胰臟癌治療研討會

活動時間： 109 年 10 月 29 日（星期四）晚上 18:30-21:00
活動地點： 麗尊酒店
活動住址: 高雄市五福一路 105 號 07-2295000

時間	主題	講師	主持人
18:30~18:40	Opening	饒坤銘 理事長 南屏癌症防治衛教學會	
18:40~20:00	Pancreatic cancer	江佳駿醫師 阮綜合醫院血液腫瘤科	饒坤銘 理事長 南屏癌症防治衛教學會
20:00~20:10	Break		
20:10~21:00	Chemotherapy obstacles toward pancreatic cancer	All	饒坤銘 理事長 南屏癌症防治衛教學會

江佳駿醫師
學歷：台北醫學大學醫學系醫學士

經歷：署立桃園醫院內科部住院醫師
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國立台灣大學附設醫院腫瘤醫學部臨床研修住院醫師
衛生福利部澎湖醫院腫瘤內科專科醫師

現任：阮綜合醫院血液腫瘤科科主任

專長 ：
1.一般內科學
2.腫瘤內科學：含癌症諮詢、治療計劃、術後輔助治療評估、 固態性腫瘤之化學治療及標靶治療、生命終期照顧

Pancreatic cancer is a lethal disease characterized by highly dense stroma fibrosis. Only 15–20% of patients with pancreatic cancer have resectable tumors, and only around 20% of them survive to 5 years. Traditional cancer treatments have little effect on their prognosis, and successful surgical resection combined with effective perioperative therapy is the main method for maximizing long-term survival. For this reason, chemotherapy is an adjunct treatment for resectable cancer and is the main therapy for incurable pancreatic cancer, including metastatic pancreatic adenocarcinoma. However, there are various side effects of chemotherapeutic medicine and low drug penetration because the complex tumor microenvironment limits the application of chemotherapy. As a novel strategy, polymer nanoparticles make it possible to target the tumor microenvironment, release cytotoxic agents through various responsive reactions, and thus overcome the treatment barrier. As drug carriers, polymer nanoparticles show marked advantages, such as increased drug delivery and efficiency, controlled drug release, decreased side effects, prolonged half-life, and evasion of immunogenic blockade. In this review, we discuss the factors that cause chemotherapy obstacles in pancreatic cancer, and introduce the application of polymer nanoparticles to treat pancreatic cancer.

Chemotherapy obstacles toward pancreatic cancer

PSCs are normally quiescent and are termed qPSCs. The qPSCs can transit into an activated myofibroblast-like phenotype, termed as aPSCs. The aPSCs express fibroblast activation proteins, such as α-smooth muscle actin (α-SMA). It is now commonly accepted that aPSCs are essential for the desmoplastic reaction in pancreatic cancer. Amrutkar and coworkers cocultured PSCs with seven different PCC lines, respectively, by both direct and indirect means, and various degrees of chemoresistance were detected among all PCC lines. By examining PSC-conditioned medium, they found several proteins that participated in the construction of ECM, such as fibronectin and collagen.Koikawa and colleagues found that PSCs and PCCs frequently invaded the collagen matrix successively to establish three-dimensional matrix remodeling. PCCs invade after the PSCs because PSCs remodeled the ECM and increased parallel fibers along the direction of invading PSCs. In addition to CAFs, PSCs are another main source of fibronectin and collagen in pancreatic cancer and they contribute to the chemotherapy obstacles.