Frontiers in Medical Sciences (FIMS'2014): Diabetes, Cancer and Their Connection.
Istanbul, Turkey. July 6-8, 2014

JOP. J Pancreas (Online) 2014 Sep 28; 15(5):524

Diabetes and Cancer: The Problem of Reverse Causality and Missing Links

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Several epidemiology studies have observed that there is a higher than expected association between diabetes mellitus (DM) and pancreatic ductal adenocarcinoma (PDAC). However, because no plausible mechanistic link exists between high glucose levels and carcinogenesis, additional risk factors are likely involved. Evaluating these epidemiologic data has been confounded by difficulty in identifying appropriate control populations and in replicating the demographics and risk found in previous studies. Furthermore, a significant subset of patients developed DM up to 2 years before the detection of cancer, suggesting that the glucose intolerance in these patients is a paraneoplastic syndrome linked to factors released from the tumor rather than typical type 1 or type 2 DM. Indeed, DM is a major co-morbidity of PDAC and is often reversed following resection of the cancer and a major part of the pancreas, while in other cases, pancreatic resection leads to DM. Finally, the DM may be associated with previous acute pancreatitis and/or subclinical chronic pancreatitis, so that the diagnosis of pancreatitis is often missed. The pancreatitis factor is important since the link between pancreatitis and pancreatic cancer is well established and a close temporal link between pancreatitis and diabetes in known. In these cases the primary epidemiology association is between DM and pancreatitis rather, and secondarily, there is a mechanistic association between pancreatitis and PDAC. To date, though, pancreatitis and pancreatogenic DM (Type 3c) has not been adequately assessed in a major epidemiology study. These observations indicate that glucose intolerance is non-specific, that PDAC can cause DM, and that pancreatitis may be an important missing link between DM and PDAC. Thus; new carefully controlled studies are needed to better understand the underlying cause of the association of PDAC with DM.