Assessment of Carcinoembryonic Antigen-Related Cell Adhesion Molecules (CEACAM6) in Pancreatic Ductal Adenocarcinoma, A Combined Serum and Immunohistochemical Study

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Abstract

Background: Pancreatic cancer is a disease with an extremely poor prognosis. Carcinoembryonic antigen-related cell adhesion molecule 6 (CEACAM6) is emerging as an important determinant of the malignant phenotype in a range of cancers, including pancreatic cancer. Therefore, the aim of this study was to evaluate the potential involvement of CEACAM6 in the development, invasion and metastasis of pancreatic cancer cells.

Patients and Methods: This prospective study included 7 cases of chronic pancreatitis and 25 cases of pancreatic duct adenocarcinoma, which were collected from General Surgery Department, Faculty of Medicine, Benha University and Egyptian National Cancer Institute (NCI), in the period 2010-2014. Carcinoembryonic antigen-related cell adhesion molecule 6 (CEACAM6) immunohistochemical staining and soluble CEACAM6 were estimated in all cases and the pattern of expression in tissue was analyzed.

Results: Eighty eight percent of pancreatic ductal adenocarcinoma cases were positive for CEACAM6, while only 14.3% of chronic pancreatitis were positive, which was statistically significant (p<0.01). Patients with PDAC showed significantly elevated CEACAM6 serum expression compared to chronic pancreatitis (p<0.01). Tissue expression of CEACAM6 was also significantly correlated to presence of lymph node metastases (p<0.01). A significant correlation was found between grade of pancreatic carcinoma, nodal involvement and serum CEACAM6 level (p<0.05). Neither tumor grade nor depth of invasion were correlated to immunohistochemical CEACAM6 expression (p>0.05).

Conclusion: CEACAM6 expression appears to be an early event in pancreatic carcinogenesis, and its expression may aid in the diagnosis and in detection of disease progression in patients undergoing resection for pancreatic ductal adenocarcinoma. Serum levels of CEACAM6 could be valuable in follow-up of patients after surgery.

Key Words: Chronic pancreatitis (CP) – Pancreatic ductal adenocarcinoma (PDAC) – Carcinoembryonic antigen-related cell adhesion molecule-6 (CEACAM6).

Introduction

In Egypt, malignant pancreatic tumors constitute 2.9% of digestive tract malignancy, of them, Pancreatic Ductal Adenocarcinoma (PDAC) constituted 65.85% [1].

In the west, it constitutes 4% of all cancers. It ranks the 4th cause of cancer mortality (5%). Only 2-3% of patients survive 5 years after a diagnosis. One important reason for this poor survival is that only 10-15% of patients are diagnosed with small, resectable cancers. About 85% of carcinomas are located in the head of pancreas and often produce obstruction of biliary and pancreatic ducts which appear dilated. Ductal adenocarcinoma predominated, comprising 95% of cases of pancreatic carcinomas [2,3].

Despite the availability of new targeted therapies, ductal pancreatic adenocarcinoma continues to carry a poor prognosis. In addition, pancreatic cancer responds poorly to most chemotherapeutic agents. Hence, there is an urgent need for a better understanding of the molecular mechanisms that contribute to pancreatic cancer development and progression as well as for new potential diagnostic and prognostic tumor markers [4,5].

The discovery of the Carcinoembryonic Antigen (CEA) as a tumor marker for colorectal cancer some 50 years ago became the first step in the identification of a much larger family of 12 carci-
noembryonic antigen-related cell adhesion molecules (CEACAMs) with surprisingly diverse functions in cell adhesion, in intracellular and intercellular signaling, and during complex biological processes such as cancer progression, inflammation, angiogenesis, and metastasis [6,7].

The development of proper molecular and biochemical tools and mouse models has enabled bidirectional translation of the CEACAM network biology. Subtypes 1 and 6 are described to be under- or overexpressed in several tumor entities like lung cancer, colon cancer and melanoma [8-10].

Carcinoembryonic antigen-related cell adhesion molecule 6 (CEACAM6; 19q13.2) is a glycosylphosphatidylinositol (GPI)-linked immunoglobulin superfamily member. There is accumulating evidence that CEACAM6 is overexpressed in several epithelial carcinomas including colon, breast, non-small cell lung cancer and intrahepatic cholangiocarcinoma. In addition, it is involved in many crucial cellular events such as migration, invasion and tumorigenicity. Recent studies have suggested that CEACAM6 plays important roles in pancreatic cancer development and progression [9,11-13].

