Table 1

	Group 1[n=90]	Group 2[n=64]	p-value
Mean number of leiomyomata resected	3.5±3.6	2.3±2.1	<0.05
Median uterine size(cm)	15.4 [10.2-18.8]	9 [7.4-10]	<0.05
Median myoma weight(g)	244.1 [107.2-387.9]	58.2 [34.7-112]	<0.05
Median EBL(ml)	100 [100-200]	100 [50-125]	NS
Time to conception (days)	307 [234-751]	299 [213-465]	NS
Patients that conceived(%)	21.1	29.7	NS
Spontaneous conception(%)	32	58	NS
Vaginal bleeding(%)	5.3	10.5	NS
Subchorionic hematoma(%)	5.3	15.8	NS

CONCLUSION: No difference exists in time to conception following abdominal versus robotic myomectomy. Median myoma size of 9 cm and weight of 58 g were safely resected robotically with outcomes comparable to abdominal myomectomy. No statistical difference was found in mode of conception between the two surgical modalities. However, the rate of spontaneous conception was higher following robotic myomectomy with a trend toward significance. This finding may be explained by a lower chance of adhesion formation. Future directions include evaluation of delivery outcomes.

ENDOMETRIOSIS

P-453 Wednesday, October 22, 2014

HIGH MOBILITY GROUP BOX 1 PROMOTES CELL PROLIFERATION THROUGH TOLL-LIKE RECEPTOR 4 AND NF-κB PATHWAY IN ENDOMETRIAL STROMAL CELLS. Y. J. Lee,[a,c] E.-J. Han,[a,c] B. H. Yun,[a,c] S. J. Chon,[a,c] S. Cho,[b,c] Y. S. Choi,[a,c] B. S. Lee.[b,c] [a]Department of Obstetrics and Gynecology, Severance Hospital, Seoul, Republic of Korea; [b]Department of Obstetrics and Gynecology, Gangnam Severance Hospital, Seoul, Republic of Korea; [c]Institute of Women's Life Medical Science, Yonsei University College of Medicines, Seoul, Republic of Korea.

OBJECTIVE: Cell death through necrosis activates the innate immune system and induces sterile inflammation. High mobility group box 1 (HMGB-1) is a DNA-binding nuclear protein, however, mediates inflammatory reaction in extracellular condition whether actively released by endotoxin or passively by cell injury. The aim of the study was to examine the HMGB-1 existance, and HMGB-1 signaling through toll like receptor (TLR) 4 in endometrium, which activates NF-κB pathway that might play a pathogenic role in endometriosis.

DESIGN: Laboratory study using endometrial cell culture.

MATERIALS AND METHODS: From March 2012 to March 2014, 69 patients who had undergone hysterectomy were included; 39 patients with endometriosis were enrolled as the case group, 30 patients without endometriosis were enrolled as the control. Endometrial tissue was obtained from the each patient and expression of HMGB-1 in endometrium was examined by immunohistochemistry. From endometriosis patient, endomtrioma tissue was obtained and used to culture the endometrial cell in vitro. The cultured human endometrial stromal cells (HESCs) were treated with recombinant HMGB-1(15ng/mL) for 48 hours. Cell proliferation was assessed by a CCK-8 proliferation assay kit. Real time PCR and western blot were used to quanitify TLR4 mRNA and protein levels. Specific inhibitor of NF-κB signaling pathway (Bay 11-7082) was used to explore the role of NF-κB signaling pathway in HESCs proliferation.

RESULTS: NAG-1 immunostaining varied over the menstrual cycle in both groups. NAG-1 expression was significantly higher in both glandular epithelial cells and stromal cells of the endometriosis group, compared to the controls. HMGB-1 significantly enhanced cell proliferation in HESCs and increased TLR4 expression by dose dependent fashion. Inhibiting TLR4 by LPS-RS, cell proliferation in HESCs and expression of TLR4 were significantly decreased comparing to the controls. After blocking NF-κB pathway with Bay 11-7082, cell proliferation in HESCs was significantly decreased. HMGB-1 signaling was associated with regulation on the activation of NF-κB signaling pathway.

