Research Institute Ville : Research Institute Pays : BRAZIL Présentateur : Non
Abrao Mauricio - email : msabrao@mac.com Etablissement : Faculdade de Medicina Universidade de São Paulo Service : Obstetrics and Gynecology Ville : São Paulo Pays : BRAZIL Présentateur : Non