Table 2  Morphometry by age group

<table>
<thead>
<tr>
<th>Age group</th>
<th>N =</th>
<th>Minimum</th>
<th>Lower quartile</th>
<th>Median</th>
<th>Upper quartile</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area epithelium (%) &lt;70 y</td>
<td>18</td>
<td>19-0</td>
<td>29-8</td>
<td>34-0</td>
<td>35-0</td>
<td>39-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>19</td>
<td>28-0</td>
<td>30-0</td>
<td>32-0</td>
<td>33-0</td>
<td>41-0</td>
</tr>
<tr>
<td>Area crypt (%) &lt;70 y</td>
<td>18</td>
<td>15-0</td>
<td>19-0</td>
<td>20-0</td>
<td>19-0</td>
<td>22-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>19</td>
<td>13-0</td>
<td>19-0</td>
<td>20-0</td>
<td>19-0</td>
<td>22-0</td>
</tr>
<tr>
<td>Area L.P (%) &lt;70 y</td>
<td>19</td>
<td>42-0</td>
<td>44-0</td>
<td>47-0</td>
<td>50-0</td>
<td>53-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>20</td>
<td>41-0</td>
<td>46-0</td>
<td>47-3</td>
<td>49-0</td>
<td>53-1</td>
</tr>
<tr>
<td>Villus height (µm) &lt;70 y</td>
<td>18</td>
<td>125-0</td>
<td>42-1</td>
<td>487-6</td>
<td>541-5</td>
<td>623-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>17</td>
<td>374-0</td>
<td>394-5</td>
<td>432-0</td>
<td>490-5</td>
<td>585-0</td>
</tr>
<tr>
<td>Crypt depth (µm) &lt;70 y</td>
<td>18</td>
<td>122-0</td>
<td>136-2</td>
<td>161-0</td>
<td>196-5</td>
<td>548-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>17</td>
<td>110-0</td>
<td>125-5</td>
<td>142-0</td>
<td>165-5</td>
<td>198-0</td>
</tr>
<tr>
<td>Epithelial height (µm) &lt;70 y</td>
<td>19</td>
<td>24-9</td>
<td>27-0</td>
<td>28-7</td>
<td>30-9</td>
<td>32-8</td>
</tr>
<tr>
<td>≥70 y</td>
<td>18</td>
<td>24-8</td>
<td>26-0</td>
<td>27-9</td>
<td>29-2</td>
<td>31-5</td>
</tr>
<tr>
<td>CV ratio &lt;70 y</td>
<td>18</td>
<td>2-3</td>
<td>3-1</td>
<td>3-2</td>
<td>3-5</td>
<td>4-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>17</td>
<td>2-5</td>
<td>2-9</td>
<td>3-1</td>
<td>3-4</td>
<td>3-6</td>
</tr>
<tr>
<td>Lymphocyte count &lt;70 y</td>
<td>19</td>
<td>6-0</td>
<td>8-0</td>
<td>11-3</td>
<td>15-0</td>
<td>21-0</td>
</tr>
<tr>
<td>≥70 y</td>
<td>19</td>
<td>5-0</td>
<td>8-0</td>
<td>11-9</td>
<td>16-0</td>
<td>20-0</td>
</tr>
</tbody>
</table>

number of subjects studied and the age ranges were too restrictive to make predictions about the effects of ageing. Their other findings that mean villus height, enteroocyte height, and intra-epithelial lymphocyte counts were not significantly different in young and old subjects were similar to ours. The study by Corazza et al.,4 found that mean surface to volume ratios and enterocyte height in 16 well nourished elderly patients were not significantly different from 22 younger controls. In summary, we did not find any significant difference in duodenal morphometry with age. Thus there was no evidence to support the hypothesis that possibly impaired absorption in normal old age may relate to morphometric changes in the upper small bowel mucosa.

We thank Drs Ian Turner and Alison Brind for the endoscopic biopsy specimens and "Research into Ageing" for their generous grant.


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Signet-ring cell carcinoma of the prostate mimicking primary gastric carcinoma

O Ben-Izhak, C Lichtig

Abstract

A post mortem examination of a 70 year old man, who died three years after a poorly differentiated adenocarcinoma of prostate had been diagnosed, showed widespread signet-ring cell carcinoma, with an associated limitis plastica. The signet-ring cells stained positively with prostatic specific antigen and with prostatic specific acid phosphatase, but failed to react with mucopolysaccharide staining. The electron microscopic appearance of the signet-ring cell tumour was due to the presence of large cytoplasmic vacuoles.

This case emphasises the possibility that cases of metastatic signet-ring cell carcinoma may be prostatic in origin. This can be confirmed by specific immunohistochemical studies.

