Short report

Alpha₁-antitrypsin bodies, Pi² phenotype, and peptic ulcer

W. K. BLENKINSOPP

From St. Mary's Hospital, Praed St., London

SUMMARY An association between chronic peptic ulcer and heterozygous a₁-antitrypsin deficiency has been reported: this study found no evidence of such an association. The prevalence of a₁-antitrypsin bodies in the liver was compared with the known prevalence of Pi² phenotype in the population: there was no significant difference.

The production of a₁-antitrypsin, the major trypsin inhibitor in the blood, is controlled by alleles—the normal being Pi⁰. Probably all persons with a Pi² allele have typical periodic acid-Schiff positive, diastase resistant, rounded bodies in the cytoplasm of their hepatocytes (Blenkinsopp and Haffenden, 1977), and these bodies rarely occur in persons with normal Pi⁰ phenotype (Bradfield and Blenkinsopp, 1977).

Andre et al. (1974) reported that 10.5% of 114 patients with chronic duodenal ulcer, and 9.6% of 83 with chronic gastric ulcer, had levels of a₁-antitrypsin in the heterozygous (Pi⁰Pi²) range, compared with 3.3% of 118 controls. The figure for patients with duodenal ulcer was statistically significantly different from that for controls, but that for gastric ulcer was not. However, normal levels of a₁-antitrypsin in the serum are scattered over a wide range, and the arbitrary choice of a cut-off at 60% of the mean normal value may be unsatisfactory. The presence of a₁-antitrypsin bodies in the liver provides a sharp discriminant, and this was used to investigate in necropsy material whether there was an increased prevalence of a₁-antitrypsin deficiency in patients with peptic ulcer.

Methods

Paraffin sections of formalin-fixed liver tissue taken at necropsy from patients with chronic gastric ulcer (72), chronic duodenal ulcer (52), or acute gastric ulcer (25), and from 159 patients without peptic ulceration were stained with periodic acid-Schiff after diastase. Patients with chronic liver disease were excluded. All positive cases were checked by immunoperoxidase, using anti-human a₁-antitrypsin (Behringwerke) and the peroxidase-antiperoxidase method. All cases positive on periodic acid-Schiff/diastase staining were also positive on immunoperoxidase staining.

Table Prevalence of livers with a₁-antitrypsin bodies

<table>
<thead>
<tr>
<th></th>
<th>Number of cases</th>
<th>With bodies</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>418</td>
<td>16</td>
<td>3.8</td>
</tr>
<tr>
<td>Chronic gastric ulcer</td>
<td>72</td>
<td>1</td>
<td>1.4</td>
</tr>
<tr>
<td>Chronic duodenal ulcer</td>
<td>52</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Acute gastric ulcer</td>
<td>25</td>
<td>2</td>
<td>8.0</td>
</tr>
<tr>
<td>Controls</td>
<td>159</td>
<td>8</td>
<td>5.1</td>
</tr>
<tr>
<td>Previous control group</td>
<td>110</td>
<td>4</td>
<td>3.6</td>
</tr>
</tbody>
</table>

Results and comment

The results are shown in the Table, which also gives the results in a previous control series (Blenkinsopp and Haffenden, 1977). Chi-square tests revealed no significant difference between any test group and the controls, and thus no evidence of an association between a₁-antitrypsin deficiency and peptic ulceration.

Summation of the results gave a prevalence of a₁-antitrypsin bodies of 3.8% in 418 cases without liver disease. Addition of the estimated number of cases with liver disease (Blenkinsopp and Haffenden, 1977) gave a prevalence of a₁-antitrypsin bodies in the hospital necropsy population of 4.1% in 424. This frequency was compared with that of 26 in 700 patients.
necropsies found by Eriksson et al. (1975) and with the frequency (3.23%) of PiZ allele found on serum phenotyping of about 10 000 samples in England (Cook, 1974), and chi-square tests showed no significant differences. This study therefore suggests that most persons with α1-antitrypsin bodies in the liver have a PiZ phenotype.

References

The January 1978 Issue

THE JANUARY 1978 ISSUE CONTAINS THE FOLLOWING PAPERS

Comparison of ‘early gastric cancer’ in Britain and Japan D. M. D. EVANS, J. L. CRAVEN, F. MURPHY, AND B. K. CLEARY

Effect of glucocorticoids on gastrin secretion in man S. SEINO, Y. SEINO, S. MATSUURA, H. KURAHACHI, M. IKEDA, M. YAWATA, AND H. IMURA

Effect of secretogogues on mucosal blood flow in the antrum and corpus of the stomach T. V. TAYLOR, B. R. PULLAN, J. GODDARD, AND B. TORRANCE

Clinical presentation of patients with ‘Dyspepsia’ JANE C. HORROCKS AND F. T. DE DOMBAL

Inhibitory effect of cimetidine on gastric acid secretion vagally activated by physiological means in duodenal ulcer patients I. M. SCHÖN AND L. OLBE

Physiological factors influencing serum bile acid levels M. PONZ DE LEON, G. M. MURPHY, AND R. HERMON DOWLING

Assessment of the (14C) aminopyrine breath test in liver disease J. GALIZZI, R. G. LONG, BARBARA H. BILLING, AND SHEILA SHERLOCK

Prevalence of α1-antitrypsin deficiency in patients with gastric or duodenal ulcer. Biomedicine, 21, 222-224.


Jejunal monosaccharide, water, and electrolyte transport in patients with chronic pancreatitis C. A. HELMAN, G. O. BARBEZAT, AND S. BANK

Hyposplenism in inflammatory bowel disease F. P. RYAN, R. C. SMART, C. D. HOLDSWORTH, AND F. E. PRESTON

Relationship between splenic size and splenic function R. C. SMART, F. P. RYAN, C. D. HOLDSWORTH, AND F. E. PRESTON

Effect of bran particle size on stool weight A. J. M. BRODRIBB AND CHRIS GROVES

Sensibility of the rectum to distension and the anorectal distension reflex in ulcerative colitis M. J. G. FARTHING AND J. E. LEANDER-JONES

Progress report Portal circulation and portal hypertension SHEILA SHERLOCK

Notes and activities

Books

Copies are still available and may be obtained from the PUBLISHING MANAGER, BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON WC1H 9JR, price £2.75, including postage.