Another function of CEACAM6 were concluded by Ieta, et al. [14], in their study on cholangiocarcinoma cases, they reported that CEACAM6 overexpressed cells were resistant to anoikis and could reside in the liver and contribute to recurrence. Anoikis (a subtype of apoptosis) is a programmed cell death induced upon cell detachment from extracellular matrix, behaving as a critical mechanism in preventing adherent-independent cell growth and attachment to an inappropriate matrix, thus avoiding colonizing of distant organs [15]. Resistance to anoikis is a property of transformed cells that is associated with greater cellular invasive ability and in vivo metastatic potential [14].

Neither reliable serum nor tissue markers predicting the clinical course of patients after diagnosis of PDAC are available. Furthermore, the molecular interactions of the tumor with the host and the local factors that allow PDAC to display such an aggressive progression are poorly understood. Therefore, there is an imperative need for a better understanding of the tumor biology with respect to the mechanisms of local tumor invasion and recurrence [16]. The aim of this study is to assess the clinical relevance of CEACAM6 expression by both, immunohistochemical and serum analysis in patients with PDAC as it is considered the major type of pancreatic cancer in Egypt.

### Patients and Methods

This prospective study included selected 25 pancreatic ductal adenocarcinoma biopsies, obtained through surgery, or during interventional radiology. Seven cases of Chronic Pancreatitis (CP) were taken as a control. Cases were collected from General Surgery Department, Benha Faculty of Medicine-Benha University and the Egyptian National Cancer Institute (NCI) in the period (January 2010-December 2014). Paraffin-embedded tissue sections were prepared from obtained specimens. Hematoxylin and Eosin sections were reviewed by two pathologists to confirm diagnosis.

#### Inclusion criteria:

**Group 1**: (Pancreatic cancer: N=20) included men and women with established diagnosis of pancreatic cancer. Diagnosis is established with histological evidence of pancreatic adenocarcinoma on biopsy, obtained through surgery, or during interventional radiology.

**Group 2**: (Chronic pancreatitis: N=7) included patients with prior episodes of pancreatitis, defined clinically or radiologically as calcifications on computed tomography, or dilatation of pancreatic duct on endoscopic retrograde cholangiopancreatography, now presenting with recurrent abdominal pain and elevated serum lipase/amylase levels with no evidence of pancreatic adenocarcinoma.

#### Exclusion criteria: All the patients were diagnosed with cancer for the first time, and none previously received chemotherapy or radiation therapy.

Histologic grade of PDAC is based on the extent of glandular differentiation. If 95% of the tumor is composed of glands then it is classified as being well differentiated, 50%-95% is moderately differentiated, and 50% is poorly differentiated [17]. Mitotic activity was taken into consideration. In grade 1 mitoses were ≤5/HPF, in grade 2, mitoses were 6-10/HPF and in grade 3, mitoses were >10/HPF [18]. Current staging for pancreatic cancer is based on the 7th edition of the AJCC Cancer Staging Manual, published in 2009 [19].

**Immunohistochemical staining**: Tissue sections were mounted on positively-charged slides, steps of staining followed the standard ABC (avidin-biotin complex) procedure using the Ultra Vision Detection System (Anti-polyvalent, HRP/DAB, ready-to-use, Lab Vision corporation). Antigen retrieval with microwave treatment in 10mM citrate buffer (Neo-Markers, Cat. #AP-9003), pH 6.0. Sections were incubated with rabbit polyclonal
Statistical analysis:

Data analysis was performed with the statistical package for social sciences (version 16.0.1; SPSS Inc., Chicago, Illinois, USA). Descriptive analysis of the variables and statistical significance of the tests were expressed in $p$-value. $p$-value less than 0.05 ($<0.05$) was considered significant and $<0.01$ was highly significant.

Results

A- Histologically:

Grade 1: PDAC (well-differentiated) were characterized by large duct-like structures combined with medium-sized neoplastic glands. The glandular structures are surrounded by concentric desmoplastic stroma. Mitotic activity is low Fig. (2A).

Grade 2: PCAC (moderately-differentiated) showed mixture of medium-sized duct-like and tubular structures of variable shapes impeded in desmoplastic stroma. Incompletely formed glands were seen. A greater variation in nuclear size, chromatin structure and prominence of nucleoli were detected. Mitotic figures are more frequent Fig. (3A).

Grade 3: PDAC (poorly differentiated) were composed of mixture of densely packed, small irregular glands and solid tumor cell sheets and nests. The neoplastic cells showed marked pleomorphism and brisk mitotic activity Fig. (4A).