Prostatic adenocarcinoma composed of signet-ring cells is very rare,1,2 and reports of these as metastases are even less common.2 Metastases of any kind to the stomach are rare,3 and when they present as limitis plastica, they usually arise from lobular carcinoma of the breast.4

We describe a case of disseminated signet-ring cell carcinoma that produced a limitis plastica

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Accepted for publication 19 September 1991
and hormonal treatment (both drugs and orchidectomy) because of bone metastases. He died three years after the initial diagnosis with widespread disease.

Pathology
A necropsy showed widespread tumour deposits in pleura, bone, liver, abdominal lymph nodes, peritoneum and pelvis. Most of the gastric wall was uniformly thickened up to 2 cm, and on section, comprised grey-white, firm tissue, not dissimilar to the tumour masses seen elsewhere. Tissue sections showed infiltrative signet-ring cell carcinoma affecting the stomach (fig 1), bones, pleura, lymph nodes, liver, peritoneum and pelvis. Mucopolysaccharide techniques including periodic acid Schiff, alcian blue, and mucicarmine failed to stain the signet-ring cells. Immunoperoxidase staining of paraffin wax embedded sections for prostate specific antigen (PSA) and for prostate specific acid phosphatase (PSAP) (both prediluted sera from Diagnostic products Corporation, Los Angeles, California) was performed using the streptavidin-biotin complex technique. The signet-ring cells stained strongly with both PSA and PSAP. Staining was seen in cytoplasmic vacuoles, especially along their luminal borders (fig 2). Electron-microscopical examination of the formalin fixed, paraffin wax embedded tissue showed that the signet-ring cells were the result of intracytoplasmic vacuoles surrounded by an ill defined membrane (caused by poor preservation of the paraffin wax embedded tissue) (fig 3).

Cases of prostatic adenocarcinoma and gastric signet-ring cell carcinoma serviced as positive and negative controls, respectively.

Discussion
The signet-ring cell variant of prostatic adenocarcinoma has been rarely reported. In a recent series Ro et al described eight cases with the signet-ring cell variant of poorly differentiated prostatic adenocarcinoma. Our case, which developed the histological features of signet-ring cells only after prolonged treatment, differs from Ro’s patients, who all presented with the signet-ring cell histology before treatment.

Our case showed the same clinically aggressive behaviour that had been reported previously in cases of signet-ring cell prostatic carcinoma, where five of the 10 cases (including ours) presented with stage D and five presented with stage C.

It is suggested that signet-ring cell prostatic carcinoma should be classified separately from the mucinous-colloid variant of prostatic carcinoma. The latter, according to some reports, disseminates less often to distant organs and has a better prognosis than the usual prostate cancer.

The signet-ring cells in both our case and Ro’s cases showed negative staining for
neutral and acid mucopolysaccharides but stained positively with PSA nd PSAP. These cases contrast with Giltman’s case,1 where the signet-ring cells stained positively with periodic acid Schiff with and without diastase digestion. Electronmicroscopically, our findings showed that the signet-ring cell appearance was due an intracytoplasmic vacuole of uncertain origin. Smaller intracytoplasmic vacuoles have been described in prostatic adenocarcinoma.8 Tannenbaum showed collections of empty, membrane-lined vacuoles in prostatic carcinoma cells in patients treated with diethylstilbestrol,9 which were not present in biopsy specimens before treatment. Our patient was indeed treated with diethylstilbestrol, but the other reported patients with signet-ring cell prostatic carcinoma were not.1,2

As stated above, the stomach is rarely the site of metastatic tumour.3 Diffuse infiltration of the gastric wall by metastatic tumour, giving a limitis plastica appearance, can be due to metastatic lobular carcinoma of breast.4 In two of the 31 cases reported by Cormier,4 the infiltrating lobular carcinoma of the breast contained signet-ring cells, but the histology of the gastric metastases was not detailed. In our case the diagnosis of metastatic prostatic carcinoma in the stomach rather than primary gastric carcinoma in a patient with prostatic carcinoma was made possible because of specific prostatic immunohistochemical staining. Discrimination between these two possibilities is important in terms of therapeutic implications. We agree with the previously suggested conclusion1,2 that any metastatic signet-ring cell carcinoma of unknown origin in a male can be of prostatic origin and requires immunohistochemical study using PSA and PSAP to confirm or preclude this possibility.

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c-erbB-2 oncogene product expression and prognosis in gastric carcinoma

D A Hilton, K P West

Abstract
The prognostic value of c-erbB-2 protein expression was assessed retrospectively in 87 “curative” gastrectomy specimens from patients with gastric carcinoma. Tumours were stained immunohistochemically with the specific antibody 21N. Eight (9%) cases had strong membrane staining, all of which were of the intestinal type, and lymph node metastases, which showed concordance of staining in seven cases. In contrast to studies in breast cancer, positive cases showed a trend towards better five year survival, but this did not reach significance.