J Cancer Sci Ther 2013, 5:10
http://dx.doi.org/10.4172/1948-5956.S1.030

OMICS Group Conferences
Accelerating Scientific Discovery

3rd World Congress on
Cancer Science & Therapy

October 21-23, 2013 DoubleTree by Hilton Hotel San Francisco Airport, CA, USA

Efficacy of different regimens of intraperitoneal chemoperfusions with cisplatin in a rat model of ovarian cancer peritoneal carcinomatosis

V. G. Bespalov, O. A. Belyaeva, A. N. Stukov, K. Yu. Senchik, G. S. Kireeva and V. A. Aristova
N.N. Petrov Research Institute of Oncology of the Russian Ministry of Health, Russia

Currently hyperthermic intraperitoneal chemoperfusion (HIPEC) becomes an important part of peritoneal carcinomatosis treatment in patients with ovarian cancer. Cisplatin is commonly used at this treatment; however doses of the drug and chemoperfusion regimens are discussed. Comparative study of HIPEC and normothermal intraperitoneal chemotherapy (IPEC) with high-doses of cisplatin in a rat model of ovarian cancer peritoneal carcinomatosis was our purpose. Ovarian cancer cells were inoculated intraperitoneally (i.p.) in 99 Wistar female rats. We used cisplatin 48 hours after the i.p. tumor inoculation in maximal tolerable doses of 4 mg/kg for i.p. injection, 40 mg/kg for IPEC, and 20 mg/kg for HIPEC. In the rats of control group saline solution was administered i.p. at a volume of 0.5 ml. The antitumor effect of cisplatin was estimated by increasing median survival time (MST). In the control group MST was 9.0 days. Single i.p. injection of cisplatin increased MST by 116% (MST=19.5, p=0.008) compared with the control group. IPEC and HIPEC with cisplatin increased MST by 317% (MST=37.5, p<0.001) and by 183% (MST=25.5, p<0,003), correspondingly, compared with the control group. In comparison with IPEC without drug (MST=16.0) IPEC with cisplatin increased MST by 134% (p=0,002). Thus cisplatin administered with intraperitoneal chemoperfusions had the most significant cytostatic effects; however at IPEC regimen cisplatin had higher maximal tolerable dose and antitumor activity than at HIPEC regimen. IPEC with cisplatin may be more effective and less toxic than HIPEC with the drug and further clinical trials of these treatment regimens are warranted in ovarian cancer patients.

Biography

V. G. Bespalov, is as Doctor of Medical Sciences, Head of Laboratory of Cancer Chemoprevention and Oncopharmacology, has been working in the N.N. Petrov Research Institute of Oncology during 30 years. He is member of an administrative Academic Council and a dissertational Academic Council of the N.N. Petrov Research Institute of Oncology, member of the Russian Oncological Society and the Russian Oncourological Society, scientific editor of Russian medical journals. He has published more than 130 papers in reputed journals and 20 monographies and books on the topics of cancer prevention, cancer chemoprevention, cancer chemotherapy, treatment of precancerous diseases, dietology and rehabilitation.

Hep88 mAb Initiated paraptosis-like PCD pathway in hepatocellular carcinoma cell line through the binding of mortalin (HSPA9) and α-enolase

Panadda Rojpibulstit[1], Suthathip Kittisenachai[2], Songchan Puthong[3], Sirikul Manochantr[4], Pornpen Gamnarai[1], Sasichai Kangsadalampai[1] and Sittiruk Roitrakul[2]
[1]Thammasat University (Rangsit Campus), Thailand
[2]National Center for Genetic Engineering and Biotechnology, Thailand
[3]Chulalongkorn University, Thailand
[4]Mahidol University, Thailand

Hepatocellular carcinoma (HCC) is the most primary hepatic cancer worldwide. Nowadays a targeted therapy via monoclonal antibodies (mAbs) specific to tumor-associated antigen is continually developed in HCC treatment. In this regard, after establishing and consequently exploring Hep88 mAb's tumoricidal effect on hepatocellular carcinoma cell line (HepG2 cell line), the Hep88 mAb's specific Ag from both membrane and cytoplasmic fractions of HepG2 cell line was identified by 2-D gel electrophoresis and western blot analysis. After in-gel digestion and subsequently analysis by liquid chromatography-mass spectrometry (LC-MS), mortalin (HSPA9) and α-enolase were identified. The recombinant proteins specific to Hep88 mAb were cloned and expressed in *E.coli* BL21 (DE3). Moreover, alteration of HepG2 and Chang liver cell line after induced by Hep88 mAb for 1-3 days were investigated using a transmission electron microscope.

The result demonstrated that Hep88 mAb can bind to the recombinant mortalin (HSPA9) and α-enolase. In addition, the gradually appearing of mitochondria vacuolization and endoplasmic reticulum dilatation were observed. Taken together, paraptosis-like program cell death (PCD) of HepG2 is induced by binding of mortalin (HSPA9) and α-enolase to Hep88 mAb. Mortalin depletion by formation of Hep88 mAb-mortalin (HSPA9) complex might initiate transcription-independent of p53-mediated apoptosis. Additionally, Hep88 mAb-α-enolase complex might initiate HepG2 cells energy exhaustion by glycolysis pathway obstruction. These fascinating results imply that Hep88 mAb might be a promising tool to a development of an effective treatment of HCC in the next decade.