CONCLUSION: This study showed that HMGB-1 may play an important role in establishment of endometriosis through TLR4 and NF-κB pathway and initiation of the proinflammatory cascade in endometriosis.

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EXPRESSION OF PERIOSTIN AND SYNDECAN-1 IN ENDOMETRIOSIS. P. Orecchia,[a,b] S. Ferrero,[c,d] L. Odetto,[c,d] D. Croxatto,[a,b] V. Remorgida,[c] P. L. Venturini,[c,d] M. C. Mingari,[a,b] B. Carnemolla.[a] [a]Laboratory of Immunology, IRCCS AOU San Martino-IST, Genova, GE, Italy; [b]Department of Experimental Medicine, University of Genova, Genova, GE, Italy; [c]Department of Obstetrics and Gynecology, IRCCS AOU San Martino - IST, Genoa, GE, Italy; [d]DINOGMI, University of Genova, Genova, GE, Italy.

OBJECTIVE: Angiogenesis plays a pivotal role in endometriosis, and antiangiogenic therapies have been proposed as therapeutic approach. The objective of this study is to investigate the expression in endometriosis of two proteins involved in the angiogenic process, periostin and syndecan-1.

DESIGN: Laboratory study.

MATERIALS AND METHODS: Eutopic endometrium and ectopic endometriotic lesions (endometriotic cysts and rectovaginal nodules) were obtained from premenopausal women undergoing laparoscopy because of endometriosis. Criteria for exclusion from the study were: menstrual bleeding on the day of surgery, signs of pelvic inflammatory disease, use of hormonal therapies in the 3 months prior to surgery, use of intrauterine device in the 3 months prior to surgery, pregnancy or breastfeeding in the 6 months prior to surgery. All patients included in the study had histological diagnosis of endometriosis. The expression of periostin and syndecan-1 were evaluated by immunofluorescence techniques. The expression of periostin was assessed by using the murine monoclonal antibody OC-20, a function-blocking anti-periostin antibody (1). The expression of syndecan-1 was assessed by using the human recombinant OC-46F2 antibody that is specific for the extracellular domain of syndecan-1 (2).

RESULTS: Ten patients with rASRM stage III-IV disease were included in the study. Periostin and syndecan-1 were highly expressed in the stroma of eutopic and ectopic endometrium of patients with endometriosis. Both OC-20 and OC-46F2 antibodies were able to recognize very well vascular structures, as shown by the colocalization with antibodies specific to several endothelial (CD31, CD34, VE-Cadherin) and pericitic (smooth muscle actin) markers.

CONCLUSION: Eutopic and ectopic endometrium of patients with endometriosis highly express periostin and syndecan-1. Previous studies showed that OC-20 and OC-46F2 antibodies are able to inhibit angiogenesis during tumor growth in pre-clinical in vivo models (1,2). These observations may contribute to the employment of novel anti-angiogenesis biological drugs in endometriosis.

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P-455 Wednesday, October 22, 2014

AQP1-DEPENDENT ANGIOGENESIS PLAYS A CRUCIAL ROLE IN THE PATHOGENESIS OF ENDOMETRIOSIS. L. Zou,[a] S. Shi,[a] J. Sheng,[b] H. Huang.[c] [a]People's Hospital of Jinhua City, Jinhua, Zhejiang Province, China; [b]Department of Pathology and Pathophysiology, School of Medicine, Zhejiang University, Hangzhou, Zhejaing Province, China; [c]International Peace Maternity and Child Health Hospital, Shanghai, China.

OBJECTIVE: 1)to investigate the expression of Aquaporin-1(AQP1) in the blood vessel endothelium of ectopic endometrium of endometriosis; 2) to investigate the effect of AQP1 in the angiogenesis and the underlying mechanism.

DESIGN: Clinical sample was used to research the location of AQP1 on the endometrium and expression difference of AQP1 between the normal and ectopic endometrium. Cell model(Human Umbilical Vein Endothelial Cells, HUVECs) was used to research the effect of AQP1 on the angiogenesis and the mechanism underlying. Animal model was used to research the effect of technically up-or down-regulation of AQP1 on the angiogenesis in vivo.

MATERIALS AND METHODS: Materials:
Cell lines:normal Human Umbilical Vein Endothelial Cells(HUVECs)