Pancreatic cancer: What is new?

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Pancreatic cancer is the fifth leading cause of cancer death in the Western world. Despite improvement in operative mortality rates, little impact has made on overall 5-year survival.

Over the past 20 years great strides have been made in our understanding of the molecular basis of disease. Pancreatic cancer is almost unique among solid gastrointestinal malignancies in its frequency of genetic abnormalities, such as K-ras activation, and p53 and DPC4 loss. K-ras mutation analysis of pancreatic juice could be used to help discriminate chronic pancreatitis from pancreatic cancer. The diagnosis of pancreatic cancer by fine needle aspiration cytology (FNAC) has a sensitivity of 90% in the best hands, but difficulty can occur in patients with inflammatory atypia. The addition of K-ras mutation analysis to standard FNAC can increase its sensitivity from 69% to 92%. DPC4 is lost in 55% of the pancreatic cancers and its loss often predicts a poorer prognosis.

Recent data described an mRNA biding protein called Hu protein antigen R (HuR or ELAV1) as a predictive marker for Gemcitabine efficacy in pancreatic cancer patients. Modulation of the expression of deoxycytidine kinase (dCK) through HuR overexpression dramatically sensitized pancreatic cancer cells to gemcitabine in vitro. Accordingly, the HuR cytolasmatic status in patient's tumor cells strongly correlates with gemcitabine response. Finally, novel radiation therapy technique, namely stereotactic body radiation therapy (SBRT) is a minimally invasive treatment option that has been shown to be safe, quick and feasible in locally advanced pancreatic cancer minimally interfering with delivering subsequent systemic chemotherapy.