Letters to the Editor

Serum tissue polypeptide antigen in pancreatic cancer and other gastrointestinal diseases

Tissue polypeptide antigen (TPA) is a glycoprotein produced by several tissues with keratin-like sites.\(^1\) It possesses an excellent sensitivity for diagnosing pancreatic cancer;\(^2\) in this neoplasm, as in other malignancies, it reflects tumour growth and extent.\(^3,4\) It is not specific, however, and raised TPA values have often been found in chronic pancreatitis and other inflammatory states.\(^4\)

The liver probably has a key role in influencing circulating TPA by two different mechanisms; (i) by releasing it from hepatic or metastatic cells into the bloodstream; and (ii) by decreasing its metabolism.

We attempted to evaluate the influence of different types of liver damage on serum TPA in patients suspected of having a pancreatic malignancy. We studied 135 subjects: 32 controls (20 men, 12 women, aged 19–60); 23 affected by histologically confirmed pancreatic cancer of duct cell origin (18 men, five women, aged 43–73); (16 had hepatic metastases); and 13 with chronic pancreatitis (12 men, one woman, aged 26–65) in whom the diagnosis was based on previously reported criteria.\(^6\) Sixty seven patients (43 men, 24 women, aged 23–81) had extra-pancreatic diseases including primary liver cell cancer (26 cases), gastric cancer (n = 3), carcinoma of the colon (n = 2), carcinoma of the hepatic bilus (n = 2), retroperitoneal sarcoma (n = 1), liver cirrhosis (n = 11), gallstones (n = 5), primary biliary cirrhosis (n = 3), irritable colon (n = 3), benign stenosis of the papilla of Vater (n = 2), duodenal ulcer (n = 2), erosive gastritis (n = 2), liver steatosis (n = 1), chronic cholecystitis (n = 1), hiatus hernia (n = 1), ulcerative colitis (n = 1) and retroperitoneal haematoma (n = 1).

The patients were divided into two groups: group A (n = 60) included those with anatomical liver damage (primary or metastatic cancers, cirrhosis, steatofibrosis) of whom 16 had pancreatic cancer, one had chronic pancreatitis, and 43 had extra-pancreatic diseases. Group B (n = 43) comprised all the remaining patients of whom five had pancreatic cancer, two had chronic pancreatitis, and three had extra-pancreatic diseases with a raised serum bilirubin value (>15 μg/l).

Serum TPA was assayed by an RIA technique using a commercial kit (Prolifigen, AB Sangtec Medical, Bromma, Sweden). Due to the scattered distribution of TPA values a logarithmic transformation of the data was used for statistical analysis.

The table reports mean values, standard errors, and statistical evaluation of the data. Group A patients had higher values than group B (t = 42.3, p < 0.001). Patients with raised values of total bilirubin, alamine transaminase, and alkaline phosphatase had higher TPA values compared with those with normal values (Student’s t test: t = 15.05, p < 0.001; t = 13.71, p < 0.001; t = 61.53, p < 0.001, respectively).

We found grossly increased concentrations of serum TPA in patients with pancreatic cancer as previously described;\(^2\) we also found that concentrations were raised in other malignant or benign diseases, especially of the liver and biliary tract.\(^5\)

The presence of anatomical hepatic damage is a factor of great importance in influencing serum TPA; our group A patients had higher concentrations than group B.

Patients with abnormal liver function tests had higher mean TPA concentrations, which suggests that liver dysfunction can increase circulating TPA. In addition to the presence of liver damage, cholestasis alone is associated with an increase in circulating TPA.

It may therefore be concluded that different types of liver pathologies may influence circulating TPA in patients with pancreatic cancer and other gastrointestinal diseases.

<table>
<thead>
<tr>
<th>TPA (U/l)</th>
<th>Mean</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control subjects (n = 32)</td>
<td>4-20</td>
<td>0-05</td>
</tr>
<tr>
<td>Pancreatic cancer (n = 23)</td>
<td>5-76*</td>
<td>0-17</td>
</tr>
<tr>
<td>Chronic pancreatitis (n = 13)</td>
<td>4-01†</td>
<td>0-15</td>
</tr>
<tr>
<td>Extra-pancreatic diseases (n = 67)</td>
<td>5-00‡</td>
<td>0-10</td>
</tr>
</tbody>
</table>

Analysis of variance: F = 24-29, p < 0.001

Bonferroni’s test for pairwise comparisons:

- *p < 0.001 compared with control subjects, chronic pancreatitis, extra-pancreatic diseases.
- t < 0.001 compared with control subjects.
- t < 0.05 compared with extra-pancreatic diseases.

References


Trisomy 11 in acute lymphoblastic leukaemia

The study of non-random, clonal chromosomal abnormalities in leukaemia has led to well established associations between a specific abnormality and a particular subtype of leukaemia. Trisomy 11 is emerging as a rare finding in clonal haemopoietic disease and thus far seems to have been associated with non-lymphoid disorders.\(^1,4\) We present details of a child with acute lymphoblastic leukaemia (ALL) in whom trisomy 11 was found in the bone marrow cells.

A 12 year old white boy presented with oral herpes and pallor. Liver and spleen were not enlarged. Chest x-ray picture showed hilar and paratracheal lymphadenopathy. The thymus was not enlarged. A full blood count showed a haemoglobin concentration of 5.8 g/dl, white cell count of 1.7 × 10^9/l (10% blasts) and a platelet count of 50 × 10^9/l. Many poikilocytes were present on the blood film and the mean red cell volume was 101 fl. Plasma folate and B12 and red cell folate concentrations were normal.