LETTERS TO THE EDITOR

Colorectal neoplasia in acromegaly: the reported increased prevalence is overestimated

EDITOR,—We read with interest a recent paper by Jenkins et al (Gut 1998;46:440–442) and in a larger study of 1362 patients with acromegaly, had at least one adenoma and six (5%) had adenocarcinomas. However, we are concerned with their assertion that acromegaly is a high risk condition for colorectal neoplasia, and their recommended advice on colonoscopic screening and surveillance. Jenkins and colleagues had found that 35 (26%) of 129 patients (updated to 155), treated for acromegaly at St Bartholomew's Hospital, had at least one adenoma and six (5%) had adenocarcinomas.

We feel that the choice of controls in this study was inappropriate because although there is no ideal control population, the authors used comparative data on the incidence of adenomatous polyps from only two cohorts: a published study of left sided adenomas1 and colonoscopic records of all patients without acromegaly that had been examined by one of the authors. Matched for age (and side), the relative risk of adenomas was higher in patients with acromegaly when compared with data from the first study, but not when compared with data from the second.

In an attempt to estimate more appropriately the prevalence of adenomas in the normal population, we have carried out a comprehensive review of the literature on adenoma prevalence per decade of life from which two groups of studies emerged. The first group comprised six necropsy studies (n=2914), and the second comprised three screening colonoscopy studies of asymptomatic average risk volunteers (n=720, table 1).1,2 With the exception of those patients between 50–59 years old, the prevalence rates of adenomas in patients with acromegaly are remarkably similar to those for individuals in screening colonoscopy studies, and less than those from necropsy studies. We find no evidence that patients with acromegaly are at increased risk of developing adenomas.

For colorectal cancer, Jenkins and colleagues used comparative data from a regional cancer registry and estimated increased relative risks of 13–90. These figures are exaggerated in comparison with studies using standardised age and sex adjusted population data. Ron and colleagues3 reported 13 colorectal cancers in 1041 male veterans with acromegaly (standardised incidence ratio (SIR) 3.1; 95% CI 1.7 to 5.1) and, using uniform methods of ascertainment of cases and comparison groups, Orme and colleagues4 found 12 cases of colorectal cancer in a larger study of 1362 patients with acromegaly (SIR 1.68; p=0.06).

There are approximately 1500 patients with acromegaly in the United Kingdom, and it would seem sensible for strategies for large bowel screening to be evidence based. The data given above suggest that the reported increased prevalence of colorectal neoplasia in patients with acromegaly is overestimated and thus, the recommendations given by these authors for early colonoscopic screening and subsequent regular surveillance above that of the normal population cannot be supported by the evidence currently available.


Table 1 Prevalence (%) of adenomatous polyps by decade of life

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>n</th>
<th>&lt;40</th>
<th>40–49</th>
<th>50–59</th>
<th>60–69</th>
<th>70+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autopsy studies*</td>
<td></td>
<td>446</td>
<td>0</td>
<td>21</td>
<td>34</td>
<td>34</td>
<td>44</td>
</tr>
<tr>
<td>Blatt</td>
<td></td>
<td>1000</td>
<td>20</td>
<td>19</td>
<td>27</td>
<td>35</td>
<td>27</td>
</tr>
<tr>
<td>Arminski and McLean</td>
<td></td>
<td>202</td>
<td>43</td>
<td>72</td>
<td>59</td>
<td>63</td>
<td>62</td>
</tr>
<tr>
<td>Stemmermann and Yatani</td>
<td></td>
<td>518</td>
<td>175</td>
<td>35</td>
<td>56</td>
<td>58</td>
<td>47</td>
</tr>
<tr>
<td>Rickert and colleagues</td>
<td></td>
<td>443</td>
<td>65</td>
<td>233</td>
<td>31</td>
<td>46</td>
<td>33</td>
</tr>
<tr>
<td>Jass and colleagues</td>
<td></td>
<td>303</td>
<td>31</td>
<td>30</td>
<td>65</td>
<td>36</td>
<td>24</td>
</tr>
<tr>
<td>Colorectal cancers</td>
<td></td>
<td>119</td>
<td>0</td>
<td>21</td>
<td>45</td>
<td>53</td>
<td>43</td>
</tr>
<tr>
<td>DiSario and colleagues</td>
<td></td>
<td>105</td>
<td>0</td>
<td>28</td>
<td>41</td>
<td>58</td>
<td>41</td>
</tr>
<tr>
<td>Lieberman et al</td>
<td></td>
<td>496</td>
<td>0</td>
<td>20</td>
<td>33</td>
<td>31</td>
<td>26</td>
</tr>
<tr>
<td>Weighted averages</td>
<td></td>
<td>2914</td>
<td>15</td>
<td>31</td>
<td>40</td>
<td>48</td>
<td>37</td>
</tr>
<tr>
<td>Necropsy studies</td>
<td></td>
<td>720</td>
<td>20</td>
<td>38</td>
<td>37</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td>Screening studies</td>
<td></td>
<td>129</td>
<td>8</td>
<td>12</td>
<td>29</td>
<td>36</td>
<td>39</td>
</tr>
<tr>
<td>Acromegalic (Jenkins and colleagues)</td>
<td></td>
<td>440</td>
<td>0</td>
<td>21</td>
<td>34</td>
<td>34</td>
<td>44</td>
</tr>
<tr>
<td>*Three other autopsy studies (Correa and colleagues; Eide and colleagues; Williams and colleagues) were considered but not included as the age bands in these studies did not correspond with those used by Jenkins et al.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| †Patients aged 60–64; ‡patients aged 65–75. 

