IL-22 promotes angiogenesis of adenomyosis by strengthening crosstalk between vascular endothelial and endometrial stromal cells

Abstract ID : 1040
Soumis par : Mingqing Li Le 2015-12-05 10:26:58
Nom de la catégorie : SEUD CONGRESS
Typologie : Communication orale / Oral communication
Statut : validé
Autorisation de diffusion : Yes/Oui

Our previous work has demonstrated that interleukin-22 (IL-22) enhances the invasiveness of endometrial stromal cells (ESCs) of adenomyosis in an autocrine manner. In the present study, we further investigated whether IL-22 mediated crosstalk between vascular endothelial cells (VECs) and ESCs in vitro and in vivo. Here we found that VECs in ectopic lesion from women with adenomyosis highly expressed IL-22 receptors IL-22R1 and IL-10R2. Both recombinant human IL-22 (rhIL-22) and IL-22 from ESCs increased IL-22R1 and IL-10R2 expression on human umbilical vein endothelial cells (HUVECs). Treatment with rhIL-22 led to an elevation of HUVECs viability, but did not influence HUVECs apoptosis. In contrast, anti-human IL-22 neutralizing antibody (?-IL-22) inhibited HUVECs viability induced by supernatants of ESCs. Stimulation with rhIL-22 or ESCs up-regulated CD105 expression on HUVECs and promoted angiogenesis, and ?-IL-22 could reverse these effect induced by ESC. Compared to non-treated HUVECs, HUVECs educated by rh-IL-22 or ESCs could further up-regulate Ki-67 and proliferating cell nuclear antigen (PCNA) expression, and down-regulate Fas ligand (FasL) expression in ESCs. However, these effects induced by ESC-educated HUVECs were inhibited by ?-IL-22. Injection with IL-22 neutralizing antibody led to decreases of blood vessel density and proliferation in ectopic lesion, and uterus weight of AD mouse. These results suggest that IL-22 derived from ESC promotes IL-22 receptors expression and enhances the viability, activation and angiogenesis of HUVEC. In turn, the educated HUVEC may further stimulate proliferation and restricts apoptosis of ESC. The integral effect may contribute to the progress of adenomyosis. Blocking IL-22 can disturb crosstalk between ESC and VEC mediated by IL-22, suggesting that blocking IL-22 may be a potential treatment strategy for adenomyosis.

Mots clefs : IL-22, endometrial stromal cells, vascular endothelial cells, angiogenesis, adenomyosis
Auteurs :
Références : , , ,

Auteurs

Mingqing Li 1, Jun Shao 1, Liping Jin 1,

1. Obstetrics and Gynecology Hospital of Fudan University, Laboratory for Reproductive Immunology, Shanghai, CHINA

Auteurs (raw format)

Li Mingqing - email : mqli@fudan.edu.cn Etablissement : Laboratory for Reproductive Immunology Service : Obstetrics and Gynecology Hospital of Fudan University Ville : Shanghai Pays : CHINA Présentateur : Oui
Shao Jun - email : shaojun700523@163.com Etablissement : Laboratory for Reproductive Immunology Service : Obstetrics and Gynecology Hospital of Fudan University Ville : Shanghai Pays : CHINA Présentateur : Non
Jin Liping - email : jinlp01@163.com Etablissement : Laboratory for Reproductive Immunology Service : Obstetrics and Gynecology Hospital of Fudan University Ville : Shanghai Pays : CHINA Présentateur : Non