

A.V.Evseyev

Zaporizhzhia State
Medical University

Key words:
pancreatic cancer,
neoplasm
invasiveness, cell
proliferation,
apoptosis.

UDC: 616.37-006.6-036.4-091.8

FEATURES OF PROLIFERATION AND APOPTOSIS OF INVASIVE PANCREATIC DUCTAL ADENOCARCINOMA

The study was performed as a part of research work "Studying the invasive and metastatic tumor properties and their early prognosis on the patients' biopsies" (state registration number 0114U000967).

ABSTRACT. Background. Current immunohistochemical identification of prognostic markers of the pancreatic ductal adenocarcinoma needs to be improved, as it helps to estimate the rate of invasion and innidiation, and consequently give an opportunity to predict the individual behavior of tumor in every case. **Objective.** The purpose of the work was to investigate the proliferative and apoptotic activity of invasive pancreatic ductal adenocarcinoma and adenocarcinoma without invasion with the help of immunocytochemical markers. **Methods.** Operational materials of pancreato-duodenal resections from 80 patients with invasive and non-invasive pancreatic ductal adenocarcinoma were used to examine the expression of Ki-67, p53, p16, p21 and Caspase-3 immunocytochemical markers. **Results.** Ductal adenocarcinoma is characterized by the low level of proliferation marker Ki-67 expression. An invasive ductal adenocarcinoma differs from an adenocarcinoma without invasion by the significantly higher level of nuclear expression of Ki-67 and p53 and cytoplasmic expression of caspase-3 by the atypical epithelium of glands at the identically high level of p21^{WAF1} and low level of p16^{INK4A} expression. **Conclusion.** 1. Pancreatic ductal adenocarcinoma without invasion is distinguished by the prominent tissue-cellular atypism, presence of rich desmoplastic stroma, low level of nuclear expression of Ki-67 (1,42±0,06 point) by the epithelium of malignant ductular cells, tubular and glandular structures, presence of pathological mitoses and absence of perineural invasion loci and invasion of the tumor into surrounding organs. 2. The invasion of pancreatic ductal adenocarcinoma is diagnosed much more often, it differs from an adenocarcinoma without invasion by its expansion on perineural spaces and surrounding organs, and also by the significantly higher level of nuclear expression of Ki-67 and p53 and cytoplasmic expression of caspase-3 in cancer cells at the identically high level of p21^{WAF1} and low level of p16^{INK4A} expression. 3. The invasion zone of ductal adenocarcinoma to duodenum is characterized by moderate proliferative activity of tumor cells (2,24±0,26 point), lower, than in the main mass of malignant tumor. At the same time, levels of p53 (30,06±4,2%) and caspase-3 expression in these zones substantially were not differ (81,2±2,8%) in atypical cells from expression of these markers in the main tumor mass of invasive adenocarcinoma.

Received: 11.09.2014

Accepted: 23.09.2014

© A.V.Evseyev, 2014

✉ evseevanton@ukr.net

Citation:

Evseyev AV. [Features of proliferation and apoptosis of invasive pancreatic ductal adenocarcinoma]. Morphologia. 2014;8(3):23-6. Ukrainian.

References:

1. Sobin LH, Gospodarowicz MK, Wittekind C, editors. TNM Classification of Malignant Tumours. 7th ed. Hoboken, NJ: Wiley-Blackwell; 2010. 276 p. ISBN 978-5-98657-025-9.
2. Attri J, Srinivasan R, Majumdar S, Radotra BD, Wig J. Alterations of tumor suppressor gene p16INK4a in pancreatic ductal carcinoma. *BMC Gastroenterology*. 2005;5(22):(Open access e-Pub <http://www.biomedcentral.com/1471-230X/5/22>).
3. Meggiato T, Calabrese F, De Cesare CM, Baliello E, Valente M, Del Favero G. C-jun and CPP32 (Caspase 3) in human pancreatic cancer: relation to cell proliferation and death. *Pancreas*. 2003;26(1):65–70.
4. Hu H-Y, Liu H, Zhang J-W, Hu K, Lin Y. Clinical significance of Smac and Ki-67 expression in pancreatic cancer. *Hepato-Gastroenterology*. 2012;59:2640–3.
5. Jeong S, Lee DH, Lee JI, Lee JW, Kwon KS, Kim PS, Kim HG, Shin YW, Kim YS, Kim YB. Expression of Ki-67, p53, and K-ras in chronic pancreatitis and pancreatic ductal adenocarcinoma. *World J Gastroenterol*. 2005 Nov 21;11(43):6765-9. PMID: 16425381.
6. Cheng F, McLaughlin PJ, Verderame MF, Zagon IS. The OGF-OGFr axis utilizes the p21 pathway to restrict progression of human pancreatic cancer. *Mol Cancer*. 2008 Jan 11;7:5. doi: 10.1186/1476-4598-7-5. PMID: 18190706; PMCID: PMC2253554.
7. Dabbs DJ. *Diagnostic Immunohistochemistry: Theranostic and Genomic Applications*. 3rd ed. New York: Saunders; 2010. 941 p.