Dong Tang, Pancreat Disord Ther 2017, 7:3 (Suppl)
DOI: 10.4172/2165-7092-C1-005

High expression of galectin-1 in pancreatic stellate cells induced T cell apoptosis and skewed Th1/Th2 balance

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Pancreatic cancer microenvironment is composed by stromal cells and extracellular components, in which the main stromal cell includes activated pancreatic stellate cells (PSC, one of the most important stromal cells). PSC in pancreatic cancer microenvironment can promote tumor cell growth, and increase the tumor cells resistance to chemical drugs in vitro. Galectin-1 is a lectin protein with high affinity to β-galactose, which can inhibit T cell proliferation and induce tumor infiltrating T-cell apoptosis. Recently, it has founded that Galectin-1 was significantly expressed in cultured activated PSC. As a lot of activated PSC existing in pancreatic cancer microenvironment, the relationship between endogenous Galectin-1 of PSC and the pancreatic cancer immunosuppression is unclear. PSC were isolated from resected fresh pancreatic tissue and Galectin-1 was knocked down using a small hairpin RNA (sh RNA) or overexpressed using recombinant lentiviruses. In order to investigate the relationship between Galectin-1 expression and tumor immune suppression in pancreatic cancer, the impacts on T cells function and apoptosis by primary PSC with different levels of Galectin-1 expression were studied, and the expression of Galectin-1 and CD3 in pathological specimens of pancreatic cancer, chronic pancreatitis and normal pancreas tissues were analyzed. Compared with normal PSCs, PSCs with Galectin-1 over-expression significantly induced apoptosis of CD3+T cells ($p<0.01$), CD4+T cells ($p<0.01$) and CD8+T cells ($p<0.05$), and CD3, CD4 and CD8 T cells apoptosis was significantly decreased in PSCs with Galectin-1 silenced ($p<0.05$). Compared with normal PSCs, PSCs with Galectin-1 over-expression significantly inhibited secretion of Th1 cytokines (IL-2 and INF-γ) ($p<0.01$), and induced secretion of Th2 cytokines (IL-4 and IL-5) ($p<0.01$), and PSCs with Galectin-1 silenced increased Th1 cytokines (IL-2 and INF-γ) secretion ($p<0.01$) and decreased Th2 cytokines (IL-4 and IL-5) secretion ($p<0.01$ and $p <0.05$, respectively). Expression of Galectin-1 and CD3 in pancreatic cancer tissues were located around the pancreatic cancer cells and significantly high than chronic pancreatitis and normal pancreas tissues ($p<0.01$). Our study suggests that PSC with Galectin-1 high expression promoted the T-cell apoptosis, and significantly inhibited the secretion of Th1 cytokines (IL-2 and INF-γ) and induced secretion of Th2 cytokines (IL-4 and IL-5) which skewed Th1/Th2 balance. High expressed Galectin-1 of PSC in pancreatic cancer microenvironment might form a tumor immunosuppression barricade which induced apoptosis of T cells and inhibited the infiltration of T cells, and results in development of immunosuppression of pancreatic cancer.

Biography

Dong Tang has completed his PhD from Nanjing Medical University and Post-doctoral studies from Nanjing University of Medicine. He is Supervisor and Assistant Chief Physician of Clinical Medical College of Yangzhou University (Subei People's Hospital of Jiangsu Provinc). He got a chance of being a Visiting Scholar in the National Cancer Center in Japan from December 2012 to January 2013. He has published more than 20 papers in reputed international journals.

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