sprouting of neovessels but also can provoke net destruction of collagen in the shoulder regions of fibro-atheromas and thus contribute to the weakening of the fibrous cap and precipitate transition to an unstable lesion, plaque rupture, leading to myocardial infarctions or strokes. Furthermore, specific MMPs have been shown to enhance angiogenesis by releasing ECM-bound angiogenic growth factors.
Conclusions. By providing pathological angiogenesis MMPs may induce plaque growth, maintenance or destabilizing of the atherosclerotic plaque.
Key words: atherosclerosis, angiogenesis, matrix metalloproteinases

232. STUDY OF THE EPITHELIO-MESENCHYMAL TRANSITION PROCESS IN THE PATHOGENESIS OF GASTROINTESTINAL TRACT ENDOMETRIOSIS

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Introduction. Epithelial – mesenchymal transition (EMT) endows cells with migratory and invasive proprieties, a prerequisite for the establishment of endometriotic lesions. The role EMT might play in the pathophysiology of endometriosis is still unknow. Therefore, we examined four markers for EMT in endometrium and endometriosis: E – cadherin + Vimentin, double reactions and simple reactions Twist and N – cadherin.
Aim of the study. The role EMT in the pathophysiology of endometriosis.
Materials and methods. During a period of five years (2012 – 2017) we analyzed 7 cases of gastrointestinal tract endometriosis: appendix (1case), colon (5 cases), ileum (1case). The material was processed according to the classic histological technique by inclusion in paraffin. The 3 μm sections obtained were stained with Hematoxylin – Eosin and Masson's trichrome stains. Another sections were dewaxed, rehydrated and processed for immunohistochemistry using as primary antibodies monoclonal antibodies Vimentin and mouse monoclonal antibody N – cadtherin, E – cadherin, Twist.
Results. Immunohistochemically, we aimed to change the immunophenotype from epithelial to mesenchyme in gastrointestinal endometriosis by analyzing the most important markers of the transition process. In endometriosis and endometrium E – cadherin, Vimentin, N – cadherin and Twist were expressed on protein level. Investigation of E – cadherin / Vimentin coexpression revealed a decrease in E – cadherin reactivity at the site of invasion of gastrointestinal endometriosis with an increase in reactivity to Vimentin together with the increase of the invasion pattern and the increase of the stage of the disease respectively. Twist transcription factor immunoexpression revealed a highly positive expression on the mesenchymal lineage, proving involvement of this transcriptional factor in the invasion process of gastrointestinal endometriosis. N – cadherin was positive in the endometrial glands, showing their differentiation into a mesenchymal phenotype and their migratory potential.
Conclusion: The results of our study confirm involvement of the epithelial – mesenchymal transition process in the pathogenesis.
Key words: endometriosis, gastrointestinal tract, mesenchymal transition

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233. THE DEVELOPMENT OF ANTIBIOTIC RESISTANT BACTERIA IN HOSPITALS