

Caveolin-1 expression as a possible biomarker in pancreatic cancer diagnosis

C. Tanase*, E. Raducan*, L. Albulescu*, E. Codorean*, M.I. Nicolescu*, D.I. Popescu*, M.L. Cruceru***, A.C.Popa***, S.O. Dima**, M. Leabu*, L.M. Popescu*, M.E. Hinescu*, I. Popescu**

* "Victor Babes" National Institute of Pathology, Bucharest, Romania

** "Fundeni" Clinical Institute, Bucharest, Romania

***"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

INTRODUCTION

Caveolin1 (Cav-1) the structural protein of caveolae, is recognized as a key switch, functioning either as a tumor suppressor or as a promoter of metastasis and tumor cell survival.

AIM

Our goal was to reveal the immunohistochemical expression of Cav-1 in different pancreatic tumors, in comparisson with Ki67 and p53, in order to evaluate their involvement in tumour aggressivness.

MATERIALS & METHODS

We studied 12 pair samples (tumoral/peri-tumoral), harvested from patients with pancreatic cancer, that underwent surgical procedures.

The antibodies used for this project were:

IHC:

Ki67 – MIB-1, Dako, 1:50
p53 – D0-7, Biogenex, 1:100
Cav-1 – sc-894, Santa Cruz, 1:200

serum (ELFA)

CEA – BioMerieux
CA19-9 – BioMerieux

(XMapArray)

VEGF – Upstate
b-FGF – Upstate

western blot

Cav-1 - sc-894, Santa Cruz, 1:200
beta-actin - sc-47778 - Santa Cruz, 1:200

The labelling index for Ki67 was 20% in tumor tissue specimens, but around 3.5% in their matched peritumoral samples. Staining for p53 was positive in 66% of tumor samples and negative results in the peritumoral samples.

A positive expression of Cav-1 was associated with positivity for Ki67, p53 in tumor and endothelial cells (tumor tissue). By contrast, in peritumoral samples Cav-1 expression was confined to some endothelial cells.

Similar results were obtained using **Western Blot** in certain cases - Cav-1 selectively increases activities in tumor tissue/peritumoral.

RESULTS

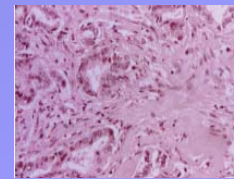


Figure 1. Ductal pancreatic adenocarcinoma. HE, 10x

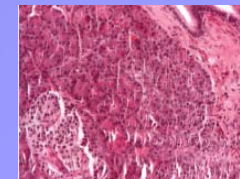


Figure 2. Peritumoral pancreatic parenchyma. HE, 10x

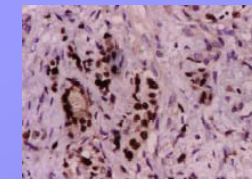


Figure 3. Positive expression for Ki67 in frequent tumor cells (tumoral tissue). IHC, 20x

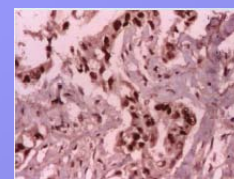


Figure 4. Positive expression for p53 in frequent tumor cells (tumoral tissue). IHC, 20x

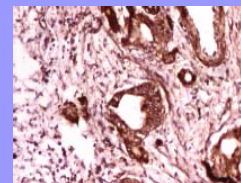


Figure 5. Cav-1 positive in tumor cells and in smooth arterial muscular wall. IHC, 10x

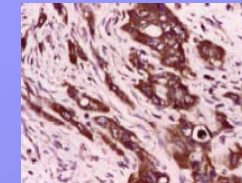


Figure 6. Intense Cav-1 positivity in tumor cells (tumoral tissue) IHC, 20x

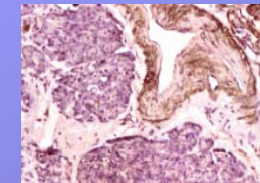


Figure 7. Cav-1 positive in blood vessels (smooth muscular cells) and in dispersed acinary cells (peritumoral tissue) IHC, 10x

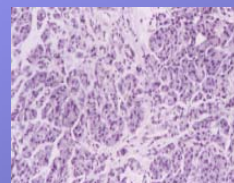


Figure 8. Negative results for Cav-1 in cells from peritumoral samples IHC, 10x

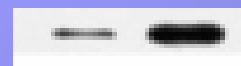


Figure 9. Caveolin-1 activity .

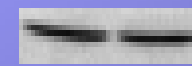


Figure 10. Control.

Serum level for tumoral markers was also increased in accordance with Cav-1 expression:

CA19.9 – avg. 109.18 UI/mL (vs. 4.00 UI/mL normal)

CEA – avg. 1.50 ng/mL (vs. 0.83 ng/mL normal)

bFGF – avg. 13.27 pg/mL (vs. 5.3 pg/mL in ctrl group)

CONCLUSIONS

- A positive corelation between the proliferative activity and overexpression of Cav-1, in concordance with tumoral grading were noted.
- Moreover, Cav-1 expression in tumors was in accordance with increased level of serum tumor markers (CEA, CA19.9) and angiogenic marker (VEGF, bFGF).
- Further research may settle if the clinical outcome can be predicted by measuring Cav-1 expression in pancreatic tumors.