B- Immunohistochemical staining results:

Positive CEACAM6 expression was dominant in PDAC in the form of diffuse brown cytoplasmic/membranous staining. It was detected in 22 (88%) of carcinoma cases but only one case (14.3%) of CP showed positive expression. This correlation was statistically significant ($p$-value $<0.01$).

The expression of CEACAM6 among different tumor grades was positive in 4 (66.7%), 13 (100%), 5 (83.3%) cases of grade 1, 2 and 3 respectively. There was no statistically significant correlation between CEACAM6 expression and tumor grade ($p$>0.05) Figs. (2-4).

Regarding depth of invasion, CEACAM6 was positive in 5 (83.3%), 8 (80%) and 9 (100%) of cases of pT1, pT2, pT3 respectively; which was a statistically non significant correlation ($p$>0.05).

All PDAC cases (100%) which were accompanied by nodal invasion were positive to CEACAM6 expression while 4 cases (57.1%) of tumors with absent nodal metastases showed positive immunostaining. A statistically significant correlation
was detected between CEACAM6 expression and lymph node metastases \( (p<0.01) \) (Table 1).

C- CEACAM6 in serum:

Patients with PDAC showed elevated CEACAM6 serum expression compared to chronic pancreatitis (CP median 1.45\( \mu \)g/l, range 0.8-2.1; PDAC median 3.35\( \mu \)g/l, range 1.5-5.2\( \mu \)g/l) and this was a statistically significant correlation \( (p<0.01) \). Sensitivity of CEACAM6 was 47.0\% with a specificity of 82.6\%.

For all cases, the mean CEACAM6 value was 2.986\( \mu \)g/l. This value was used as a cut-off point to distinguish the cases as 2 groups; negative group with CEACAM6 \( \leq 2.986 \mu \)g/l and positive group with CEACAM6 \( > 2.986 \mu \)g/l.

The positivity of CEACAM6 was increased in correlation to higher grades of PDAC. There was a statistically significant correlation between tumor grade and CEACAM6 level in the serum \( (p<0.05) \).

Also, a significant correlation was found between nodal involvement and serum CEACAM6 level \( (p<0.05) \), as 72.2\% of PDAC cases with lymph node metastases were associated with positive serum CEACAM6.

A statistically non significant correlation was detected between serum CEACAM6 and depth of tumor invasion \( (p>0.05) \). Sixty percent of cases with pT2 were positive to CEACAM6 while 77.8\% of cases with pT3 were positive (Table 2).

D- Correlation between immunohistochemical CEACAM6 expression and its serum values:

A statistically significant correlation was found between CEACAM6 tissue expression and elevated levels in the blood serum of these patients \( (p<0.05) \). Twenty three cases (71.9\%) examined in this study showed positive immunostaining for CEACAM6; of them, 60.9\% showed positive serum values for CEACAM6 (Table 3).

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Fig. (1): Chronic pancreatitis shows (A): Irregularly distributed fibrosis around and within the pancreatic lobules with reduced number and size of acini. The fibrous tissue shows lymphocytic infiltration (H & E x 400). (B): Negative CEACAM6 expression; score 2, (ABC x 400).

Fig. (2): PDAC grade 1 shows (A): Large duct-like structures impeded in concentric desmoplastic stroma and lined by neoplastic ductal cells with mild pleomorphism and low mitotic activity (H & E x 400). (B): Positive cytoplasmic CEACAM6 expression; score 6, (ABC x 400).
Fig. (3): PDAC grade 2 shows (A): Malignant acini lined by neoplastic epithelial cells with variable nuclear size, variable chromatin pattern and prominent nucleoli (H & E x 400). (B): Malignant acini with positive cytoplasmic/membranous CEACAM6 expression; score 6, (ABC x 400).

Fig. (4): PDAC grade 3 shows (A): Solid tumor sheets entirely replacing the acinar tissue. The neoplastic epithelial cells show marked pleomorphism (H & E x 400). (B): Densely packed small irregular glands with positive cytoplasmic/membranous CEACAM6 expression; score 5, (ABC x 400).

Table (1): Correlation between immunohistochemical CEACAM6 expression and studied clinico pathological parameters.

