

tumor-free omentum, indicating a niche for ovarian cancer cells toward omental metastasis.

Conclusion Our data demonstrate that $\alpha 2,6$ sialylation on integrin $\alpha 2$ triggers ovarian cancer cell adhesion to metastatic sites. Therefore, blocking sialylation and integrin $\alpha 2$ may be a therapeutic target for preventing ovarian cancer metastasis in the future.

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EVALUATION OF IMMUNOHISTOCHEMICAL EXPRESSION OF MICROFIBRILLAR-ASSOCIATED PROTEIN 5 (MFAP5) IN INVASIVE BREAST CARCINOMA OF NO SPECIAL TYPE

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Introduction Breast cancer (BC) remains the most prevalent female cancer in Egypt and worldwide. Microfibrillar-associated protein 5 (MFAP5) is a multifunctional glycoprotein. Although MFAP5 gene was among the genes that found globally expressed in human cancers, it had been only recently reported in few cancer research studies.

Material and methods This is a retrospective study that has been conducted on 66 Egyptian patients who had invasive carcinoma of no special type (IC-NST). Immunohistochemical staining for MFAP5 was applied on the archival formalin-fixed paraffin-embedded blocks. Staining was assessed semiquantitatively and correlated with the available clinicopathological parameters and immunohistochemical subtypes of BC.

Results and discussions MFAP5 epithelial cytoplasmic expression was observed in 89.4% (59/66) of cases. In contrast, nuclear expression was seen in normal breast lobules and premalignant lesions adjacent to tumours that also exhibited constant staining in myoepithelial layer. Statistical analysis of epithelial cytoplasmic expression revealed association of MFAP5 expression with tumour size ($p=0.046$), high histological grade ($p=0.007$), presence of lymph node (LN) metastasis ($p=0.014$), poor Nottingham Prognostic Index (NPI) ($p=0.001$), late stage ($p=0.008$), immunohistochemical subtypes of BC ($p=0.018$), and increased MVD using CD34 immunostaining ($p=0.04$). MFAP5 cytoplasmic expression was also observed in an adjacent DCIS component in 37/45 cases (82.2%).

Conclusion This study showed that MFAP5 is a novel myoepithelial cell marker that appears to be up-regulated in duct epithelium in DCIS and IC-NST during tumourogenesis and that its cytoplasmic expression in invasive tumours seems to have a poor prognostic role manifested by its association with poor prognostic parameters such as high grade, late stage, lymph node invasion and increased MVD.

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THE SECRETARY MIR-141 FACILITATES OVARIAN CANCER METASTASIS THROUGH REPROGRAMMING STROMAL FIBROBLAST CELLS IN PRE-METASTATIC NICHES FORMATION

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Introduction Regardless of the modern advances in cancer therapeutics, cancer metastasis is still a major obstacle in the clinical management of ovarian cancer. Emerging evidence discloses exosomal miRNAs act as critical roles in cancer development. One of the possibility is the secretary miRNAs mediating communications between tumour cells and their tumour microenvironment. However, the functions and mechanisms of miRNAs in regulating cancer metastasis are not fully understood. We have previously identified that Hsa-miR-141 (miR-141) is not only aberrantly expressed in aggressive ovarian cancer but also enhances anoikis resistance in metastatic progression of ovarian cancer through targeting KLF12/SP1/Survivin axis. Here, we report that miR-141 is also a tumour secretary miRNA which can remodel the stromal cells to facilitate the formation of pre-metastatic niches for ovarian cancer metastasis.

Material and methods QPCR analysis was used to evaluate the level of exosomal miR-141 in the conditioned medium of ovarian cancer cells. T-HESCs and WPMY-1 were used for miR-141 mediated reprogramming stromal cells. Cytokine array, ELISA, QPCR and Western blot analyses were used for measuring the levels of EMMPRIN and GRO- α . LC-MS/MS and proteomic analyses to identify miR-141-mediated target, Yes Associated Protein 1(YAP1).

Results and discussions miR-141 was frequently overexpressed in ovarian cancer cells and also secreted to the surroundings through exosomal pathway. Functional analyses revealed miR-141 enables to reprogram the stromal cells because miR-141-expressing stromal cells showed an escalating secretion of cytokines GRO- α and EMMPRIN in cultured media. By co-culture with ovarian cancer cells with above conditioned media, or the recombinant proteins of GRO- α and EMMPRIN, ovarian cancer cells exhibited increased cell proliferation. Proteomic profiling of miR-141 reprogrammed stromal cells revealed YAP1, a key downstream effector of the Hippo pathway was suppressed. Depletion of YAP1 in stromal cells also increased the expression of GRO- α and EMMPRIN in the conditioned media. On the contrary, restoration of YAP1 attenuated the escalated secretion of GRO- α and EMMPRIN, indicating the reduction of YAP1 might be required for increased YAP/TAZ-mediated transcriptional activities of GRO- α and EMMPRIN.

Conclusion Our preliminary findings suggest the exosomal miR-141 could reprogram stromal cells through altering Hippo/YAP1 signalling in production of GRO- α and EMMPRIN, to facilitate metastatic colonisation of ovarian cancer.

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HEPATOCTE GROWTH FACTOR ACTIVATOR INHIBITOR-2 SUPPRESSES HUMAN PROSTATE AND LUNG CANCER CELL INVASION AND METASTASIS

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Introduction Cancer metastasis is a main cause for mortality. Dysregulation of pericellular proteolysis usually accounts for cancer cell invasion and metastasis. In this study, we are interested in delineating the role of hepatocyte growth factor activator inhibitor-2 (HAI-2) in prostate and lung cancer cell invasion and metastasis.

Material and methods We used different prostate and lung cancer cells and animal models to examine the role of HAI-2 in prostate and lung cancer cell invasion and metastasis.