Reply

EDITOR,—Our conclusion that acromegaly may be a high risk condition for the development of colorectal cancer was based on our own data (we have now discovered 10 patients with cancer from approximately 210 patients who had had a colonoscopy), but also on those of several other studies. In the prospective studies carried out by Mourovex and colleagues1 and Iruarte et al.,2 cancers were discovered in 12.5% and 20% respectively of patients with acromegaly. Retrospective epidemiological surveys may have failed to show an increased risk of cancer because of differences in methodology—for example, in one study case ascertainment depended upon death certificate entries and cancer registrations which may have been incomplete.3 Furthermore, these studies did not discuss the relevance of the age of the patients. The mean and range of age during follow up were not stated,1 and in another study the mean age of the patients at diagnosis of acromegaly was 52 years old and that at follow up was only 61 years old.4 Our results clearly show that colorectal cancer is a late complication of acromegaly, as the mean age of affected patients was 67 years old.

The situation for adenomas is less clear and we agree that there is a lack of proper control groups. However, many of the prevalence figures given by Renihan and colleagues were obtained from necropsy studies and therefore cannot provide a valid comparison because the resected bowel was thoroughly washed on up to three occasions, repeatedly examined under magnification in optimal lighting, and lesions as small as 1 mm were classified as adenomas. This gives an increased prevalence of the disease compared with incidences of neoplasia revealed by colonoscopic screening. Furthermore, these studies were of populations with very different demographic, socioeconomic and dietary influences, which are factors known to influence the incidence and prevalence of colorectal adenomas. By contrast, our control groups were taken from similar populations to the patients with acromegaly, and in one group, the colono-
scopies were performed by the same operator. A comparison between these groups showed a significant increase in the relative risk of adenomas in patients with acromegaly, although we accept that this risk is not as high as that for cancer, and this raises intriguing questions about the cause of colorectal cancer in patients with acromegaly. It is possible that the adenoma–carcinoma sequence in patients with acromegaly differs from that in the nonacromegalic population, or that the cancers arise de novo without an adenoma-stage.

We fully concur that strategies for large bowel screening should be evidence based. Our initial screening recommendations are based on current data and will be modified according to our continuing prospective studies. Subsequent strategies might take into account not only the age of the patients but also their acromegaly, because preliminary data suggest that those patients in whom the disease is more active (elevated serum IGF-I) are more likely to develop adenomas. Until more multicentre studies involving large numbers of patients undertaking careful and total colonscopy allow the risks to be better quantified, it seems prudent for patients to undergo colonscopic screening every five years, or every three years if an adenoma is found.

P JENKINS
P FAIRCLOUGH
J M BESSER
Departments of Endocrinology and Gastroenterology,
St Bartholomew’s Hospital,
Witney, Oxfordshire

Gastric antral vascular ectasia and its relation with portal hypertension

EDITOR,—Spahr and colleagues recently published a case series which described the poor response of a haemorrhage from gastric antral vascular ectasia (GAVE) to portal decompression by insertion of a transjugular intrahepatic portosystemic shunt (TIPS) (Gut 1999;44:727–28). We have recently encountered a fourth patient with the same rare condition. A 66 year old non-smoking white man presented in 1997 with large bowel obstruction. His mother had a history of colorectal carcinoma and polyps in acromegaly [abstract]. At his 80th Annual meeting of The Endocrine Society: 1996 June 24-27; New Orleans. Bethesda, MD: The Endocrine Society, 1998:504.


Replay

EDITOR,—We appreciate Dr Fisher’s comments on our recent paper, in which we provided evidence against the role of portal hypertension in the pathogenesis of GAVE.

Firstly, lowering or normalisation of portal pressure was not followed by any patient surgical end to side portocaval shunt (portocaval gradient 2 mm Hg). Furthermore, the degree of residual portal hypertension was not correlated with clinical and endoscopic evolution in patients treated by TIPS. Interestingly, the only patient that responded to TIPS still had an increased gradient after the treatment (14 mm Hg); in this patient, the favourable outcome of GAVE was paralleled by a noticeable improvement in liver function. One study has suggested that arteriopanography may have a diagnostic value in GAVE. However, typical findings were shown on the arterial phase (hypervascularisation of the antrum and early arteriovenous shunting), and none of our patients had a coeliac axis arteriogram. On direct portography, it was impossible to show dilated muscular blood vessels in the antrum. In all non-responders, bleeding recurred despite a
reversed portal blood flow observed after TIPS or surgical shunt. In addition, splenic vein thrombosis was not observed in any of our patients, obviously because such a finding would contraindicate TIPS, which is not a treatment of segmental portal hypertension. Therefore, we are still convinced that porto-systemic shunting and liver function could both influence liver metabolism of the vasodilating substances that contribute to the pathogenesis of GAVE, whereas portal hypertension alone has no influence.