<table>
<thead>
<tr>
<th>Type of lesion:</th>
<th>CEACAM6 score</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>Negative (&lt;3)</td>
<td>Positive (3)</td>
</tr>
<tr>
<td>CP</td>
<td>7</td>
<td>6 (85.7%)</td>
</tr>
<tr>
<td>PDAC</td>
<td>25</td>
<td>3 (12%)</td>
</tr>
<tr>
<td>Tumor grade:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1</td>
<td>6</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>G2</td>
<td>13</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>G3</td>
<td>6</td>
<td>1 (16.7%)</td>
</tr>
<tr>
<td>Depth of invasion:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PT1</td>
<td>6</td>
<td>1 (16.7%)</td>
</tr>
<tr>
<td>PT2</td>
<td>10</td>
<td>2 (20%)</td>
</tr>
<tr>
<td>PT3</td>
<td>9</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Nodal metastases:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N0</td>
<td>7</td>
<td>3 (42.9%)</td>
</tr>
<tr>
<td>N1</td>
<td>18</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

CP: Chronic pancreatitis.
PDAC: Pancreatic ductal adenocarcinoma.

Table (2): Serum levels of soluble CEACAM6 in studied cases.

<table>
<thead>
<tr>
<th>Type of lesion:</th>
<th>Total</th>
<th>Serum CEACAM6</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>CP</td>
<td>7</td>
<td>7 (100%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>PDAC</td>
<td>25</td>
<td>10 (40%)</td>
<td>15 (60%)</td>
</tr>
<tr>
<td>Tumor grade:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G1</td>
<td>6</td>
<td>4 (66.7%)</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>G2</td>
<td>13</td>
<td>6 (46.2%)</td>
<td>7 (53.8%)</td>
</tr>
<tr>
<td>G3</td>
<td>6</td>
<td>0 (0%)</td>
<td>6 (100%)</td>
</tr>
<tr>
<td>Depth of invasion:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PT1</td>
<td>6</td>
<td>4 (66.7%)</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>PT2</td>
<td>10</td>
<td>4 (40%)</td>
<td>6 (60%)</td>
</tr>
<tr>
<td>PT3</td>
<td>9</td>
<td>2 (22.2%)</td>
<td>7 (77.8%)</td>
</tr>
<tr>
<td>Nodal metastases:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N0</td>
<td>7</td>
<td>5 (71.4%)</td>
<td>2 (28.6%)</td>
</tr>
<tr>
<td>N1</td>
<td>18</td>
<td>5 (27.8%)</td>
<td>13 (72.2%)</td>
</tr>
</tbody>
</table>

CP: Chronic pancreatitis.
PDAC: Pancreatic ductal adenocarcinoma.
Assessment of CEACAM6 in Pancreatic Ductal Adenocarcinoma

Table (3): Correlation between immunohistochemical CEACAM6 expression and its serum values.

<table>
<thead>
<tr>
<th>CEACAM6 score:</th>
<th>Total</th>
<th>Serum CEACAM6</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative (&lt;3)</td>
<td>9</td>
<td>8 (88.9%)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Positive (&gt;3)</td>
<td>23</td>
<td>14 (60.9%)</td>
<td></td>
</tr>
</tbody>
</table>

Discussion

CEACAMs are cell surface glycoproteins involved in intercellular binding and belong to the immunoglobulin superfamily. They are implicated in various cellular functions governing growth and differentiation. Some CEACAM members have been also implicated in various intracellular signaling-mediated effects involved in the growth and differentiation of cancerous cells thus playing a key role in the modulation of various types of cancers [22]. Interestingly, recent studies found CEACAM 1 and 6 expressions in primary PDAC correlated with a shortened overall patient survival [6,23]. This work tried to assess the CEACAM6 expression in patients with PDAC using both immunohistochemical and serum analyses.

In this work, 88% of studied PDAC cases were positive to CEACAM6 with cytoplasmic/membranous expression, while only 12% of CP cases were positive. This was a statistically significant correlation (p<0.01). Also, there was a statistically significant difference in the serum level of CEACAM6 between PDAC cases and CP cases (p<0.01) as the median level in PDAC was 3.35µg/l, range 1.5-5.2µg/l; CP median 1.45µg/l, range 0.8-2.1).

This result agreed with Gebauer, et al. [16] who reported that CEACAM6 expression was observed in 72.3% of pancreatic carcinoma patients. In addition, they reported elevated CEACAM6 serum expression compared to CP (PDAC median 2.90 µg/l, range 1.34-5.46µg/l; CP median 2.25µg/l, range 0.77-5.15; p=0.06).

