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 1999;44:585–587). 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 have found that 33 (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 adenoma2 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 colonoscopic studies of asymptomatic average risk volunteers (n=720, table 1). 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 colleagues1 used comparative data from a region cancer registry and estimated increased relative risks of 13–90. These figures are exaggerated in comparison with studies using regional age and sex adjusted population data. Ron and colleagues2 reported 13 colonic cancers in 1041 male veterans. Acromegalics (standardised incidence ratio (SIR) 3.1; 95% CI 1.7 to 5.1) and, using uniform methods of ascertainment of case and comparison groups, Orme and colleagues3 found 12 cases of colonic 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.

A G RENEHAN
Department of Surgery,
Christie Hospital NHS Trust,
Wilmslow Road, Withington,
Manchester M20 4BX, UK

S M SHALET
Department of Endocrinology,
Christie Hospital NHS Trust


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

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>n&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>446</td>
<td>40</td>
<td>21</td>
<td>34</td>
<td>34</td>
<td>214</td>
</tr>
<tr>
<td>Blatt</td>
<td>1000</td>
<td>27</td>
<td>19</td>
<td>34</td>
<td>27</td>
<td>297</td>
</tr>
<tr>
<td>Arminski and McLean</td>
<td>202</td>
<td>43</td>
<td>72</td>
<td>59</td>
<td>63</td>
<td>202</td>
</tr>
<tr>
<td>Stemmermann and Yatani†</td>
<td>518</td>
<td>17</td>
<td>35</td>
<td>56</td>
<td>58</td>
<td>518</td>
</tr>
<tr>
<td>Rickert and colleagues‡</td>
<td>443</td>
<td>6</td>
<td>23</td>
<td>31</td>
<td>46</td>
<td>443</td>
</tr>
<tr>
<td>Van and Stalsberg§</td>
<td>303</td>
<td>3</td>
<td>11</td>
<td>30</td>
<td>36</td>
<td>303</td>
</tr>
<tr>
<td>Colonoscopy studies in screened populations</td>
<td>119</td>
<td>–</td>
<td>21</td>
<td>45</td>
<td>53</td>
<td>119</td>
</tr>
<tr>
<td>DiSario and colleagues**</td>
<td>105</td>
<td>–</td>
<td>28</td>
<td>41</td>
<td>58</td>
<td>105</td>
</tr>
<tr>
<td>Rex and colleagues**</td>
<td>496</td>
<td>–</td>
<td>20</td>
<td>33†</td>
<td>31‡</td>
<td>496</td>
</tr>
<tr>
<td>Weighted averages</td>
<td>2914</td>
<td>15</td>
<td>31</td>
<td>40</td>
<td>48</td>
<td>2914</td>
</tr>
<tr>
<td>Necropsy studies</td>
<td>720</td>
<td>20</td>
<td>38</td>
<td>37</td>
<td>31</td>
<td>720</td>
</tr>
<tr>
<td>Screening studies</td>
<td>12</td>
<td>29</td>
<td>36</td>
<td>39</td>
<td>26</td>
<td>12</td>
</tr>
</tbody>
</table>

1* Three other autopsy studies (Cottere and colleagues; Elde 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.
2†Patients aged 60–64; ‡patients aged 65–75.
stump appeared mildly inflamed. Biopsy samples of the rectal stump were consistent with active colitis and a putative diagnosis of diversion colitis was made. Treatment with steroid enemas was effective. Six months later the patient developed bleeding and mucous discharge per rectum. Finally, it was thought that the patient needed total colectomy with ileostomy. The biopsy samples on this occasion were consistent with ulcerative colitis with chronic inflammation in the lamina propria, cryptitis and crypt abscess formation. The patient responded well to treatment with oral corticosteroids and mesalazine.

We have postulated previously that the development of ulcerative colitis may be initiated by inflammation of another aetiology at an anatomically discontinuous site of the bowel. This hypothesis would support the hypothesis that diversion colitis might be one such trigger for ulcerative colitis. The mechanisms underlying this remain speculative but may involve the recruitment of T lymphocytes from the diverted colon to the phenotypically similar vascular endothelium of the intransm colon.

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;44:739–742). However, the authors’ claim that this indicates the absence of a relation between GAVE and portal hypertension is seriously flawed. The failure of this condition to respond to portal decompression cannot exclude a primary role for portal hypertension in the pathogenesis of the disorder. Furthermore, this study would have been more informative if aortoportography that is, superior mesenteric and splenic angiography with venous phase imaging, had been performed in order to determine the pattern of portosystemic shunting before and after TIPS insertion. The authors have not addressed the question of whether portosystemic shunts supply the vascular lesions of GAVE; it would have been helpful if they had performed angiography in at least some of the patients. In our recent paper, in which we published preliminary data suggest that those patients in whom the disease is more active (elevated serum IgF-I) are more likely to develop adenomas, we have stated whether splenic vein thrombosis was excluded in all patients. If present, this could explain why the patients with diathesis continued to respond to TIPS insertion. The authors have not provided evidence against the role of portal hypertension in some of those patients. The possible association of GAVE with chronic renal failure cited by Spahr et al is probably not be classified as GAVE by current criteria, therefore this association is questionable.

