Correlative dependence on the frequency of cancer and diabetes mellitus

Diabetes 2 type is associated with increasing risk of the internal organs tumor development, such as hepatocellular carcinoma, pancreatic adenoma, colorectal cancer, breast cancer.

Possible mechanisms of direct relation between diabetes and tumor growth include hyperinsulinemia, hyperglycemia and inflammation.

Insulin is the main regulator of cell metabolism, in addition, it refers to tissue growth factors. The effects of insulin in target cells are mediated through insulin receptors, which are transmembrane proteins and belong to tyrosine kinase receptors.

Hyperinsulinemia increases the risk of cancer development. There are endogenous (prediabetes, metabolic syndrome, obesity, diabetes 2 type) and exogenous (insulin therapy SD) hyperinsulinemia. A lot of cancer cells requires insulin for extracorporeal growth. The following mechanisms for the oncogenic potential manifestation in diabetes mellitus are suggested:

1. insulin stimulates the growth of cancer cells mainly through its own receptor;
2. in many cancer cells, hyperexpression of insulin receptors occurs, while its A-isoforms predominate, which have a more evident mitogenic effect than B-isoforms. This provides a selective advantage for the growth of malignant cells under the influence of insulin;
3. in addition to hyperinsulinemia, the risk of developing cancer with diabetes may increase for other reasons. Most patients with diabetes 2 type have an excess of adipose tissue. Adipose tissue is an active endocrine organ that produces free fatty acids, interleukin-6 (IL-6), plasminogen-1 activator inhibitor (IAP-1), adiponectin, leptin and tumor necrosis factor-alpha.Each of these factors can play an etiological role in malignant transformation or progression of the cancer.

Adenocarcinoma of the pancreas occurs associated with the diabetes 2 type more often. Adenocarcinoma of the pancreas is the most prognostically unfavorable cancer - with a 5-year survival rate of less than 5%. At the same time in the world there are increasing incidences of mortality from pancreatic cancer, which is the cause of death in about 227 thousand people a year.

About 85% of tumors are already inoperable on having diagnosed, so prevention of this type of cancer becomes particularly important by modifying its risk factors. More than 90% of cases are cancer, which develops from ductal epithelium (mainly adenocarcinoma), 5-10% - on cancer, which develops from islet cells. The head of the pancreas is affected in 70%, the body - in 20%, the tail - in about 10% of cases.

The risk of pancreas cancer in patients with diabetes increases up to 94%, regardless of place of residence, sex, alcohol consumption, BMI and smoking. At the same time, the risk of cancer is the maximum in duration of DM less 1 year, then it gradually decreases. There is a hypothesis that this is due to a mix of cause and effect. Clinical, epidemiological and experimental findings indicate that pancreatic cancer causes diabetes, secreting mediators that disrupt beta cells function and metabolism of glucose in the liver and muscles. The frequency of diabetes diagnosis in patients with pancreatic adenocarcinoma gradually and continuously increases for 3 years preceding the detection of pancreatic cancer. The occurrence of hyperglycemia does not depend on the stage of pancreatic cancer (PC). In this case pancreatogenic diabetes should be distinguished from type 2 diabetes. As a rule, such patients do not have family history of diabetes, BMI <25 kg / m2, age over 65, weight loss> 2 kg.

     Nevertheless, the likelihood that long-term diabetes is a risk factor for the development of pancreatic cancer cannot be excluded. In the duration of diabetes 2 type more than 10 years there is increasing risk of cancer, which can hardly be explained by its untimely diagnosis.

In diabetes 2 type, pancreatic exocrine cells that are very close to insulin secreting cells are exposed to very high concentrations of insulin. Experimental data that insulin stimulates proliferation and reduces apoptosis in cancer cells of the pancreas both directly and indirectly due to increasing in the bioavailability of IGF-1. Hyperinsulinemia and hyperglycemia (fasting blood glucose level 6.1-6.9 mmol / l, after loading blood glucose level - 7.8-11.0 mmol / l, HbA1c - 5.7-6.4%) , have already been present at the pre-diabetes stage. Thus, it is possible that prediabetes can also increase the risk of developing PC.

The rate of development of pancreatic cancer increases linearly by 14% with increasing in fasting blood glucose for every 0.56 mmol / l. Early revealing of pre-diabetes and lifestyle changes that promote glucose metabolism can be an effective strategy to contain the growing incidence of PC.

The risk of PC increases significantly in combination with diabetes 2 type and chronic pancreatitis. Other risk factors for developing prostate cancer are smoking, age over 50, male sex.

     In older people with diabetes suspected of PC, the following symptoms can be observed:

     - jaundice, weight loss, abdominal pain, fat in the stool (pancreatic head cancer);

    - pain in the abdomen, weight loss (cancer of the tail and body of the pancreas).

     However, these symptoms are also found in other gastrointestinal diseases.

To prevent the development of PC, a systematic and dynamic control of glucose and insulin concentration in the blood is necessary. In constant increasing level of insulin, there is a need to prescribe medicines that can dispose of insulin, as well as antiinsulinic hormones. The anti-insulin action of the anterior lobe of the pituitary gland hormones has been shown with particular clarity on de-pancreatic (i.e., pancreatic-deprived) dogs with severe diabetes. As it turned out, such operated animals can sharply reduce the concentration of sugar in the blood not only by introducing insulin, but also by removing the pituitary gland. So in long-term diabetes 2 ензу, as the hyperinsulinaemia and hyperglycemia decrease, the risk of cancer also decreases.