G POMIER-LAYARGUES Liver Unit, Hôpital Saint-Luc, 1058, rue Saint-Denis, Montréal, Québec H2X 3J4, Canada.


BOOK REVIEWS


The series of ABC articles in the *BMJ* is always enjoyable. This book brings together articles that were published several years ago and were well received in the first edition of 1995. These articles have been updated and eight additional chapters have been added on constipation, diarrhoea, irritable bowel syndrome, inflammatory bowel disease, anal cancer, colorectal trauma, tropical colonic diseases, and paediatric problems.

The editor (DJ Jones) has turned vision into reality to produce a basic and useful coloproctology text. He has also worked hard as he has written half of the chapters (nine solo and three shared). The authors of the first chapter on Anatomy and Physiology of the Colon, Rectum and Anus are Mr Hill and Professor Irving: the former runs the im manual and useful M62 Coloproctology Course and the latter needs little introduction and is President of the Association of Coloproctology of Great Britain and Ireland. Not surprisingly they combine forces to produce three pages with all the basic facts and 10 superb illustrations.

Chapter 2 takes the reader through examination (I appreciated time spent on how to perform a rectal examination) and tests from proctoscopy to colon transit studies. Chapters 3–23 then cover all aspects of colorectal disease before the final chapter on drugs. Each chapter is concise (3–6 pages) and packed with really good photographs and illustrations. All credit to the various departments of medical illustration involved in this venture!

For me the prize chapter is on inflammatory bowel disease: the authors (Mr Scott and Professor Thompson) bridge the gap between a presentation of the vital facts and the thorny management issues so eloquently.

Who should read this book? Let us first consider the exciting and challenging times we are living in. At long last colorectal cancer is receiving due recognition from the Government, primary care physicians, managers, and the general public. Attention is upon us and our daily work load is ever increasing. How encouraging that at a time like this the arms of colorectal surgeons from Poznan to Vancouver (as the saying has been or is soon to be strengthened by the colorectal nurse specialist. This book will be very useful to them.

Who else will find this book useful? Well certainly medical students, trainees in gastroenterology and surgery, and our general practitioner colleagues. And dare I also suggest gastroenterologists and colorectal surgeons. Not because we'll learn anything of course. Actually, that's rubbish because I was full of the new facts I learnt over this last weekend and I was educating everyone in theatre this Monday afternoon! I definitely will be using this book in preparing for both undergraduate and postgraduate teaching.

A LEATHER


Simon Anderson has put a lot of work into this book. He writes clearly, concisely and well. But, does the world need another handbook on gastroenterology?

What is the role of professional books, large or small, nowadays? Down at your local bookshop, Delia Smith will tell you how to make an omelette, Charlie Dimmock how to build that water feature; there's no real alternative. So, in the hospital who will tell you about haemachromatosis? The professional book has always held pride of place as the font of all knowledge. Owning such book gave native. So, in the hospital who will tell you about haemachromatosis? The professional book has always held pride of place as the font of all knowledge. Owning such book gave peace of mind, but this was at a hefty price and that has always been the problem. How often will people keep paying the ransom for the latest edition, or the newest series? It is not surprising that cutting edge books on neurology book. The case for buying this book is a modest one. A book must gather together definite clinical paradigms for how to approach the GI patient. £21 is still a lot of money for something that doesn't provide immediate gratification; however, in these days when even the Sun is buying books for schools, if pharmaceutical companies changed their mission from "chicken korma for all by 2000" to something of more tangible benefit, perhaps this book would reach a wider audience as it should.

J MEENAN

NOTES

Second Annual Gastrointestinal Cancer Update: A Multidisciplinary Approach

The Second Annual Gastrointestinal Cancer Update conference will be held at the Yarrow Hotel and Conference Centre, Park City, Utah, USA, on 15–19 March 2000. Further information from: Rosalie Lammle. Tel: +1 801 581 5664; fax: +1 801 581 3647; email: rosalie.lammle@hsc.utah.edu

European Courses on Laparoscopic Surgery

The European Courses on Laparoscopic Surgery will be held at the University Hospital Saint Pierre, Brussels, Belgium, on 4–7 April 2000 and 21–24 November 2000. Further information from: Conference Services S.A., Drève des Tumuli, B-1170 Brussels, Belgium, Tel: +32 2 375 1648; fax: +32 2 375 3299; email: conference.services@skynet.be
Diversion colitis: a trigger for ulcerative colitis in the instream colon

A G Lim and W Lim

Gut 2000 46: 440
doi: 10.1136/gut.46.3.440a

Updated information and services can be found at:
http://gut.bmj.com/content/46/3/440.2

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/