Thus, the pathogenesis of this interesting disorder remains uncertain but the strong association of portal hypertension with this condition (overt or covert) with the majority of cases of GAVE means that this is likely to be a key contributory factor.

N.C. Fisher
Specialist Registrar in Gastroenterology, Manor Hospital, Walsall, West Midlands WS2 9PS, UK


Reply

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 improvement in liver function. In this case series, one patient had to be transfused repeatedly for five years despite a patent 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, 16 patients responding 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 arterioporo- tography may have a diagnostic value in GAVE. However, typical findings were shown on the arterial phase (hypervascularisation of the antrum and early arterovenous shunting), and none of our patients had a coeliac axis arteriogram. On direct portography, it was impossible to show dilated mucosal blood vessels in the antrum. In all non-responders, bleeding recurred despite a


Diagnostic colitis: a trigger for ulcerative colitis in the intransm colon

Editor,—We published in 1999, to our knowledge, the first description of diversion colitis appearing to trigger instream ulcerative colitis (Gut 1999;44:279–282). We have recently encountered a fourth patient with a similar presentation. The patient developed bleeding and mucus per rectum at the age of 71 years. There was no family history of inflammatory bowel disease. An in-service colonoscopy revealed ulcerative colitis. The biopsy samples on this occasion were consistent with ulcerative colitis with chronic inflammation in the lamina propria, cryptitis and crypt abscess formation. The patient responded well to treatment with oral corticosteroids and mesalazine.

We have postulated previously that the development of ulcerative colitis may be initiated by inflammation of another aetiology at an anatomically discontinuous site of the bowel. This hypothesis would support the hypothesis that diversion colitis might be one such trigger for ulcerative colitis. The mechanisms underlying this remain speculative but may involve the recruitment of T lymphocytes from the diverted colon to the phenotypically similar vascular endothelium of the intransm colon.

A G LIM
W LIM
Department of Gastroenterology, Epson General Hospital, Epson, Surrey KT19 7EG, UK

Letters, Book reviews, Notes
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 vaso-dilating 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, Montreal, Quebec 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, diarrhea, 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 immense 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 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 colonic 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 that 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 London have been or are 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 practice colleagues. And dare I also suggest gastroenterologists and colorectal surgeons. Not because we’d 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 a book gave me 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 cimetidine still adorn many bookshelves. Time to mention the “I” word.

If anything, the problem currently for professional books is that knowledge is cheap, plentiful and easily obtained. Case reviews, subject overviews and clinical slides are painlessly easy to obtain through the Internet. They are free and bang up to date. A 1999 review on haemachromatosis can be found and printed off within 15 minutes. Game set and match to the Internet, Woolworth vouchers to the poor runner up, books. Not quite. Whereas books are cheap, tidy, handy, and look good, the Internet is disparate and its out-pourings are a mess. Books have editors, the Internet doesn’t. Today, to be worth buying, a book must address either a very specialised audience or a very broad one. A book must gather together information that is difficult to obtain elsewhere in a coherent form (e.g. clinical classifications and research scoring systems, etc.), or that is timeless (clinical manifestations).

The Key Topics series does reflect some of these qualities, though weakly.

The stated church for Key Topics in Gastroenterology is senior house officers (SHO) and registrars preparing for the MRCP and all doctors wishing to keep up to date—the usual publishers’ non-specific audience of anyone that can walk upright, do joined up writing and has a medical degree. The format is lists of key points, many of which are expanded to short paragraphs. Topics are presented alphabetically which leads to a strange contents page with Nausea and vomiting coming before Neuroendocrine tumours and after Menetrier’s disease, but that is a quirk of the Key Topics series and of little importance.

All the major areas of gastroenterology are covered and although I wouldn’t agree with several of the statements made, these areas are covered well. The section on small bowel transplantation lacks any great substance, however, though it does at least serve to highlight the fact that such work is being carried out. So, who should buy it?

Dr Anderson has used up to date references throughout, but as he and his collaborators point out, this book is not intended to be a complete textbook on gastroenterology. If you need a reference book, gastroenterologist or general physician alike, this is not for you. Go and look it up on the Internet. If you are a gastroenterology house-officer, SHO or registrar consider it, although you should look around first, particularly at the Little Brown series. If you are a non-GI SHO you’re probably better off buying a radiology or neurology book. The case for buying this book would be stronger if it provided more 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 Swo 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 Lamml. Tel: +1 801 581 8664; fax: +1 801 581 3647; email: rosalie.lammlle@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, 18, B-1170 Brussels, Belgium. Tel: +32 2 375 1648; fax: +32 2 375 3299; email: conference.services@skynet.be