Epigenetic change induced by reactive oxygen species in endometriosis

Young Eun Jeon*,1, Kyung Jin Lim2, Kyung Eun Lee1, Hyo In Yang2, Hye Yeon Kim3, Seok Kyo Seo2,
Young Sik Choi2, SiHyun Cho1, Byung Seok Lee1

Obstetrics and Gynecology, Yonsei University College of Medicine, Gangnam Severance Hospital1,
Severance Hospital2, Yongin Severance Hospital3

목적: In addition to genetic alterations, several epigenetic aberrations have been reported in endometriosis. Promoter methylation is associated with gene silencing. Reactive oxygen species (ROS) is considered to be one of the most important factors affecting the development of endometriosis. However, the relationship between ROS and epigenetic changes in endometriosis is not clear. We sought to determine whether ROS promote hypermethylation of the promoter region of E-cadherin, a regulator of the epithelial-to-mesenchymal transition, in endometriosis.

방법: Primary culture cell of endometiotic stromal cell and endometrial stromal cell were exposed to H2O2. E-cadherin expression level was examined by real-time reverse-transcriptase polymerase chain reaction. The methylation status of E-cadherin was examined by bisulfate sequencing.

결과: We found that ROS induced hypermethylation of the E-cadherin promoter in endometriosis.

결론: These finding provide novel mechanistic insights into epigenetic modulations induced by ROS in endometriosis.