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Gemcitabine promoting CCL2 secretion by bladder cancer cell recruited MDSCs into tumor microenvironment

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Statement of the Problem: Bladder cancer (BC) is one of the most common malignant tumors of the urinary tract in China, 75% to 85% of which are grouped as non-muscle invasive bladder tumors. Gemcitabine (GEM), mainly known as chemotherapeutic drug, is known to enhance the resistance potential of cancer cells to chemotherapy. However, the mechanism of that remains unclear.

Method of Study: In this study, 5637 and T24 bladder cancer cell lines were divided into control groups and GEM treated groups. The cells treated drug concentration was tested by CCK-8 experiment. The chemotaxis of myeloid-derived suppressor cells (MDSC) from peripheral blood was evaluated by flow cytometry assay. The mRNA and protein level of CCL2 in BC cells was analyzed by qRT-PCR, ELISA and flow cytometry assay. The expression of the key molecules in GEM treated BC cells was assessed by in-cell Western assays. The effects of MDSCs on the proliferation of BC cells *in vitro* were assessed using flow cytometry assay.

Results: We have found that though GEM treatment can kill and wound BC cells, which leads to higher expression of CCL2 in the GEM treated groups than the control groups. High level of CCL2 significantly enhances the chemotaxis of MDSCs in the microenvironment of BC cells. The accumulation of MDSCs can promote the progression of BC, such as the proliferation, invasion and migration of the BC cells. This phenomenon can be inhibited by using CCR2 antagonist.

Conclusions: GEM can enhance BC cells generate much more CCL2, which can recruit MDSC to tumor site. This phenomenon can partly explain the mechanism of GEM related chemotherapy resistance.

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