Duxbury, et al. [24] reported that CEACAM6 expression was observed in more than 90% of pancreatic adenocarcinoma specimens while was very weakly expressed in normal pancreatic tissue. Chen, et al. [25] found that positive CEACAM6 immunohistochemical reaction was localized to the membrane and cytoplasm of pancreatic cancer cells and was detected in 90.9% of cases. Also, Zhang, et al. [26] reported that the average expression level of CEACAM6 in the gastric carcinoma was significantly increased in tumor tissues when compared with matched non-tumor tissues with 78.2% of studied cases showing increased CEACAM6 expression in tumor tissues. This may suggest a possible role of CEACAM6 in the process of carcinogenesis in PDAC.

Several studies agreed with this suggestion. In their study on gastric carcinoma, Deng, et al. [13] reported that CEACAM6 may play a role in human gastric carcinogenesis and participate in the malignant transformation of neoplastic cells in gastric adenocarcinomas. Yasui, et al. [27] and Jass [28] reported that CEACAM6 expression is an early event in human colorectal cancers and plays a role in both early carcinogenesis and subsequent tumor progression.

In a study by Blumenthal, et al. [11], on multiple organs, CEACAM6 expression was similar in prostate cancer and normal tissues, while its expression in lung, breast, ovarian, colon and pancreatic cancer were significantly higher than non-neoplastic tissues (p<0.001). The authors suggested that CEACAM6 expression is strongly dependent on the histotype of the tumor. Antigen expression in some tumor subtypes is 2-4-fold higher than in normal tissues, while in others, expression is similar to non-neoplastic tissues.

In this study, the immunohistochemical expression of CEACAM6 in different tumor grades was 66.7%, 100% and 83.3% in grade 1, grade 2, and grade 3 respectively. There was no significant statistical correlation between CEACAM6 expression and tumor grade (p>0.05). On contrary, a statistically significant correlation between serum CEACAM6 level and grade of PDAC was found (p<0.05). This controversy may be attributed to the difference in antibody clones used in this study. Immunohistochemical marker was a polyclonal one while the serum marker was monoclonal; hence, more specific.

In their work, Deng, et al. [13] found that there were no relationships between immunohistochemical CEACAM6 expression and histological differentiation. Conversely Chen, et al. [25] reported that CEACAM6 expression correlated with tumor differentiation (p<0.05). Gebauer, et al. [16] reported such significant correlation between serum CEACAM6 and grade of PDAC.

In their study, Singer, et al. [29] indicated that CEACAM6 expression on the cell surface abolishes the cell-cell contact-triggered inhibitory signal mediated by CEACAM 1. Consequently, they concluded that CEACAM6 expression on the cell surface acts as a potent inducer of cellular prolif-
positive correlation between serum CEACAM6 positive immunostaining. This was a statistically positive to CEACAM6 expression, while 57.1% positive immunostaining. Serum values of between CEACAM6 expression and depth of tumor play between different CEACAMs is crucial for regulation of cell proliferation and growth arrest.

In this work there was no significant correlation between CEACAM6 expression and depth of tumor invasion ($p > 0.05$). Among cases of pT1, 83.3% were positive to CEACAM6. Eighty percent of cases of pT2 and 100% of cases of pT3 showed positive immunostaining. Serum values of CEACAM6 also showed a statistically insignificant correlation to depth of tumor invasion ($p > 0.05$). Chen, et al. [25] found that no correlation of CEACAM6 expression and tumor size, or T stage ($p > 0.05$).

Regarding nodal involvement, all cases (100%) of PDAC associated with nodal metastases were positive to CEACAM6 expression, while 57.1% of cases without lymph node involvement showed positive immunostaining. This was a statistically significant correlation ($p < 0.01$). Also a statistically positive correlation between serum CEACAM6 and nodal involvement was found ($p > 0.05$).

Depending on this significant correlation with nodal involvement beside the absent significant correlations with depth of invasion, this study suggests another possible role for CEACAM6 in distant tumor progression.

Parallel to such results Duxbury, et al. [24] found that negative tumoral CEACAM6 expression was associated with absence of lymph node metastases ($p = 0.012$). Deng, et al. [13] reported that gastric adenocarcinomas with elevated CEACAM6 expression were significantly associated with lymph node metastases as it was found that CEACAM6 in 63.33% gastric adenocarcinoma specimens was observed to be associated with lymph node metastasis ($p = 0.003$) and advanced stage ($p = 0.001$). These observations indicated that CEACAM6 is a specific determinant of malignant cellular behavior and progression in gastric adenocarcinoma.

