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Genetic Alterations of Pancreatic Cancer In North Indian Population

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Background & Aim: Pancreatic cancer is one of the most lethal human cancers; less than 5% of patients survive 5 years. The etiology of pancreatic cancer is poorly understood, and pancreatic tumors respond unfavorably to chemotherapy, radiation therapy, and surgery. Therefore, the objectives of this study were to analyze the molecular aspects (methylation status, mutational analysis and expression of p16 & K-ras gene) of pancreatic cancer; and then focus on studying different risk factors in order to shed light on the etiology of this deadly disease.

Materials & Methods: The study population included 65 cases of pancreatic cancer and a control group of 50 cases of chronic pancreatitis who underwent surgery for pancreaticoduodenectomy An attempt was also made to work on autopsy of pancreatic tissue from 50 healthy individuals who had accidentally died. p16 methylation status, mutational analysis of K-ras were examined using the two step polymerase chain reaction (PCR) RFLP. Immunohistochemistry and other risk factors are assessed by standard questionnaire.

Results: A total of 65 samples of pancreatic cancer, 50 chronic pancreatitis tissues and 50 normal autopsy tissues were analyzed with MSPCR. In pancreatic cancer 58.46% (38/65) and 8% (4/50) in chronic pancreatitis samples showed amplification with methylated primer. This shows a statistically significant correlation between methylation status of p16 gene promoter in pancreatic cancer, chronic pancreatitis and control.($P<0.05$) with OR = 16.19 (95% CI 4.92-67.51). In our study K–ras positivity was observed in 47/65 and mutations were observed in 31/65 and the remaining 34 were wild type, while in chronic pancreatitis it was observed that 3/50 showed mutations and remaining 47 were wild type, which shows statistical significance ($p<0.05$) with OR=14.28 (95% CI 3.88-77.25). The prevalence of p16 gene and k-ras was found be statistically higher in men with pancreatic cancer compared to chronic pancreatitis ($P<0.05$*). In this study, men showed more prevalence of p16 methylation status and mutation analysis of K-ras gene compared to women. Patients age ≥ 40 yrs showed statistically higher incidence of pancreatic cancer than chronic pancreatitis ($P<0.05$*). Smoking was found to be one of the risk factor for pancreatic cancer compared to chronic pancreatitis ($p<0.05$*). Other possible associated risk factors like alcohol consumption, diabetes, tea, coffee, milk products and dietary habits were found to be statistically non-significant.

Conclusion: This study shows evidence of p16 methylation and mutation of K-ras gene to be statistically higher in pancreatic cancer compared to chronic pancreatitis and this suggests that inactivation of cell cycle regulator and tumor suppressor p16 and k-ras gene represent a critical early step in the genesis of pancreatic cancer.