P12 EFFECT OF PRIMARY HUMAN CHOLANGIOCARCINOMA CELL ON PLATELET FUNCTIONS.

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ABSTRACT

Tumor cell induced platelet aggregation (TCIPA) played an importance role in early state of thrombosis in cancer patients. In addition, TCIPA was recognized as one important step in metastatic cascade. Many investigations in the past were found that the ability of tumor cells to activate platelets depend on type and state of cancer cells. Several mechanisms of TCIPA have been described, such as adenosine diphosphate (ADP), thrombin, thromboxane-dependent mechanism and secretion of proteinase. Cholangiocarcinoma, one of the most cancer in the north-eastern part of Thailand, associated with thrombosis have been reported. However, the mechanism by which cholangiocarcinoma facilitated platelet function is unknown. Here, we have investigated how cholangiocarcinoma cell from cell culture effect on platelet function as measured by platelet aggregation. Primary human chlolangiocarcinoma (HuCCA) cells were established in our laboratory. Cells were cultured in T-75 Flasks with Dulbeco Modified Eargle's Medium (DMEM) containing 15 % fetal bovine serum, 100 unit/ml penicillin G, 100 µg/ml streptomycin and grown to confluence until uses. After which cell were replaced with fresh medium (DMEM) without serum for 24, 48 and 72 hours. Then, the conditioned medium (CM) was collected. To study the whole cell effect, HuCCA were grown to confluence and extracted using trypsinization. Cells were resuspended in DMEM to yield a concentration 10^7 cells/ml. 150 µl of cell suspension or CM, therefore, were added to 850 µl of platelet rich plasma (PRP) and platelet aggregation was measured by aggregometer. CM from HuCCA failed to aggregate in PRP,whereas direct HuCCA pellets induced platelet aggregation and potentiated platelet aggregation induced by submaximal concentration of thrombin. Interestingly, the effect of HuCCA induced platelet aggregation were variated in each groups of platelet status of subject (63.76, 47.41 and 44.71 % maximum aggregation in groups hyper, normal and dis-aggregation, respectively). This finding suggest that cholangiocarcinoma cell is able to induced platelet aggregation in a direct tumor cell-platelet contacts. This results might explain the pathogenesis of thrombosis in cholangiocarcinoma and support metastasis in this cancer. However, the actual mechanism remains to further elucidated.