This conclusion was also supported by studies in pancreatic and cholangiocarcinoma cancers showing that overexpression of CEACAM6 correlates with aggressive growth [14,23] and those adhesion molecules play a role in the steps leading to malignant cell metastasis.

Since CEACAM expression in normal tissue has pro-angiogenetic effects, regulation of the cell adhesion and may play a part in apoptosis, these effects could also be of importance in tumor progression when CEACAM expression is up-regulated within the tumor cells [16].

The role of CEACAM6 in tumor progression and aggressiveness could also be attributed to its role in anoikis. It has been suggested that CEACAM6 overexpression in breast cancer was associated with greater resistance to anoikis and high cellular invasion potential in vitro as well as higher metastatic potential in vivo [30]. In pancreatic ductal adenocarcinoma, deregulated overexpression of CEACAM6 has been shown to inhibit differentiation and anoikis. Conversely, knockdown of CEACAM6 has been shown to reverse anoikis resistance and inhibit the metastatic potential in pancreatic cancer mouse xenograft models in vivo by enhancing caspase-3-mediated apoptosis [31,32].

This study found a statistically significant correlation between CEACAM6 tissue expression and elevated levels in the blood serum of these patients. This might have several reasons: For example, the expression of the proteins may lead to an increased shedding of them. Furthermore, flushing of the shedded molecule into the blood stream might be a consequence of the disruption of anatomical barriers surrounding host tissues and endothelial cells [16]. In their study on non-small cell lung carcinoma, Zhou, et al. [33] suggested that soluble CEACAM 1 may originate from shedding or dead cells in addition to active secretion.

In conclusion: CEACAM6 expression appears to be an early event in pancreatic carcinogenesis, and its expression is associated with adverse pathologic features and prognosis in patients undergoing resection for pancreatic ductal adenocarcinoma. This study demonstrates that combined CEACAM6 expression in serum and tissue may represent a useful biomarker that may aid in the diagnosis and detection of disease progression. However, serum level of CEACAM6 alone could be used in follow-up of patients after surgery as a simple non-invasive technique. It is appropriate to include larger number of cases and to provide more quantitative support with other techniques on biopsy tissues in future studies.

References


الملخص العربي

المقدمة: يعتبر سرطان البنكرياس من أكثر السرطانات ذات التطور السريع، ويعتبر ال (سي كام 1) محدد هام لبعض السرطانات ومنهم سرطان البنكرياس.

الهدف من البحث: تهدف هذه الدراسة إلى فحص دور (سي كام 1) في نشأة واختراق وحذف ثانويات سرطان البنكرياس.

طريقة البحث: وقد تم تطبيق هذه الدراسة على شرحات موجبة من بالغات السمنة لعينات تشير إلى 25 حالة لسرطان البنكرياس و 1 حالة انتفاضة البنكرياس المزمن. والتي تم جمعها من قسم الجراحة العامة بكلية الطب - جامعة بني امرد ومختبر الدم المصري للدورة في الفترة ما بين 2011 - 2014. وقد تم معالجة هذه الشرحات بكميات مختلفة من النباتات القهوة النسجية للكشف عن ظهور الدلالا (سي كام 1) في نسبه البنكرياس المختلفة. وقد تم تحديد قيمة ال (سي كام 1) في مصل الدم.

نتائج البحث: وجد أن 80% من حالات سرطان البنكرياس كانت إيجابية لـ (سي كام 1) و 14.2% من حالات الانتفاضة البنكرياس المزمن كانت إيجابية لهذه الدلالا. وهذا الاختلاف يوحي أن النباتات القهوة النسجية عاملة، وقد وجد أن هناك علاقة احصائية طردية هامة بين درجة شدة الدلالات (سي كام 1) وبين درجة تطور الورم وكذلك انتشار الورم في الغدد الليمفاوية.

وقد وجد أن مرضى سرطان البنكرياس ظهروا قيمة أعلى لل (سي كام 1) في مصل الدم مقارنة بمرضى الانتفاضة البنكرياس المزمن. وقد وجد أن هناك علاقة احصائية طردية عالية بين نسبة ال (سي كام 1) في مصل الدم وبين درجة تطور الورم والانتشار الثاني للورم في الغدد الليمفاوية.

خلاصة البحث: وجدت هذه الدراسة أن ظهور ال (سي كام 1) مبكراً في بنكهة سرطان البنكرياس وأن ظهوره يمكن أن يساعد في تشخيص وتحديد تطور الورم.